Using Neural Metrics to Determine Optimal Hearing Aid Gains for Individual Patients

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***Abstract*—**Hearing aids have been tremendously successful at restoring some hearing abilities for people with hearing impairments. However, even with some of today's most advanced technologies, patients still have an abnormal degree of difficulty understanding speech in the presence of competing talkers. Hearing scientists believe that listening in complex acoustic environments such as this may require the use of details in the acoustic waveform (known as Temporal Fine Structure) that might otherwise be unimportant. This paper evaluates the importance of this *fine structure* information in terms of the underlying physiology, hearing aid amplification strategies, and the resulting neural responses. By combining ideas from previous behavioral and neurophysiological experiments, I present a study that identifies how the gain may be adjusted optimally to improve the encoding of either the slowly varying *envelope* or the more rapidly varying *fine structure* information for patients based on their individual physiological impairments. Results indicate that, although fine structure coding does not appear to be affected substantially, envelope coding depends greatly on the underlying physiology.

I. Introduction

In a normally functioning cochlea, several rows of outer hair cells actively amplify the vibration of the basilar membrane and a single row of inner hair cells transduces this vibration to electrical signals for the brain. One common type of hearing loss is the result of impaired hair cells (often due to noise exposure or aging) and is referred to as sensorineural hearing loss (SNHL). The typical model of SNHL assumes that most of the impairment is due to damaged outer hair cells, which normally amplify low intensity sounds but apply less gain to sounds that are already high intensity [1]. (However, note that several studies [2-4] suggest that a more typical scenario may involve both inner and outer hair cell dysfunction.) A hearing aid often attempts to restore this gain, compressing the dynamic range to make soft sounds audible while keeping loud sounds relatively comfortable. Although hearing aids have been tremendously successful in many situations, patients still have an abnormal degree of difficulty in acoustically complex environments (for example, a crowded restaurant) [5].

Biondi [6] suggested that, by comparing normal and *aided+ impaired* neural codes, a hearing aid might be designed that minimizes the difference (see Figure 1). Several scientists have used computational models of auditory physiology to implement such a system [7-10]. For example, Bondy et al [7, 10, 11] attempted to minimize the difference between normal and impaired coding. However, the authors considered only the rate-place encoding of the auditory signals and did not calculate any measure of phase locking. (Phase locking refers to the fact that auditory neurons tend to fire in sync with a particular phase of the stimulus waveform.) More recently, Bruce [12] claims to have calculated neural information based on both average discharge rate and

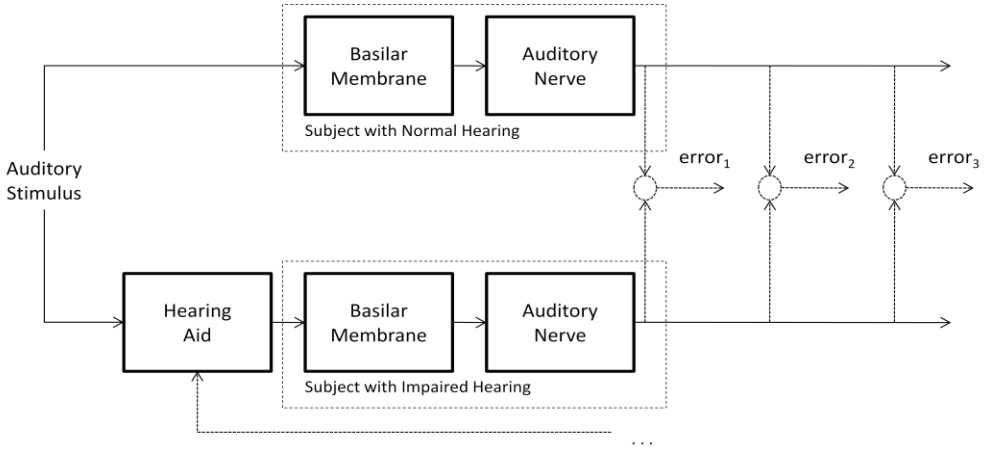

Figure 1. Framework for calculating optimal hearing aid parameters (adapted from Biondi 1978)

spike timing. However, the only difference between these two measures was the length of the averaging window; the authors used a very short window size to determine timing. They averaged spike counts using a Hamming window length of 256μs, which has the effect of attenuating fluctuations faster than approximately 2.5kHz. This metric might therefore measure timing (e.g. phase locking) in response to low frequencies, but it may not be sufficient because synchronous timing can be measured up to at least 5kHz in the auditory nerve [13]. Because precise timing may be important for hearing in complex situations, future physiologically-based designs should consider metrics that include both long-term rate and precise temporal coding.

