THE PROBLEMS OF INNER-HAIR-CELL IMPAIRMENT FOR HEARING AID GAIN PRESCRIPTIONS

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Abstract

Historically, impairment of outer hair cells (OHCs) in the cochlea was thought to be the predominant contributor to sensorineural hearing loss (SNHL). The loss of cochlear compression and broadening of cochlear filtering that are produced by OHC impairment have consequently guided the development of compression algorithms in hearing aids and prompted the investigation of spectral sharpening schemes. More recently there has been an interest in the consequences of cochlear dead regions, that is regions of complete inner hair cell (IHC) dysfunction, for speech intelligibility and hearing aid amplification prescriptions. However, there is now growing evidence from physiological studies in animals and psychophysical studies in humans that partial IHC impairment is a substantial component of SNHL in most cases. Estimates of the IHC contribution to threshold shifts are typically around 1/3 to 1/2 of the total threshold shift. Furthermore, a recent animal study has shown that significant dysfunction of the IHC/auditory-nerve-fiber (ANF) synapse and subsequent degeneration of ANFs can occur even in cases of a temporary threshold shift [1].

In this study we have utilized a computational model of the auditory periphery [2, 3] to study the individual and combined effects of OHC and IHC impairment on optimal hearing aid amplification gains. The mean discharge rate and spike-timing information in response to speech stimuli were both taken into consideration when analyzing the neural response patterns across the model ANFs. Simulation results show that optimal gains predicted by the model for mild impairment of just the OHCs match the NAL-R and NAL-NL1 prescriptions. In contrast, any degree of IHC impairment leads to a mismatch between optimal gains for restoring ANF mean rates and spiking-timing, and the best that linear prescriptions and wide-dynamic-range-compression schemes can achieve is a balance between restoring these two aspects of the ANF response. These results prompt the development of more sophisticated amplification schemes to better deal with the distortions in ANF spike-timing information arising from IHC and OHC impairment.

I. INTRODUCTION

- Early observations in hearing aid prescription procedures showed that the preferred gain at a particular frequency equaled approximately half the hearing threshold at the same frequency. This is referred to as the "half-gain" rule [4].
- Popular linear hearing aid prescriptions, including the NAL-R (National Acoustic Laboratories-Revised) and DSL (Desired Sensation Level) prescriptions, are based on variations of the half-gain rule and judgments of speech intelligibility, sound comfort, and loudness equalization [4].
- The goal of this study was to find optimal single-band gain adjustments around the NAL-R and DSL prescribed gains by using the neural representation of speech rather than perceptual feedback.
- The independent and combined effects of outer hair cell (OHC) and inner hair cell (IHC) impairment were investigated.

II. METHODS

A. The Auditory-Periphery Model

- The auditory-periphery model used in this study (Fig. 1) was that of Zilany and Bruce [2,3]. This phenomenological model describes the cat auditory pathway from the middle ear through to the auditory nerve.
- In this study, the real-ear unaided gain is modelled after the adult head-related transfer function described by Wiener and Ross [5].
- Speech waveforms, with instantaneous pressures in Pascal and sampled at 100 kHz, are delivered to the model to derive an auditory nerve spike train for a fiber with a specific characteristic frequency (CF).
- Model parameters, C_{IHC} and C_{OHC}, which control the level of inner and outer hair cell impairment respectively, can be adjusted to provide a desired hearing threshold shift at a specific CF. A C_{IHC} or C_{OHC} of 0 produces full impairment whereas 1 produces normal function.

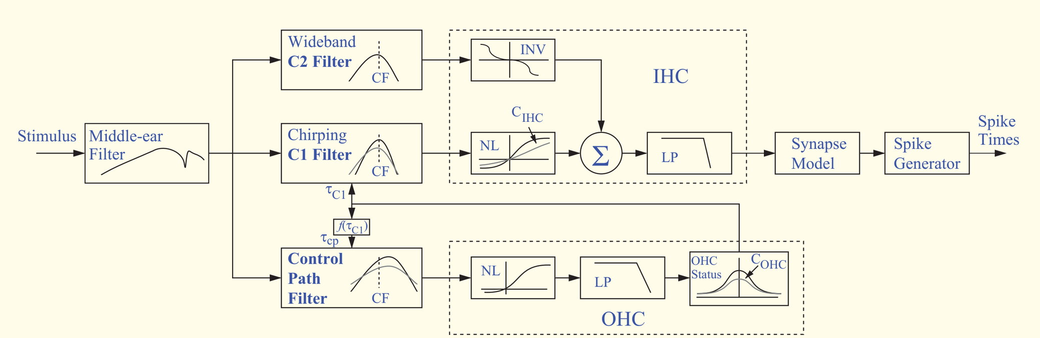

Figure 1: *Zilany and Bruce cat auditory nerve model [2, 3].*

B. Stimuli

- Speech recordings were taken from the TIMIT database.
- For consistency and good sound pressure level coverage, the speech signals were normalized to 45, 65, or 85 dB SPL before being presented to the model.
- For the investigations of the independent effect of OHC and IHC impairment, the synthesized vowel /ε/ with the spectrum shown in Fig. 2 was utilized.

