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UTILIZATION OF A CLINICAL TESTING BATTERY TO HELP IDENTIFY SUSPECTED HIDDEN HEARING LOSS (HHL) IN HUMANS

by

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Thesis Approval Page

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THESIS APPROVAL PAGE

This is to certify that the thesis prepared by Larry Taylor, B.S., entitled "Utilization of a Clinical Testing Battery to Help Identify Suspected Hidden Hearing Loss (HHL) in Humans" has been approved by the thesis committee as satisfactorily completing the thesis requirement for the degree of Doctor of Audiology.

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Abstract

Utilization of a Clinical Testing Battery to Help Identify Suspected Hidden Hearing Loss (HHL) in Humans

Larry Taylor

Objective

The goal of the current study was to investigate the potential presence of hidden hearing loss (HHL) in adult participants with and without a reported history of noise exposure.

Methods

Six normal hearing adults, three with and three without a reported history of noise exposure, were recruited as participants in the study and were able to complete the entire test battery consisting of audiometric testing, central auditory processing disorder ((C)APD) testing, and threshold seeking auditory brainstem response (ABR). Comprehensive audiometric testing included otoscopy, tympanometry, ARTs, pure tone audiometry from 250-8000 Hz, speech audiometry, DPOAEs, and TEOAEs. (C)APD testing included a comprehensive test battery of dichotic listening, temporal processing, monaural low redundancy, and binaural interaction tests. Threshold seeking ABR was then performed at 500, 1000, 2000, and 4000 Hz at descending intensities from 80-20 dB nHL. ABR wave V amplitudes and latencies were measured and compared. Non-parametric one way ANOVAs and T-tests were used to analyze the results.

Results

Each participant demonstrated normal hearing sensitivity with healthy middle ear function, as demonstrated by normal audiometric results. The control group tended to

demonstrate higher average DPOAE and TEOAE SNRs and higher average scores on (C)APD tests. The experimental group, surprisingly, tended to have comparable or higher average wave V amplitudes and faster latencies. However, few significant differences for these tests among the test groups were obtained and these significant differences could not be extrapolated toward a conclusion of presence of HHL in the participant groups.

Conclusions

The hallmark complaints of HHL are very real for the individuals who experience them and more research into this suspected phenomenon should continue. Further research on extended high-frequency audiometry, analysis of ABR wave I, and potential individual behavioral differences is necessary before clinical testing for suspected HHL can become a clinical reality.

Table of Contents

List of Tables

List of Figures

Utilization of a Clinical Testing Battery to Help Identify Suspected Hidden Hearing Loss (HHL) in Humans

Introduction

Hearing is an integral part of facilitating oral communication. Approximately 37.5 million American adults over the age of 18 years struggle with difficulty hearing (Blackwell, Lucas, & Clark, 2012). Untreated hearing loss has been associated with decreased cognitive, emotional, social, and physical wellbeing (World Health Organization, 2016). Hearing loss has always been measured clinically by elevated thresholds on the audiogram, the gold standard of hearing testing (Kujawa & Liberman, 2009). Recently, however, seminal neurophysiological studies in animals and humans have reshaped our understanding of human hearing, and expanded our definition of hearing loss to include a new term: "hidden hearing loss". Hidden hearing loss (HHL) refers to reduced supra-threshold speech discrimination in competing background noise or speech babble in the absence of an abnormal pure tone audiogram (Kujawa & Liberman, 2009; Zeng, 2015). Hidden hearing loss is theorized to be the result of auditory nerve damage to the afferent synapses ("cochlear neuropathy" or "synaptopathy") following a period of noise exposure (Kujawa & Liberman, 2009). The synaptic damage to the auditory nerve does not typically affect hearing sensitivity, but does impact suprathreshold speech perception, particularly in adverse listening conditions. HHL consequent to cochlear synaptopathy has been studied and documented in rodents using a combination of behavioral and electrophysiological tests, and histological analyses. While functional deficits in hearing have been documented in noise-exposed humans with normal audiometric thresholds, researchers are yet to demonstrate test results that link suprathreshold speech perception deficits to specific neural metrics.

Literature Review

The purpose of this thesis is to determine a clinical test battery for humans so that the underlying mechanisms driving abnormal suprathreshold perception in a human listener with normal hearing thresholds can be better understood.

HHL in the animal model

It has been theorized that the underlying cause of hidden hearing loss is the breakdown of the synaptic connection between the afferent auditory nerve and the inner hair cell while the hair cells themselves remain intact (Liberman, 2015). To measure and confirm the afferent cochlear synaptopathy in animals, a combination of behavioral tests, electrophysiological tests, and histological sample analysis examining the central and peripheral auditory nervous systems has been utilized.

Researchers interested in studying HHL in animals have often employed the auditory brainstem response (ABR), distortion product otoacoustic emissions (DPOAEs), and histological procedures to determine with greater accuracy the source of HHL in noise-exposed mice. The auditory brainstem response (ABR) is an objective electrophysiological tool used to assess the integrity of the central auditory system from the distal portion of the auditory nerve up to the lateral lemniscus and/or inferior colliculus (Ponton, Moore, & Eggermont, 1996). The subject's responses from the ABR can be represented visually as waves that are correlated with a specific anatomical sequence and time sequence. For example, wave I has been linked to the processing of the auditory signal by the distal portion of the auditory nerve (Ponton, Moore, & Eggermont, 1996). Although the presence of all five waves signifies efficient transmission of the signal, researchers have been primarily concerned with wave I due to its origin in the distal auditory

nerve and wave V because of its robustness (Ponton, Moore, & Eggermont, 1996). Along with ascertaining a picture of neural synchrony and overall neural function, the ABR can also be used as a tool to estimate hearing thresholds (Hood, 1995). The ABR's capability for hearing threshold estimation is an invaluable tool for evaluating those who cannot provide a behavioral response to hearing a signal or for cases where non-organic hearing loss is suspected (Hood, 1995). DPOAEs are used clinically and in research as a means of determining outer hair cell integrity. Although measurement of present and robust DPOAEs indicates the healthy function of the cochlear outer hair cells, DPOAEs are not a measure of hearing threshold estimation. DPOAEs are measured in the ear by introducing two simultaneous primary tones to the ear (Abdala & Visser-Dumont, 2003). The first primary is of a lower frequency than the second primary so that the intensity of the first primary tone is 65 dB SPL and the second primary is 55 dB SPL. Stover, Gorga, Neely, and Montoya (1996) advised that, although equal and high intensity primaries yield the largest DPOAE response, the most clinically applicable DPOAEs are recorded using 65 dB SPL and 55 dB SPL. In a healthy cochlea, the interaction of the two primary tones creates a distortion product that is of a different frequency and intensity than the primary tones. The measurement of the distortion product reflects the non-linearity of the basilar membrane, which is an indicator of overall outer hair cell health (Abdala & Visser Dumont, 2003). Histology samples of the affected mice auditory neurons were included for research analysis because, when specifically stained, the afferent synapses of interest were readily and easily interpreted.

Kujawa and Liberman conducted the seminal study beginning the recent conversation on HHL in 2009. The researchers exposed mice to a 100 dB SPL octave-wide sound (8-16k Hz) for two hours. At 24 hours post-exposure, threshold sensitive ABR and DPOAE measures showed

an expected temporary threshold shift. At eight weeks post-noise exposure, the threshold sensitive ABR and DPOAE results from the subject mice had returned to almost baseline and remained stable. However, at suprathreshold levels, the ABRs of the affected mice were measured at 40% of their baseline ABR thresholds. Conversely, DPOAE thresholds remained stable and did not change significantly post noise-exposure. Histological examination of the test group's auditory neuron cells revealed that noise-damaged mice had a significant loss of the afferent cochlear synaptic terminals (cochlear synaptopathy), coupled with intact outer and inner hair cell bodies, when compared to the control ears. This cochlear synaptopathy was especially apparent in high frequency cochlear areas (32k Hz) where the strongest suprathreshold shift was seen in the affected mice (Kujawa & Liberman, 2009). The authors concluded that the affected afferent auditory synapses did not recover, as the outer hair cells did, and rendered the afferent connections inefficient at transmitting supra-threshold intensity levels. This was concluded based upon the observation that the destroyed connections did not regenerate after time, as seen in the consistently increased suprathreshold ABR results post-noise exposure.

Fernandez, Jeffers, Lall, Liberman, and Kujawa (2015) took research of HHL in animals a step further and monitored the responses and histology of mice that had been exposed to two different noise levels from 8-16k Hz: 100 dB SPL and 91 dB SPL. Two different stimulus intensities were utilized in different test groups because 100 dB SPL was found to induce cochlear synaptopathy, while the 91 dB SPL stimulus merely caused a temporary threshold shift without permanent or progressive afferent auditory nerve synapse damage. The two groups of mice were monitored from one hour post exposure to 20 months post exposure to determine if synaptopathy increased as the mice aged. The researchers found that the two groups of mice had similar 35 dB threshold shifts post-exposure with threshold recovery after two weeks post

exposure. However, the 100 dB SPL group had increased suprathreshold wave I latencies and reduced amplitudes from 24 hours to 20 months post-noise exposure. Furthermore, histology showed that the 100 dB SPL group had more auditory nerve synapse damage compared to the 91 dB SPL group. As the mice were monitored for a total of 20 months, the researchers concluded that the 100 dB SPL group had suffered a more chronic and pervasive afferent auditory nerve synaptopathy that continued with age. These mice had absolutely no signs of synapse recovery and exhibited cochlear synaptopathy. The 91 dB SPL mouse group merely showed a TTS, but no lasting DPOAE or ABR threshold shifts and no afferent cochlear synaptopathy.

Lobarinas, Spankovich, and Le Prell (2016) also conducted research considering the existence of HHL, but used rats instead of mice. The researchers in this study subjected ten adult male rats to an octave-wide 109 dB SPL sound for 2 hours. Use of ABR, DPOAE, and hearing-in- noise tests were included to determine if there was a correlation between the amount of threshold shift, subsequent auditory nerve synapse damage, and performance to hearing-in-noise. Hearing-in-noise was tested in the animals using a conditioned prepulse inhibition (PPI) to the startle response. The test rats were conditioned to associate a brief tone, in the presence of background noise, with a subsequent puff of air. The logic behind this conditioning was thought to be that when the rats heard the tone, they would come to expect the puff of air, thereby lessening or inhibiting the startle response. Based on this hypothesis, it was theorized that if a rat who had been previously conditioned to associate the tone in noise with a subsequent puff of air to the face exhibited an increased startle response post-noise exposure, the rat could no longer hear the tone in background noise. Results of this study showed that, post-noise exposure, the test mice exposed to the 109 dB sound were the worst at inhibiting their startle to puff of air response via a conditioned tone in noise. The control mice were shown to exhibit similar PPIs

throughout the experiment. The researchers of this study noticed that, when a 35 dB or more thresholds shift was recorded, the resulting ABRs showed an initial deviation in wave morphology and latency, but soon recovered. However, when suprathreshold ABRs were performed two weeks post-exposure for this group, reduced wave I latencies and amplitudes were recorded. As in the Kujawa and Liberman (2009) study, the rats' DPOAE thresholds were reduced 24 hours post noise exposure, but recovered back to baseline after two weeks. High frequency hearing in noise was determined to be the cochlear region most affected by this period of noise exposure, but the degree of hearing loss in noise did not correlate to the amount of threshold shift or reduction in ABR wave I. Lobarinas, Spankovich, and Le Prell (2016) therefore concluded that the increase in suprathreshold ABRs and reduced PPIs of the startle reflex were expected and consistent with the prior studies' conclusions; that being afferent auditory synapse degeneration following high intensity noise exposure.

Hickox and Liberman (2014) exposed mice to three noise conditions: 100 dB SPL of 8-16 kHz for 2 hours, 94 dB SPL of 8-16 kHz for 2 hours, and a control group withheld from loud noise exposure. By inducing a startle response to noise, ABR, DPOAEs, and histological examination, the researchers measured the effect of the different noise conditions on the deafferentation of the inner hair cell (IHC) synapses. The results of this study indicated that the noise group exposed to 100 dB SPL noise had an average 44% deafferentation of their IHC synapses (without damage to the IHCs themselves) and increased latencies and decreased amplitudes of ABR wave I with full recovery of their temporary threshold shift approximately two weeks post-noise exposure. DPOAEs were measured in all three noise groups as well and indicated that outer hair cell (OHC) function remained normal after the recovery of the temporary threshold shift in the noise exposed groups. The 100 dB SPL group also exhibited an

increased prepulse inhibition of the startle response to noise, which Hickox and Liberman (2014) theorized was the result of an increase in deafferentation and IHC synaptopathy. The researchers therefore concluded that the 100 dB SPL noise group suffered from cochlear synaptopathy that increased their suprathreshold ABR responses and decreased their ability to hear in noise while their outer hair cell integrity regained baseline function.

As research has shown in the mouse model, noise-induced deafferentation of the IHC synapses in mice led to decreased suprathreshold auditory nerve function and decreased hearing in noise; but does the same noise-induced deafferentation occur in humans?

HHL in Humans

As evident in the previous section, studies have established the presence HHL in various animal models (mice, cats, guinea pigs, rats, etc.…). These animals have similar auditory systems compared to human auditory systems; therefore, it is easy to assume that similar effects of noise should be seen in humans. However, HHL has proved to be difficult to study in humans because it is not possible to perform the same invasive histological exams that have proven the afferent auditory synaptic loss in animals. To make this determination more difficult, the hallmark symptom of poor speech perception with normal audiometric thresholds is also evident in other audiological conditions. Therefore, researchers have had to create, and continue to develop, non-invasive procedures that can locate the neural metrics of interest. Often, these tests are supplemented with behavioral tests outside of the routine clinical audiometric test battery that measure speech perception in adverse listening conditions.

The increased interest in HHL, coupled with the interesting results of the animal model studies, has triggered a recent conversation regarding the presence of HHL in humans. The major

conclusion of the animal-model studies was that deafferentation of the cochlear inner hair cells (cochlear synaptopathy) was the underlying cause of HHL. However, cochlear synaptopathy following noise exposure might be just one factor out of a myriad of other audiological factors influencing decreased suprathreshold hearing in noise. In addition to the interplay of comorbid or similar audiological conditions, it is impossible to study temporal bone samples in living humans who have been exposed to noise loud enough to cause hearing loss using modern histological techniques. Post-mortem histological analyses are often utilized, but the effects of noise and age are difficult to differentiate.

Suprathreshold speech understanding deficits in patients with normal audiograms are not an unusual occurrence. For example, Plack, Barker, and Prendergast (2014) noted that the characteristics of HHL are like a condition called King-Kopetzky syndrome or central auditory processing disorder ((C)APD). Stephens and Zhao (2000) broadly defined King Kopetzky syndrome as an audiological phenomenon characterized by pure tone thresholds within normal limits, but difficulty hearing in background noise. However, the underlying cause of King-Kopetzky syndrome was theorized to be influenced by family history of difficulty hearing in noise and various physiological causes. Such a broad definition with a vague cause does not lend such a condition useful for explaining HHL in humans.

Sharma, Purdy, and Kelly (2012) defined (C)APD as an impairment of the auditory system beyond the peripheral auditory system (i.e. outer, middle, and inner ear). This auditory phenomenon is conventionally diagnosed in childhood around the age of 7 years old because the child has difficulty hearing and understanding speech in noise, frequently asks “huh?” or “what?” in conversation, and exhibits poor musical and/or singing skills (ASHA, 2005). Children who have been diagnosed with (C)APD also exhibited poor performance on tests of dichotic

listening, temporal processing, auditory closure, and/or binaural interaction. Children with (C)APD usually do not have peripheral hearing loss and can perform well scholastically with environmental adjustments and with the aid of personal frequency modulation (FM) systems (ASHA, 2005). Although some of the same symptoms are present in HHL (decreased hearing in noise, poor speech comprehension), (C)APD is usually diagnosed in children age seven and older. However, adults with central auditory processing difficulties who have been misdiagnosed either through active comorbid conditions or falling through the cracks can also be diagnosed if the appropriate test results are met. Therefore, interest and exploration into HHL has increased.

Research has also shown that aging adults, regardless of hearing thresholds, experience more difficulties with auditory processing than younger adults in difficult communication situations. Tun, Williams, Small, and Hafter (2012) reviewed the literature and concluded that there are measurable and noticeable declines in auditory processing in older adults due to changes in anatomical, physiologic, and structural functions related to hearing and comprehension. More specifically, decreases in inner hair cells, outer hair cells, and spiral ganglion cells, and changes in blood flow supply to the inner ear and brainstem are predictable changes that occur because of the aging process (Tun et al., 2012). When structural changes occur at the beginning of the auditory system, the resulting signal that is sent to the brainstem is further degraded due to observed decreases in neural synchrony and frequency selectivity. These changes lead to older adults experiencing and reporting difficulty effectively hearing and communicating in situations with rapid speech, background noise, or reverberation (Tun et al., 2012). Although there are several contributing factors, researchers have focused their studies on cochlear synaptopathy in adults without the factors previously mentioned contributing to HHL.

As previously mentioned, the research using animal models points definitively to afferent cochlear synaptopathy, but this phenomenon cannot be studied the same way with human participants. Therefore, researchers utilized more non-invasive methods of analyzing for functional deficits. Somewhat contrary to the studies that used the mouse model to investigate HHL, researchers who have studied HHL in humans have traditionally used a combination of questionnaires, behavioral testing, electrophysiological testing, and limited histological assessments of afferent auditory nerve synaptopathy.

Behavioral measures considering the intricacies of HHL have included standard audiometric testing and speech discrimination tasks. Standard pure tone audiometry is commonly used to verify normal hearing thresholds and is a subjective test of hearing thresholds. Speech discrimination tasks have been used to evaluate temporal perception of speech, specifically by researching the interaural timing difference (Bharadwaj et al., 2015). Electrophysiological measures testing the intricacies of HHL include the frequency following response (FFR), envelope following response (EFR), electroencephalogram (EEG), and auditory brainstem response (ABR).

Administration of the ABR or electrocochleography (ECochG) has been used as a helpful tool in understanding the neural synchrony of the distal portion of the auditory nerve in wave I. The auditory nerve tuning curves have been used to specify the characteristic frequency of specific auditory nerve fibers along the basilar membrane (Oxenham & Shera, 2003). DPOAE input-output curves were used to verify the nonlinearity of the cochlea by verifying the distortion product emission, or 2f1-f2. DPOAEs are a measure of outer hair cell health and functionality, and therefore are not a measure of hearing sensitivity or auditory nerve health (Shera & Guinan, 1999). Finally, histological samples in post-mortem human cochlea tissue samples have proved

very useful in visualizing the amount of cochlear synaptopathy in humans with and without histories of noise exposure.

Questionnaires probing into previous noise exposure, use of hearing protection, and family history have proved useful in separating participants into test and control groups. Johnson, Cooper, Stamper, and Chertoff (2017) developed the noise exposure questionnaire (NEQ) as a means of quantifying occupational and non-occupational cases of noise exposure. In the NEQ, participants were asked to answer the questionnaire based on their participation in noise exposure but also how long the exposures lasted (Johnson et al., 2017). It is important to take this information into the analysis of noise exposure because the intensity of the noise source and the length of exposure are different for each noisy activity. Additionally, noise screeners can serve as useful tools for quickly, and possibly clinically, ascertaining the involvement of noise exposure in a patient's audiological health (Johnson et al., 2017).

The lifetime exposure of noise and solvents questionnaire (LENS-Q) identifies frequency, duration, use of hearing protection across civilian occupational, military occupational, and recreational noise. Bramhall et al. (2017) utilized the LENS-Q to categorize their participants based on their answers in their study of abnormal ABRs in normal hearing participants with previous noise exposure. It is scored by fixing an intensity value to each noise exposure activity based on publicly available databases of noise level measurements. Conversion from dBA to dB SPL was necessary for this study because most the public data was measured in dBA. For LENS-Q responses that noted hearing protection was always worn, 15 dB was reduced from the associated public data. 10 dB was reduced when answers indicated hearing protection was used most of the time and 5 dB was reduced for using hearing protection some of the time. (Bramhall et al., 2017).

In the study conducted by Liberman et al. (2016), participants completed a series of questionnaires including questions probing otological health and questions made to investigate frequency and duration of noise exposure. The researchers were also interested if those who reported previous noise exposure had utilized hearing protection in conjunction with their exposure. Again, participants were divided into groups based on their low-risk or high-risk for noise exposure damage. Liberman et al. (2016) also added questions concerning self-assessment of hearing abilities in quiet and noisy settings. Additionally, questions for loudness and annoyance of every day sounds were evaluated.

In their study on adults with poorer-than-expected speech recognition in noise, Eckert, Matthews, and Dubno (2017) utilized the Hearing Handicap Inventory for Adults (HHIA) if participants were age 60 years or younger and the Hearing Handicap Inventory for the Elderly (HHIE) if the participants were older than 60 years. A total of 162 older adults with no more than mild hearing losses were administered the HHIA/HHIE before any formal pure tone or speech audiometry tests were done. The HHIA/HHIE has a total of 25 questions that assess the total social and emotional tolls that perceived hearing loss had on the participants. There are twenty-two shared questions from both handicap inventories, but the HHIA has three more questions that are better suited for younger participants (Eckert, Matthews, & Dubno, 2017).

Although we know that these different conditions mimic the symptoms of HHL, their presence may arise due to various reasons that are not cochlear synaptopathy due to noise damage. Therefore, I am interested in deficits in suprathreshold perception with normal hearing thresholds occurring because of noise exposure and, perhaps, cochlear synaptopathy.

According to Liberman (1978) there are three groups of auditory neurons with respect to neural threshold and firing rate properties: high spontaneous rate (19-100 spikes/second),

medium spontaneous rate (0.5-18 spikes/second), and low spontaneous rate (< 0.5 spikes/second). High SRs are correlated with lower neural thresholds, while low SR fibers are correlated with higher neural thresholds (approximately 40-70 dB). Low SR nerve fibers integrity is required for listening in background noise because of their large dynamic range, higher thresholds for activation, and ability to follow quick amplitude changes in the signal (Shi, Chang, Li, Aiken, and Wang, 2016). Because of their importance, and uncertainty in their role as possible contributors in human HHL, researchers have utilized testing measures that attempt to determine the involvement of low-SR auditory nerve reduction.

Viana et al., (2015) utilized a purely histological approach to uncovering HHL in humans. In this study, the group of researchers analyzed five histological samples from post-mortem human inner ears. They made sure that these samples were from cadavers that had not suffered excessive noise exposure or a high percentage of hair cell loss. Using the same histological and immunostaining techniques employed by researchers studying HHL in mice, Viana et al., (2015) found that the afferent connections to IHCs were greatly reduced, especially in the basal (high frequency) portions of the basilar membrane and spiral ganglion samples. The researchers concluded that presbycusis may influence age-related cochlear synaptopathy, thus resulting in difficulty listening in adverse conditions with normal pure tone thresholds.

Liberman, Epstein, Cleveland, Wang, and Maison (2016) conducted a recent inquiry into assessing potential HHL in humans. In their study, they recruited 34 adults from a local university and divided them into the following two groups: high-risk for noise exposure and low-risk for noise exposure. The high-risk group was dominated with mostly music performance majors, while the low-risk group was dominated with mostly communication science and disorders majors. The subjects varied in age (18-41 years old) and gender. Each of the

participants rated their history of noise exposure, history of hearing protection, and their perceived annoyance to certain sounds such as a baby crying or someone chewing food. Next, each participant's pure tone thresholds were assessed from 250-16,000 Hz (bone conduction from 250-4000 Hz), word recognition scores (WRSs) were obtained for NU-6 ordered by difficulty 50-word lists in quiet, signal to noise ratios of 5 and 0 dB HL, and time compression of 45 or 65%. The researchers also employed DPOAEs and electrocochleography (ECochG) to assess outer hair cell integrity and the integrity of the auditory nerve. Clinicians and researchers use ECochG to record evoked electrical potentials of the cochlea. A summating potential (SP) and an action potential (AP) are recorded during ECochG testing (Ferraro, 2000). The SP represents a direct current potential generated primarily by the cochlear outer hair cells while the AP represents an alternating current potential of the summed synchronous firing of auditory nerve fibers with negative peaks (N1 and N2). The AP evoked response has contributions chiefly from high-frequency nerve fibers when elicited by transient stimuli. The comparison of the SP and the AP, called the SP/AP amplitude ratio, is used to ascertain the health of the cochlear outer hair cells and the afferent auditory nerve fibers (Ferraro, 2000).

Liberman et al., (2016) found that each of the participants had normal hearing thresholds, except for an expected reduction in the higher frequencies near 16,000 Hz in the high-risk group. The authors of this study concluded that high frequency audiometry (above 8000 Hz and up to 16000 Hz) was useful in identifying noise-induced hearing loss in the high-risk group. Not surprisingly, the high-risk group had lower WRSs in the 5 and 0 dB SNR and the time compressed conditions. The researchers found that these results correlated better with the subjective responses on the questionnaires compared to the high frequency pure tone thresholds. The high-risk group also featured much larger summating potential (SP) to action potential (AP)

ratios when compared to the low-risk group. More specifically, the AP had reduced amplitude while the SP had increased amplitude, which is consistent with selective afferent auditory nerve fiber loss.

Bharadwaj et al. (2014) measured the auditory temporal abilities of participants exposed to high intensity noise using behavioral and electrophysiological means. Temporal abilities have been shown to be reduced in those with noise exposure and in the elderly populations. The researchers in this study used amplitude modulated (AM) pure tones in background noise and the envelope following response (EFR) via electroencephalogram (EEG) to measure differences in suprathreshold temporal coding in normal hearing listeners and if those differences were resulting from cochlear neuropathy. The EFR was chosen for this study because it can display the contributions from low-SR auditory nerve fibers. This is accomplished because changes in EFR amplitude with stimulus modulation allows for interpretation of suprathreshold temporal coding. An amplitude modulated (AM) 500 Hz tone fitted with a 4000 Hz band of noise was used to obtain behavioral thresholds, while an interaural time difference discrimination task was utilized to obtain a glimpse at binaural temporal coding. Additionally, 400 ms bursts of 1000 Hz transposed tones were used as stimuli for the EFR to obtain objective results. The results of the study showed that the participants with the most noise exposure were shown to have reduced AM sensitivity on an ITD task and reduced EFR magnitudes compared to participants who had little to no noise exposure. Bharadwaj et al. (2014) noted that interaural timing differences, individual EFR slopes, and DPOAE thresholds were variables in this study. Additionally, they proposed that the reduction in ability to identify AM tones in background noise and the reduced magnitude of the EFR were due to the reduction, because of noise damage, in the low-SR auditory afferent auditory nerve fibers.