A signal may be thought of as a combination of a rapidly varying signal (the *temporal fine structure, or TFS*), modulated by a slowly varying signal (the *envelope*). Shannon et al [14] showed that the acoustic envelope of a speech signal is often sufficient for speech recognition in quiet. He later showed [15] that the number of spectral bands (of acoustic envelope) needed for speech recognition increases as the difficulty of the listening situation increases. However, as Zeng [16] pointed out, a large number of narrow auditory filters can introduce TFS cues that could potentially be transduced into the neural system. Envelope and TFS coding at the level of the auditory nerve will be referred to as *neural envelope* and *neural TFS* in this paper.

Some scientists believe that listeners with normal hearing take advantage of the acoustic waveform's temporal fine structure to listen in the short "dips" of the background noise, and perhaps use this information to improve auditory stream segregation.[17] Recent findings suggest that hearing impaired listeners are often unable to take advantage of TFS [18] , although it should be noted that even the normal hearing listeners in this study may have depended on some residual envelope cues in addition to (or even in place of) TFS cues. In any case, this inability to recognize speech sounds may be due in part to the amplification, which cannot completely restore hearing to normal. Psychophysical data in the literature suggests that there is a wide variability among individual patients, with some hearing impaired subjects showing a near-normal response to fine structure information [19]. One possible reason for this unexplained variability is the fact that patients are often characterized by a rather crude approximation of their hearing thresholds. This does not take into consideration the many variations of physiological impairments that may result in similar audiometric thresholds.

The following study extends the Bruce et al [12] experiment in two ways. First, metrics for calculating neural envelope and TFS coding are considered. This allows us to evaluate the neural coding from a different perspective, without imposing any limitations on the temporal resolution of our measurements. Additionally, responses were measured with three different types of simulated impairments. This allows us to evaluate the hypothetical range of gains that would be appropriate for patients with different sensorineural impairments.

II. Methods

A. Experimental Design

The basic experimental design is based on the framework proposed by Biondi [6] and extended by Bruce et al [12]. In short, a mild hearing impairment is simulated, with thresholds sloping from 20dBHL at 250Hz to 50dBHL at 6kHz. A linear hearing aid prescription (NAL-R) is calculated, which provides a frequency-dependent gain. The overall gain was adjusted by ±40dB in 5dB steps. The resulting acoustic signal (the word "dark", extracted from a sentence in the TIMIT database) was passed through a computational model of the ear and auditory nerve [20, 21]. The model simulated fibers at 30 characteristic frequencies (CFs), and 3 spontaneous rates per CF. The spontaneous rates were chosen to be representative of the physiological distribution [22], and the responses were weighted accordingly.

A neurogram (firing rate –vs- characteristic frequency –vs- time) was calculated for the normal case and for each of the impaired cases. The gain which produced a neurogram with the least mean absolute error (re normal) was chosen as the optimal gain for a given input level. In the present study, as in the Bruce et al study, neurograms were smoothed with two different Hamming windows. The first window was 8ms in duration and was designed to represent the average discharge rate. The shorter window was 256μs in duration and was designed to represent the spike timing information.

B. Envelope & TFS

In addition to the neurograms, the present study calculated neural envelope and TFS coding. The metrics are based on work by Joris et al [23, 24], in which they used *shuffled autocorrelations* (SACs) to quantify the response components due to the envelope and the response components due to the fine structure. An SAC is similar to a conventional autocorrelation function, except that the time between every pair of spikes in represented, even across multiple presentations. Spikes can be compared across multiple repetitions of the same stimulus (referred to as an SAC) or across a condition using a stimulus and another condition using an inverted-polarity version of the same stimulus (referred to as a *cross-polarity autocorrelation*, or X_PAC). It is worth noting that, because spikes are compared across different presentations, the resulting correlation functions are able to measure very fine time intervals that would otherwise be impossible because of the refractory period of a neuron. Similarly, we can calculate cross correlations, where two different stimuli are used. In this analysis (discussed by Heinz and Swaminathan [25]), we can calculate a *shuffled cross-correlation* (SCC) between two stimuli or a *cross-polarity cross-correlation* (X_PCC) between one stimulus and a polarity-inverted second stimulus.

