Identifying and Quantifying Hidden Hearing Loss

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by

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Abstract

Objectives: The purpose of this study was to identify and quantify a so-called ‘Hidden Hearing Loss,’ which is defined as degraded speech perception in background noise with no hearing threshold elevation. We hypothesized that 1) the loss of auditory nerve fibers, or cochlear synaptopathy, causes hidden hearing loss, and 2) the level of medial olivocochlear reflex (MOCR) reflects cochlear synaptopathy, thus predicting hidden hearing loss. Design: Eighteen subjects were recruited for this study and complained of difficulty understanding speech in environments with high noise levels. All participants completed behavioral and MOCR testing, followed by a speech-in-noise understanding test. A modified Noise Exposure Questionnaire (NEQ) was also completed by each subject in order to account for their noise exposure history. Results: The level of MOCR did account for variance in SiN accuracy. Age and pure-tone audiometry also contributed significantly to SiN accuracy, but in a redundant manner. However, noise exposure history did not seem to have any correlation to SiN understanding within this present study.
Identifying and Quantifying Hidden Hearing Loss

The ability to understand speech is a crucial factor for human communication. With poor speech perception, an individual is less likely to understand a message being portrayed from the speaker, and their lifestyle and social interactions are thus directly affected. In the past, speech understanding was believed to be a byproduct of an individual’s hearing ability, therefore concluding that the more residual hearing you have, the better your speech perception will be. However, recent studies suggest that this is not the case and that accurate speech perception is not simply the product of hearing thresholds (Huetting & Altmann, 2005).

Cochlear sensory hair cell damage can be seen in an audiogram hours after noise exposure, with a brief elevation in hearing thresholds that in most cases, returns to normal with no noticeable permanent hearing impairment as a result. However, the discovery of Hidden Hearing Loss (HHL) presents a different outcome. Even though an individual’s audiogram may suggest that their hearing is normal, they may still have difficulty with speech-in-noise understanding and other complex signal detection (Liberman, 2015). In fact, individuals with HHL may even have affected speech perception in moderate levels of noise, around 70 to 80 dB, not just in environments with higher noise levels. Since this hearing loss tends to be missed by those in hearing healthcare, it has thus been referred to ‘Hidden Hearing Loss’ due to its lack of identification.

HHL may have a lack of sensitivity towards threshold measures due to different spontaneous rates each individual neuron has in the neural pathway. For every hair cell in the inner ear, there is a plethora of nerve fibers that connect to these hair cells, all with different spontaneous firing rates and frequency dependencies. The fibers with high spontaneous rates (HSR) tend to bare lower threshold while those with low spontaneous rates (LSR) bare higher
threshold and are the most vulnerable to damage when exposed to moderate to loud noise. These two spontaneous rates act as an overlapping function, where the HSR initially acts as a protective layer towards the LSR, in lower levels of noise. However, as noise level increases, HSR begins to plateau while the LSR continues to grow linearly, being more and more exposed to the impact of noise. Therefore, when an individual is exposed to moderate to loud noise levels, these LSR nerve fibers are the ones that begin to degenerate and could result in HHL. According to Liberman, Epstein, Cleveland, Wang & Maison (2016), even with the return of a normal threshold, up to 50% of the synapses between inner hair cells and cochlear neurons are lost. This is referred to as cochlear synaptopathy: the loss of auditory-nerve connections between the inner hair cells of the cochlea and the synapses of the auditory nerve fibers.

Previously, outer and inner hair cells in a human cochlea were deemed to be the most vulnerable aspects of the inner ear. However, recent studies suggest that in fact, this may not be the case and instead, the synapses between hair cells and cochlear nerves within the neural pathways of a human ear may degenerate and deteriorate prior to an individuals hair cells, when stimulated with moderate to high levels of noise. This degeneration located at the hair cell synapses or somewhere further along the neural pathway cannot be detected in any current clinically used hearing tests, particularly because it does not have an affect on the hearing thresholds presented by a clinical audiogram. These thresholds presented in an audiogram focus on the population of nerve fibers in the inner ear that have been less damaged. However, in order to quantify HHL, attention needs to be given to the damaged nerve fibers in these neural pathways.

In order to try identifying and quantifying HHL in this study, a medial olivocochlear reflex (MOCR) test was selected for use, in conjunction with a speech-in-noise (SiN) test and
noise exposure questionnaire (NEQ). The MOC reflex begins in the superior olive within the brainstem. This reflex has many roles, but its ability to decrease cochlear amplifier gain and improve signal detection in noisy situations are the most important functions for the purposes of this research study. Thus with proper MOC activation, a decrease in the cochlear amplifier gain would allow an individual to supply more selective attention to a desired stimulus, especially in noisy situations where background noise can interfere (Redfern & Goodman, 2017). Kujawa & Liberman (1997) also state that cochlear efferent feedback pathways are known to protect against hair-cell loss and threshold elevation following exposure to very loud sounds. Even though the MOCR is not applied within audiology clinics, it is an important tool that may give us more insight on the auditory functioning of humans. Therefore this method was selected for this study, in order to see if individual differences would arise in efferent strength and MOCR.