Paul, Bruce, and Roberts (2016) hypothesized that tinnitus is another possible symptom of HHL. As demonstrated in the animal model, and proposed in humans, low SR afferent auditory nerve fiber loss because of noise exposure or aging can reduce wave I of the ABR. The researchers in this study recruited participants with and without tinnitus and measured their abilities to detect the presence of AM in pure tones in the presence of different levels of background noise. They also measured the magnitude of their EFRs by the same AM tones in quiet and at different AM depths in background noise. The purpose of testing a set AM pure tone in background noise was to assess the contribution of the low-SR fibers for AM coding. The purpose of assessing the EFR by the same AM tone in quiet was to investigate the contributions of low and high-SR involvement, while the purpose of assessing different AM depths in background noise was to investigate purely low-SR involvement. Additionally, Paul et al. (2016) utilized a computer model to simulate human hearing with different combinations of low and high-SR nerve losses. What they found was that the participants in the control group who exhibited EFR magnitude resistant to background noise had better AM detection in background noise. The researchers also found that computer simulated auditory nerve responses showed that low-SR fibers were better preserved in the control group. Conversely, participants in the tinnitus group exhibited poorer AM detection and smaller EFR magnitudes. The computer-generated algorithm for the same pattern of responses indicated that reduced low-SR involvement was at play. Paul, Bruce, and Roberts (2016) concluded that the symptoms of HHL (difficulty hearing in background noise and reduced suprathreshold signal comprehension with normal hearing thresholds) seem to follow the same pattern of responses in the tinnitus participant groups; therefore, reduction in low-SR fibers might be a contributor to the deficits seen in HHL.

Bramhall, Konrad-Martin, McMillan, & Griest (2017) recruited military veteran and civilians with normal hearing thresholds and different histories and levels of noise exposure. The level of previous noise exposure was determined using the Lifetime Exposure of Noise and Solvents Questionnaire (LENS-Q) and participants were divided into military with high noise exposure, military low noise exposure, civilian high noise exposure, and civilian low noise exposure. ABR wave I amplitude recorded at 1000, 3000, 4000, and 6000 Hz at ascending intensities up to 90 dB SPL and DPOAE input/output (L1 = 65, L2 = 55 dB SPL) functions were assessed. Bramhall et al. (2017) established that ABR wave I amplitudes across 1000, 3000, 4000, and 6000 Hz were reduced for the groups with the highest noise exposures even when DPOAE amplitude and gender were controlled for. Essentially, normal OHC function was obtained in all participants because of their normal hearing thresholds, but the afferent auditory nerve connections were faulty at some point in the distal portion of the auditory nerve. Bramhall et al. (2017) noted that similar results were obtained in studies for animal models, but without human temporal bone excision and analysis, presence of cochlear synaptopathy could not be concluded.

Research into the effects of participants who have been exposed to blasts (usually because of military deployment) and/or traumatic brain injury (TBI) are also linked with the possibility of HHL in humans. Fausti, Wilmington, Gallun, Myers, and Henry (2009) noted that blast exposure is a more extreme type of noise exposure that causes temporary or permanent threshold shifts and chemical changes at the neuronal level. These chemical changes are akin to sustained noise exposures that cause permanent long term afferent auditory nerve damage, or afferent cochlear synaptopathy. Fausti et al. (2009) noted that blast exposure is often a physically traumatic event that causes concussions to the brain. The effects of the concussion can

lead to swelling, hemorrhaging, or central auditory dysfunction in the temporal lobe. The researchers explained that the effects of cochlear synaptopathy may be coupled with TBI negatively influencing speech understanding in noise, localization, and reduced temporal characteristics and auditory patterns for speech perception.

Fausti et al. (2009) and Myers, Wilmington, Gallun, Henry, and Fausti (2009) recommend full auditory diagnostic evaluations, (C)APD assessments, and multidisciplinary interventions for victims of blast exposure. Standard behavioral audiometry accompanied by tympanometry, acoustic reflex thresholds (ARTs), OAEs, and electrophysiological assessments were recommended to obtain a comprehensive assessment of hearing after the blast exposure. Because temporal processing is often affected after blast exposure, (C)APD tests of temporal patterning are of utmost importance, along with a standard clinical (C)APD battery of assessments. Both Fausti et al. (2009) and Myers et al. (2009) recommended multidisciplinary approaches to treatment of victims of blast exposure. They recommended collaboration between audiologists, physical therapists, counselors/psychologists, and other rehabilitative professionals. Although victims of blast exposure and those with sustained noise exposure can have similar audiological symptoms (difficulty hearing in noise, reduced temporal processing), the incidence of permanent sensorineural hearing loss ranges from 35-100% for blast exposure victims (Fausti et al., 2009). Although hearing loss documented by an audiogram is not a characteristic of HHL, it is difficult to rule out obvious cochlear synaptopathy as a characteristic of their hearing impairment.

Plack et al. (2016) made the case for the development of a diagnostic test battery for HHL in humans and noted the following barriers to this endeavor: human variability and test validation. In their paper, the researchers noted that electrophysiological auditory evoked

potentials in humans are highly variable between participants. When researchers use electrophysiological tests on rodent subjects, they use near-field electrode recordings to increase the amplitude of the response. In humans, far-field recording is the much less invasive and inexpensive way to record auditory evoked responses, but signal averaging with the help of a preamplifier to boost the already small response is sometimes not enough to definitively mark peaks and call responses. Gender, age, head size, neural synchronization, and amount of electrophysiological noise are all other factors that further confound electrophysiological tests and their inter-subject and intra-subject variability (Plack et al., 2016).

Regarding test validation, researchers who used rodent subjects to study HHL could perform histological analysis of the affected neuronal regions and compare these findings with the electrophysiological and behavioral tests that were utilized. However, excising auditory neuronal samples from human subjects is not possible unless the subject has already expired (Plack et al., 2016). It is not feasible or ethical to utilize histological analysis of human auditory neuron samples in HHL studies. The histological analysis that has already been done regarding HHL was performed on post-mortem samples. However, age related synaptopathy could also be a contributing factor to the decrease in afferent auditory neuron connections. Plack et al. (2016) discussed in their paper ways to determine the validity of electrophysiological and behavioral tests for HHL in humans. Their suggestions included the use of animal comparisons, computer modeling of cochlear synaptopathy, auditory nerve imaging via magnetic resonance imaging, and human temporal bone histology on long term study participants. Because of the current inability to determine cochlear synaptopathy in humans, researchers have instead focused their efforts in studying functional hearing deficits and strived to develop a more global approach for identifying HHL.

Because of the complexities involved in determining the presence of HHL in humans, Plack et al. (2016) proposed that level and frequency dependent testing ought to give researchers more information about the nature of HHL in humans. More specifically, electrophysiological tests performed at different intensities and low and high frequencies could be useful in noise-exposed groups and control groups. In their paper, Plack et al. (2016) explained that, when participants were grouped by hearing thresholds (audiogram matched), noise exposed groups had reduced FFR amplitudes for high frequency signals, while low frequency signals were comparable between the noise exposed and the control groups. These results were expected because of the convention that noise exposure usually causes high frequency hearing loss. When participants were compared across intensity, researchers also found that high intensity signals, such as 100 dB nHL, yielded lower ABR wave 1 amplitudes and increased latencies compared to lower intensity signals. This finding works under the assumption that low spontaneous rate auditory fibers, which have increased thresholds already, are affected after noise exposure and make suprathreshold understanding more difficult if they are compromised (Plack et al., 2016).

Based on the literature review above, the presence of HHL in humans has yet to be concretely proven due to the limitations in ethical human histological analysis and because of the lack of a clinically feasible test battery. Afferent cochlear synaptopathy has been proven in the animal model because of the quick peripheral and central maturation and in the feasibility of post-mortem histological temporal bone analysis. Mice can be subject to noise that induces a TTS and their electrophysiological and other objective responses can be measured throughout their short life span. After their death, the afferent connections to the IHCs can be studied. Following a group of human participants with noise exposure until their natural deaths to analyze histological samples from temporal bones would be a lengthy and expensive process.

Additionally, the standard clinical test battery does not include the same listening in background noise and electrophysiological measures mentioned previously. Because the same methods for detecting HHL in humans is different from animals, there is a need for clinically feasible test battery that can determine with some confidence what anatomical sites are faulty. There are still many unknown variables regarding HHL in humans and there is a great need for more research building upon the findings of previous studies in this field.

Methodology

Participants

Participants with previous noise exposure through occupational or recreational means were recruited for the current study. Additionally, a control group consisting of individuals with normal hearing, little to no noise exposure, and no perceived hearing handicap were recruited. Participation was voluntary and enrollment was based on the following criteria: history of noise exposure, normal audiometric hearing thresholds, and normal middle ear pressure and compliance. Participant recruiting for the noise exposed group focused on music majors at Towson University, current or previous recreational hunters, and those with lawn care equipment (e.g. lawnmower, weed eater, leaf blower) use. Questionnaires were administered to each participant to assess their subjective hearing capabilities, their previous noise exposure, and their use of hearing protective devices. All participants had their hearing tested in both ears using insert earphones. Thresholds from 250-8,000 Hz were 20 dB HL or better for continuation in the current study. Additionally, participants had Jerger type A tympanograms, indicating normal

middle ear function, for inclusion in the current study. A (C)APD testing battery and a threshold seeking ABR were also administered for analysis in the current study. All participants gave informed consent in compliance with a protocol approved by the Institutional Review Board at Towson University.

Questionnaire

The questionnaires administered to participants included questions from the Goldsmiths Musical Sophistication Index v0.9, the Hearing Handicap Inventory for Adults, and Questionnaire 2 from Appendix I from the Liberman et al. (2016) study. These questionnaires, respectfully, probed the participants' musical background and experience, their own perceived handicap from noise exposure, and their conversational confidence in different hypothetical but practical communicative situations. The responses to the questionnaire were scored and used to categorize participants on their noise exposure history, duration of noise exposure, and their perceived handicap.

The Goldsmiths Musical Sophistication Index v1.0 is a questionnaire that focuses on musical interest, musical talent, and musical background. There are 71 total questions and five demographic questions. The first 62 questions allow participants to rate their answers for musical-based questions on a scale of 1 (completely disagree) to 7 (completely agree). Questions 63-71 ask the participant about their musical practice history, their attendance of live concerts, and their musical formal training. Finally, the last five questions pertain to occupational status, favorite musical genre, highest level of education, and personal identification information that is optional to fill out. The complete Goldsmiths Musical Sophistication Index v1.0 also includes genre sorting tasks, melody memory tasks, and beat alignment perception and production tasks; however, the current study did include the musical tasks and only administered the self-report

questionnaire to participants. The summed total of an individual's responses to the first 62 questions was calculated. This total score was referred to as the "overall score of musical sophistication" and a higher total score correlates to greater musical training and interest.

The HHIA consists of 25 questions in which the participants answered "yes", "sometimes", or "no". The "yes", "sometimes", and "no" responses have a point value of four, two, and zero, respectively. The total number of points was divided by 100 to obtain a total score percentage. Additionally, twelve out of the twenty-five questions probed into the social aspect of hearing loss, while thirteen out of the 25 questions probed the emotional aspect of a perceived hearing handicap. The total of the social responses was divided by 48, while the total number of the emotional question responses was divided by 52. The total score, the social score, and the emotional score all use the same scale in which 0-16% indicates no handicap, 18-42% indicates a mild-moderate handicap, while 44% or greater indicates a significant handicap.

Questionnaire 2 from appendix I in the Liberman et al. (2016) study focused on the participants' abilities to hear and comprehend in different hypothetical communicative environments. There are eight questions in which the participant responded by marking on a scale from zero to ten how applicable the question applies to them (zero being "not at all" and ten being "perfectly"). There is also an option for the participant to mark "not applicable" if needed. The numerical responses were summed and compared between the control and the experimental groups. A higher summed response score correlates to more communicative difficulty (Liberman et al., 2016).

Test Battery

A combination of peripheral and central measures were employed in the current study to better understand the mechanisms of HHL in humans. These tests included a full audiologic evaluation, DPOAEs, TEOAEs, administration of tests of (C)APD, and a threshold seeking ABR.

Audiologic evaluation. Participants completed a full audiologic evaluation including otoscopic examination, tympanometry, acoustic reflex thresholds (ARTs), pure tone audiometry, and speech audiometry. Tympanometry was performed using a GSI Tympstar immittance bridge. A 226 Hz probe tone was used and Jerger type A tympanograms were obtained to rule out middle ear involvement. Ipsilateral and contralateral ARTs were obtained bilaterally at 500, 1000, and 2000 Hz. ARTs of 95 dB HL or less were used for inclusion in the current study. According to Gelfand (2016), ARTs of 95 dB HL and below are considered within normal limits for those with normal hearing. Pure tone audiometry was tested in each participant using ER3A insert earphones from 250-8000 Hz using a GSI 61 audiometer. Thresholds of 20 dB HL or better were obtained within this frequency range. Speech testing included obtaining speech recognition scores using spondee words and word recognition scores using NU-6 ordered by difficulty lists.

DPOAEs and TEOAEs. Because outer hair cell integrity is essential for inclusion in the current study, DPOAEs were recorded in each ear from 1000-4000 Hz and TEOAEs were recorded from 1000-6000 Hz using the ILOv6 software. Recording for DPOAEs used the 65/55 dB intensity paradigm because of its clinical utility in assessing distortion products and outer hair cell integrity from the cochlea. DPOAEs and TEOAEs with signal-to-noise ratios of 6 dB HL or better at each testing frequency must be obtained to continue testing in the current study.

Behavioral Tests. The goal of the current study was not to assess listeners for the presence of (C)APD. Instead, the goal for introducing tests of (C)APD was to examine participants' abilities to process complex auditory signals. There are four test categories for screening for (C)APD: dichotic listening, temporal processing, monaural low redundancy speech tests, and binaural interaction. To aid in the clinical search for HHL in humans, tests of (C)APD were administered to examine any auditory processing breakdowns.

The following subtests were included in administration and scoring: The Screening Test for Auditory Processing Disorders for Adults (SCAN-3A), dichotic digits test (DDT), pitch pattern sequence (PPS), duration pattern sequence (DPS), random gap detection test (RGDT), and masking level difference (MLD).

Dichotic listening. Tests of dichotic listening involve the presentation of two competing words or sentences simultaneously to both ears (Musiek & Chermak, 2014). The dichotic word stimuli can be a combination of consonant and vowel units, digits, or monosyllabic words (Musiek & Chermak, 2014). Dichotic listening tasks ask the participant to either separate or combine the stimuli presented to each ear.

Binaural integration vs. separation. Combining the binaural stimuli is referred to as binaural integration, while separating binaural stimuli is referred as binaural separation (Chermak & Musiek, 2014). Tasks of binaural integration require the participant to focus their attention on both ears and to repeat all the binaural stimuli. Conversely, tasks of binaural separation require the participant to divide their attention to the stimuli presented in one ear while ignoring the stimuli from the contralateral ear.

DDT. The screening task assessing binaural integration to be used in the current study is the DDT. The participant was presented with simultaneous spoken digit pairs in each ear (ex. "two" and "seven" in the right ear and "four" and "three" in the left ear). The participant must repeat all four digits that are heard and are encouraged to make a guess if they are unsure. Repeating the digits in any specified order is not required. The participant's responses were marked and a percent-correct score was calculated for each ear and compared to age-appropriate normative data. (Musiek & Chermak, 2014).

Temporal Processing. Musiek and Chermak (2014) defined temporal processing as an umbrella term for several different interacting central processes involved in processing all the different components of a speech signal. These components included temporal sequencing, temporal resolution, temporal integration, and temporal masking. Temporal processing abilities are crucial for adequate and rapid comprehension of speech, especially in less than ideal communication environments such as in background noise (Musiek & Chermak, 2014).

Temporal Sequencing. Assessments of temporal sequencing include stimuli feature detection, duration discrimination, pitch discrimination, and acoustical pattern contour recognition (Chermak & Musiek, 2014). The screening tasks that were used in the current study that assess temporal patterning are the PPT and DPT.

PPT and DPT. The PPT is an assessment consisting of three tone bursts presented monaurally (Chermak & Musiek, 2014). Two of the tones are either the same high pitch (1122 Hz) or low pitch (880 Hz) while the third tone is the opposite pitch of the two same-pitch bursts. The presentation pattern of the three pitches is random and the participant verbally labeled the pitch pattern that is presented (ex. "low-high-low" or "high-high-low"). Thirty sequences were presented in one ear at a time and a percent-correct score was calculated for each ear. The DPT is

similar in structure to the PPT, but instead of pitch, the participant is listening for three 1000 Hz tone burst durations of either 250 ms (short) or 500 ms (long) duration with a 300 ms intertone interval (Musiek & Chermak, 2014). The participant verbally labeled the tone patterns (ex. "short-long-short" or "long-short-long") as they are presented to each ear individually. Again, thirty sequences were presented monaurally and a percent correct score was calculated. Percent correct scores were compared to appropriate age normative data (Musiek & Chermak, 2014).

Temporal Resolution. The shortest duration of time in which a participant can correctly discriminate between two auditory signals is referred to as temporal resolution. For listeners with normal hearing, this duration can be as short as two to three milliseconds (Phillips, 1999). For this area of temporal processing, the RGDT was administered.

RGDT. This subtest requires the participant to indicate the presence of one or two tones or clicks presented binaurally. Tone duration is 15 ms while click duration is 1 ms of white noise (Musiek & Chermak, 2014). The RGDT consists of four subtests using nine interpulse intervals ranging from 2-40 ms. The test administrator kept track of correct vs. incorrect responses from the participant and the interpulse interval. The scores for each ear were compared to normative values for the stimuli range in milliseconds. (Musiek & Chermak, 2014).

Monaural Low Redundancy. The peripheral and central auditory system is created in such a way so that there are multiple redundancies to help facilitate auditory processing (Musiek & Chermak, 2014). The system uses extrinsic redundancies of the auditory signal (ex. Pitch, intensity, duration) and intrinsic redundancies of the physiologic structure of the auditory pathways such that parallel pathways send information to the CANS simultaneously (Musiek & Chermak, 2014). Tests of monaural low redundancy utilize degraded extrinsic redundancies to assess the integrity of the participant's intrinsic redundancies.

Spectral degradation vs. temporal degradation. The two branches of monaural low redundancy tests include spectral degradation and temporal degradation (Musiek & Chermak, 2014). Spectrally degraded stimuli are either altered by pitch or are presented in background noise. Temporally degraded stimuli are altered in the time domain. This means that the stimuli, usually speech, are either sped up or slowed down. The SCAN-3A is a test popular test that assesses both branches of monaural low redundancy in adults (Musiek & Chermak, 2014).

SCAN-3A. The subtests included in the SCAN-3A include filtered words (FW), auditory figure ground (AFG), competing words (CW) and competing sentences (CS) (Musiek & Chermak, 2014). The FW subtest features words filtered with a low-pass filter with a cut-off at 750 Hz. The words are presented monaurally and a percent-correct score is calculated for each ear which is then compared to appropriate norms. The AFG subtest uses a +4 dB signal to noise ratio in which the participant is instructed to listen for a word in multi-talker babble noise and repeat or guess the word that was presented. This test was administered and scored monaurally by calculating a percent correct score and comparing that score to normative data (Musiek & Chermak, 2014).

Although the CS and CW subtests are tests of binaural separation, an auditory process of dichotic listening, they are unique in the current study because they are linguistically loaded (Musiek & Chermak, 2014). In the CS subtest, a total of twenty trials were presented, ten in which the participant attended to and repeated the sentence that was said in the right ear and then ten trials in which sentences only in the left ear were repeated. The same testing paradigm was used for the CW test, except singular words were used instead of sentences (Musiek & Chermak, 2014).

Binaural Interaction. Binaural interaction is the combined effect of listening with both ears as opposed to hearing with restriction to only one ear (Musiek & Chermak, 2014). Binaural interaction is not a phenomenon that begins in the peripheral auditory system; rather, it begins in the brainstem at the level of the superior olivary complex. Abilities such as localization and hearing in background noise require the interaction of balanced and functional binaural integration.

Homophasic vs. antiphasic. The most common test of binaural integration is the MLD test. This test refers to the intensity difference of signal detection thresholds in homophasic and antiphasic masking paradigms (Musiek & Chermak, 2014). Homophasic masking is known as the reference condition because the signals are in phase with one another and the masking noise is in phase with one another, respectively (Musiek & Chermak, 2014). This is the most difficult testing condition due to the very low signal to noise phase ratio. Conversely, antiphasic masking is a condition in which the either the signal or the noise masker are 180 degrees out of phase with respect to each other. This condition exemplifies the greatest release from masking coupled with the best signal to noise ratio (Musiek & Chermak, 2014).

MLD. The MLD was administered using a 500 Hz tone and a narrow-band masker centered around this pitch (Musiek & Chermak, 2014). The participant was instructed to listen for a tone and pressed the button when the tone was heard while also being instructed to ignore the masking noise as best as possible. The homophasic masking condition was tested first, followed by the antiphasic masking conditions. Once the threshold for the antiphasic threshold had been subtracted from the homophasic threshold, that masking level difference was compared to normative data to determine if there is a concern for binaural integration (Musiek & Chermak, 2014).

Threshold seeking ABR. Finally, threshold seeking ABR was performed on each subject as a validation of their normal hearing thresholds and to demonstrate objectively any suprathreshold declines in hearing. In the animal model discussion, researchers used threshold seeking ABR on noise-exposed mice and found that after the effects of a TTS had dissipated, suprathreshold ABR recordings were found to have increased latencies and abnormal morphology. Suprathreshold ABR wave V latencies and amplitudes will be examined to see if similar differences exist in human listeners with and without noise exposure. Threshold seeking ABRs were recorded to tone burst stimuli at 500, 2000, and 4000 Hz. The intensities will sweep from 80 dB nHL down to 20 dB nHL.

In the Kujawa and Liberman study (2009), click-evoked threshold-seeking ABRs were used to test mice that were exposed to noise. It was hypothesized by Kujawa and Liberman (2009) that as the TTS was reversed, when click-evoked ABRs were tested from approximately 10 dB nHL below threshold up to 90 dB nHL, increased wave V latencies and decreased wave V amplitudes would be recorded due the effects of cochlear synaptopathy. In the current study, a threshold-seeking ABR utilizing a click stimulus will also be used. ABR wave V amplitudes and latencies were recorded. The electrode montage used in the current study included a non-inverting (Cz) lead on the top of the forehead, two inverting leads on each mastoid (A1 and A2), and one common ground lead on the forehead just above the midline of the eyebrows (Fpz).

Analysis

Analysis of the data collected in the current study incorporated non-parametric t-tests and analysis of variance (ANOVA) for making comparisons between the control and experimental group along each of the subjective and objective tests. Non-parametric T-Tests (Mann Whitney U) and one way ANOVAs (Kruskal Wallis) were used to compare each test and subtest of

(C)APD between the control and experimental groups. Finally, non-parametric tests such as the Kruskal Wallis and Mann Whitney U were used to compare the threshold-seeking ABR results between the control and experimental groups.

Results

Due to the low number of total completed participants (n = 6), normal distribution of the data could not be assumed; therefore, non-parametric equivalent tests of the one-way ANOVA (Kruskal-Wallis) and t-Test (Mann-Whitney U) were executed in the SPSS software to analyze the data. Ear-specific TEOAE, DPOAE, APD test battery, and threshold ABR results will be discussed.

Otoacoustic Emissions

TEOAEs. Table 1 displays the mean TEOAE dB SNR values and standard deviations for each participant group at each test frequency and for each ear.

Table 1

Mean TEOAE SNR Values (dB SNR) and Standard Deviation for Left and Right Ears as a Function of Test Frequency

Group by ear	1000 Hz (SD)	1400 Hz (SD)	2000 Hz (SD)	2800 Hz (SD)	4000 Hz (SD)
Control Left	14.97 (5.51)	17.17 (2.87)	11.00 (4.57)	12.53 (5.97)	6.67 (6.95)
Control Right	17.53 (2.27)	15.23 (2.68)	12.13 (2.10)	8.73 (3.54)	12.93 (7.53)
Experimental Left	-1.47 (11.60)	-0.03 (9.07)	3.87 (11.42)	1.47 (10.50)	2.27 (3.13)
Experimental Right	-0.53 (8.90)	4.87 (13.31)	9.54 (12.72)	8.57 (5.17)	6.20 (3.28)

Right ear.

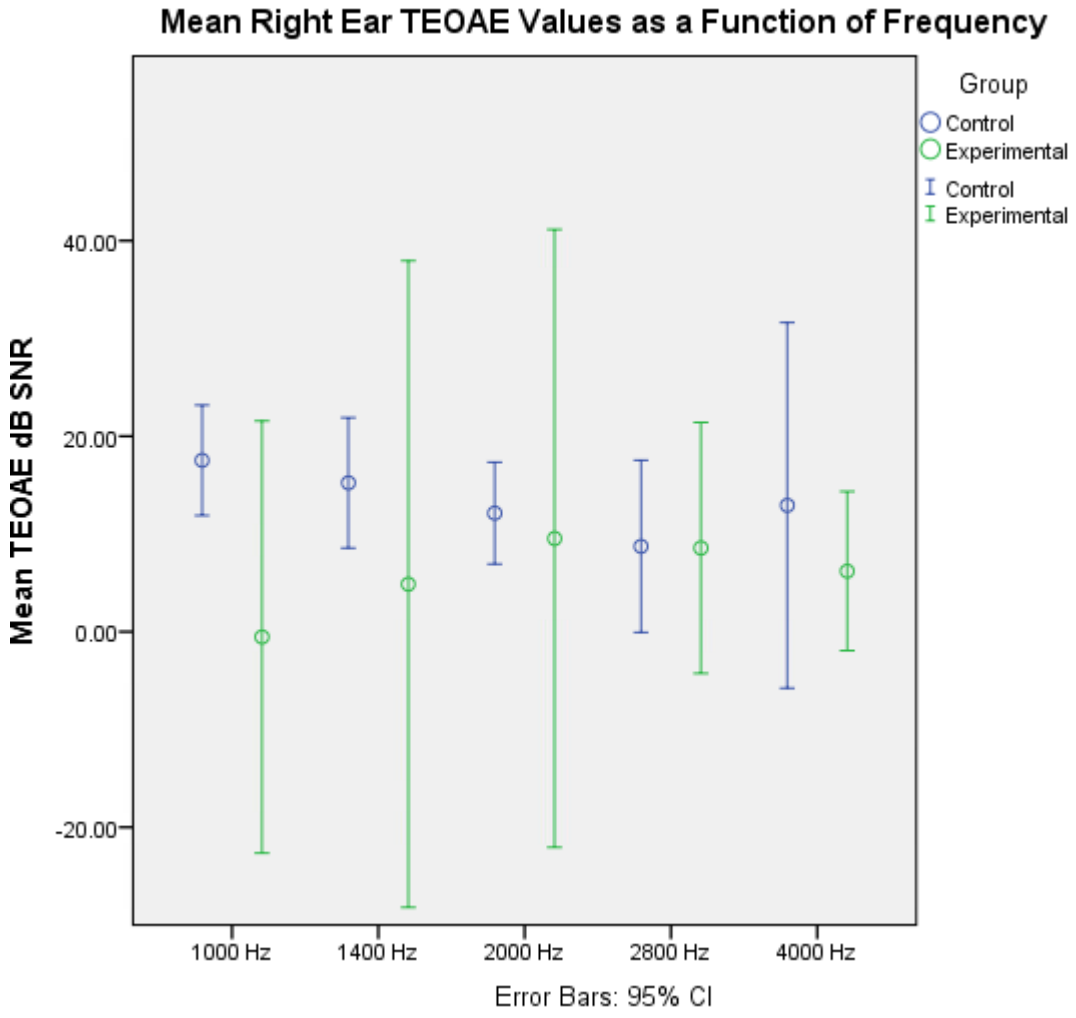


Figure 1. Right ear TEOAE mean SNR values from 1000 Hz through 4000 Hz for control and experimental group participants. Error bars represent a 95% confidence interval around the mean.