Because the envelope of a band-limited signal is symmetric in amplitude, inverting the polarity of a stimulus will only change the fine structure. Therefore, adding the SAC and X_PAC (effectively looking at what is similar between the two) will provide a measure of the envelope coding. This is known as the SumCor. Calculating the difference between the SAC and X_PAC (known as a DifCor) will provide a measure of what is different, i.e. the temporal fine structure. Similarly, we can add the SCC and the X_PCC to obtain a metric of the envelope coding (ρ_{env}) that is common between two stimuli. We can also subtract the X_PCC from the SCC to obtain a metric (ρ_{tfs}) of the TFS coding that is common between the two stimuli. The peaks of these correlation functions are used as a single numerical representation of the envelope or TFS coding.

In the framework of the current experiment, we want to maximize either the envelope of TFS coding *common* to both the normal and aided+impaired conditions. Therefore, the two stimuli used for the SCC and X_PAC metrics are in fact the same acoustic stimulus but passed through either the normal system or the aided-impaired system.

C. Simulated Impairments

The auditory nerve model used in this experiment [20, 21] allows selective control of both inner (IHC) and outer hair cell (OHC) functionality. Because various combinations of IHC and OHC damage can result in the same threshold levels, we can adjust the parameters of the model to simulate varying degrees of IHC and OHC functionality. This experiment simulated three types of impairment- mixed damage (2/3 of impairment due to OHC damage, 1/3 due to IHC damage), pure IHC damage, and nearly all OHC damage. As shown in Figure 2, the amount of hearing loss due to OHC damage is controlled to be either 2/3 of total loss, zero, or nearly the full loss. Because the outer hair cells provide limited gain at low frequencies, some IHC damage was needed at the lowest frequencies to achieve the full 20dB loss.

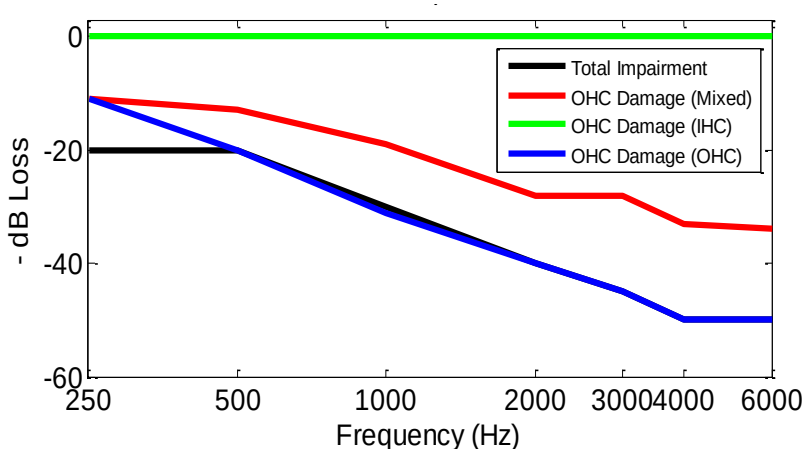

Figure 2. Three types of impairment. The lines show the amount of hearing loss due to outer hair cells (OHC) for each case. (Note that, in the "pure" OHC case, some hearing loss at low frequencies is in fact due to IHC damage.)

III. RESULTS

After confirming that the optimal gains for a single phone, /a/, were similar to the results achieved by Bruce et al [12], I needed to extend the duration of the stimulus in order to accurately measure the correlations (see the Discussion for more details on this limitation). The following analyses are based on gains applied to an entire word.

The mixed-damage experiment was first repeated with the long- and short-term averaged neurograms (see Figure 3A,B). The results look similar to the single phone case in which optimal gains for average discharge rate were generally above the prescribed gain while the optimal gains for spike timing information were at or below the prescribed gain. (Further reduced gains were generally optimal for increased sound levels when spike timing information was used.) The optimal gains for TFS coding (Figure 3D) are similar to the results for the short-term discharge rate (Figure 3B). However, the optimal gains for envelope coding (Figure 3C) differed somewhat from the results for average discharge rate (Figure 3A). Upon further analysis, it was noted that the higher gains tended to saturate the response of many of the nerve fibers, thus reducing the envelope coding at these levels.