Research Questions

The study at hand presents two research questions: Where in the auditory system does the problem of HHL occur? Also, is there an objective test that can identify individuals with this type of problem and quantify the severity of the problem?

Methods

Participants

Eighteen participants (8 female) between the ages of 18 and 66 years participated in this study (mean age = 32.4 year; SD = 3.83 years). All subjects reported an excessive amount of noise exposure in their lifetime and difficulty understanding speech in noisy situations. None of the subjects reported prior hearing device use. Participants included in the study had otoscopic screening measures and 226-Hz tympanograms within normal limits bilaterally. Using a clinical audiometer (GSI-61, Grasson Stadler), pure-tone air-conduction thresholds were also measured
at nine audiometric frequencies: .25, .5, 1, 2, 3, 4, 6, 8 and 12 kHz. Inclusion criteria consisted of having air-conduction thresholds $\leq 20$ dB HL at .25-4 kHz and any type of hearing thresholds past 4 kHz.

Individuals with thresholds $>20$ dB HL between .25-4 kHz were excluded from participation in this study for various reasons. First, almost all speech information is at 4 kHz or below so having a hearing loss above 4 kHz is not expected to have a direct impact on speech recognition, and thus would not have significant effects on the data collected in this study. Second, we ultimately wanted to establish that all of our subjects had normal audibility for speech based on their audiometric thresholds. By allowing subjects to have high-frequency (>4 kHz) hearing losses, it maximized the chance of obtaining a sample of subjects with “hidden” hearing loss at lower frequencies (.25 – 4 kHz) that are important for understanding speech. The Institutional Review Board at the University of Iowa approved all experimental protocol and written informed consent was obtained from all subjects.

**Equipment**

All testing occurred in two laboratories at the Wendell Johnson Speech and Hearing Clinic at the University of Iowa with all experiments taking place inside double-walled sound-treated booths. During MOCR testing, subjects were seated in a reclining chair. Stimulus playback and data acquisition were accomplished using a custom-written MATLAB (The MathWorks Inc.) software package. The software ran on a Windows desktop computer connected to a 24-bit sound card (RME Fireface). The stimuli were then routed to channels of an OAE probe-microphone system (ER-10X, Etymotic Research). Recorded OAE’s were analyzed post-hoc using custom MATLAB software.

Within 24 hours of each subject, MOCR equipment was calibrated in order to verify that
equipment was working properly. Calibration included the use of cavity recordings based on fixed lengths.

During SiN testing, a single loudspeaker, situated at a 0° azimuth angle was used to present stimuli (model #LOFT40, JBL). Stimuli were presented from another custom-made MATLAB software (The MathWorks Inc.), and were implemented using a PsychToolbox 3 package (Brainard, 1997; Pelli, 1997). A computer monitor was also used inside the sound booth to display testing information, which was placed at eye level for each subject.

**Procedures**

1. MOCR

The effects of contralateral noise on transient-evoked OAE’s (TEOAEs) were examined by obtaining input-output functions across a wide range of reflex activator noise levels. Click stimuli were presented ipsilaterally at a rate of 50/s in the right ear. Contralateral noise was presented to the left ear in order to activate the MOCR. To start, there was no noise present. Over the course of one second, the noise increased linearly to 80 dB SPL and over the course of another second, it then decreased back down the to starting level. The stimulus was then followed by two seconds of no contralateral noise. This sequence was repeated 255 times per subject and took approximately twenty minutes to complete.

Subjects were awake for the duration of the experiment, and were asked to remain as quiet and still as possible during each recording. It was important that each participant did not make any extra behavioral noise such as swallowing, coughing or popping of their ears since these behaviors would interfere with the data being collected. Thus, subjects were instructed to watch the display of a computer screen in order to monitor their excess movements, and keep
them to a minimum. Because research suggests that MOCR activity may be modified by attention (Delano et al., 2007), subjects were asked to perform a simple key-press task throughout the procedure to help them maintain relative alertness.