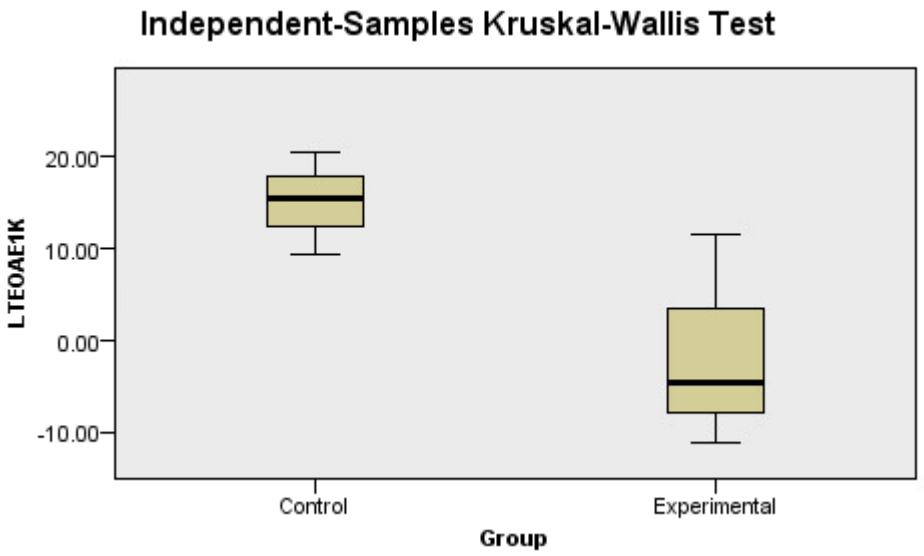


Figure 2. Box plot of right ear TEOAE SNRs at 1000 Hz for both participant groups.

Figure 1 plots the mean dB SNR values of both participant groups' right ear TEOAEs as a function of test frequency. Visually, the control group achieved higher mean TEOAE dB SNR values for this ear as compared to the experimental group across the frequency range; this excludes 2800 Hz where the mean dB SNR values are almost identical. A Kruskal-Wallis H test was run to determine if there were any differences in right ear TEOAE SNR values for the control (n=3) and experimental (n=3) groups at the different test frequencies. Distribution of SNR values was similar at 1400, 2000, 2800, and 4000 Hz and were not statistically significant, $p>0.05$. However, distribution of the right ear TEOAE SNR values was not similar at 1000 Hz, as visualized by boxplot inspection in figure 2. The distribution of the SNR values for 1000 Hz in the right ear was statistically lower for the experimental group compared to the control group, χ^2 (1) = 3.857, p = .05.

Left ear.

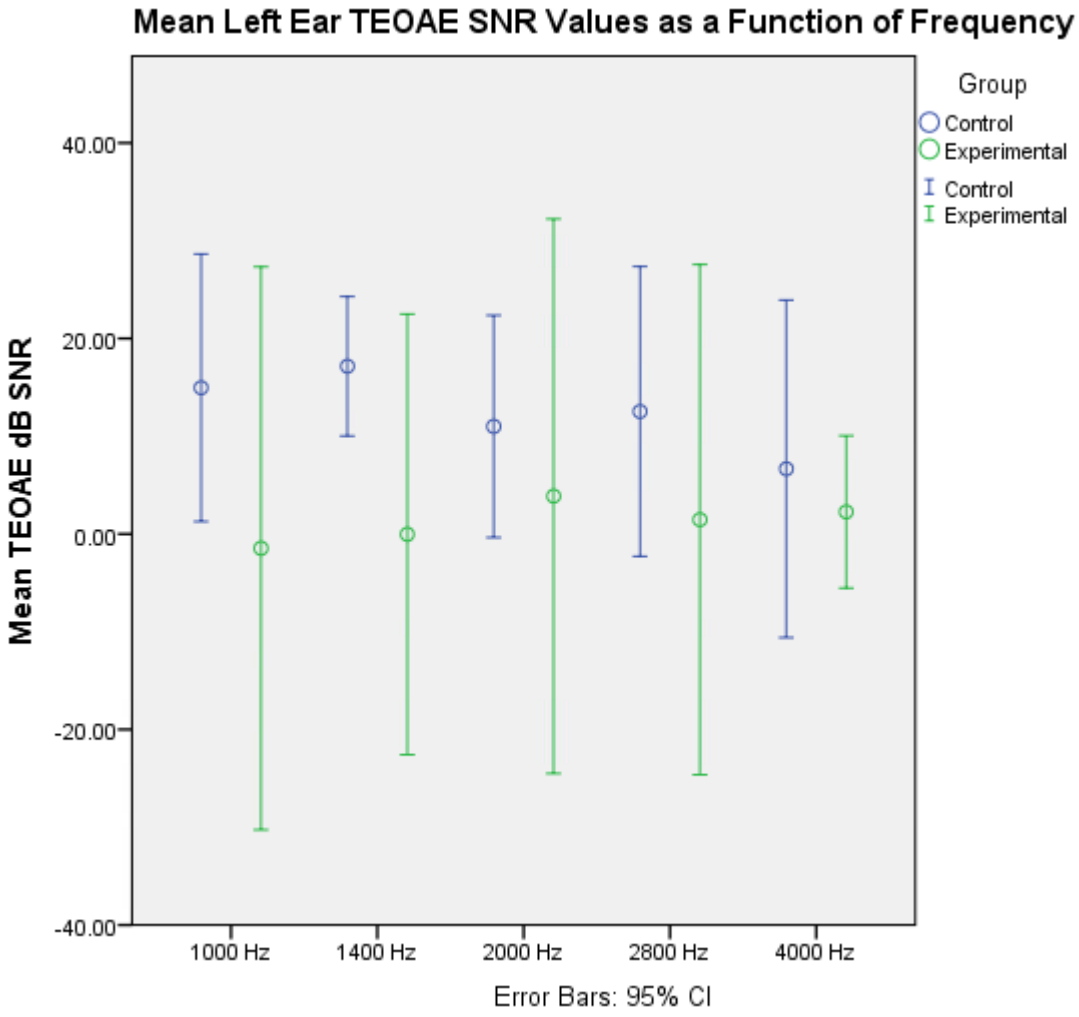


Figure 3. Left ear TEOAE mean SNR values from 1000 Hz through 4000 Hz for control and experimental group participants. Error bars represent a 95% confidence interval around the mean.

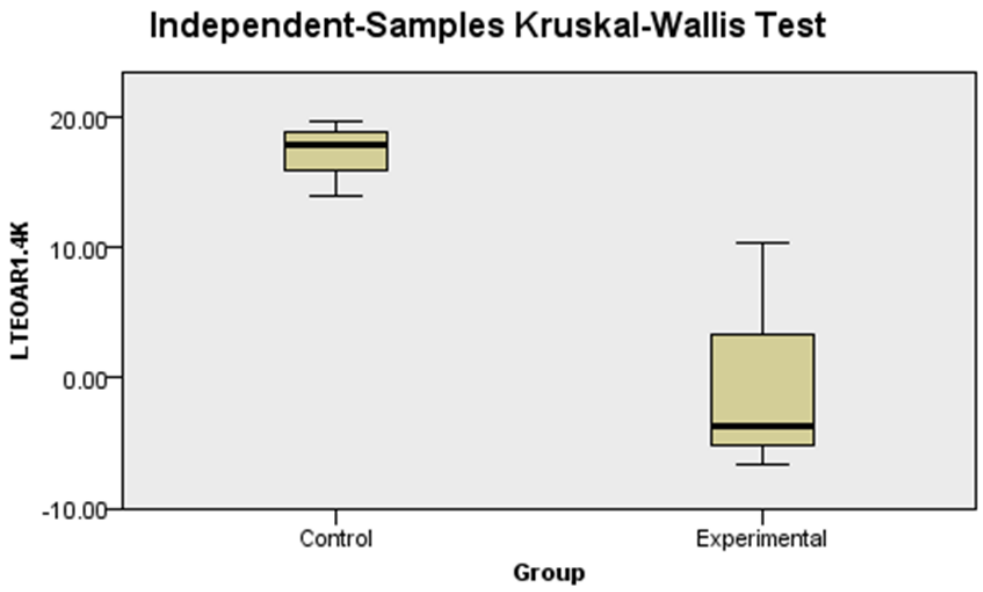


Figure 4. Box plot of left ear TEOAE SNRs at 1400 Hz for both participant groups.

Figure 3 plots the mean dB SNR values of both participant groups' left ear TEOAEs as a function of test frequency. As seen, the control group on average achieved higher dB SNR values across the test frequency range. A Kruskal-Wallis H test was run to determine if there were any differences in left ear TEOAE SNR values for the control (n=3) and experimental (n=3) groups at the different test frequencies. Distribution of SNR values was similar at 1000, 2000, 2800, and 4000 Hz and were not statistically significant, $p>0.05$. However, distribution of the left ear TEOAE SNRs was not similar at 1400 Hz, as visualized by boxplot inspection in figure 4. The distribution of the SNR values for 1400 Hz in the left ear was statistically significantly lower for the experimental group compared to the control group, χ^2 (1) = 3.857, p = .05.

DPOAEs. Table 2 displays the mean DPOAE SNR values and standard deviations for each participant group at each test frequency and for each ear.

Table 2

Mean DPOAE SNR Values (dB SNR) and Standard Deviation for Left and Right Ears as a Function of Test Frequency

Group by Ear	1000 Hz (SD)	1400 Hz (SD)	2000 Hz (SD)	2800 Hz (SD)	4000 Hz (SD)	6000 Hz (SD)
Control Left	9.20 (8.84)	18.43 (6.23)	20.03 (0.84)	14.47 (8.01)	21.80 (8.93)	25.00 (1.83)
Control Right	6.30 (2.76)	15.40 (6.35)	19.87 (1.21)	17.17 (6.48)	20.03 (8.78)	22.03 (2.95)
Experimental Left	8.20 (8.76)	10.87 (5.20)	12.00 (10.84)	13.73 (8.31)	11.73 (9.40)	8.90 (3.03)
Experimental Right	10.07 (7.11)	11.33 (4.84)	21.27 (3.84)	14.80 (8.15)	20.80 (7.57)	20.60 (0.30)

Right ear.

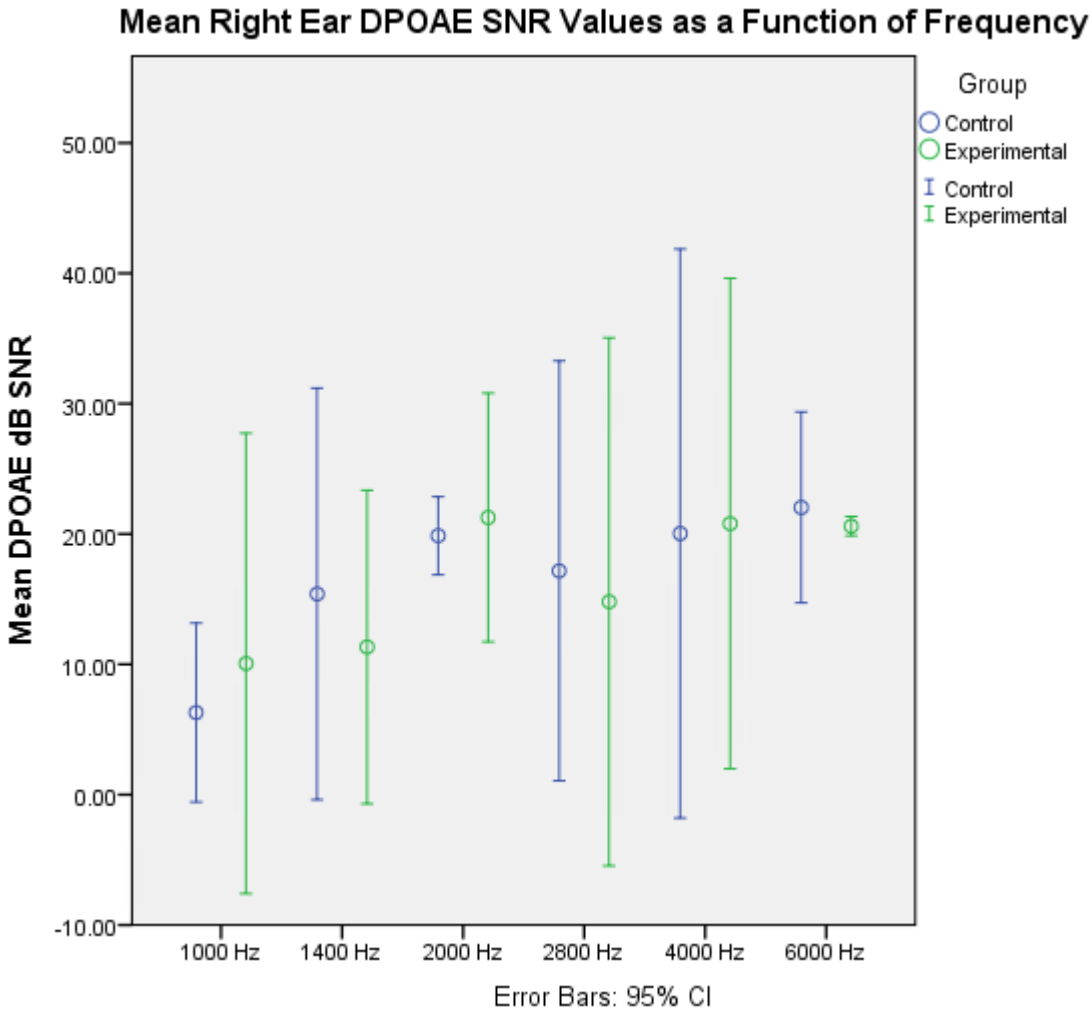


Figure 5. Right ear DPOAE mean SNR values from 1000 Hz through 6000 Hz for control and experimental group participants. Error bars represent a 95% confidence interval around the mean.

Figure 5 plots the mean dB SNR values of both participant groups' right ear DPOAEs as a function of test frequency. This figure demonstrates a much more similar spread of mean dB SNR values for this ear from 2000-6000 Hz. At 1000 Hz the experimental group achieved higher mean dB SNR values compared to the control group, but this result pattern is reversed at the

subsequent 1400 Hz. A Kruskal-Wallis H test was run to determine if there were any differences in right ear DPOAE SNR values for the control (n=3) and experimental (n=3) groups at the different test frequencies. Distribution of the right ear DPOAE scores was similar at all test frequencies for both groups, as visualized by boxplot inspection. Distribution of right ear DPOAE SNRs at 1000 Hz, χ^2 (1) = 0.784, p = .376, at 1400 Hz, χ^2 (1) = 1.190, p = .275, at 2000 Hz, χ^2 (1) = 0.048, p = .827, at 2800 Hz, χ^2 (1) = 0.429, p = .513, at 4000 Hz, χ^2 (1) = 0.048, p = .827, and at 6000 Hz, χ^2 (1) = 0.429, p =.513, was not statistically significantly different.

Left ear.

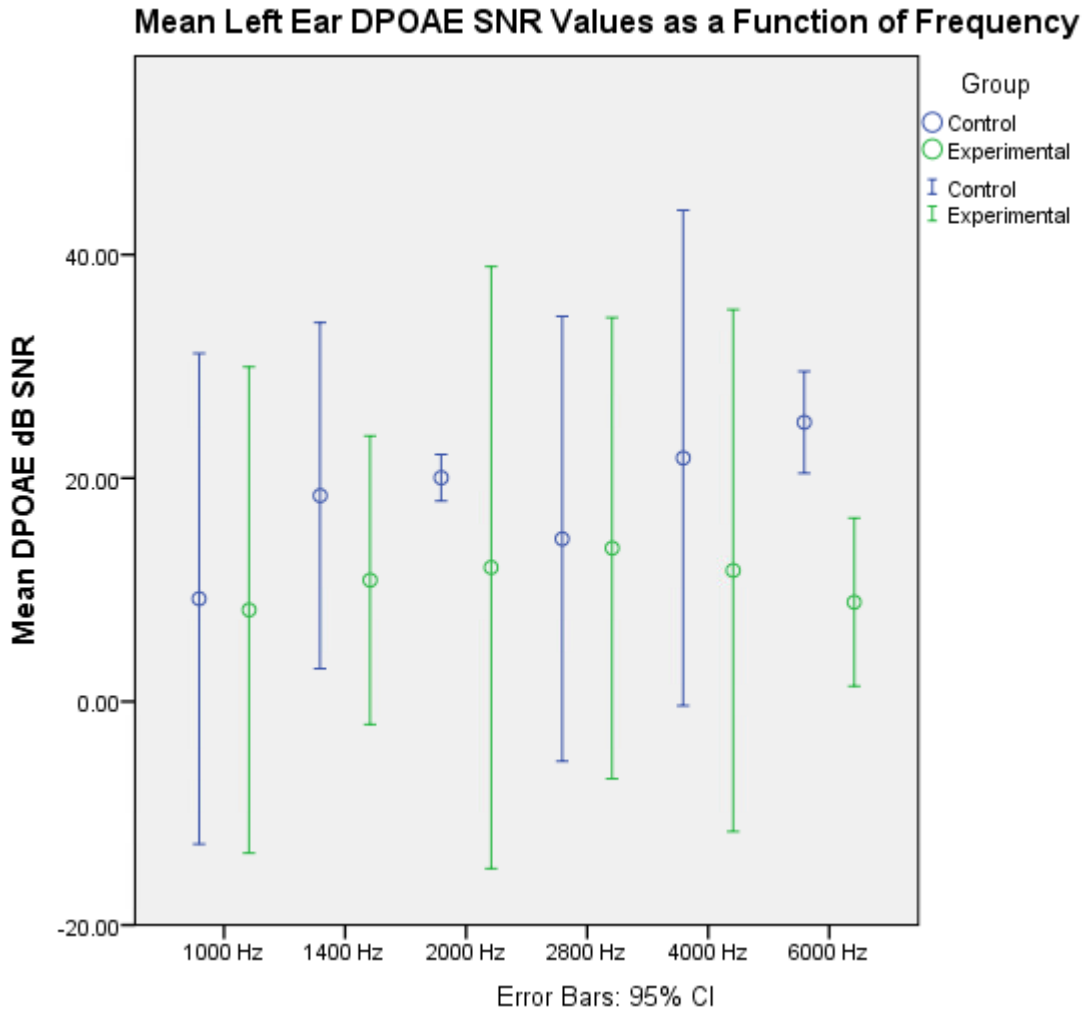


Figure 6. Left ear DPOAE mean SNR values from 1000 Hz through 6000 Hz for control and experimental group participants. Error bars represent a 95% confidence interval around the mean.

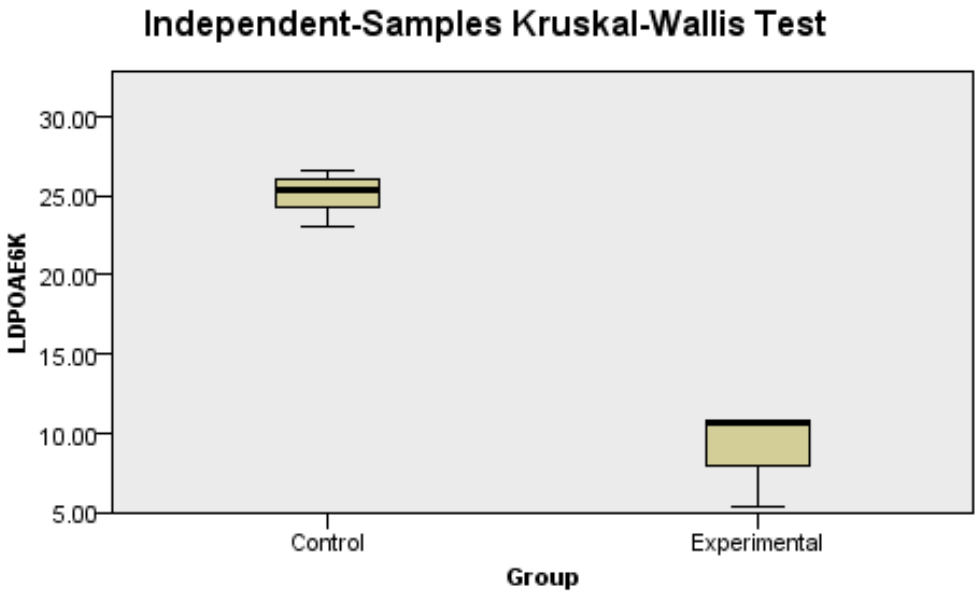


Figure 7. Box plot of left ear DPOAE SNRs at 6000 Hz for both participant groups.

Figure 6 plots the mean dB SNR values of both participant groups' right ear DPOAEs as a function of test frequency. Similar mean dB SNR value distributions are observed at 1000 and 2800 Hz; for the remaining test frequencies, the control group achieved higher mean dB SNR ratios for this ear. A Kruskal-Wallis H test was run to determine if there were any differences in left ear DPOAE SNR values for the control (n=3) and experimental (n=3) groups at the different test frequencies. Distribution of SNR values was similar at 1000, 1400, 2000, 2800, and 4000 Hz and were not statistically significant, $p>.05$. However, distribution of the left ear DPOAE SNR values was not similar at 6000 Hz, as visualized by boxplot inspection in figure 7. The distribution of the SNR values for 6000 Hz in the left ear was statistically lower for the experimental group compared to the control group, χ^2 (1) = 3.857, p = .05.

(C)APD Tests

Dichotic Listening. Table 3 displays the mean score percentages and standard deviations of dichotic listening for the DDT for each participant in the right and left ears.

Table 3

Mean Ear-Specific Scores (%) and Standard Deviation for Dichotic Listening

Group	Right Ear DDT	Left Ear DDT
Control	98.33 (1.44)	100.00 (0.00)
Experimental	92.50 (0.00)	95.00 (2.50)

Dichotic digits test.

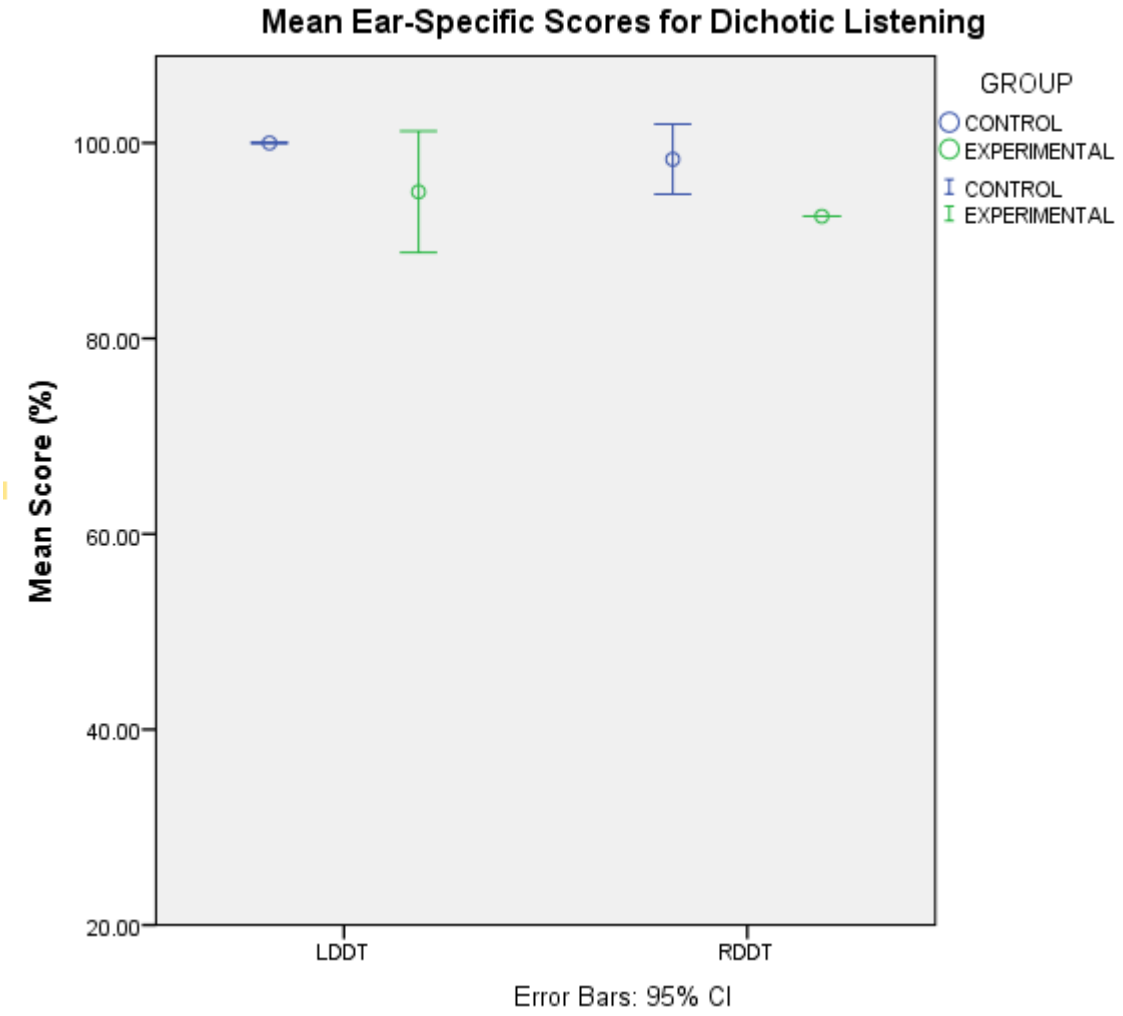


Figure 8. Right and left ear DDT scores (%) for the control and experimental groups. Error bars represent a 95% confidence interval around the mean.

Figure 8 illustrates the similar average of DDT scores of the right and left ears between the two participant groups. Mann Whitney U tests were run to determine if there were differences in scores for right and left ears between each group. Both groups scored very high and performed exceptionally well.

Right ear. There was not a statistically significant difference between the right ear DDT scores for control vs. experimental group, as evident by Mann Whitney U analysis (U = 0.00, z = -2.121, p = .100).

Left ear. There was not a statistically significant difference between the left ear DDT scores for control vs. experimental group, as evident by Mann Whitney U analysis (U = 0.000, z = -2.087, p = .100).

Temporal Processing. Table 4 displays the mean score percentages and standard deviations of temporal processing tests for each participant group in the right and left ears.

Table 4

Mean Ear-Specific Scores (%) and Standard Deviation for Temporal Processing

Group	Right Ear PPS	Left Ear PPS	Right Ear DPS	Left Ear DPS
Control	99.00 (1.73)	99.90 (0.17)	94.33 (5.13)	99.00 (1.73)
Experimental	97.90 (1.82)	96.57 (3.50)	94.67 (4.04)	94.87 (2.98)

Pitch pattern sequence.