A similar simulation was performed with primarily outer hair cell damage. The optimal gains for all four metrics were very similar to the mixed-damage case, indicating that the average discharge rate and envelope coding generally required gains above prescription but the short-term discharge rate and TFS coding generally required gains at or below prescription.

When the same simulation was run with only inner hair cell damage, the results for average discharge rate, short-term discharge rate, and TFS coding (Figure 4A,B,D) were similar to the previous simulation. However, the optimal gains for improving envelope coding (Figure 4C) were drastically different (as compared to Figure 3C). A cochlea with IHC damage appears to require much lower gains than one with OHC damage.

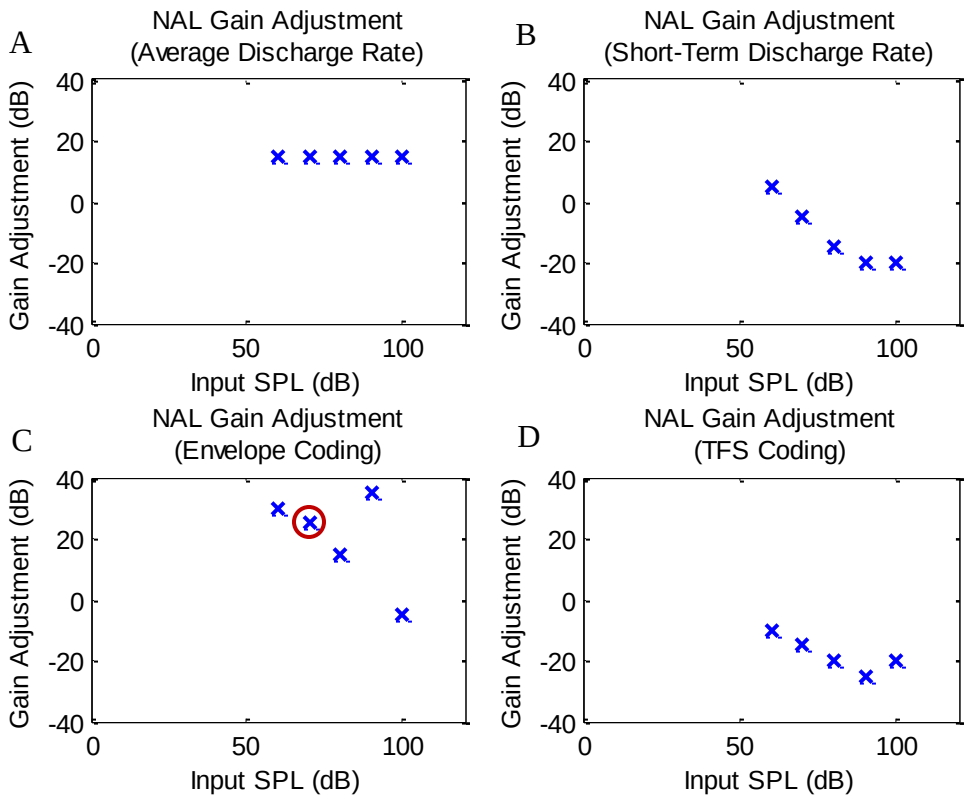

Figure 3. Optimal gains for *mixed hair cell damage*, based on average discharge rate (A), short-term discharge rate (B), envelope coding (C), and TFS coding (D). Note that gains above NAL are generally better for long term metrics, but gains below NAL are better for the short term metrics. Details regarding the circled data point are shown in Figure 5.