2. Speech-In-Noise Test

The Speech-in-Noise (SiN) recognition task used was based on the California Consonant Test (CCT) (Owens & Schubert, 1977). During this paradigm, monosyllabic consonant-vowel-consonant (CVC) words were projected from a loudspeaker approximately 1.2 meters away from the listener. A male speaker, at a consistent level of 65 dB SPL, spoke these words in General American English. However, these English words were spoken in the presence of varying types and levels of background noise. Two SNRs were created including a +3 dB (high SNR) and -3 dB (low SNR) for multi-talker babble background noise. Based on prior experiments, the low SNR condition was used in order to provide a more difficult listening condition of approximately 70% accuracy (Sarampalis et al., 2009). The high SNR on the other hand provided an easy listening condition, and would thus result in higher speech perception scores. In our manipulated version of the CCT, we also included trials where the background noise included rhythmic and non-rhythmic music, in order to see individual differences across subjects and various background noises. Therefore, the speech perception task included a total of 200 trials including 50 high SNR multi-talker babble, 50 low SNR multi-talker babble, 50 non-rhythmic music and 50 rhythmic music conditions, presented in a randomized order.

Participants started each trial by fixing their gaze on a plus symbol located in the middle of the computer monitor. As their gaze was fixed, a cue phrase “check the word” was presented followed by two seconds of one of the four background noise conditions. After one second of background noise, the target word was then spoken by the male voice. Four answer choices that
differed by one consonant in either the initial or final position were then displayed on the computer monitor for the subject to select (e.g., “1. THAN, 2. VAN, 3. BAN, 4. PAN” for the target word “ban”) by clicking a corresponding key on a keyboard. No feedback was given to the subject once a trial was completed.

3. Noise Exposure Questionnaire

A modified version of the Noise Exposure Questionnaire (NEQ) taken from Johnson, Cooper, Stamper and Chertoff (2017) was used as part of this study. The NEQ took about 5-10 minutes to complete, and consisted of 17 questions. This NEQ established whether or not an individual was at risk for Noise-Induced Hearing Loss (NIHL), based on their prior exposure to noise in occupational and non-occupational settings from the past 12 months. Subjects were asked to recall participation from the past year to gather information on seasonal and infrequent activities and events.

This NEQ consisted of three individual sections. The first section included demographic information (gender and age). The second section consisted of screening questions (#1-6) that could be used to determine individuals with a high-risk of NIHL. The rest of the questions included in this NEQ (#7-17) were detailed questions that referred to each individual’s participation in specific, noisy activities. These final eleven questions were used to calculate each subject’s annual noise exposure.

Analysis

1. MOCR

All analyses for MOCR data were completed on a Windows PC computer with custom-made MATLAB software, written by Dr. Shawn Goodman (The MathWorks, Inc.). Protocol for this portion of the analysis used the same structure and steps as Mertes & Goodman (2016).
2. Speech-in-Noise Test

Analysis for SiN accuracy was based on a percent correct calculation. Out of the trials completed by each subject, the amount of trials they answered correctly was then divided by the total amount of 200 trials, resulting in a percentage. These total percentages can be seen in Fig. 1, resulting in a normal distribution with a slight tail in the negative direction.

3. Noise Exposure Questionnaire

Calculation was almost identical to the protocol used in Johnson, Cooper, Stamper and Chertoff (2017). Episodic (occasional) and routine (daily) noise exposures were calculated separately and then combined for a total annual noise exposure score, represented in $L_{Aeq8760h}$. In this metric, “$L$” expresses sound pressure level in dB, “A” represents use of an A-weighted frequency response, “eq” is used to express a 3-dB exchange rate for calculation of the time/level relationship and finally, “8760h” indicates the total duration of noise exposure in hours. Any scores $\geq 74 L_{Aeq8760h}$ were considered at risk for NIHL while any score $<74 L_{Aeq8760h}$ was not of concern for potential NIHL. For all questions in the NEQ, the frequency of occurrence was obtained as well.

One modification used for the NEQ version used in this study included obtaining the percentage of hearing protection used for each episodic question (#7-17). By obtaining information about hearing protection, we were able to calculate the appropriate sound level of each activity based on time with hearing protection and time without hearing protection and factor this information into the final score for each subject.

Results
MOCR results showed variable degrees of neural feedback across individuals. While some subjects had ideal MOC reflexes, other subjects had poor MOC reflexes, with some even being non-existent. MOC reflex results from a few subjects from this study can be seen in Fig. 2.

**Figure 2**: Fig. 2A is an example of a subject who had a stronger MOCR. Fig. 2B shows a subject that had weaker MOCR. Fig. 2C on the other hand is an example of a subject that lacked an MOCR. The top rows in these figures show changes in OAE magnitudes as a function of time, the middle rows display OAE phase change as a function of time and the bottom rows show the level of a contralateral noise activator. The contralateral noise rises for one second, falls in level for another second and then is absent for two seconds. Dashed vertical and horizontal lines show differences in MOCR threshold as well as temporal offset of the MOCR relative to the activator.

After analysis, three independent variables were deemed important for this study: age, pure-tone audiometry at 4 kHz, and MOC reflexes. These independent variables were compared to one another as well as to the dependent variable of each subject’s SiN accuracy. One initial finding was that age and pure-tone audiometry at 4 kHz were directly correlated, as seen in Fig. 3.