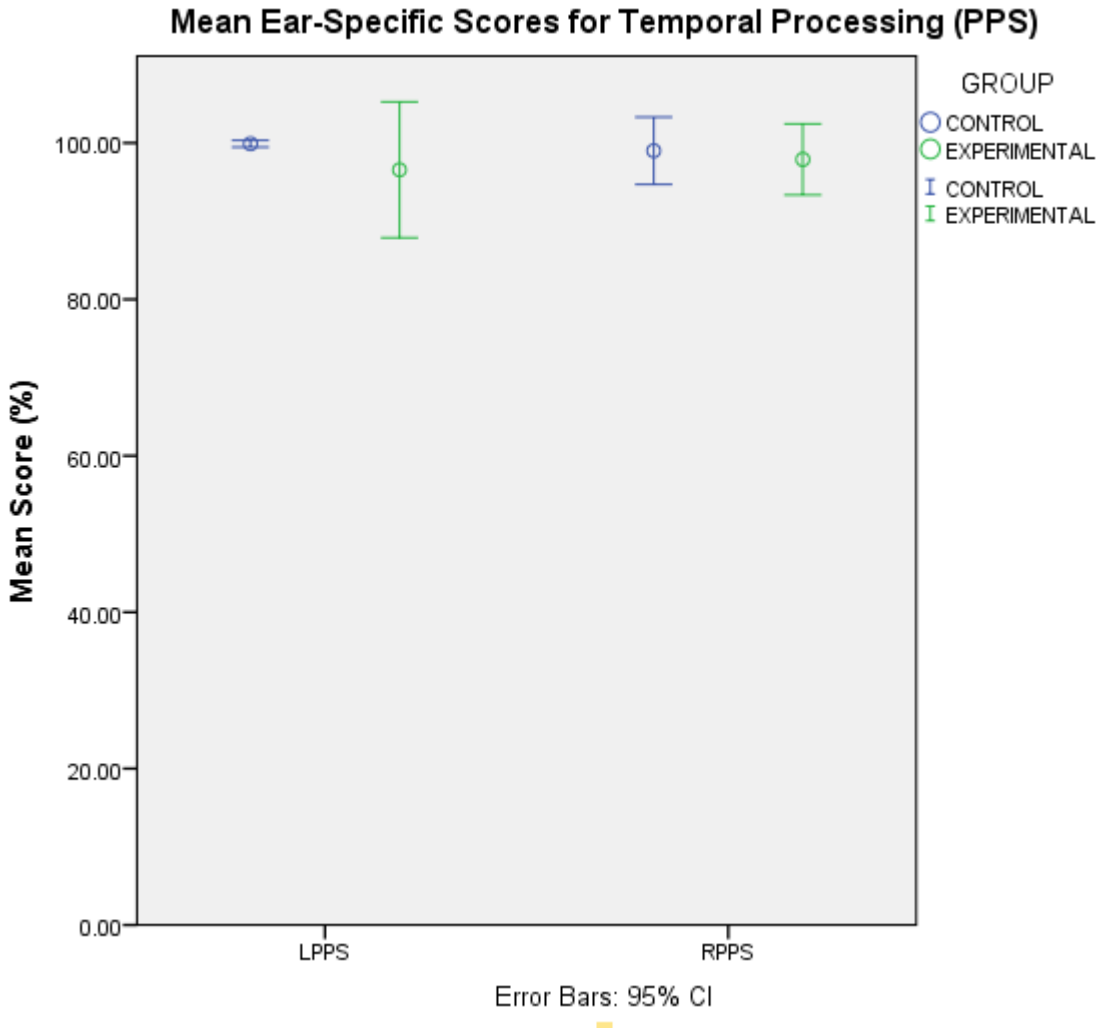


Figure 9. Right and left ear PPS scores for the control and experimental groups. Error bars represent a 95% confidence interval around the mean.

Figure 9 also illustrates the similar average of PPS scores of the right and left ears between the two participant groups. Mann Whitney U tests were run to determine if there were differences in scores for right and left ears between each group. Both groups scored very high and performed exceptionally well.

Right ear. There was not a statistically significant difference between the right ear PPS scores for control vs. experimental group, as evident from Mann Whitney U analysis (U = 2.500, z = -0.943, p = .400).

Left ear. There was not a statistically significant difference between the left ear PPS scores for control vs. experimental group, as evident by Mann Whitney U analysis (U = 2.000, z = -1.159, p = .400).

Duration pattern sequence.

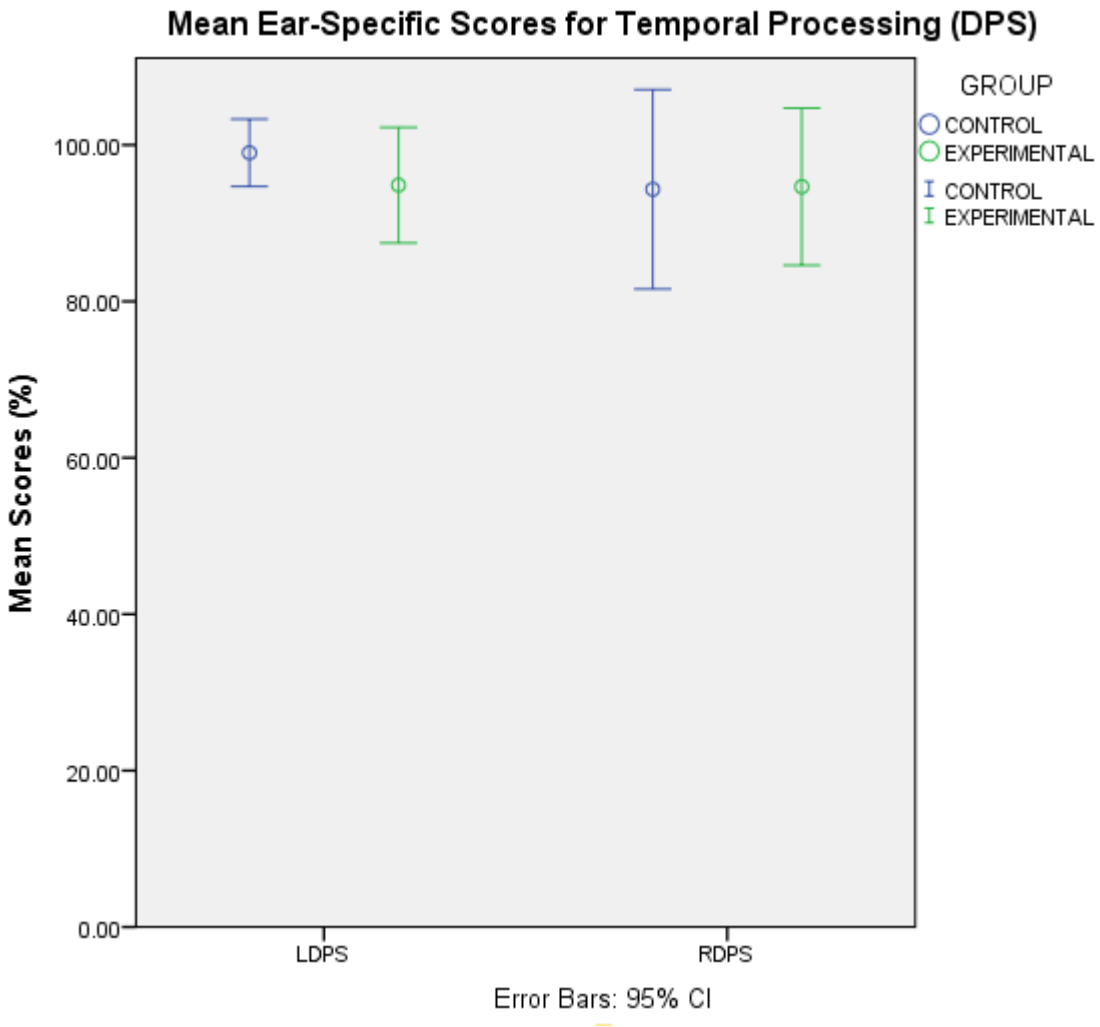


Figure 10. Right and left ear DPS scores for control and experimental groups. Error bars represent a 95% confidence interval around the mean.

Again, figure 10 illustrates the similar average of DPS scores of the right and left ears between the two participant groups. Mann Whitney U tests were run to determine if there were differences in scores for right and left ears between each group. Both groups scored very high and performed exceptionally well.

Right ear. There was not a statistically significant difference between the right ear DPS scores for control vs. experimental group, as evident by Mann Whitney U analysis (U = 4.500, z = 0.000, p = 1.000).

Left ear. There was not a statistically significant difference between the left ear DPS scores for control vs. experimental group, as evident by Mann Whitney U analysis (U = 1.000, z = -1.550, p = .200).

Temporal Resolution. Table 5 displays the mean random gap detection times and standard deviations of the RGDT for each participant.

Table 5

Mean Scores and Standard Deviation for Binaural RGDT times (ms) for Each Group

Group	500 Hz (SD)	1000 Hz (SD)	2000 Hz (SD)	4000 Hz (SD)	Click (SD)
Control	4.00 (1.73)	4.00 (1.73)	6.67 (2.89)	6.67 (2.89)	5.67 (4.04)
Experimental	8.33 (2.89)	11.67 (7.64)	13.33 (5.77)	8.33 (2.89)	8.33 (2.89)

Random gap detection test.

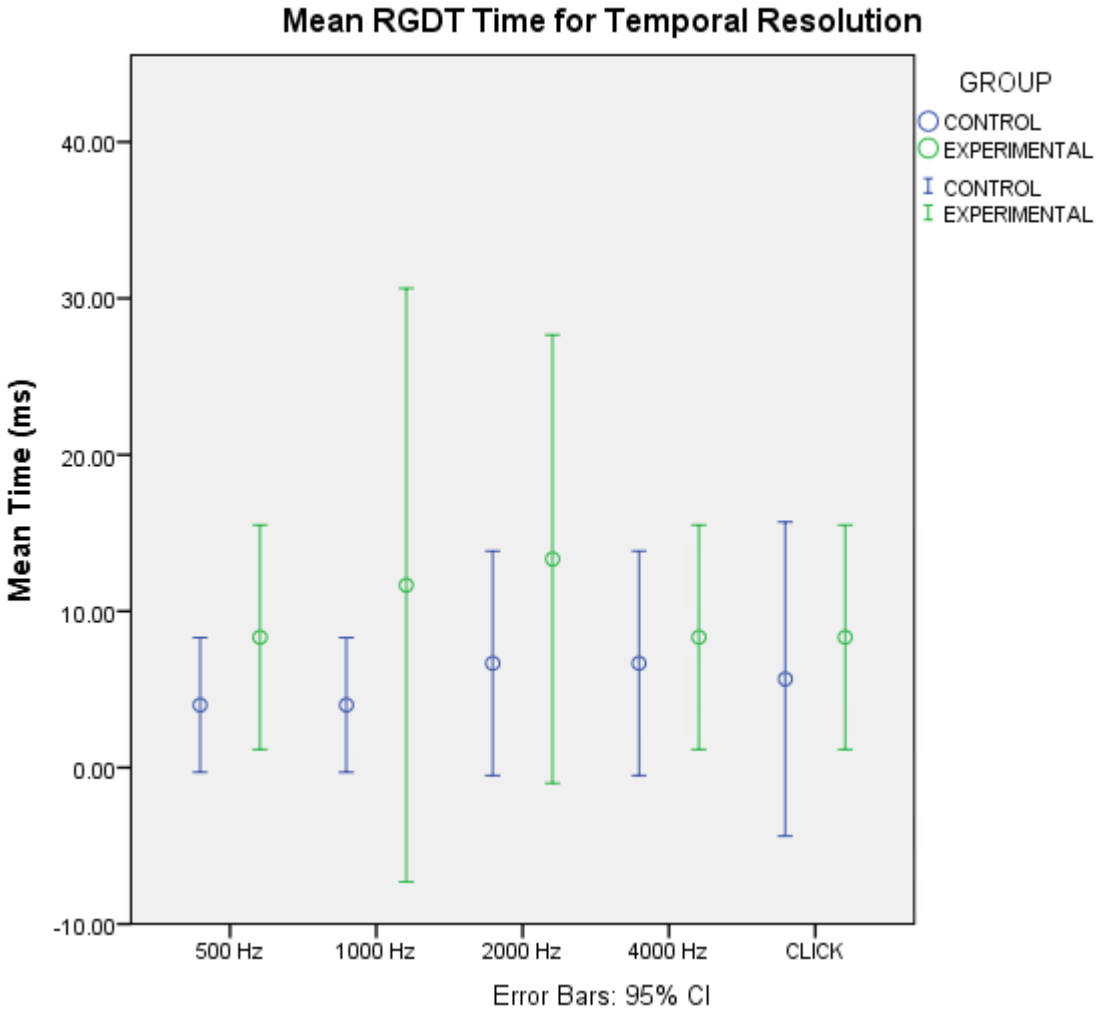


Figure 11. Mean time (ms) for each participant to detect the lowest random gap in stimuli at 500 Hz, 1000 Hz, 2000 Hz, 4000 Hz, and a click stimulus. Error bars represent a 95% confidence interval around the mean.

For this test of temporal resolution, a lower detection time is desirable. It is evident in this figure that the control group did achieve lower gap detection times as compared to the experimental group. A Mann-Whitney U test was run to determine there were any differences in scores for the random gap detection test in the binaural condition for the control (n=3) and

experimental (n=3) groups. Scores for 500 Hz (U = 8.000, z = 1.650, p = .200), 1000 Hz (U = 8.000, z = 1.623, p = .200), 2000 Hz (U = 8.000, z = 1.623, p = .200), 4000 Hz (U = 6.000, z = 0.745, p =.700), and click stimulus (U = 6.500, z = 0.943, p = .400) were all not statistically significantly different between groups using an exact sampling distribution for U.

Monaural Low Redundancy. Table 6 displays the mean SCAN 3A auditory composite scores and standard deviations for each participant in the right and left ears.

Table 6

Mean Composite Scores and Standard Deviation for Monaural Low Redundancy

Group	SCAN 3A
Control	121 (4.58)
Experimental	109 (4.58)

SCAN 3A.

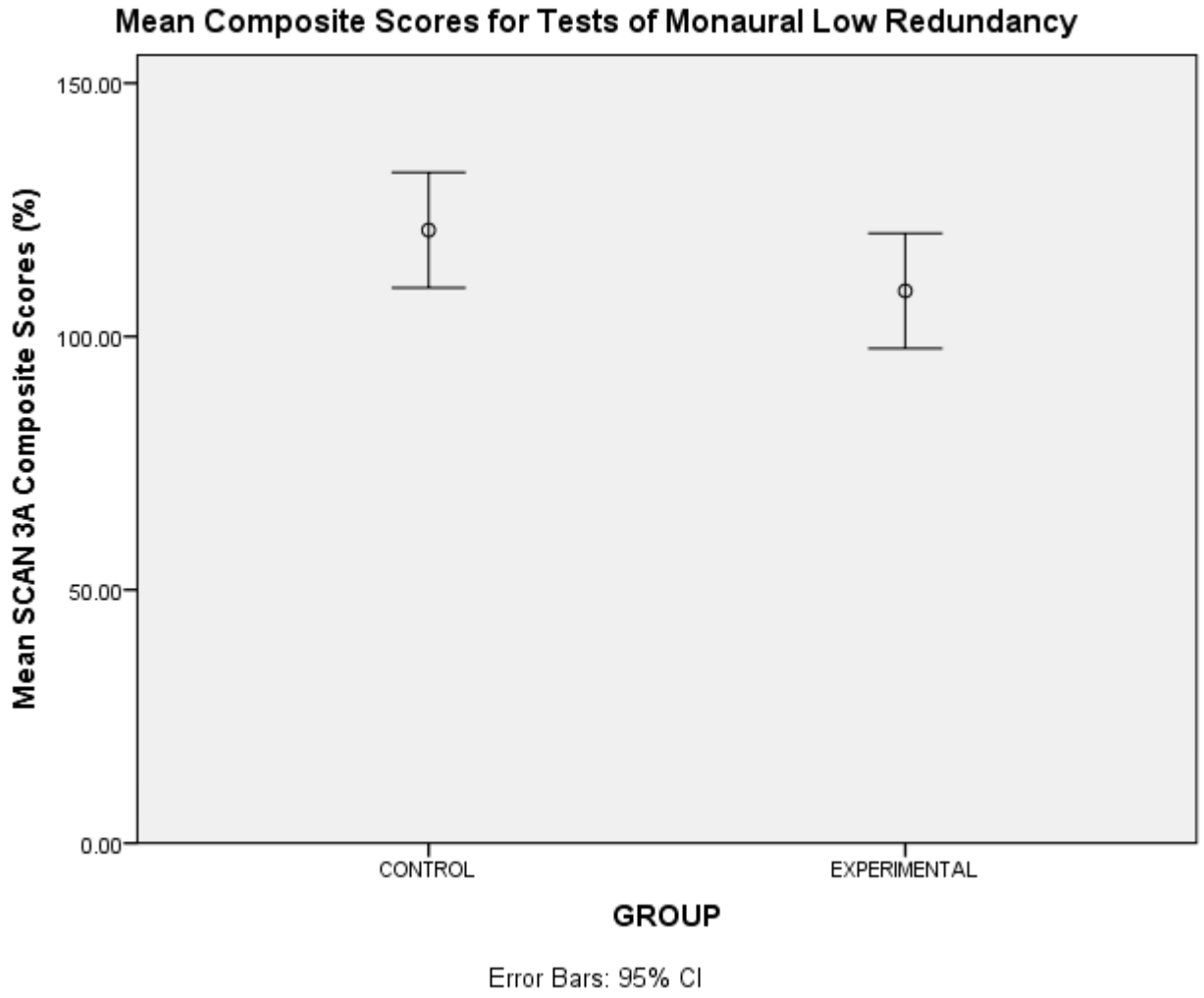


Figure 12. Mean age-normed auditory composite scores resulting from the different SCAN 3A tests for each participant group. Error bars represent a 95% confidence interval around the mean.

The control group achieved higher (better) SCAN 3A auditory composite scores when visually compared to the experimental group. A Mann-Whitney U was run to determine if there was any difference in scores for the SCAN 3 test auditory composite score for the control (n=3) and experimental (n=3) groups. There was no statistically significant difference in scores between both groups, U = 0.000, z = -1.964, p = .100, using an exact sampling distribution for U.

Binaural Interaction. Table 7 displays the mean masking level difference scores and standard deviations for each participant group in the right and left ears.

Table 7

Mean Composite Scores and Standard Deviation for Binaural Interaction in dB

Group	MLD
Control	15.33 (2.31)
Experimental	15.33 (3.06)

Masking level difference.

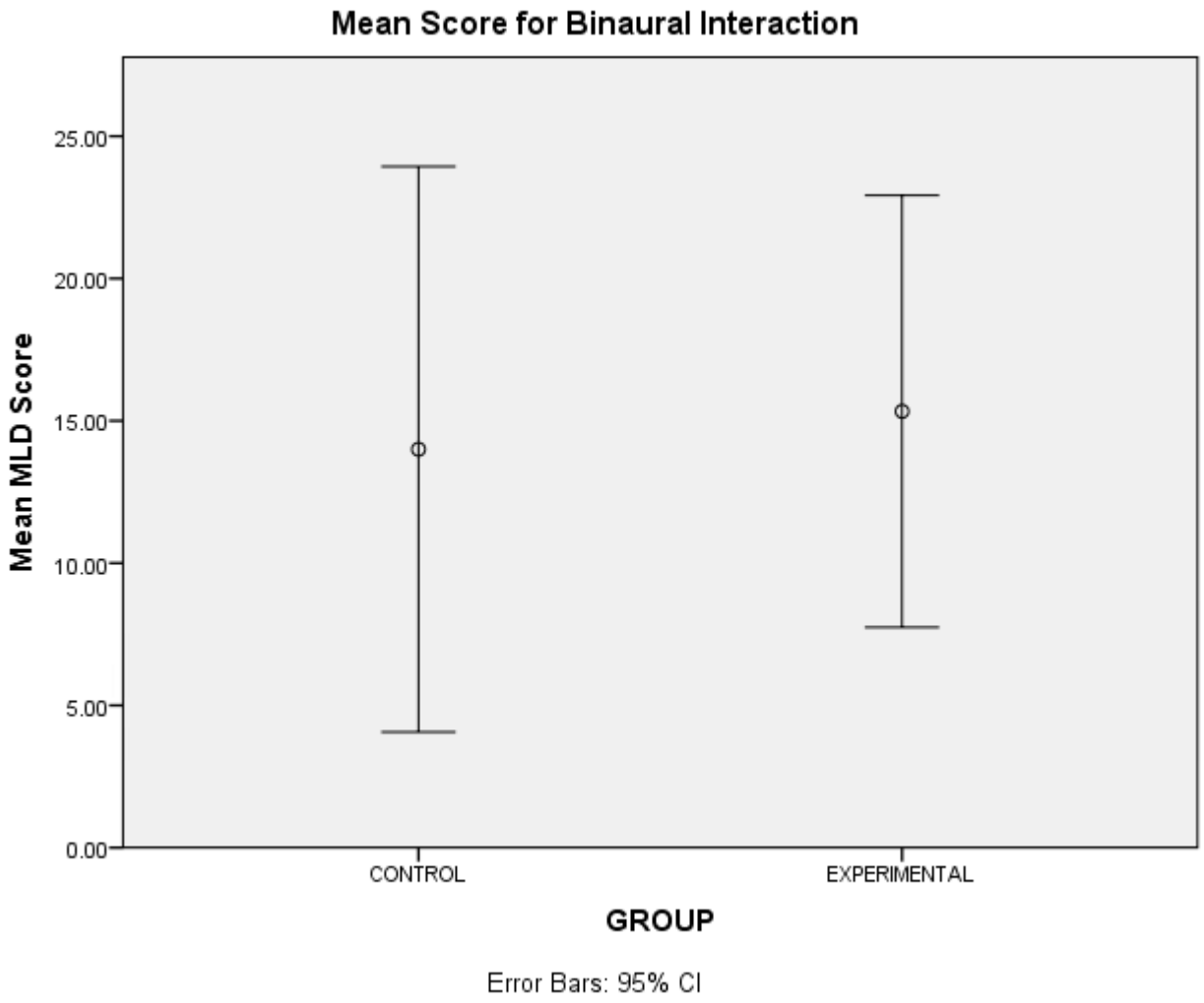


Figure 13. Mean MLD scores for the control and experimental groups. Error bars represent a 95% confidence interval around the mean.

In figure 13, the control group achieved lower (better) masking level difference scores compared to the experimental groups. Error bars for both groups exhibited a rather large range. A Mann-Whitney U was run to determine if there was any difference in scores for the masking level difference test in the binaural condition for the control (n=3) and experimental (n=3)

groups. There was no statistically significant difference in scores between both groups, U = 5.500, z = 0.443, p = .700, using an exact sampling distribution for U.

Threshold ABR

Amplitude. Tables 8, 9, 10, and 11 display the mean amplitudes (μV) and standard deviations for wave V in the right and left ears as tonal stimulus intensity decreases.

Table 8

500 Hz Threshold ABR Mean Amplitudes as a Function of Intensity, Ear, and Participant Group

Participant Group	80 dB nHL (SD)	60 dB nHL (SD)	40 dB nHL (SD)	20 dB nHL (SD)
Control Right	0.33 (0.15)	0.30 (0.13)	0.20 (0.06)	NA
Experimental Right	0.32 (0.10)	0.34 (0.21)	0.52 (NA)	NA
Control Left	0.25 (0.09)	0.25 (0.15)	NA	NA
Experimental Left	0.40 (0.04)	0.27 (0.13)	0.12 (0.04)	NA

Table 9

1000 Hz Threshold ABR Mean Amplitudes as a Function of Intensity, Ear, and Participant Group

Participant Group	80 dB nHL (SD)	60 dB nHL (SD)	40 dB nHL (SD)	20 dB nHL (SD)
Control Right	0.44 (0.25)	0.25 (0.06)	0.21 (NA)	NA
Experimental Right	0.30 (0.02)	0.34 (0.20)	0.20 (0.05)	NA
Control Left	0.31 (0.06)	0.15 (0.07)	0.15 (0.06)	NA
Experimental Left	0.37 (0.01)	0.40 (0.22)	0.15 (0.07)	NA

Table 10

2000 Hz Threshold ABR Mean Amplitudes as a Function of Intensity, Ear, and Participant Group

Participant Group	80 dB nHL (SD)	60 dB nHL (SD)	40 dB nHL (SD)	20 dB nHL (SD)
Control Right	0.31 (0.11)	0.23 (0.05)	0.14 (0.04)	0.16 (NA)
Experimental Right	0.41 (0.08)	0.20 (0.03)	0.20 (0.05)	NA
Control Left	0.30 (0.04)	0.17 (0.03)	0.17 (0.04)	NA
Experimental Left	0.25 (0.01)	0.16 (0.02)	0.17 (0.03)	0.12 (NA)

Table 11

4000 Hz Threshold ABR Mean Amplitudes as a Function of Intensity, Ear, and Participant Group

Participant Group	80 dB nHL (SD)	60 dB nHL (SD)	40 dB nHL (SD)	20 dB nHL (SD)
Control Right	0.35 (0.05)	0.25 (0.12)	0.17 (0.07)	0.12 (NA)
Experimental Right	0.38 (0.07)	0.28 (0.09)	0.18 (0.03)	0.10 (NA)
Control Left	0.35 (0.06)	0.24 (0.07)	0.16 (0.01)	0.05 (NA)
Experimental Left	0.41 (0.02)	0.29 (0.01)	0.16 (0.03)	0.12 (0.04)

Right ear.