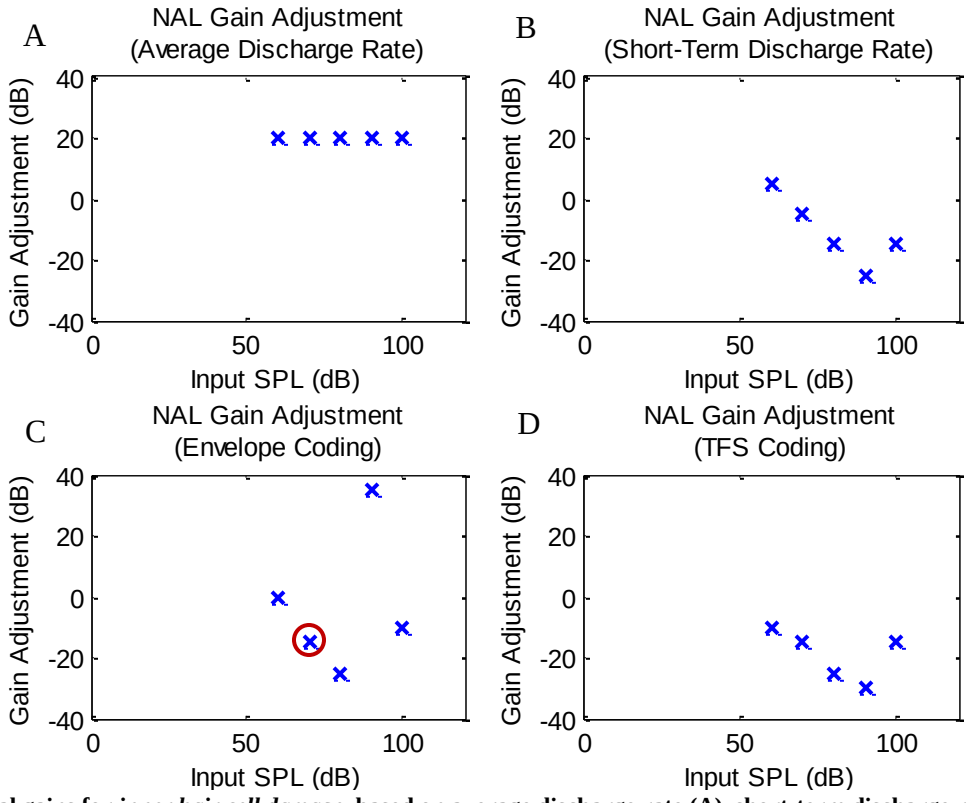

Figure 4. Optimal gains for *inner hair cell damage*, based on average discharge rate (A), short-term discharge rate (B), envelope coding (C), and TFS coding (D). Note that gains above NAL are still best for average discharge rate, but gains below NAL are better for all other metrics (including envelope coding). Details regarding the circled data point are shown in Figure 5.

Upon closer inspection (see Figure 5), we can see that envelope coding is particularly enhanced in the cases with impaired outer hair cells or in cases of IHC damage with moderately high gain. In general, we expect an increase in gain to result in an increase in envelope coding (assuming minimal neural saturation). However, because we are interested in maximizing the envelope *common to both normal and impaired systems*, minimal gain may be required in some cases.

For example, Figure 5E shows the SumCor (SAC + X_PAC) for the impaired systems with a gain 20dB below the prescribed gain. Notice that the peak of the IHC curve has lowered substantially, as compared to Figure 5B, to be much closer to normal envelope coding (see Figure 5A,D). Also notice than the SumCor of the cross-correlation (Figure 5C,F) is reduced as the gain is reduced for the mixed damage case (indicating envelope coding further from normal), but the SumCor has increased as the gain is reduced for the case with only IHC damage (indicating envelope coding closer to normal).