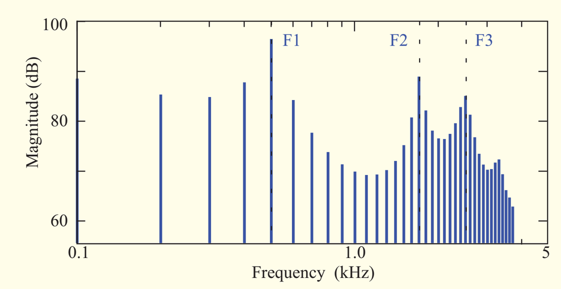

Figure 2: *Spectrum of the synthesized vowel /ε/.*

C. Analysis of Neural Responses

- The neural representation of speech in the auditory nerve is visualized by a "neurogram". A neurogram is similar to the spectrogram (Fig. 3B), except that it displays the neural response as a function of CF and time.
- The neurogram can exclude the spike timing information by computing the moving average with a window of several milliseconds to give only the mean discharge rate as a function of time (Fig. 3C), or the neurogram can include the spike-timing information of the neural responses by maintaining a small time bin size (Fig. 3D).
- In this study, 30 CFs spaced logarithmically between 250 and 8000 Hz were chosen. The neural response at each CF is composed of 50 AN fiber responses, with 60% of fibers being high spontaneous rate fibers, 20% being medium, and 20% being low.

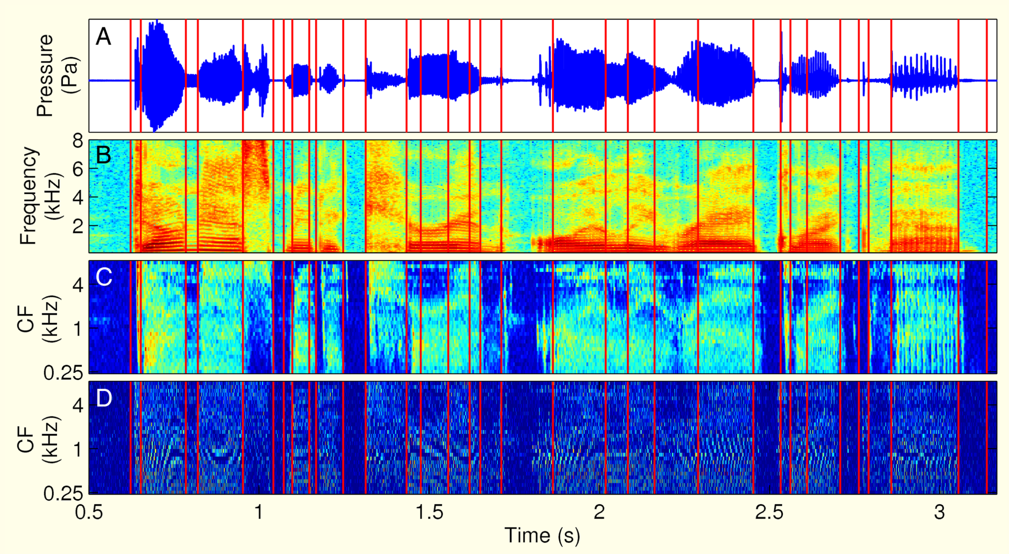

Figure 3: *An example sentence from the TIMIT database and the corresponding spectrogram and neurograms. (A) Time-domain pressure waveform; (B) Spectrogram; (C) Neurogram based on the mean discharge rate; (D) Neurogram based on the spiking-timing information. Phoneme boundaries are indicated by the vertical red lines. Reprinted from [6, 7].*

- Vowel responses were also analyzed by taking Fourier transforms of the spike trains to determine i) the phase and ii) the strength of synchrony to each of the vowel formants, referred to as the power ratio (PR) [3, 8].