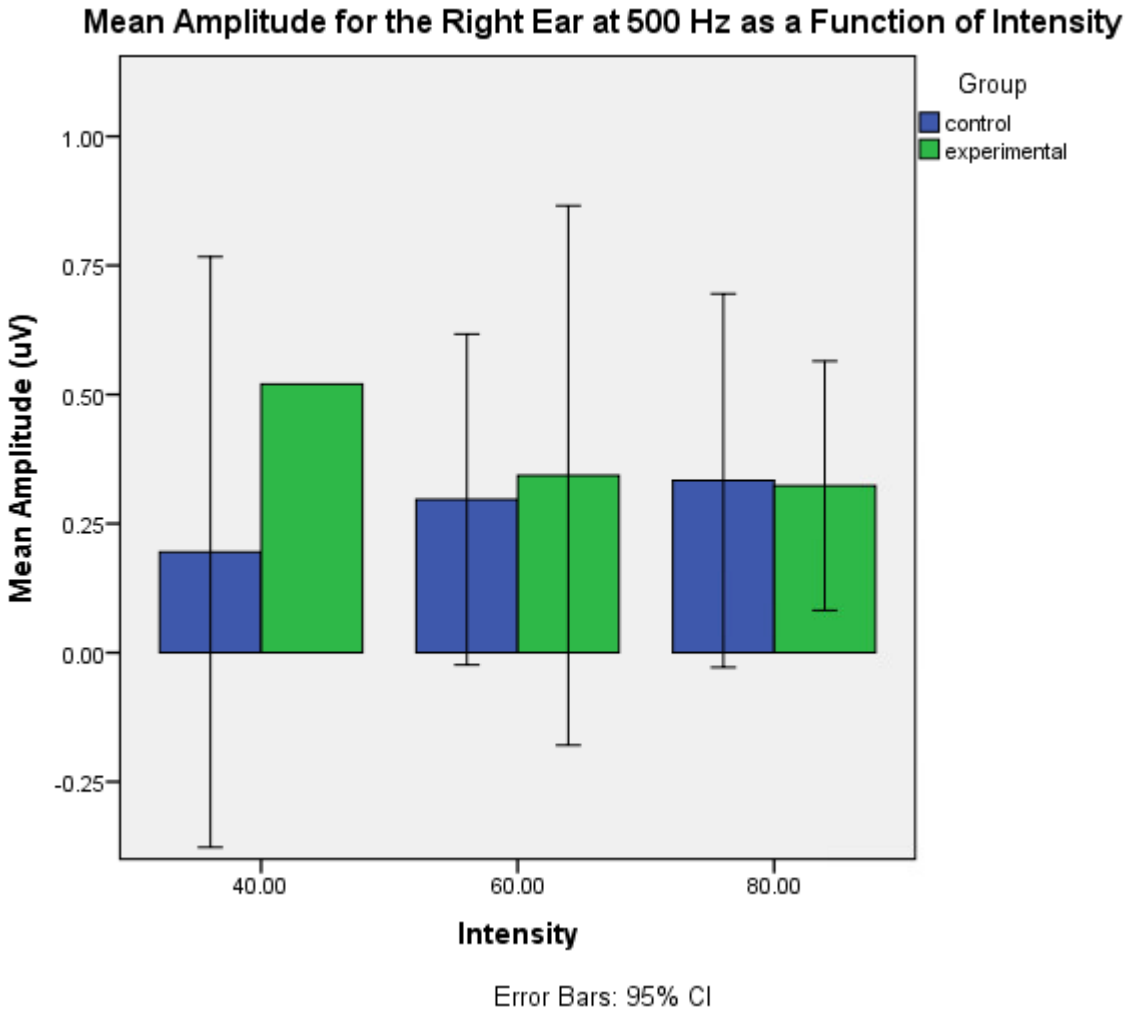


Figure 14. Mean amplitude (μV) of threshold ABR wave V at 500 Hz as intensity decreases from 80 dB nHL. Error bars represent a 95% confidence interval around the mean.

At 500 Hz in the right ear, ABR tracings exhibited poor morphology and amplitude of wave V across the participant groups, although a few participants did have measurable and repeatable wave V tracings. Amplitude stayed approximately the same from 80 to 60 dB nHL, and then decreased in the right ear for the control group and increased in the experimental group. A Kruskal-Wallis H test was run to determine if there were any differences in 500 Hz threshold

ABR amplitude values at 80, 60, 40, and 20 dB nHL in the right ear for the control (n=3) and experimental (n=3) groups. Distribution of the right ear 500 Hz threshold ABR amplitude values was similar for both groups, as visualized by boxplot inspection. The differences between the control and experimental group amplitude values were not statistically significantly different at 80 dB nHL, χ^2 (1) = 0.48, p = .827, at 60 dB nHL, χ^2 (1) = 0.048, p =.827, and at 40 dB nHL, χ^2 (1) = 1.500, p = .221. Comparisons could not be performed for 20 dB nHL because wave V latencies and amplitudes could not be measured at this intensity.

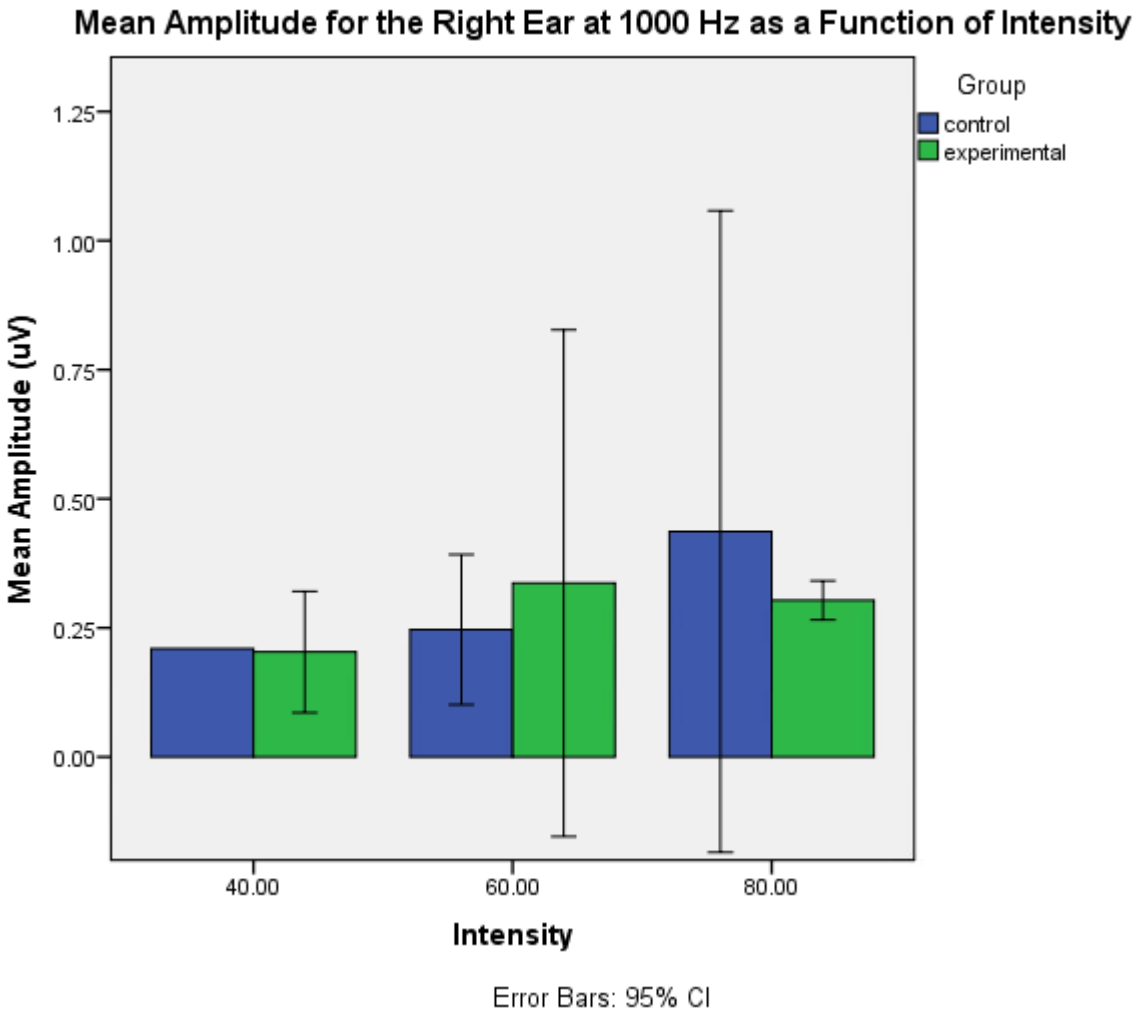


Figure 15. Mean amplitude (μV) of threshold ABR wave V at 1000 Hz in the right ear as intensity decreases from 80 dB nHL. Error bars represent a 95% confidence interval around the mean.

Mean amplitudes for this frequency in the right ear decrease from 80 dB nHL to 60 dB nHL and plateau at 40 dB nHL for the control group. Experimental group amplitude means stay approximately the same as intensity decreases. A Kruskal-Wallis H test was run to determine if there were any differences in 1000 Hz threshold ABR amplitude values at 80, 60, 40, and 20 dB nHL in the right ear for the control (n=3) and experimental (n=3) groups. Distribution of the

right ear 1000 Hz threshold ABR amplitude values was similar for both groups, as visualized by boxplot inspection. The differences between the control and experimental group amplitude values were not statistically significantly different at 80 dB nHL, χ^2 (1) = 0.429, p = .513, at 60 dB nHL, χ^2 (1) = 0.429, p = .513, and at 40 dB nHL, χ^2 (1) = 0.200, p = .655. Comparisons could not be performed for 20 dB nHL because wave V latencies and amplitudes could not be measured at this intensity.

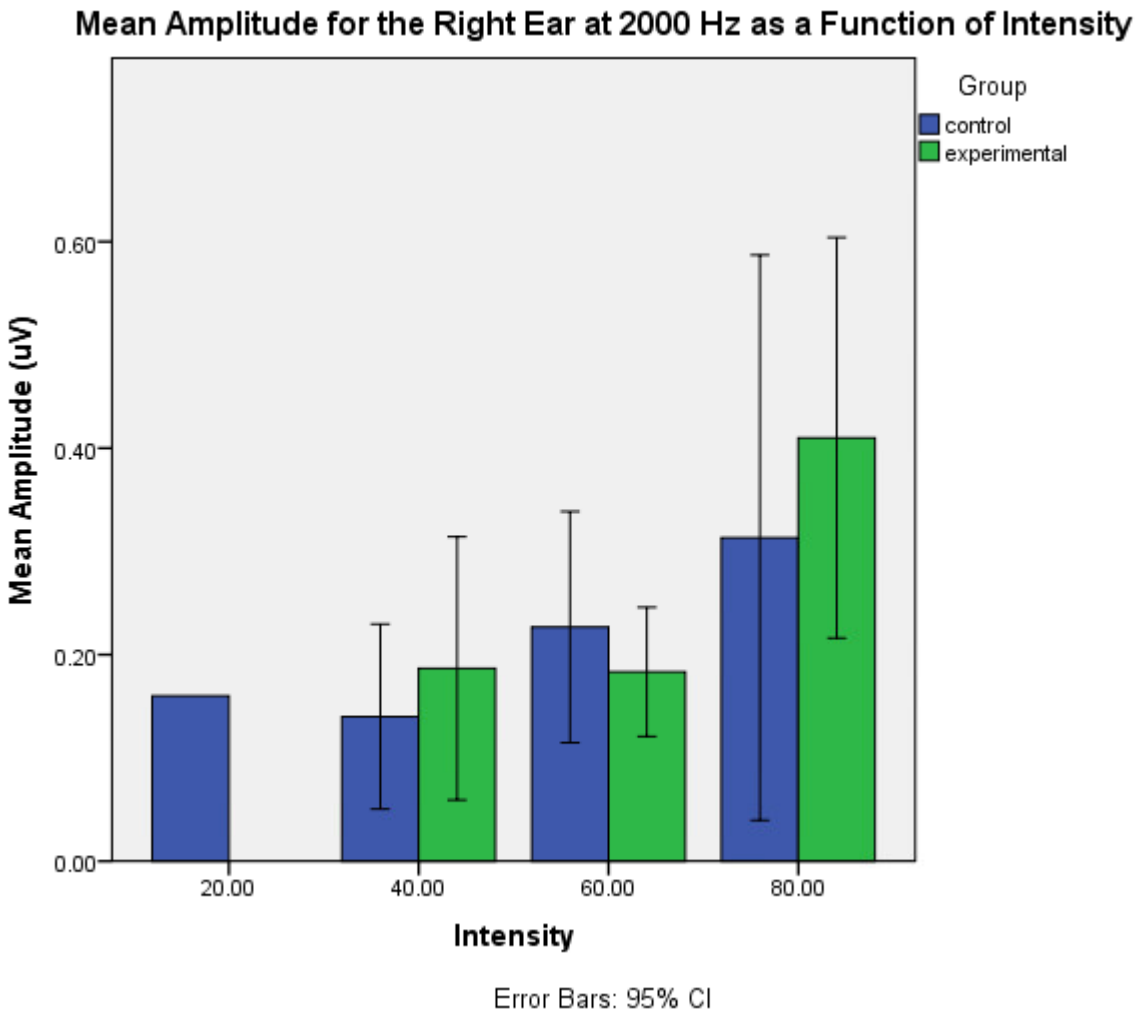


Figure 16. Mean amplitude (μV) of threshold ABR wave V for the right ear at 2000 Hz as intensity decreases from 80 dB nHL. Error bars represent a 95% confidence interval around the mean.

As seen, mean wave V amplitudes decrease for both participant groups from 80 to 60 dB nHL. From 60 to 40 dB nHL, amplitudes for the control group continue to decrease while amplitudes increase slightly for the experimental group. At 20 dB nHL in the control group the wave V amplitudes does not decrease from 40 dB nHL. A Kruskal-Wallis H test was run to determine if there were any differences in 2000 Hz threshold ABR amplitude values at 80, 60,

40, and 20 dB nHL in the right ear for the control (n=3) and experimental (n=3) groups. Distribution of the right ear 2000 Hz threshold ABR amplitude values was similar for both groups, as visualized by boxplot inspection. The differences between the control and experimental group amplitude values were not statistically significantly different at 80 dB nHL, χ^2 (1) = 2.333, p = .127, at 60 dB nHL, χ^2 (1) = 1.765, p = .184, and at 40 dB nHL, χ^2 (1) = 1.190, p = .275. Comparisons could not be performed for 20 dB nHL because wave V latencies and amplitudes could not be measured at this intensity.

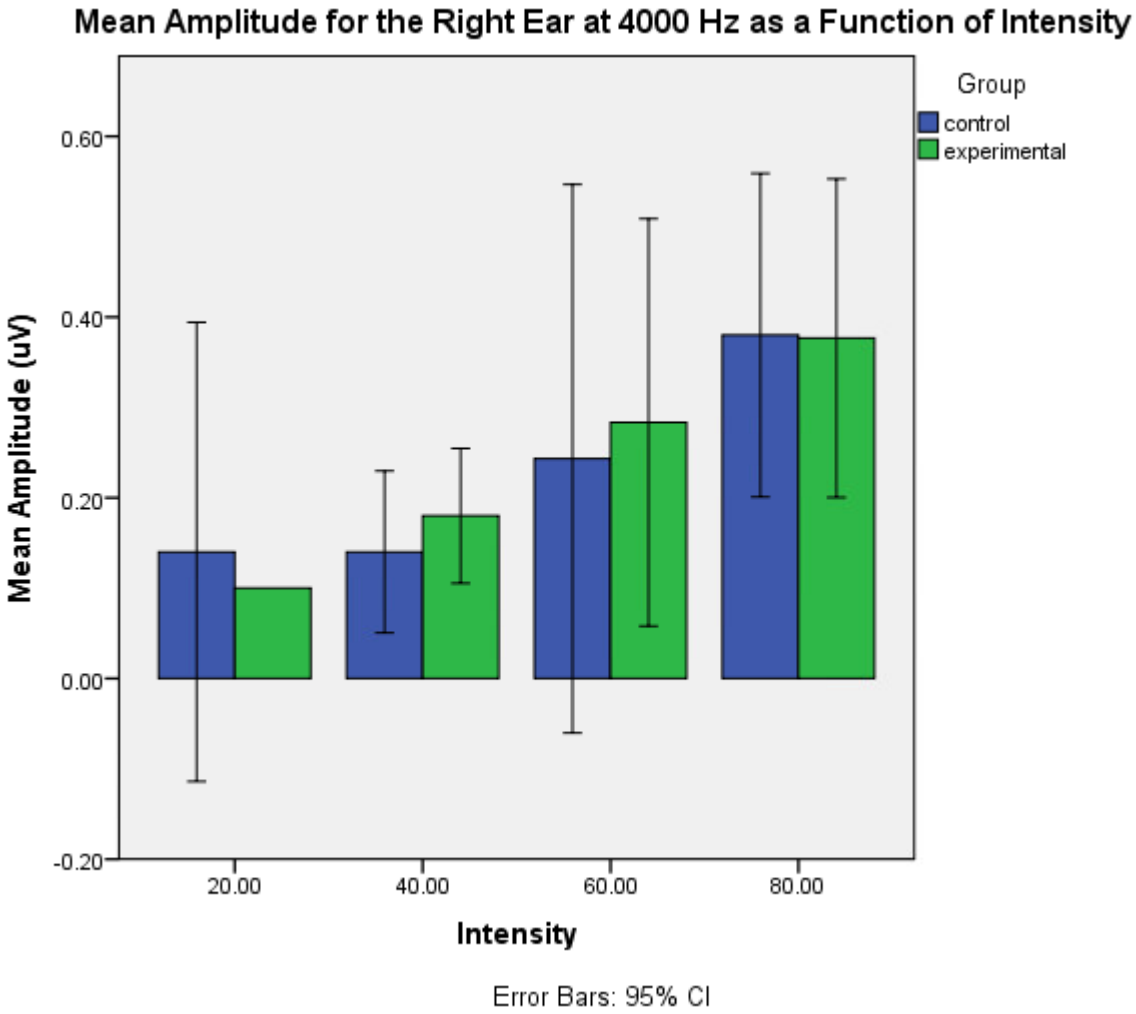


Figure 17. Mean amplitude (μV) of threshold ABR wave V at 4000 Hz in the right ear as intensity decreases from 80 dB nHL. Error bars represent a 95% confidence interval around the mean.

At 4000 Hz, mean right ear wave V amplitudes are almost the same at 80 dB nHL for the control and experimental groups; however, as intensity decreases to 40 dB nHL, the experimental group mean wave V amplitudes are slightly higher compared to the control group. At 20 dB nHL, this reverses as the control group attains slightly higher mean wave V amplitudes. A Kruskal-Wallis H test was run to determine if there were any differences in 4000 Hz threshold

ABR amplitude values at 80, 60, 40, and 20 dB nHL in the right ear for the control (n=3) and experimental (n=3) groups. Distribution of the right ear 4000 Hz threshold ABR amplitude values was similar for both groups, as visualized by boxplot inspection. The differences between the control and experimental group amplitude values were not statistically significantly different for 80 dB nHL, χ^2 (1) = 0.196, p = .658, at 60 dB nHL, χ^2 (1) = 0.048, p = .827, at 40 dB nHL, χ^2 (1) = 0.048, p = .827, and at 20 dB nHL, χ^2 (1) = 1.000, p = .317.

Left ear.

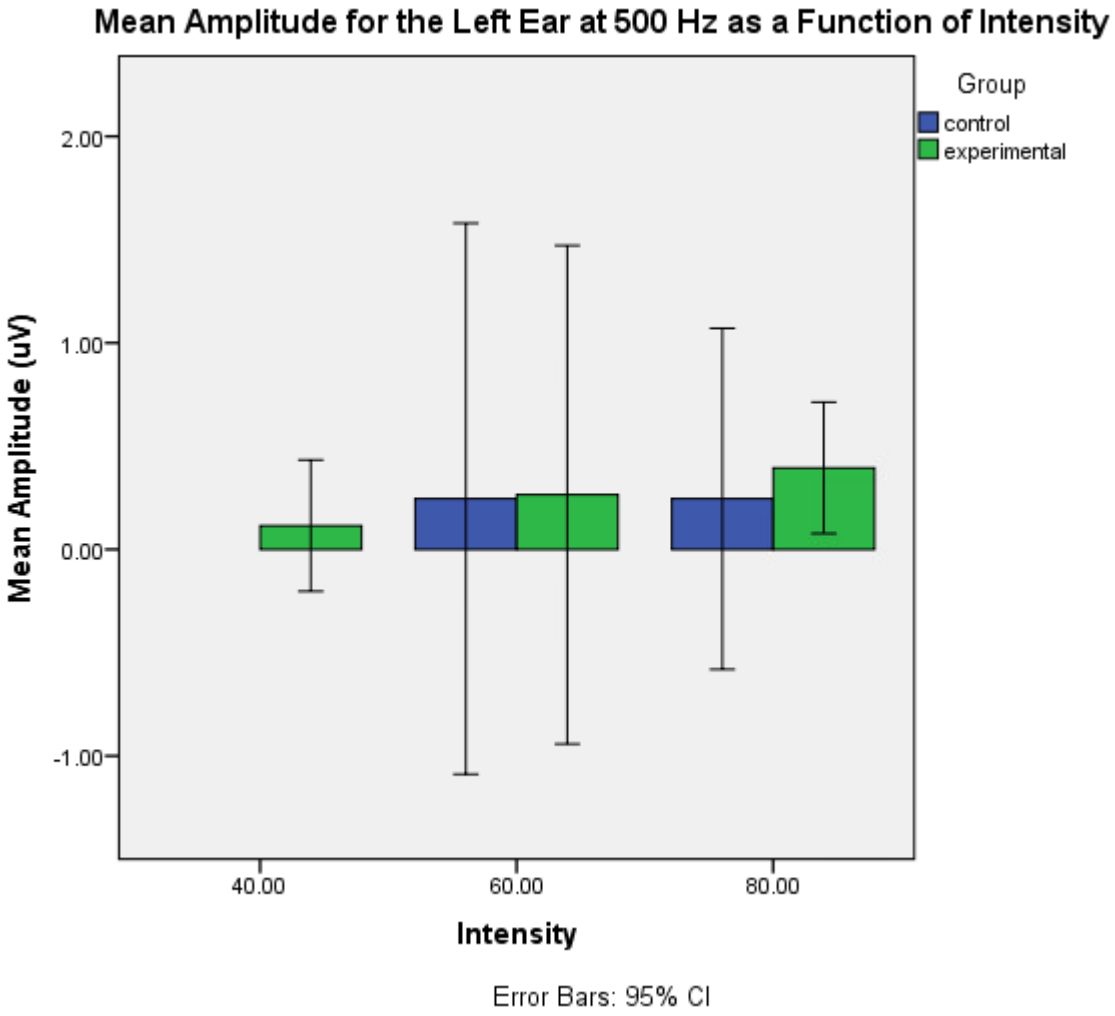


Figure 18. Mean amplitude (μV) of threshold ABR wave V at 500 Hz for the left ear as intensity decreases from 80 dB nHL. Error bars represent a 95% confidence interval around the mean.

At 500 Hz in the left ear, ABR tracings exhibited poor morphology and amplitude of wave V across the participant groups, although a few participants did have measurable and repeatable wave V tracings. As intensity decreases from 80 to 40 dB nHL, the experimental group mean wave V amplitudes are seen to decrease; for the control group, amplitudes stay about the same from 80 to 60 dB nHL and then could not be measured onward. A Kruskal-Wallis H

test was run to determine if there were any differences in 500 Hz threshold ABR amplitude values at 80, 60, 40, and 20 dB nHL in the left ear for the control (n=3) and experimental (n=3) groups. Distribution of the left ear 500 Hz threshold ABR amplitude values was similar for both groups, as visualized by boxplot inspection. The differences between the control and experimental group amplitude values were not statistically significantly different for 80 dB nHL, χ^2 (1) = 2.400, p = .121, and at 60 dB nHL, χ^2 (1) = 0.600, p = .439. Comparisons could not be performed for 40 and 20 dB nHL because some wave V latencies and amplitudes could not be measured at this intensity.

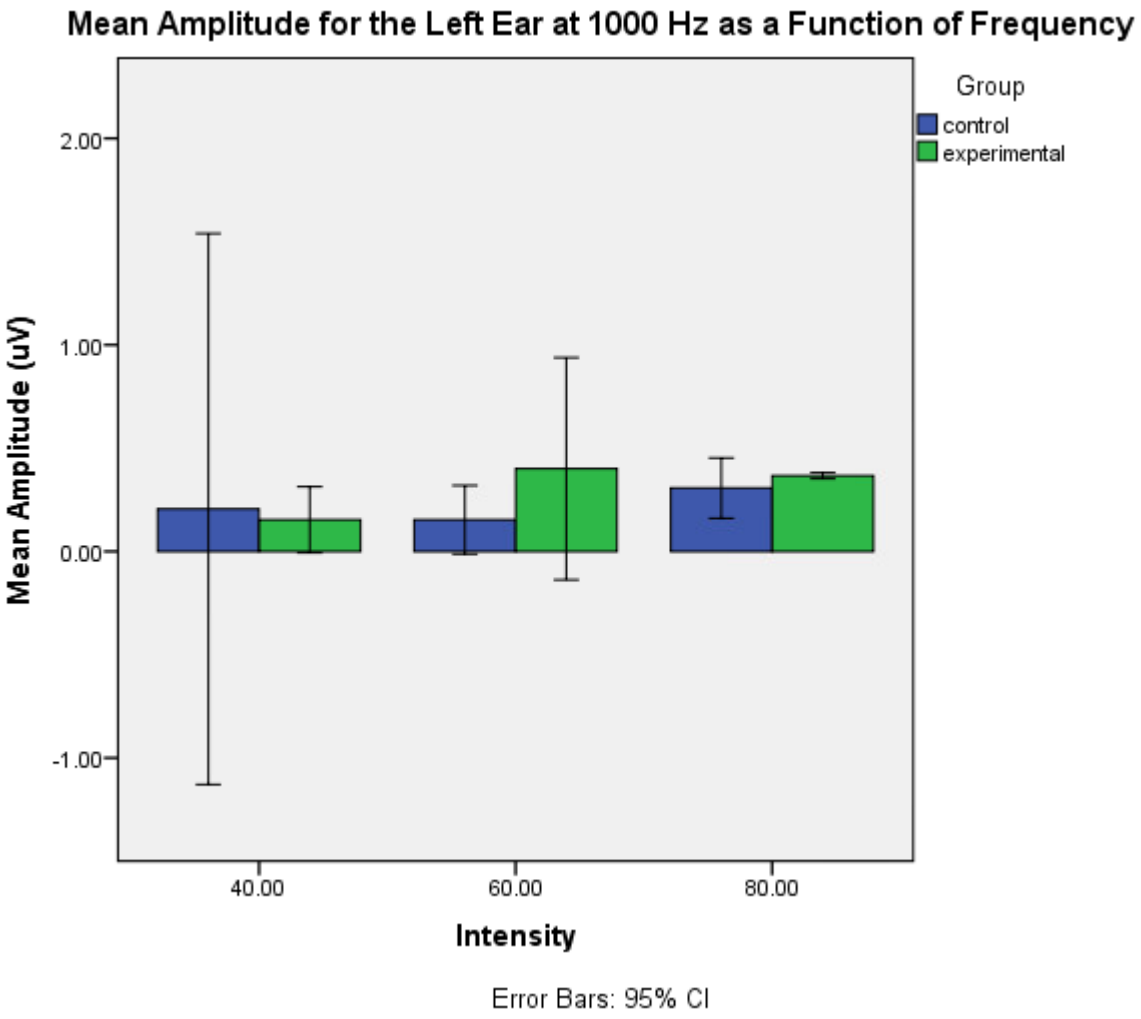


Figure 19. Mean amplitude (μV) of threshold ABR wave V at 1000 Hz in the left ear as intensity decreases from 80 dB nHL. Error bars represent a 95% confidence interval around the mean.

Mean wave V amplitudes for the control group decrease from 80 to 60 dB nHL and then increase slightly as intensity decreases to 40 dB nHL; the experimental group wave V amplitude means stay approximately similar from 80 to 60 dB nHL and then decrease as intensity is lowered to 40 dB nHL. A Kruskal-Wallis H test was run to determine if there were any differences in 1000 Hz threshold ABR amplitude values at 80, 60, 40, and 20 dB nHL in the left ear for the control (n=3) and experimental (n=3) groups. Distribution of the left ear 1000 Hz

threshold ABR amplitude values was similar for both groups at 60 and 40 dB nHL, as visualized by boxplot inspection. The differences between the control and experimental group amplitude values were not statistically significantly different for 60 dB nHL, χ^2 (1) = 2.333, p = .127, and at 40 dB nHL, χ^2 (1) = 0.333, p = .564. However, control group amplitudes at 80 dB nHL were statistically lower compared to the experimental group, χ^2 (1) = 3.971, p = .046. Comparisons could not be performed for 20 dB nHL because some wave V latencies and amplitudes could not be measured at this intensity.