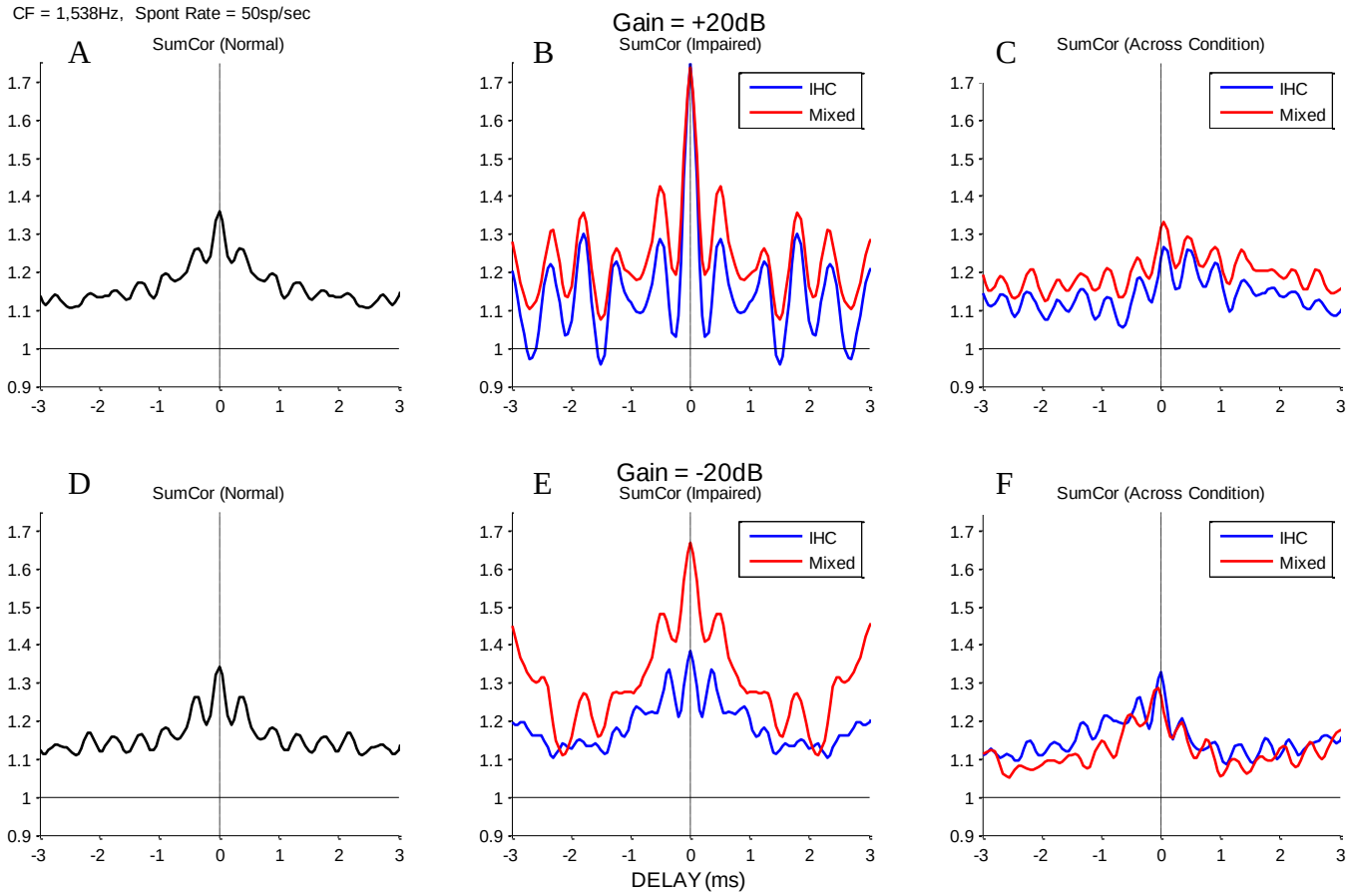

Figure 5. Envelope Coding for an input level of 70dBSPL (CF=1,538Hz). The SumCor function (SAC+ X_PAC) for the normal system (A, D). A comparison of two impaired systems with a gain of 20dB greater than prescription (B). The SumCor function (SCC+X_PCC) for the normal and impaired systems, with a gain of +20dB (C). A comparison of two impaired systems with a gain of 20dB less than prescription (E). The SumCor function (SCC+X_PCC) for the normal and impaired systems, with a gain of -20dB (F).

IV. Discussion

It is interesting to note that compression, a reduction in gain with increasing level, is needed to preserve timing information, in terms of both short-term rate (based on the neurogram) and temporal fine structure (as calculated using the correlation metrics). Little or no compression seems to be necessary for preserving rate information <100Hz (given an 8ms Hamming window), but it appears that some compression may in fact be necessary for envelope coding (which was only limited in frequency by the characteristic frequency of each nerve fiber).

Of particular interest is the result suggesting that the optimal gain for preserving envelope in a patient with primarily OHC damage is drastically higher than the gain for a patient with primarily IHC damage. If this is in fact true, it would be beneficial to clinically assess a patient's OHC/IHC damage before fitting a hearing aid. Given some information about the underlying physiology, a hearing aid could be better fit for the individual patient. For example, a computational model could be used to match the patient's behavioral performance, then the hearing aid parameters could be adjusted to improve performance of the model. These optimized parameters could then be tested on the patient, thus minimizing the patient's time in the clinic but potentially maximizing performance.