D. Model Audiograms and Hearing Aid Schemes

- The two model audiograms tested are shown in Fig. 4, along with the corresponding linear prescriptions.
- For the case of mixed OHC/IHC impairment, 2/3 of the threshold shift at each CF was caused by OHC impairment and 1/3 by IHC impairment [8, 9].
- Independent OHC and IHC impairment was also investigated. Only the mild hearing loss case (left panel of Fig. 4) could be achieved by OHC impairment alone; the moderate-severe audiogram (right panel) required some level of IHC impairment. In contrast, both audiograms could be achieved by IHC impairment alone.

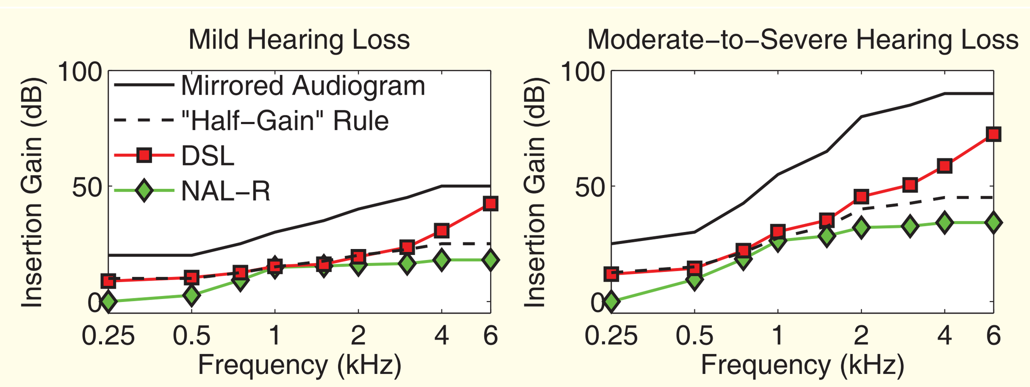

Figure 4: *The two example hearing loss profiles and corresponding insertion gains used in this poster. Left panel: a mild high-frequency hearing loss; Right panel: a moderate-to-severe high-frequency hearing loss. Adapted from [6, 7].*

E. Gain Optimization Strategy

- Optimal single-band gain adjustments around the hearing aid prescription gains were obtained though the gain adjustment strategy shown in Fig. 5 below.
- The gain adjustment strategy compares neural responses to speech sentences on a phoneme-by-phoneme basis for the impaired and normal models.

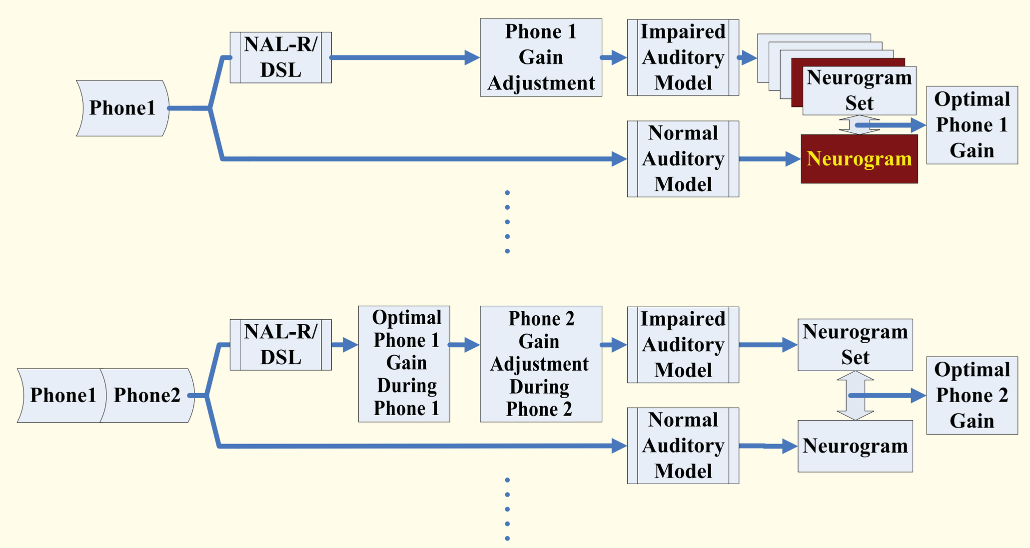

Figure 5: *Flow diagram of gain adjustment strategy. Adapted from from [6, 7].*

- The strategy begins by passing the first phone through the normal model to derive the normal neurogram. In the impaired pathway, the phone is passed though either the NAL-R or DSL amplification prescription before a single-band gain adjustment is applied. Gain adjustments range from -40 to $+40$ dB in 5-dB increments resulting in 17 uniquely amplified phones. The phones are passed through the impaired model, producing a set of 17 neurograms. The gain adjustment that minimizes the mean absolute error between the normal and impaired neurograms is deemed the optimal gain adjustment for that phone.
- For each amplification prescription, optimal gain adjustments were found by comparing either the neurograms with spike timing information or the average discharge rate neurograms.
- The second and all subsequent phones are analyzed in the same manner as the first, however, due to adaptation in the auditory-periphery model, all prior phones are prepended. The range of gain adjustments is applied only to the current phone and all previous phones are amplified with their optimal gain adjustments.