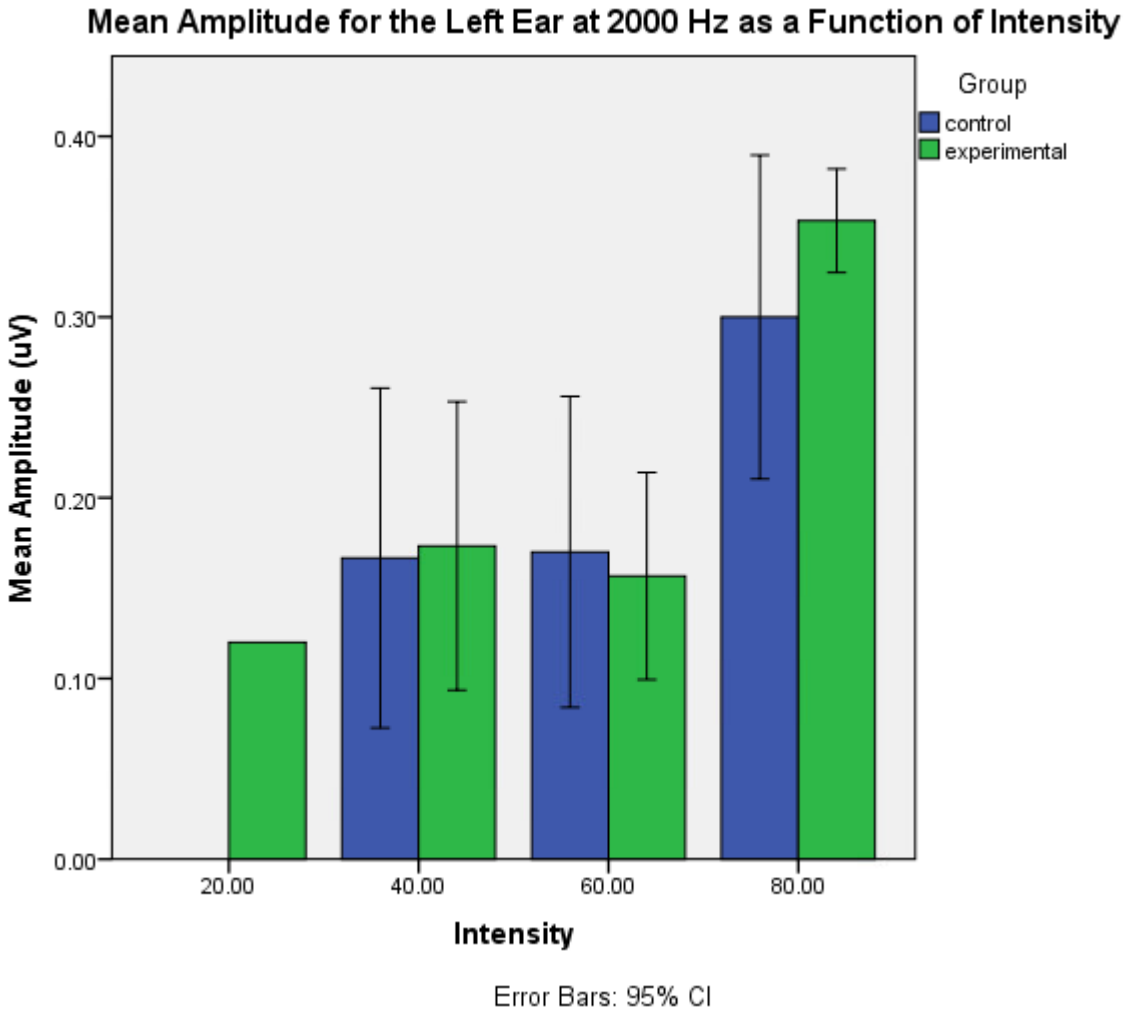


Figure 20. Mean amplitude (μV) of threshold ABR wave V at 2000 Hz in the left ear as intensity decreases from 80 dB nHL. Error bars represent a 95% confidence interval around the mean.

Mean wave V amplitudes are higher in the experimental group at 80 dB nHL, but both groups show a decrease in amplitude as intensity is decreased to 60 dB nHL. From this intensity level, the mean wave V amplitudes remain approximately the same as intensity is decreased to 40 dB nHL. At 20 dB nHL, only the experimental group had repeatable wave V tracings down to 20 dB nHL. A decrease in amplitude is also seen at this intensity level. A Kruskal-Wallis H test was run to determine if there were any differences in 2000 Hz threshold ABR amplitude values

at 80, 60, 40, and 20 dB nHL in the left ear for the control (n=3) and experimental (n=3) groups. Distribution of the left ear 2000 Hz threshold ABR amplitude values was similar for both groups, as visualized by boxplot inspection. The differences between the control and experimental group amplitude values were not statistically significantly different for 80 dB nHL, χ^2 (1) = 3.232, p = .072, at 60 dB nHL, χ^2 (1) = 0.051, p = .822, and at 40 dB nHL, χ^2 (1) = 0.455, p = .500. Comparisons could not be performed for 40 and 20 dB nHL because some wave V latencies and amplitudes could not be measured at this intensity.

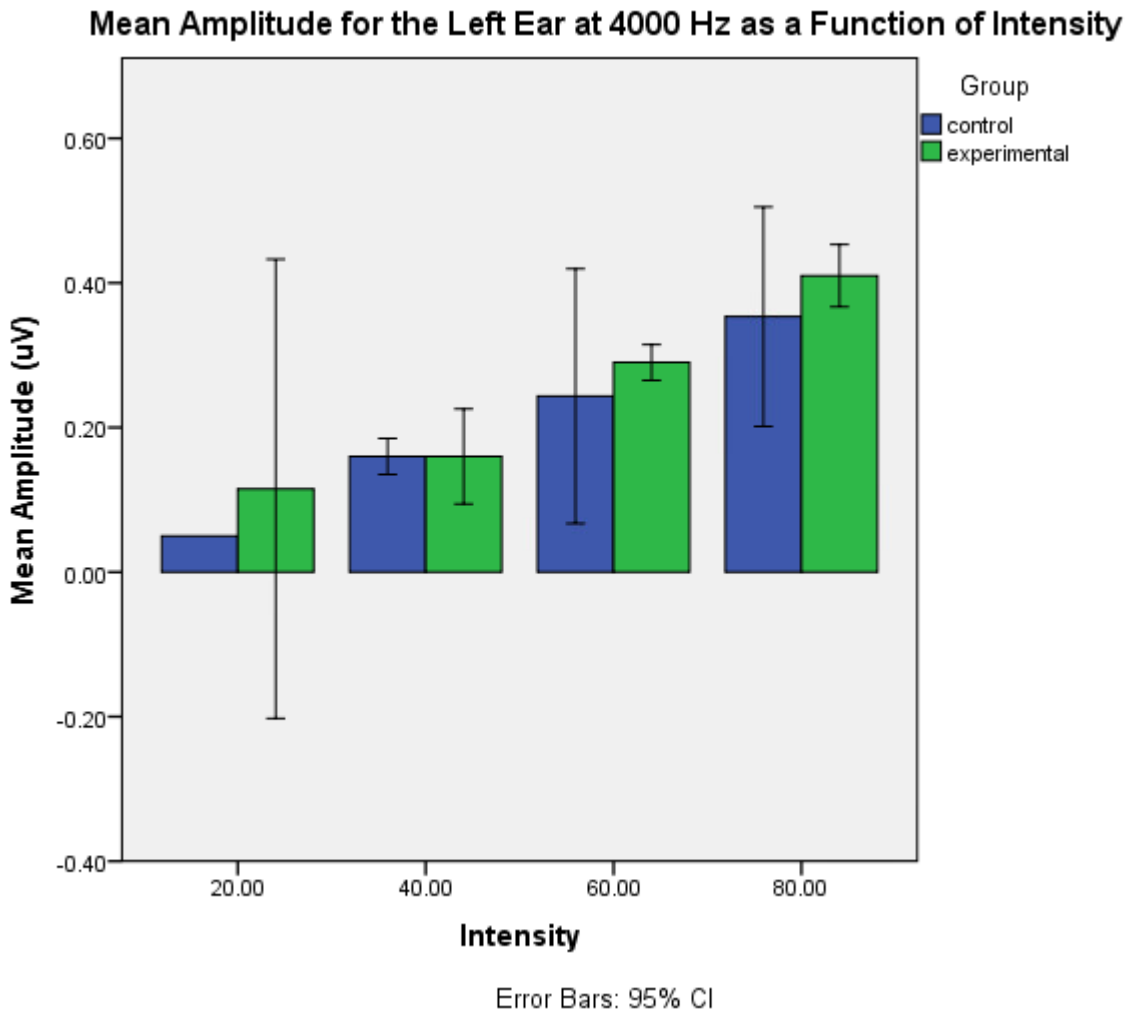


Figure 21. Mean amplitude (µV) of threshold ABR wave V at 4000 Hz in the left ear as intensity decreases from 80 dB nHL. Error bars represent a 95% confidence interval around the mean.

As intensity decreases from 80 to 20 dB nHL, both groups' mean wave V amplitudes decrease, with the experimental group having slightly higher amplitude values at 80, 60, and 20 dB nHL. A Kruskal-Wallis H test was run to determine if there were any differences in 4000 Hz threshold ABR amplitude values at 80, 60, 40, and 20 dB nHL in the left ear for the control (n=3) and experimental (n=3) groups. Distribution of the left ear 4000 Hz threshold ABR amplitude values was similar for both groups, as visualized by boxplot inspection. The differences between the control and experimental group amplitude values were not statistically significantly different

for 80 dB nHL, χ^2 (1) = 1.225, p = .268, at 60 dB nHL, χ^2 (1) = 0.429, p = .513, at 40 dB nHL, χ^2 (1) = 0.196, p = .658, and at 20 dB nHL, χ^2 (1) = 1.500, p = .221.

Latency. Tables 12, 13, 14, and 15 display the mean latencies (ms) and standard deviations for wave V in the right and left ears as tonal stimulus intensity decreases.

Table 12

500 Hz Threshold ABR Mean Latencies as a Function of Intensity, Ear, and Participant Group

Participant Group	80 dB nHL (SD)	60 dB nHL (SD)	40 dB nHL (SD)	20 dB nHL (SD)
Control Right	6.36 (1.24)	7.15 (1.23)	7.82 (0.47) NA	NA
Experimental Right	5.91 (0.96)	7.21 (0.63)	7.98 (NA) NA	NA
Control Left	6.92 (0.12)	8.50 (0.99)	NA	NA
Experimental Left	6.89 (0.34)	7.80 (0.06)	8.19 (0.93)	NA

Table 13

1000 Hz Threshold ABR Mean Latencies as a Function of Intensity, Ear, and Participant Group

Participant Group	80 dB nHL (SD)	60 dB nHL (SD)	40 dB nHL (SD)	20 dB nHL (SD)
Control Right	7.30 (0.66)	7.87 (0.69)	8.48 (NA)	NA
Experimental Right	7.77 (0.31)	7.68 (0.19)	8.92 (0.43)	NA
Control Left	6.95 (0.26)	8.23 (0.34)	9.12 (0.52)	NA
Experimental Left	6.93 (0.28)	7.81 (0.14)	8.85 (0.44)	NA

Table 14

2000 Hz Threshold ABR Mean Latencies as a Function of Intensity, Ear, and Participant Group

Participant Group	80 dB nHL (SD)	60 dB nHL (SD)	40 dB nHL (SD)	20 dB nHL (SD)
Control Right	6.57 (0.45)	7.80 (024)	8.52 (0.49)	8.93 (NA)
Experimental Right	6.26 (0.32)	7.30 (0.21)	8.32 (0.27)	NA
Control Left	6.71 (0.36)	7.65 (0.48)	8.47 (0.63)	NA
Experimental Left	6.31 (0.35)	7.05 (0.35)	8.40 (0.16)	9.23 (NA)

Table 15

4000 Hz Threshold ABR Mean Latencies as a Function of Intensity, Ear, and Participant Group

Participant Group	80 dB nHL (SD)	60 dB nHL (SD)	40 dB nHL (SD)	20 dB nHL (SD)
Control Right	6.30 (0.32)	7.05 (0.42)	7.55 (0.45)	8.45 (NA)
Experimental Right	6.05 (0.54)	6.75 (0.23)	7.52 (0.14)	8.48 (NA)
Control Left	6.29 (0.34)	7.11 (0.60)	7.70 (0.42)	8.90 (NA)
Experimental Left	6.18 (0.40)	6.69 (0.40)	7.19 (0.36)	8.24 (0.41)

Right ear.

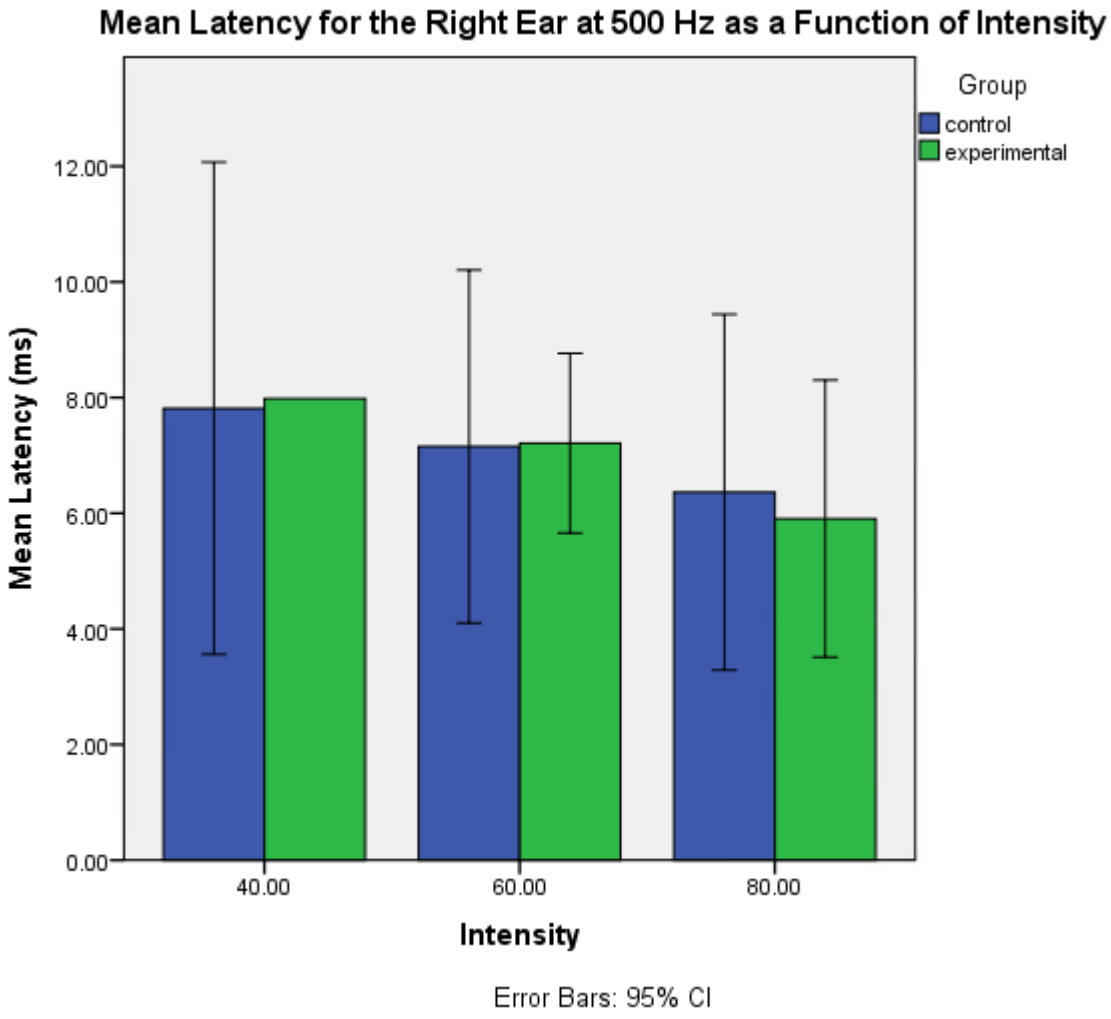


Figure 22. Mean latencies (ms) of threshold ABR wave V at 500 Hz in the right ear as intensity decreases from 80 dB nHL. Error bars represent a 95% confidence interval around the mean.

As intensity is decreased in the left ear from 80 to 40 dB nHL, latencies for wave V in both groups increase. Mean latencies are slightly lower in the experimental group at 80 dB nHL, but then become higher compared to the control group at 40 dB nHL. A Kruskal-Wallis H test was run to determine if there were any differences in 500 Hz threshold ABR latency values at 80, 60, 40, and 20 dB nHL in the right ear for the control (n=3) and experimental (n=3) groups. Distribution of the right ear 500 Hz threshold ABR latency values was similar for both groups, as visualized by boxplot inspection. The differences between the control and experimental group latency values were not statistically significantly different for 80 dB nHL, χ^2 (1) = 0.048, p = .827, at 60 dB nHL, χ^2 (1) = 0.048, p = .827, and at 40 dB nHL χ^2 (1) = 1.500, p = .221. Comparisons could not be performed for 40 and 20 dB nHL because some wave V latencies and amplitudes could not be measured at this intensity.

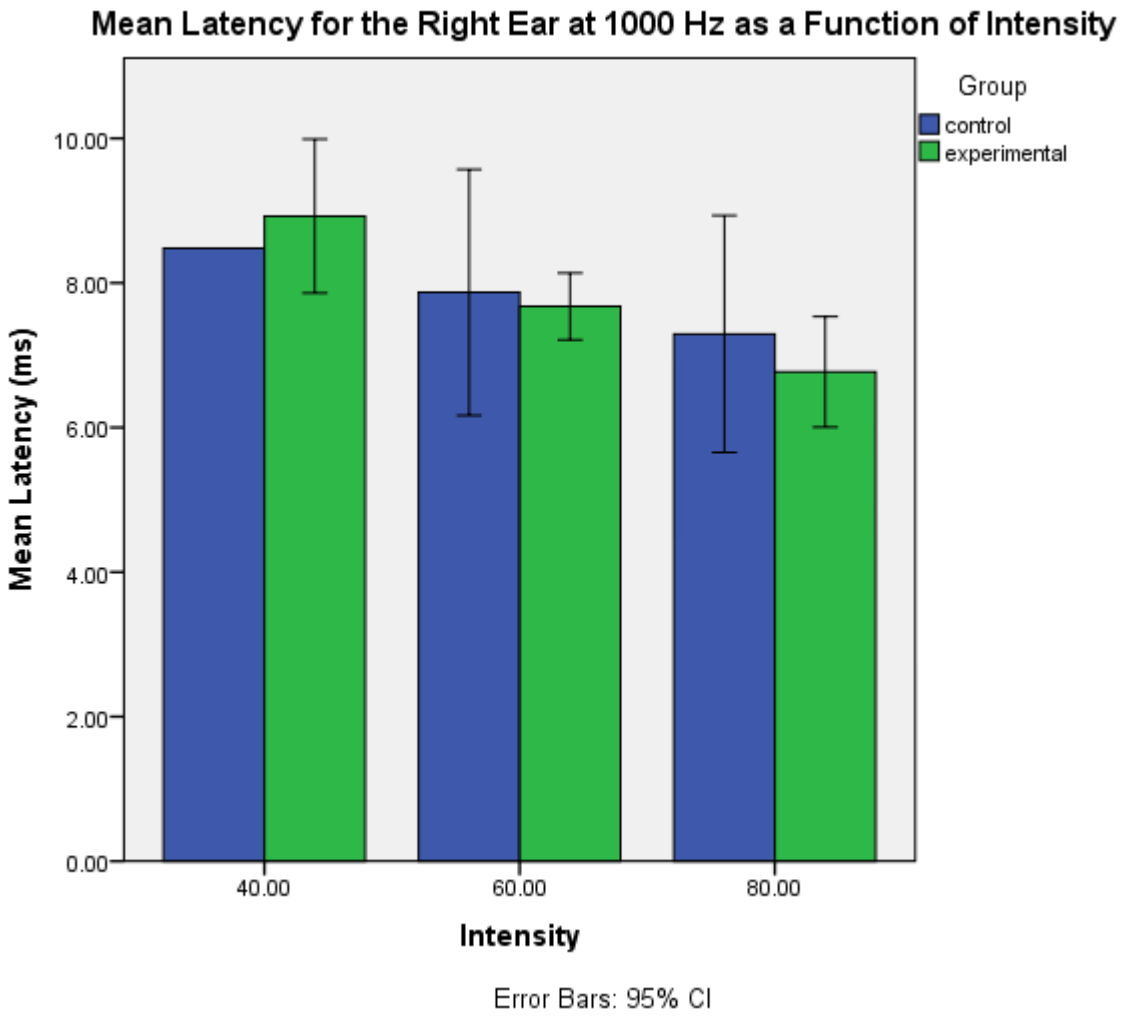


Figure 23. Mean latencies (ms) of threshold ABR wave V at 1000 Hz in the right ear as intensity decreases from 80 dB nHL. Error bars represent a 95% confidence interval around the mean.

A Kruskal-Wallis H test was run to determine if there were any differences in 1000 Hz threshold ABR latency values at 80, 60, 40, and 20 dB nHL in the right ear for the control (n=3) and experimental (n=3) groups. Distribution of the right ear 1000 Hz threshold ABR latency values was similar for both groups, as visualized by boxplot inspection. The differences between the control and experimental group latency values were not statistically significantly different for 80 dB nHL, χ^2 (1) = 1.190, p = .275, at 60 dB nHL, χ^2 (1) = 0.048, p = .827, and at 40 dB nHL,

χ^2 (1) = 1.800, p = .180. Comparisons could not be performed for 20 dB nHL because some wave V latencies and amplitudes could not be measured at this intensity.

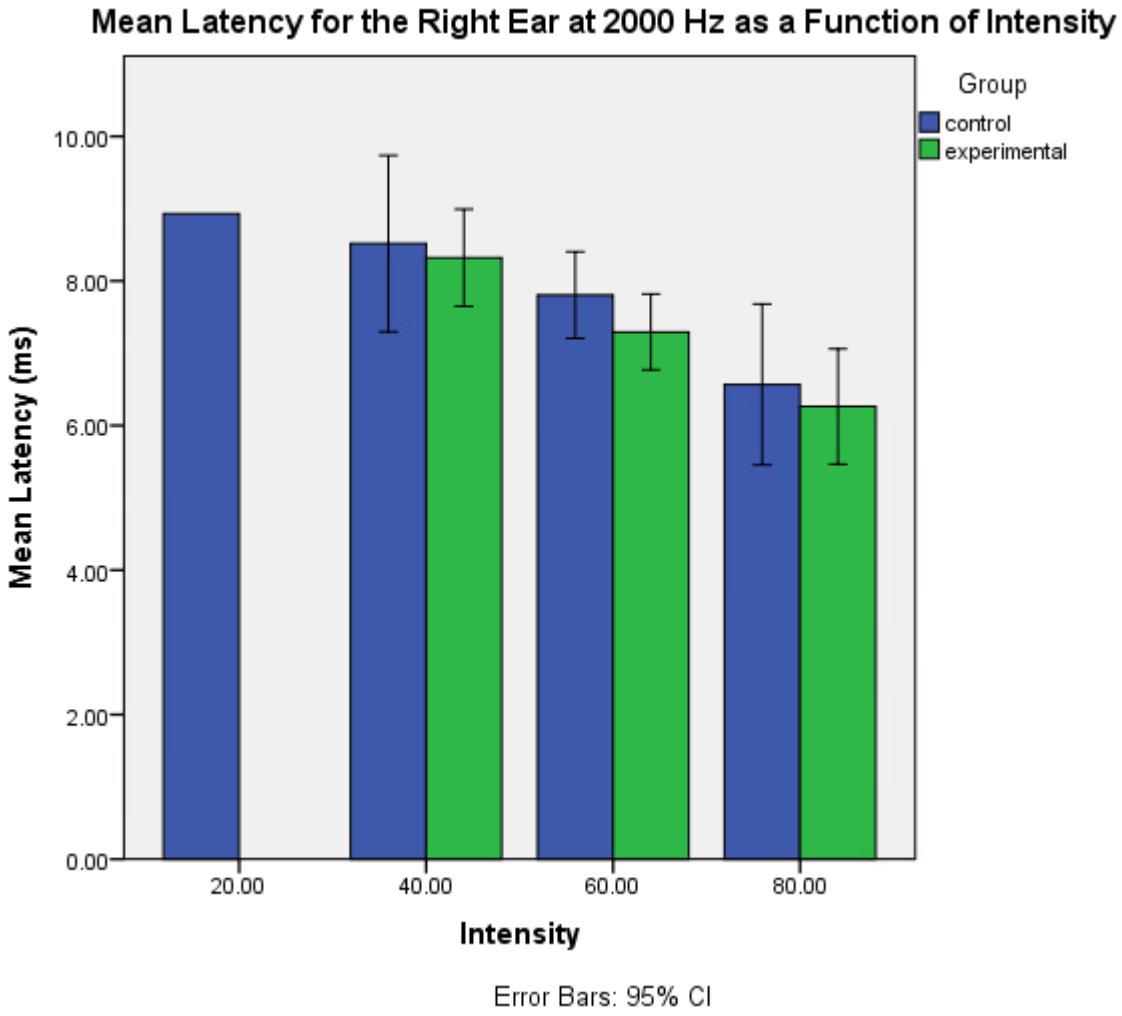


Figure 24. Mean latencies (ms) of threshold ABR wave V at 2000 Hz in the right ear as intensity decreases from 80 dB nHL. Error bars represent a 95% confidence interval around the mean.

Mean wave V latencies at this frequency for the left ear remain slightly lower for the experimental group when compared to the control group; overall, latencies increase for both groups as intensity decreases. A Kruskal-Wallis H test was run to determine if there were any differences in 2000 Hz threshold ABR latency values at 80, 60, 40, and 20 dB nHL in the right

ear for the control (n=3) and experimental (n=3) groups. Distribution of the right ear 1000 Hz threshold ABR latency values was similar for both groups at 80 and 40 dB nHL, as visualized by boxplot inspection. The differences between the control and experimental group latency values were not statistically significantly different for 80 dB nHL, χ^2 (1) = 1.190, p = .275, and at 40 dB nHL, χ^2 (1) = 0.429, p = .513. However, control group latencies at 60 dB nHL were statistically lower compared to the experimental group, χ^2 (1) = 3.857, p = .050. Comparisons could not be performed for 20 dB nHL because some wave V latencies and amplitudes could not be measured at this intensity.