There are several means by which a researcher/clinician could predict the impairment of inner and outer hair cells. For example, Moore et al [26] suggest that loudness matching, ERB (equivalent rectangular bandwidth) measurements, and growth-of-masking functions can all be used as measurements of OHC functionality. (The remaining impairment may be assumed to be related to IHC functionality.) Our lab is also working on behavioral metrics of OHC/IHC health. For example, it may be that patients with particular physiologies confuse certain sounds. By analyzing these confusions, we may be able to predict the physiological impairments that result in behavioral impairments. These methods are by no means conclusive evidence, as the relationships between physiology and behavior are still poorly understood. However, there is growing evidence that these measurements do in fact represent the active and passive responses of the cochlea.[27-31]

Unfortunately, the auditory nerve model used in this study [20, 21] has some limitations. The first limitation is that it is a model of a cat auditory system, however the primary difference between cat and human cochleae is thought to be the size. This limitation can be overcome with a simple transform [32]. A larger limitation is the exponential characteristics of the synapse model. This version of the code does not accurately model the long-term adaptation or the distribution of spontaneous rates. Because of these limitations, envelope coding may not be accurately represented. However, a new version of the auditory nerve model [33] incorporates these changes and more accurately reflects the adaptation (and therefore envelope coding) of the physiological system. Future work will incorporate this updated model.

Another limitation of the techniques used in this study is that the short duration of the stimulus limits the correlation analysis. Because the original window is rectangular, the correlation will inherently be weighted by a triangular function (i.e. reduced at the edges). This effect can be counteracted with an inverse triangular weighting, but other effects of the short duration remain. The most difficult effect to overcome in this study is the low number of neural spikes. (Also note, as the level and/or gain is reduced, the number of spikes decreases, especially for nerve fibers with low spontaneous rates.) A reduced number of spikes acts to reduce the statistical accuracy of the correlation metrics. To help overcome this limitation, the stimulus duration was increased from a single phone (<150ms) to a whole word (~380ms). In future work, it might also be beneficial to bootstrap the data to obtain a more reliable metric.

Future work might also include an analysis of the spatiotemporal coding. It has been suggested [34] that the relative phase of signals along the basilar membrane may be an important feature for level encoding. The correlation analyses used in the current study could be used to measure the *characteristic delay* (CD) at various places along the length of the cochlea. The CD would simply be the lag at which the correlation function is maximal. By selecting gains that minimize the CD of the SCC, we may be able to minimize the phase error of the aided-impaired system. Because the importance of this cue is poorly understood, more work is needed to determine the effectiveness of this approach. (The phase may also be explicitly adjusted within the gain control, but this method, as implemented previously, has met with little success [9, 35].)

The original Bruce et al study [12] calculated optimal gains for multiple phones in succession. Due to the limitations of the correlational analyses used here, individual phones were far too short in duration to analyze in this study. However, if these limitations can be overcome, it would be interesting to see how the neural coding of multiple phonemes in succession changes the results. This would also be a great place to use the newest version of the auditory nerve model, which successfully models forward masking (as caused by adaptation in the cochlea).

V. Conclusion

This extension of a previous study evaluated envelope and fine structure coding as a function of gain to determine the optimal gain for hearing aids. A computational model of the auditory nerve was used to determine the gain needed to best restore neural coding to normal. The differences between ears with mostly outer hair cell damage and those with mostly inner hair cell damage were most obvious when envelope coding was used as the criterion for optimal gain. For impaired systems with mixed damage, restoration of envelope coding required gains well *above* prescription. However, for impaired systems with only inner hair cell damage, restoration of envelope coding required gains well *below* prescription. It was demonstrated that although envelope coding may increase with gain, lower gains were chosen as optimal in the case of IHC damage because they correlate best with the original neural coding.

This work has potential clinical applications for hearing aid design and fitting. If a clinician can use some simple measurements to better understand the underlying physiological impairments, the gain can be adjusted appropriately. This work suggests that cochlear regions with primarily outer hair cell damage may need more acoustic gain than regions with primarily inner hair cell damage (given the same audiometric thresholds).

Acknowledgment

My thanks to Dr. Michael Heinz and Jayaganesh Swaninathan for providing the code to measure envelope and fine structure coding.

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