When looking at the relationship that SiN accuracy had with the mentioned three independent variables, all displayed other significant correlations as displayed in Fig. 4.
A multivariate linear-regression analysis yielded results also, when MOC reflex, age and pure-tone threshold at 4 kHz were used as independent variables that predict SiN accuracy. Although all three variables show significant correlation with SiN accuracy, threshold is strongly associated with age, with only MOC reflex explaining variances in SiN accuracy when age is factored out. These results can be seen in Fig. 5.

**Discussion**

Recent animal studies and a few human focused studies have attempted to quantify and identify the underlying physiology of HHL. Liberman, et al., (2016) claims that synaptic connections between hair cells and cochlear neurons can degenerate well before the loss of sensory hair cells. Yet this degradation is not noticeable in a clinical audiogram and likely hinders the understanding of complex stimuli, such as speech. However, past research has deemed it impossible to tell whether or not this neural degeneration is at the level of the low and high spontaneous rate fibers (LSR and HSR) or further along the neural pathway due to the lack of technology that can be used on human subjects, without harm.
Thus study thus aimed to determine whether or not there is an objective test that can quantify HHL, and where this problem specifically occurs within our auditory and neural system. The overall results of this study suggest that early neural processing has some affect on SiN understanding and accuracy and that a wide range of individual differences in early neural systems of the subjects existed, no matter their age. With the use of a unique MOC reflex testing measure and its relationship with SiN accuracy, the multivariate linear regression model suggests that approximately 25% of an individual’s SiN accuracy can be accounted for from a subject’s MOCR strength. Since the MOC reflex is an auditory processing mechanism located at the superior olive within the brainstem, the 25% of the SiN accuracy must lie somewhere physiologically between the cochlear proper, where hair cell and neural synapses are located and the superior olive. Another 45% of SiN accuracy can be accounted for by pure-tone threshold and age, which are both currently identified in audiology clinics across the world. However, there are no neural processing measures similar to our new MOCR measure being used clinically. Therefore by incorporating an early neural processing measure in clinics, 70% of an individual’s speech score can potentially be identified, instead of just 45%. This unique new measurement may also be able to quantify the severity of the problem.

Many previous studies have also focused on noise exposure and it’s relationship with the HHL phenomenon, such as Liberman (2015). This study also aimed to determine whether or not noise exposure is a true factor that potentially makes an individual more prone to the acquisition of HHL. Based on the results of the modified NEQ used from Johnson, et al., (2017), there were no significant correlations to the SiN accuracy of subjects or other variables within this study, and thus it could not be concluded that noise exposure history had any affect on an individual’s speech perception ability. This does not rule out noise exposure as a factor contributing to the
acquisition of HHL, but rather based on the use of the questionnaire within this study, concludes that noise exposure history was not important.

Many things may explain why this NEQ did not contribute to the results of this study. One reason may be that the survey was not a good indicator of actual noise exposure for each individual. With this survey, all subjects had to use their memory in order to recall events in which they were exposed to loud environments or situations. Therefore they may have been inaccurate on their recollection and thus could have skewed the data. Another reason is that this NEQ identified individuals who were at risk for NIHL, it did not indicate people who had HHL. Finally, the survey only accounted for the past 12 months of noise exposure, limiting the amount of noise subjects could record on their survey. This point is important to note since the oldest participant would have had, in general, a greater amount of noise exposure than the youngest participant solely due to age. Yet the younger subject may have recorded a noisier past year compared to the older participant and would be deemed at risk for NIHL, whereas this may not actually be the case.

Future research is needed to provide more support on the results portrayed in this study. Using a larger sample size of participants can help to verify the present results. Also, approximately 30% of the variability of SiN difficulty remains unaccounted for. Therefore measures such as electroencephalogram (EEG) that target areas of the brain, past the lower brainstem level, may be helpful. Testing working memory, attention and language may also be helpful to see if these factors have anything to do with SiN understanding and accuracy. Finally, when trying to identify the unknown 30% of variability of SiN difficulty, future research may fall short. This may be because this 30% accuracy is just random chance or based on other
factors that account for randomness such as the focus or motivation of a subject. These things need to be considered before future research is attempted in this area of study.

**Conclusion**

Ultimately, the results across subjects account for 70% of total SiN accuracy, compared to a total of 45% SiN accuracy that is currently being identified in audiology clinics. The unique MOC reflex paradigm that was used within this study was able to potentially determine the general location of this HHL phenomenon, thus suggesting it occurs anywhere from previously researched LSR fibers up to somewhere in the lower brainstem. After more research is completed using MOCR testing, a better understanding of HHL and its physiology will be better understood, potentially acquiring early neural processing measures in clinics.

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References