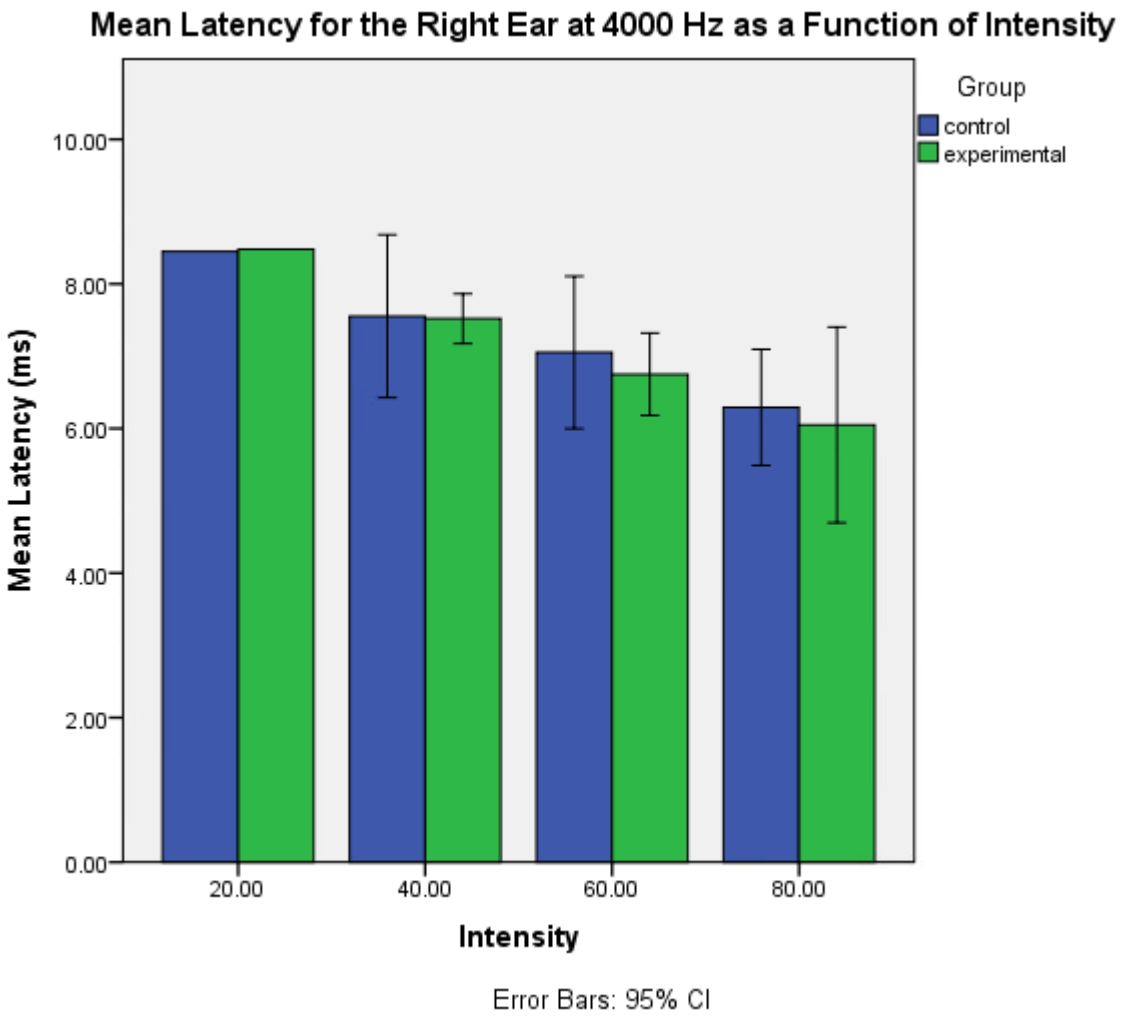


Figure 25. Mean latencies (ms) of threshold ABR wave V at 4000 Hz in the right ear as intensity decreases from 80 dB nHL. Error bars represent a 95% confidence interval around the mean.

As intensity decreases for this frequency in the left ear, mean wave V latencies remain faster at 80 and 60 dB nHL for the experimental group compared to the control group; from 40 to 20 dB nHL, mean latencies remain about the same for both groups. Overall, mean wave V latencies increase as intensity decreases. A Kruskal-Wallis H test was run to determine if there were any differences in 4000 Hz threshold ABR latency values at 80, 60, 40, and 20 dB nHL in the right ear for the control (n=3) and experimental (n=3) groups. Distribution of the right ear

4000 Hz threshold ABR latency values was similar for both groups, as visualized by boxplot inspection. The differences between the control and experimental group latency values were not statistically significantly different for 80 dB nHL, χ^2 (1) = 0.196, p = .658, at 60 dB nHL, χ^2 (1) = 1.190, p = .275, at 40 dB nHL, χ^2 (1) = 0.196, p = .658, and at 20 dB nHL, χ^2 (1) = 1.000, p = .317.

Left ear.

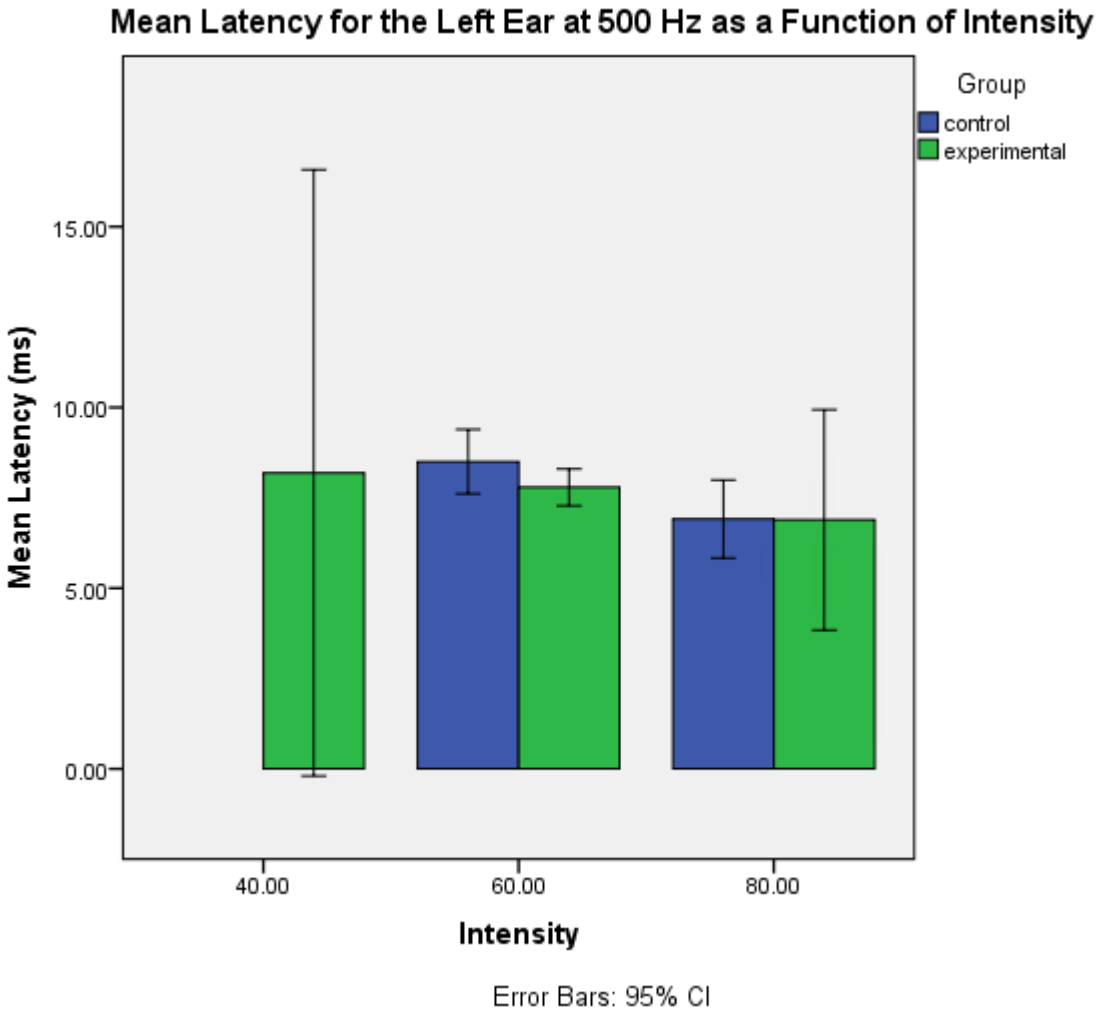


Figure 26. Mean latencies (ms) of threshold ABR wave V at 500 Hz in the left ear as intensity decreases from 80 dB nHL. Error bars represent a 95% confidence interval around the mean.

Mean wave V latencies do increase slightly for both groups as intensity decreases from 80 to 60 dB nHL, but then do not seem to increase at 40 dB nHL for the experimental group. Repeatable wave V tracings could not be recorded for the control group at 40 dB nHL and for neither group at 20 dB nHL. A Kruskal-Wallis H test was run to determine if there were any differences in 500 Hz threshold ABR latency values at 80, 60, 40, and 20 dB nHL in the left ear for the control (n=3) and experimental (n=3) groups. Distribution of the left ear 500 Hz threshold ABR latency values was similar for both groups, as visualized by boxplot inspection. The differences between the control and experimental group latency values were not statistically significantly different for 80 dB nHL, χ^2 (1) = 0.000, p = 1.000, and at 60 dB nHL, χ^2 (1) = 2.400, p = .121. Comparisons could not be performed for 40 and 20 dB nHL because some wave V latencies and amplitudes could not be measured at this intensity.

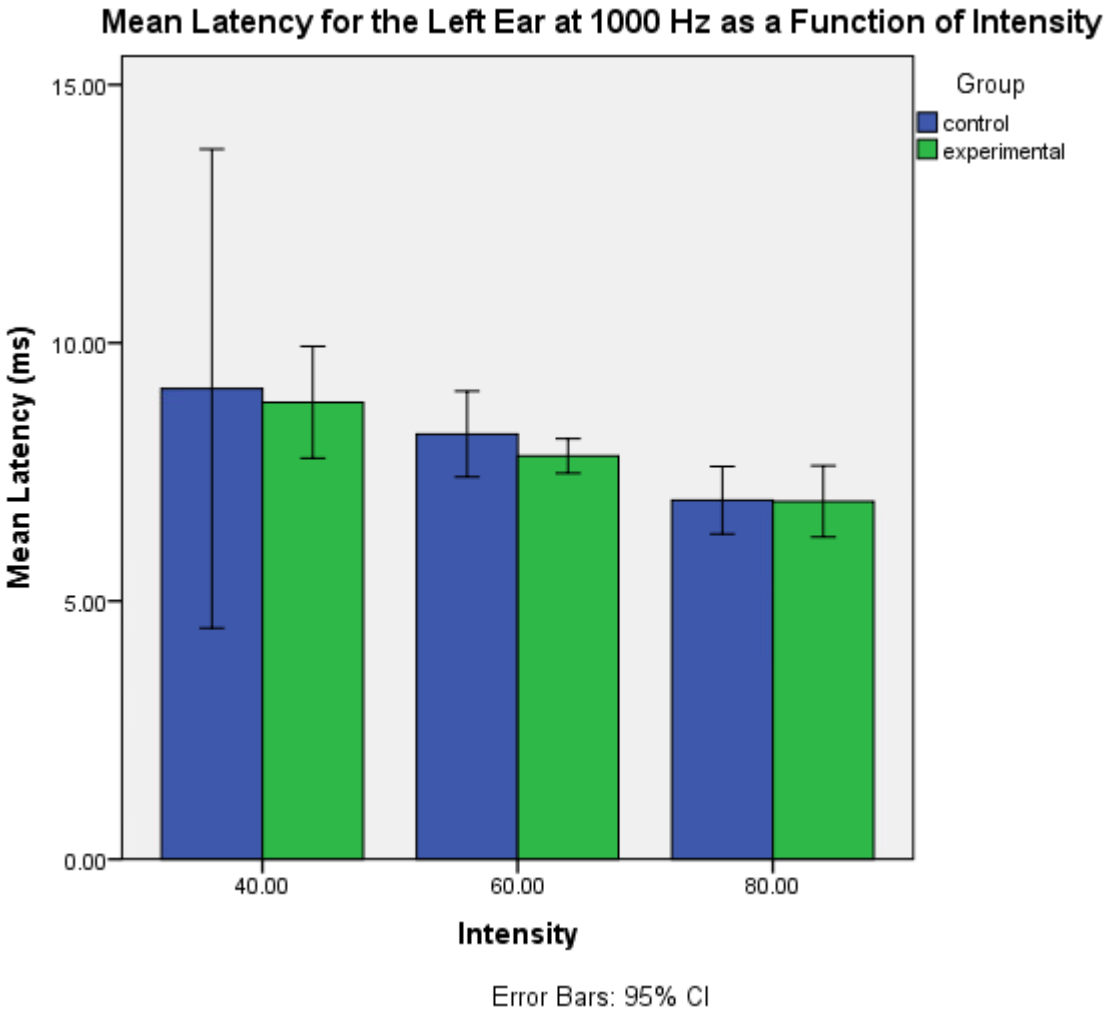


Figure 27. Mean latencies (ms) of threshold ABR wave V at 1000 Hz in the left ear as intensity decreases from 80 dB nHL. Error bars represent a 95% confidence interval around the mean.

As intensity decreases, mean wave V latencies at 1000 Hz for the left ear increase. These mean latencies are approximately the same at 80 dB nHL, but as intensity continues to decrease, mean wave V latencies for the experimental group are slightly faster compared to the control group. A Kruskal-Wallis H test was run to determine if there were any differences in 1000 Hz threshold ABR latency values at 80, 60, 40, and 20 dB nHL in the left ear for the control (n=3) and experimental (n=3) groups. Distribution of the left ear 1000 Hz threshold ABR latency values was similar for both groups, as visualized by boxplot inspection. The differences between

the control and experimental group latency values were not statistically significantly different for 80 dB nHL, χ^2 (1) = 0.048, p = .827, at 60 dB nHL, χ^2 (1) = 2.333, p = .127, and at 40 dB nHL, χ^2 (1) = 0.333, p = .564. Comparisons could not be performed for 20 dB nHL because some wave V latencies and amplitudes could not be measured at this intensity.

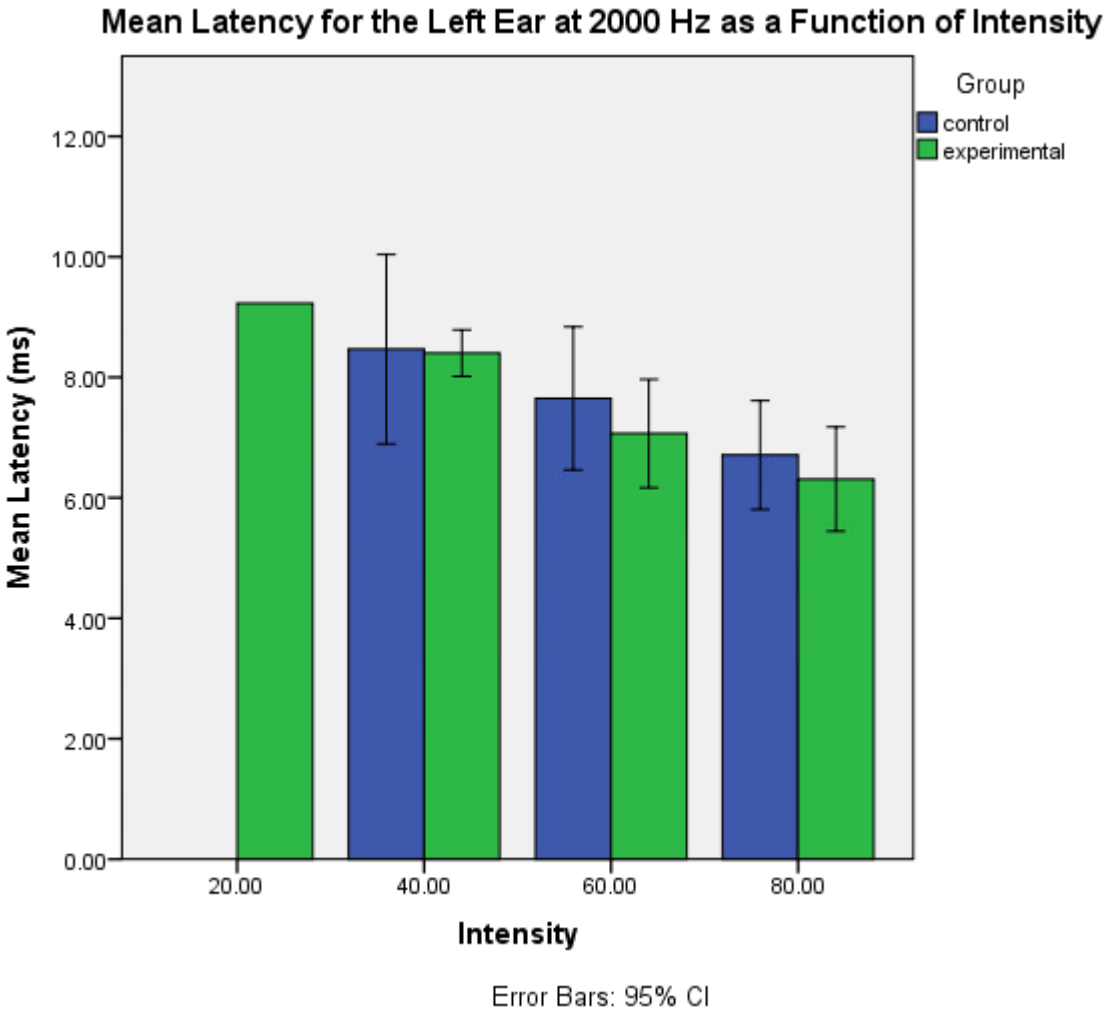


Figure 28. Mean latencies (ms) of threshold ABR wave V at 2000 Hz in the left ear as intensity decreases from 80 dB nHL. Error bars represent a 95% confidence interval around the mean.

Mean wave V latencies remain slightly faster for the experimental group compared to the control group, as intensity decreases; however, we can see that latencies increase as intensity decreases for both groups. Repeatable wave V could not be measured at 20 dB nHL for the control group. A Kruskal-Wallis H test was run to determine if there were any differences in 2000 Hz threshold ABR latency values at 80, 60, 40, and 20 dB nHL in the left ear for the control (n=3) and experimental (n=3) groups. Distribution of the left ear 2000 Hz threshold ABR

latency values was similar for both groups, as visualized by boxplot inspection. The differences between the control and experimental group latency values were not statistically significantly different for 80 dB nHL, χ^2 (1) = 1.190, p = .275, at 60 dB nHL, χ^2 (1) = 1.190, p = .275, and at 40 dB nHL, χ^2 (1) = 0.429, p = .513. Comparisons could not be performed for 20 dB nHL because some wave V latencies and amplitudes could not be measured at this intensity.

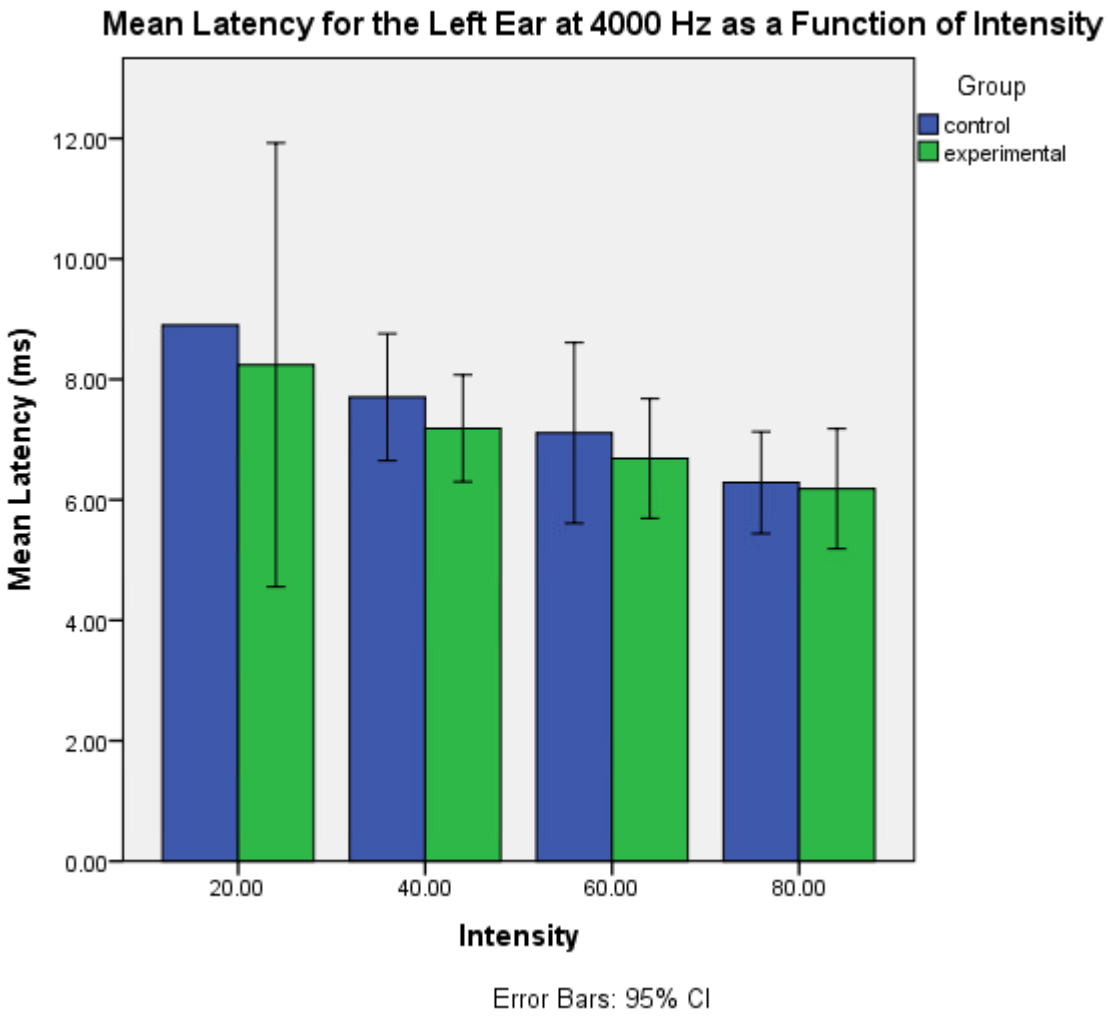


Figure 29. Mean latencies (ms) of threshold ABR wave V at 4000 Hz in the left ear as intensity decreases from 80 dB nHL. Error bars represent a 95% confidence interval around the mean.

As intensity decreases at 4000 Hz in the left ear, both participant groups show increases in their mean wave V latencies; again, wave V latencies are slightly faster across the intensity range for the experimental group compared to the control group. A Kruskal-Wallis H test was run to determine if there were any differences in 4000 Hz threshold ABR latency values at 80, 60, 40, and 20 dB nHL in the left ear for the control (n=3) and experimental (n=3) groups. Distribution of the left ear 4000 Hz threshold ABR latency values was similar for both groups, as

visualized by boxplot inspection. The differences between the control and experimental group latency values were not statistically significantly different for 80 dB nHL, χ^2 (1) = 0.429, p = .513, at 60 dB nHL, χ^2 (1) = 0.347, p = .496, at 40 dB nHL, χ^2 (1) = 1.190, p = .275, and at 20 dB nHL, χ^2 (1) = 1.500, p = .221.

Discussion

The aim of the current study was to determine if normal-hearing participants with a history of reported noise exposure (e.g. music majors, power tools, and firearm use) had test result patterns that might be indicative of cochlear synaptopathy, or HHL. A group of participants without reported noise exposure (n=3) and with reported noise exposure (n=3) were subjected to a comprehensive audiological evaluation that included pure tone air and bone thresholds (250-8000 Hz), speech audiometry, TEOAEs, and DPOAEs. Next, the participants were administered a (C)APD testing battery that included tests of dichotic listening, temporal processing, temporal resolution, monaural low redundancy, and binaural interaction. Finally, each participant had threshold seeking ABRs performed for each ear at 500, 1000, 2000, and 4000 Hz at decreasing intensities from 80-20 dB nHL. This discussion will focus on the overall findings from the current study, how these findings relate to previous studies looking at cochlear synaptopathy in humans, limitations, and future directions.

Overall Findings

The overall findings of this test battery revealed that the control group typically achieved higher average OAE dB SNR values, achieved better average scores on tests of auditory processing, and had comparable threshold ABR wave V amplitudes and latencies. However, there were not enough statistically significant differences on the test metrics between the control and experimental groups to indicate the presence of cochlear synaptopathy in the experimental group. TEOAE SNRs for the right ear were only statistically significantly lower in the experimental group at 1000 Hz and for the left ear at 1400 Hz. DPOAE SNRs were significantly lower in the experimental group for the left ear at 6000 Hz; no other significant differences were seen between groups for DPOAEs. Results for the DDT, PPS, DPS, RGDT, SCAN 3A, and

MLD all showed slightly better scores in the control groups as compared to the experimental groups; however, no statistically significant differences in results for any of the (C)APD tests were seen between groups. Threshold ABR wave V amplitudes were statistically significantly lower in the control group compared to the experimental group in the left ear at 1000 Hz for 80 dB nHL. No other statistically significant amplitude differences were noted at any frequency between the control and experimental group for either ear. Threshold ABR wave V latencies were only statistically significantly delayed in the right ear at 2000 for 60 dB nHL for the control group compared to the experimental group. No other statistically significant latency differences were observed at any of the frequencies tested between the control and experimental group for either ear.

Differences in Methodology

The results obtained in the current study were compared to results obtained in the literature. Overall, DPOAE results and (C)APD results were consistent; however, ABR results in this study were inconsistent with what other researchers had concluded, most obviously due to differences in wave analysis. Some of the methodological differences between the current study and other studies dealing with HHL in humans will be discussed.

High-frequency audiometry.

The current study did not include assessment of extended high-frequency audiometry (9000 Hz-16,000 Hz). Thus, conclusions regarding possible group differences for thresholds at extended high frequencies could not be made. Liberman et al. (2016) included these extended high-frequencies in their assessment of audiometric hearing sensitivity and found that hearing

thresholds at these extended high-frequency were significantly elevated in the participants who had a higher risk for NIHL as compared to the control group.

ABR wave I assessment.

The current study did not include assessment and measurement of wave I amplitudes and latencies; Bramhall et al. (2017) focused their efforts into analyzing wave I amplitudes for an experimental group of veterans from 1000-6000 Hz at ascending intensities up to 90 dB SPL and concluded that significant reductions in wave I amplitudes were apparent in the veteran groups, possibly because of their histories of noise exposure and hypothetical cochlear synaptopathy.

Lobarinas, Spankovich, and Le Prell (2017) utilized analysis of wave I peak-to-peak amplitudes in their animal model study. They theorized that in subjects with reduced wave I peak-to-peak amplitudes following noise exposure, this metric would be highly correlated with suprathreshold functional deficits despite recovery from a TTS.

Rather, focus was placed upon wave V amplitudes and latencies because of its anatomical correlation with the lateral lemniscus and/or the inferior colliculus and the frequency following response (FFR). Both the ABR wave V and the FFR are theorized to originate in these rostral brainstem structures. However, no significant group differences were calculated for wave V amplitude and latency measurements at the different test frequencies and at descending intensities.

FFR.

The FFR is “a sustained response to a periodic stimulus, reflecting phase-locked neural activity in the rostral brainstem (region of the lateral lemniscus/inferior colliculus)” (Plack, Barker, & Prendergast, 2014). Although is does not directly reflect activity of the auditory nerve

and its efferent connections, the robustness (or lack thereof) of FFR recordings may reflect decreased central temporal coding that may be influenced by cochlear neuropathy (Krishnan, 2006). It is the FFR's central auditory locus that provides, at present, an incomplete attempt at describing cochlear synaptopathy in isolation. Rather, the FFR better describes individual differences in efficacy and robustness of the central auditory system (Plack et al., 2016). These individual differences in central auditory processing abilities unrelated to cochlear synaptopathy make interpretation of the FFR difficult regarding research of suspected HHL in humans (Plack et al., 2016).

Mixed Findings in the Literature

Although the goal of the current study was to determine if HHL plays a role in humans who have been previously exposed to damaging noise, other studies have not found a connection between test results and evidence of possible HHL. The uses of the ABR, FFR, and behavioral tests of hearing will be briefly discussed.

ABR.

Some researchers utilized animal models to conclude that intense noise exposure, followed by a period of time without such noise exposure, contributes to reduced wave I amplitude as seen in the click-evoked ABR (Kujawa and Liberman, 2009). However, in humans with a history of noise exposure, this electrophysiological hallmark is not reliably replicable (Prendergast et al., 2017; Plack et al., 2016; Plack, Barker, & Prendergast, 2014). Wave I of the ABR is highly correlated with function of the distal portion of the auditory nerve, yet abundant variability of wave I amplitude in subjects with and without a history of noise exposure has been documented as well (Prendergast et al., 2017). Participant sex, head size, individual

synchronicity of the auditory nerve fibers, and individual differences in internal ambient noise have all been proposed as possible reasons for the variability of wave I (Plack et al., 2016).

Behavioral tests of hearing.

Behavioral measures require a response from the listener, unlike the objectivity of the ABR and FFR. Similar to the FFR, however, results from subjective measures may also be influenced by individual central auditory processing factors that are unrelated to potential cochlear synaptopathy (Plack et al., 2016). For example, results of speech comprehension in noise may differ on an individual basis based on participant age, native language, central auditory processing abilities, and attention. Studies that focused on blast-exposed participants offer some insight into these behavioral test result differences.

Myers et al. (2009) reviewed the relevant literature on peripheral and central auditory effects of blast exposure. They offer that blasts can sometimes reach traumatic acoustic levels of 140 dB SL or higher; a blast of this intensity has the potential to destroy sensory hair cells via extreme Basilar membrane displacement. The most common peripheral result of blast exposure was tympanic membrane rupture and sensorineural hearing loss. Additionally, auditory processes of the temporal lobe are also at risk for damage due to intense blasts (Myers et al., 2009). The force of the intense sound has the potential to physically harm the axons and blood vessels in the brain. However, the extent of these injuries on actual tests of (C)APD are unknown as of yet.

Limitations

There were many limitations in the current study. First and foremost, a mere total of six participants, three with and without a reported history of noise exposure, completed the entire testing battery. Reasons for the low number of total participants completed include scheduling

conflicts and timeline for data collection. Scheduling conflicts on the part of the PI and interested participants occurred from the outset and carried into the end of data collection. Some interested participants had to reschedule a set appointment for data collection due to academic responsibilities and personal conflicts. Some of these scheduling conflicts were resolved and allowed for complete testing for inclusion into analysis of the study, while the majority of scheduling conflicts persisted due to PI schedule conflicts on alternate dates proposed by the participants. IRB approval to begin data collection was granted by the Towson University IRB in late January 2018; however, data collection did not commence until early March 2018 and ended in early April 2018. As a result, almost a month of potential data collection was squandered.

Second, because of the very low number of total completed participants, the assumption of normal distribution of the data collected was violated and could not be taken into account during statistical analysis. As a result, non-parametric test equivalents of the standard one-way ANOVA (Kruskal Wallis H) and t-test (Mann Whitney U) had to be utilized instead. These statistical measures deal more with ranking of scores and with distribution of median score results as opposed to median test results.

Third, equal numbers of male and female participants were not obtained. Although almost equal, there were a total of four females and two males included in the current study. Finally, there was an abundance of self-reported data from questionnaire responses. Self-reported data cannot be independently verified and the participant's responses must be taken as a fact. Self-reported responses are sensitive to biases stemming from selective memory, attribution of internal and external forces on questionnaire results, and exaggeration of questionnaire results. However, this does not discredit the benefits of using questionnaires in formal research. The current study did not analyze the questionnaire results due to the low number of participants.

Future Directions

Future directions in the possible contribution of cochlear synaptopathy on comprehension of suprathreshold speech in background noise with otherwise normal hearing sensitivity may wish to include measures of ABR wave I amplitude, extended high-frequency audiometry, increased sample size, and other electrophysiological measures. Although discussed earlier in this section regarding its mixed findings, future studies in HHL in humans should focus on wave I of the ABR due to its proximity and anatomical correlation with the distal portion of the auditory nerve. Extended high-frequency audiometry should also be employed in future studies for two reasons. First, higher frequencies are most affected by history of noise exposure. Future participants with reported noise exposure may have lower thresholds from 9000-16,000 Hz compared to participants without a history of noise exposure. Second, extended high-frequency information may correlate better with DPOAE values at higher test frequencies.

Any future studies into the possibility of HHL in humans must have a larger sample size than the current study. It is extremely difficult to take the results of the current study and apply them to real-world clinical inquiry due to the low sample size. Larger sample sizes are more representative of the population from which the sample is acquired, and more information can be gathered from statistical analysis of more participants in each group. Other electrophysiological measures such as the FFR and slow cortical potentials may also be considered for future study of possible HHL in humans. As discussed earlier in this section, results from the FFR may provide more insight into how individuals with a history of noise exposure encode and process auditory stimuli when compared to a control group. These FFR results can then be compared to tests of other anatomical correlates of the peripheral and central auditory system to formulate a more robust conclusion.

Appendix A

The Goldsmiths Musical Sophistication Index

The Goldsmiths Musical Sophistication Index, v0.9

October 7, 2011

Please circle the most appropriate category:	1 Completely Disagree	2 Strongly Disagree	3 Disagree	4 Neither Agree nor Disagree	5 Agree	6 Strongly Agree	7 Completely Agree
1. I spend a lot of my free time doing music-related activities.	1	2	3	4	5	6	7
2. I rarely listen to music as a main activity.	1	2	3	4	5	6	7
3. Attending a whole evening of live music would bore me.	1	2	3	4	5	6	7
4. If I hear two tones played one after another I have trouble judging which of them is higher.	1	2	3	4	5	6	7
5. I don't consider music to be a central part of my identity.	1	2	3	4	5	6	7
6. I sometimes choose music that can trigger shivers down my spine.	1	2	3	4	5	6	7
7. I often sing to myself even when there's no music playing.	1	2	3	4	5	6	7
8. I enjoy writing about music, for example on blogs and forums.	1	2	3	4	5	6	7
9. I never sing or hum along when my favourite tunes are playing.	1	2	3	4	5	6	7
10. I avoid discussing music with friends.	1	2	3	4	5	6	7
11. If somebody starts singing a song I don't know, I can usually join in.	1	2	3	4	5	6	7

2

Please circle the most appropriate category:	1 Completely Disagree	2 Strongly Disagree	3 Disagree	4 Neither Agree nor Disagree	5 Agree	6 Strongly Agree	7 Completely Agree
12. I use music to calm myself when Im stressed.	1	2	3	4	5	6	7
13. I have trouble tapping along to the beat when I listen to a song.	1	2	3	4	5	6	7
14. I rarely get tunes stuck in my head.	1	2	3	4	5	6	7
15. Playing or listening to music isn't one of my favourite hobbies.	1	2	3	4	5	6	7
16. I am able to judge whether someone is a good singer or not.	1	2	3	4	5	6	7
17. I usually know when I'm hearing a song for the first time.	1	2	3	4	5	6	7
18. I can sing or play music from memory.	1	2	3	4	5	6	7
19. I never come up with new tunes in my head.	1	2	3	4	5	6	7
20. I like to listen to music of other cultures.	1	2	3	4	5	6	7
21. I'm intrigued by musical styles I'm not familiar with and want to find out more.	1	2	3	4	5	6	7
22. Pieces of music rarely evoke emotions for me.	1	2	3	4	5	6	7

Please circle the most appropriate category:	1 Completely Disagree	2 Strongly Disagree	3 Disagree	4 Neither Agree nor Disagree	5 Agree	6 Strongly Agree	7 Completely Agree
23. I am able to hit the right notes when I sing along with a recording.	1	2	3	4	5	6	7
24. I would find it difficult to tell the difference between the sound of a flute and a clarinet.	1	2	3	4	5	6	7
25. I rarely tap or clap along when listening to music.	1	2	3	4	5	6	7
26. When I listen to music, I have a hard time hearing whether one note is a different pitch to the next.	1	2	3	4	5	6	7
27. I don't think that music is very important for setting the atmosphere of an occasion.	1	2	3	4	5	6	7
28. I find it difficult to spot mistakes in a performance of a song even if I know the tune.	1	2	3	4	5	6	7
29. I never sing along to music in public (eg at a party or concert).	1	2	3	4	5	6	7
30. I'm not interested in radio or TV programmes where music is discussed.	1	2	3	4	5	6	7
31. I can compare and discuss differences between two performances or versions of the same piece of music.	1	2	3	4	5	6	7

4

Please circle the most appropriate category:	1 Completely Disagree	2 Strongly Disagree	3 Disagree	4 Neither Agree nor Disagree	5 Agree	6 Strongly Agree	7 Completely Agree
32. I have trouble recognizing a familiar song when played in a different way or by a different performer.	1	2	3	4	5	6	7
33. I hardly ever hum or sing along to music.	1	2	3	4	5	6	7
34. If I have to clap along to music in a group situation I find it difficult and have to follow other people's lead.	1	2	3	4	5	6	7
35. I have never been complimented for my talents as a musical performer.	1	2	3	4	5	6	7
36. I often read or search the internet for things related to music.	1	2	3	4	5	6	7
37. I often pick certain music to motivate or excite me.	1	2	3	4	5	6	7
38. I am not able to sing in harmony when somebody is singing a familiar tune.	1	2	3	4	5	6	7
39. Music can trigger shivers down my spine.	1	2	3	4	5	6	7
40. I can tell when people sing or play out of time with the beat.	1	2	3	4	5	6	7
41. After I have been to a concert with other people, I like to talk about the music and the performance.	1	2	3	4	5	6	7

5

Please circle the most appropriate category:	1 Completely Disagree	2 Strongly Disagree	3 Disagree	4 Neither Agree nor Disagree	5 Agree	6 Strongly Agree	7 Completely Agree
42. The ability to play music is a very valuable skill.	1	2	3	4	5	6	7
43. I am able to identify what is special about a given musical piece.	1	2	3	4	5	6	7
44. I am able to talk about the emotions that a piece of music evokes for me.	1	2	3	4	5	6	7
45. I don't spend much of my disposable income on music.	1	2	3	4	5	6	7
46. When I hear a catchy tune I find myself moving to the beat.	1	2	3	4	5	6	7
47. I have difficulties in distinguishing between musical genres.	1	2	3	4	5	6	7
48. I can tell when people sing or play out of tune.	1	2	3	4	5	6	7
49. When I sing, I have no idea whether I'm in tune or not.	1	2	3	4	5	6	7
50. I can't read a musical score.	1	2	3	4	5	6	7
51. Music is kind of an addiction for me - I couldn't live without it.	1	2	3	4	5	6	7
52. I could imagine myself living without music.	1	2	3	4	5	6	7

Please circle the most appropriate category:	1 Completely Disagree	2 Strongly Disagree	3 Disagree	4 Neither Agree nor Disagree	5 Agree	6 Strongly Agree	7 Completely Agree
53. I often listen to music to help pass the time if I'm doing something boring.	1	2	3	4	5	6	7
54. I dont like singing in public because Im afraid that I would sing wrong notes.	1	2	3	4	5	6	7
55. When I hear a music I can usually identify its genre.	1	2	3	4	5	6	7
56. I have never made up songs or musical pieces for myself or others to perform.	1	2	3	4	5	6	7
57. I would not consider myself a musician.	1	2	3	4	5	6	7
58. I keep track of new of music that I come across (e.g. new artists or recordings).	1	2	3	4	5	6	7
59. After hearing a new song two or three times, I can usually sing it by myself.	1	2	3	4	5	6	7
60. I only need to hear a new tune once and I can sing it back hours later.	1	2	3	4	5	6	7
61. Music can evoke my memories of past people and places.	1	2	3	4	5	6	7
62. I never go to concerts if I dont know the artists who are playing.	1	2	3	4	5	6	7

Please circle the most appropriate category:

63. I engaged in regular, daily practice of a musical instrument (including voice) for **0 / 1 / 2 / 3 / 4-5 / 6-9 / 10 or more** years.
64. At the peak of my interest, I practiced **0 / 0.5 / 1 / 1.5 / 2 / 3-4 / 5 or more** hours per day on my primary instrument.
65. I have played or sung in a group, band, choir, or orchestra for **0 / 1 / 2 / 3 / 4-5 / 6-9 / 10 or more** years.
66. I have attended **0 / 1 / 2 / 3 / 4-6 / 7-10 / 11 or more** live music events as an audience member in the past twelve months.
67. I have had formal training in music theory for **0 / 0.5 / 1 / 2 / 3 / 4-6 / 7 or more** years.
68. I have had **0 / 0.5 / 1 / 2 / 3-5 / 6-9 / 10 or more** years of formal training on a musical instrument (including voice) during my lifetime.
69. I can play **0 / 1 / 2 / 3 / 4 / 5 / 6 or more** musical instruments.
70. I listen attentively to music for 0-15 min / 15-30 min / 30-60 min / 60-90 min / 2 hrs / 2-3 hrs / 4 hrs or more per day.
71. The instrument I play best (including voice) is _____ **/ Not applicable**.

Please tick one of the following:

Occupational status

- ☐ Still at School
- ☐ At University
- ☐ In Full-time employment
- ☐ In Part-time employment
- ☐ Self-employed
- ☐ Homemaker/full time parent
- ☐ Unemployed
- ☐ Retired

What is the musical genre you mainly listen to?
(tick only one box)

- ☐ Rock/Pop
- ☐ Jazz
- ☐ Classical Music

What is the Highest educational qualification you have attained?

- ☐ Did not complete any school qualification
- ☐ Completed first school qualification at about 16 years (e.g. GCSE/Junior High School)
- ☐ Completed Second qualification (e.g A levels/ High School)
- ☐ Undergraduate degree or professional qualification
- ☐ Postgraduate degree
- ☐ I am still in education

If you are still in education, what is the highest qualification you expect to obtain?

- ☐ First school qualification (e.g. GCSE / Junior High School)
- ☐ Post-16 vocational course
- ☐ Second school qualification (e.g. A-levels / High School)
- ☐ Undergraduate degree or professional qualification
- ☐ Postgraduate degree
- ☐ Not applicable

Anonymous ID:_____
(please write down the last three letters of your surname, the day you were born (2 digits) and todays day (8 digits). Example: Adam Smith, born 13.02.1982, taken the test 05.03.2011 and Anonymous ID: ith1305032011)
Your age : _____ years.
Gender: **Female / Male**
Nationality:
Country in which you spent the formative years of your childhood and youth:
Country of current residency:
Email address (permanent email address, optional): _____
Name (optional): _____

☐ Please tick the box only if you dont want to be contacted about this project again in the future.

Hearing Handicap Inventory for Adults

Northwestern University Audiology Clinic
2240 Campus Drive
Evanston, IL 60208
Phone: (847) 491-3165 Fax: (847) 467-0410

HEARING HANDICAP INVENTORY FOR ADULTS (HHIA)

NAME:_____ DATE:_____

INSTRUCTIONS: The purpose of the scale is to identify the problems your hearing loss may be causing you. Check YES, SOMETIMES, or NO for each question. DO NOT skip a question if you avoid a situation because of your hearing problem. If you use a hearing aid, please answer the way you hear **WITHOUT** your aid.

		YES (4)	SOME-TIMES (2)	NO (0)
S-1.	Does a hearing problem cause you to use the phone less often than you would like?			
E-2.	Does a hearing problem cause you to feel embarrassed when meeting new people?			
S-3.	Does a hearing problem cause you to avoid groups of people?			
E-4.	Does a hearing problem make you irritable?			
E-5.	Does a hearing problem cause you to feel frustrated when talking to members of your family?			
S-6.	Does a hearing problem cause you difficulty when attending a party?			
S-7.	Does a hearing problem cause you difficulty hearing/understanding coworkers, clients, or customers?			
E-8.	Do you feel handicapped by a hearing problem?			
S-9.	Does a hearing problem cause you difficulty when visiting friends, relatives, or neighbors?			
E-10.	Does a hearing problem cause you to feel frustrated when talking to coworkers, clients or customers?			
S-11.	Does a hearing problem cause you difficulty in the movies or theater?			
E-12.	Does a hearing problem cause you to be nervous?			
S-13.	Does a hearing problem cause you to visit friends, relatives, or neighbors less often than you would like?			
E-14.	Does a hearing problem cause you to have arguments with family members?			
S-15.	Does a hearing problem cause you difficulty when listening to TV or radio?			
S-16.	Does a hearing problem cause you to go shopping less often than you would like?			
E-17.	Does any problem or difficulty with your hearing upset you at all?			
E-18.	Does a hearing problem cause you to want to be by yourself?			

		YES (4)	SOME-TIMES (2)	NO (0)
S-19.	Does a hearing problem cause you to talk to family members less often than you would like?			
E-20.	Do you feel that any difficulty with your hearing limits or hampers your personal or social life?			
S-21.	Does a hearing problem cause you difficulty when in a restaurant with relatives or friends?			
E-22.	Does a hearing problem cause you to feel depressed?			
S-23.	Does a hearing problem cause you to listen to TV or the radio less often than you would like?			
E-24.	Does a hearing problem cause you to feel uncomfortable when talking to friends?			
E-25.	Does a hearing problem cause you to feel left out when you are with a group of people?			

NO = 0 points Sometimes = 2 points YES = 4 points

Total # of points _____ / 100
Total # of points for SOCIAL____ / 48 = _____
Total # of points for EMOTIONAL _____ / 52 = _____

0 (no handicap) to 100 (total handicap)

0-16% = No handicap
18-42% = Mild-Moderate Handicap
44%+ = Significant Handicap

Adapted from Newman, C.W., Weinstein, B.E., Jacobson, G.P. and Hug, G.A., Test-retest reliability of the Hearing Handicap Inventory for Adults, *Ear Hear.*, 12, 355-357 (1991)

Liberman (2016) Questionnaire 2

Questionnaire 2

The following questions ask about your ability and experience hearing and listening in different situations.

For each question, put a mark, such as a cross (X), anywhere on the scale that runs from 0 through 10, below each question. Putting a mark at 10 means you would be perfectly able to do or experience what is described in the question. Putting a mark at 0 means you would be unable to do or experience what is described.

We expect that all the questions are relevant to your everyday experience, but if a question describes a situation that does not apply to you, put a cross in the "not applicable" box.

1) You are in a group of about five people, sitting round a table. It is an otherwise quiet place. You can see everyone else in the group. Can you follow the conversation?

Not at all 0---1---2---3---4---5---6---7---8---9---10 *Perfectly* ☐ *Not Applicable*

2) You are talking with one other person. There is continuous background noise, such as a fan or running water close by. Can you follow what the person says?

Not at all 0---1---2---3---4---5---6---7---8---9---10 *Perfectly* ☐ *Not Applicable*

3) You are in a group of about five people in a busy restaurant. You CANNOT see everyone else in the group. Can you follow the conversation?

Not at all 0---1---2---3---4---5---6---7---8---9---10 *Perfectly* ☐ *Not Applicable*

4) You are talking to someone in a place where there are a lot of echoes, such as a church or railway station. Can you follow what the other person says?

Not at all 0---1---2---3---4---5---6---7---8---9---10 *Perfectly* ☐ *Not Applicable*

5) You are having a conversation with one person in a room where there are many other people talking. Can you follow what the person you are talking to is saying?

Not at all 0---1---2---3---4---5---6---7---8---9---10 *Perfectly* ☐ *Not Applicable*

6) You are sitting around a table or at a meeting with several people. You can't see everyone. Can you tell where any person is as soon as they start speaking?

Not at all 0---1---2---3---4---5---6---7---8---9---10 *Perfectly* ☐ *Not Applicable*

7) You are sitting in between two people. One of them starts to speak. Can you tell right away whether it is the person on your left or your right, without having to look?

Not at all 0---1---2---3---4---5---6---7---8---9---10 *Perfectly* ☐*Not Applicable*

8) You are in an unfamiliar house. It is quiet. You hear a door slam. Can you tell right away where that sound came from?

Not at all 0---1---2---3---4---5---6---7---8---9---10 *Perfectly* ☐*Not Applicable*

Informed Consent Form

Informed Consent Form

Principal Investigator: _____ Phone: _____

Purpose of the Study:

The current study was designed to evaluate if there are any differences in how speech and other sounds are processed from the inner ear to the brainstem in participants who do and do not have reported noise exposure. The results obtained from participants will be used to better understand the phenomenon of suspected Hidden Hearing Loss (HHL) in humans.

Procedures:

All procedures will take place in Van Bokkelen Hall on the Towson University campus. In the first session, participants will be asked to complete 3 brief questionnaires that assess musical experience and perceived hearing handicap in different hypothetical communicative situations. Then, the participants will undergo a complete audiological evaluation that includes: otoscopic examination, tympanometric testing, acoustic reflex testing, pure tone audiometry, speech audiometry, and Distortion Product Otoacoustic Emissions (DPOAEs). Over the ear headphones will be used for pure tone and speech audiometry. Inserted earphones will be used for tympanometric testing, acoustic reflex testing, and DPOAE testing.

In the second session, participants will complete a testing battery in a sound treated booth that assesses auditory processing ability. Measures of dichotic listening, temporal processing, binaural interaction, and monaural low redundancy will be included in the auditory processing testing battery. Over the ear or inserted earphones will be used as needed.

Finally, a third session consisting of threshold-seeking Auditory Brainstem Response (ABR) will be administered to the participants. Electrodes (sensors) will be adhered to the participant's head using gel and tape. Low pitch and high pitch tones will be presented monaurally to the participant using insert earphones. The stimuli will decrease in loudness as responses from the auditory anatomical sites are recorded from the electrodes (sensors) and sent to the ABR software.

Risks/Discomfort:

There are no known risks associated with participation in the study. Breaks will be given intermittently throughout the testing procedure to account for participant fatigue. Participants may experience some discomfort when wearing over the ear headphones or with inserted earphones. Inserted earphones are placed into the outer ear at a depth of approximately 0.8 inches (standard length of a foam earplug). Some skin types may also experience irritation with the use of electrode gel and/or tape during the third session. Additionally, some participants may experience feelings of claustrophobia when in the sound booth.

Benefits:

It is hoped that the results of this study will provide some indication of clinical evidence to participants with normal hearing thresholds who struggle with communication in background noise.

Alternatives to Participation:

Participation in the study is voluntary. Participants are free to withdraw or discontinue at any time.

Cost Compensation:

Participation in this study will be compensated $10 per hour for each of the 3 sessions. If participants complete all 3 sessions faster than the scheduled 4 hours, then the amount compensated will be adjusted accordingly.

Confidentiality:

All information collected during the study period will be kept strictly confidential. You will be identified through your initials and the year of your birth. No publications from this project will include identifying information on any participant. If you agree to join this study, please sign your name below.

____ I have read and understood the information on this form.

____ I have had the information on this form explained to me.

_____ _____

Subject's Signature Date

_____ _____

Witness to Consent Procedures Date

_____ _____

Principal Investigator Date

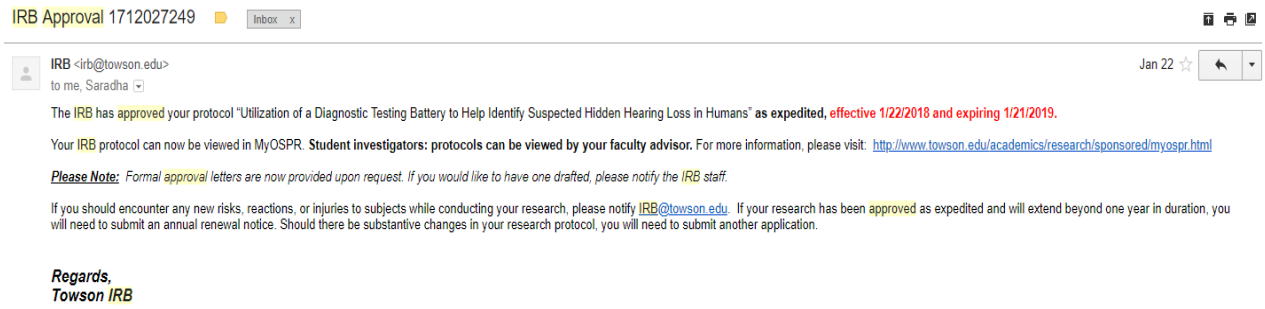
If you have any questions regarding this study, please contact the Principal Investigator, Larry Taylor, at Ltaylo19@students.towson.edu or the Institutional Review Board Chairperson, Dr. Elizabeth Katz, Office of the University Research Services, 8000 York Road, Towson University, Towson, MD, 21252; phone (410) 704-3207.

THIS PROJECT HAS BEEN REVIEWED BY THE INSTITUTIONAL REVIEW BOARD FOR THE PROTECTION OF HUMAN PARTICIPANTS AT TOWSON UNIVERSITY.

**If investigator is not the person who will witness participant's signature, then the person administering the informed consent should write his/her name and title on the "witness" line.

Appendix B

IRB Approval



IRB Approval 1712027249 Inbox x

IRB <irb@towson.edu> Jan 22
to me, Saradha

The IRB has approved your protocol "Utilization of a Diagnostic Testing Battery to Help Identify Suspected Hidden Hearing Loss in Humans" **as expedited,** effective 1/22/2018 and expiring 1/21/2019.

Your IRB protocol can now be viewed in MyOSPR. **Student investigators: protocols can be viewed by your faculty advisor.** For more information, please visit: http://www.towson.edu/academics/research/sponsored/myospr.html

Please Note: *Formal approval letters are now provided upon request. If you would like to have one drafted, please notify the IRB staff.*

If you should encounter any new risks, reactions, or injuries to subjects while conducting your research, please notify IRB@towson.edu. If your research has been approved as expedited and will extend beyond one year in duration, you will need to submit an annual renewal notice. Should there be substantive changes in your research protocol, you will need to submit another application.

Regards,
Towson IRB

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CURRICULUM VITA

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PROGRAM OF STUDY: Audiology

DEGREE AND DATE TO BE CONFERRED: Doctor of Audiology, May 2019

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Havre de Grace, MD

May, 2009

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Towson University	Fall 2015-May 2019	Doctor of Audiology	May 2018

Major: Audiology

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