III. RESULTS

A. Mixed OHC/IHC Impairment

- The optimization results for the TIMIT sentences are shown in Fig. 6.
- The neurogram error behavior for the vowel /ε/ is shown in the left panels of Figs. 7 and 8 for the mild and moderate-severe audiograms, respectively.

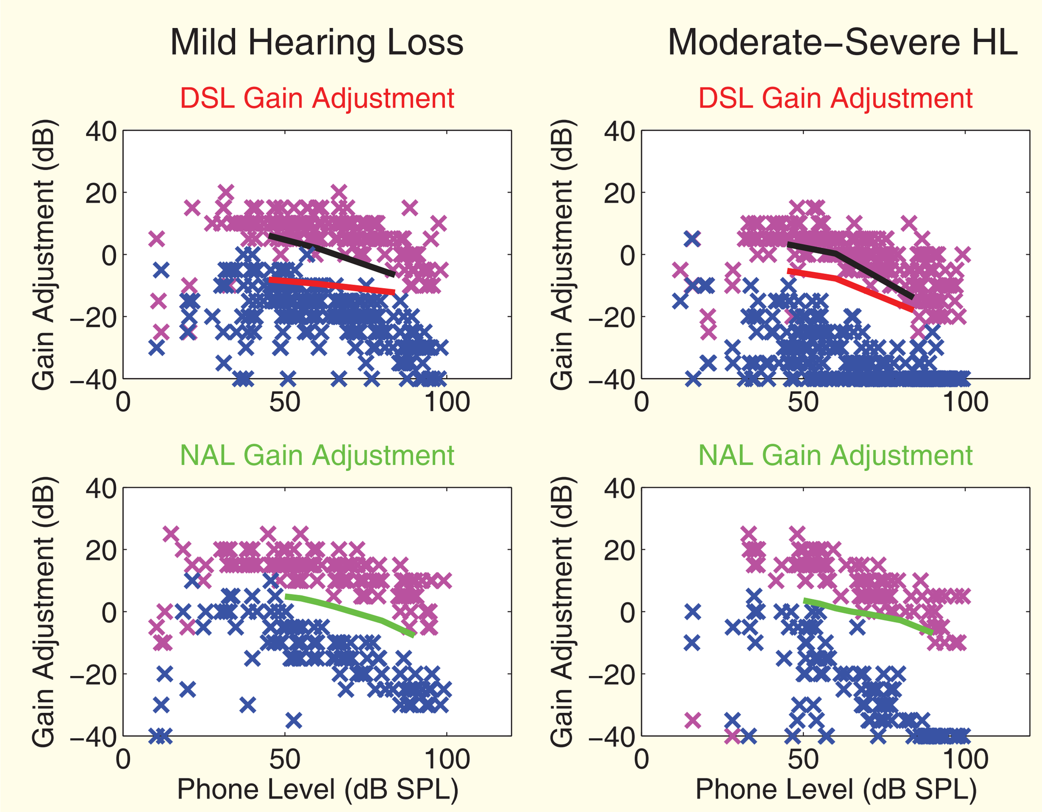

Figure 6: *Optimal gain adjustments versus phoneme input sound pressure for DSL (top panels) and NAL-RP (bottom panels) for the cases of mild (left column) and moderate-severe (right column) hearing loss [6, 7]. Magenta symbols indicate the optimal adjustment of an individual phoneme based on the mean-rate neurogram, while the blue symbols are for the spike-timing neurogram representation. The solid* **black** *and red lines indicate the DSL m[i/o] [10]* **paediatric** *and adult single-band compression prescriptions, respectively, while the solid green lines indicate the NAL-NL1 [11] single-band compression prescriptions.*

- Errors in response phase are more dominant for mild loss, while errors in synchronization frequency dominate for more severe losses. The effects of impairment and amplification are illustrated for these two cases in the right panels of Figs. 7 and 8.

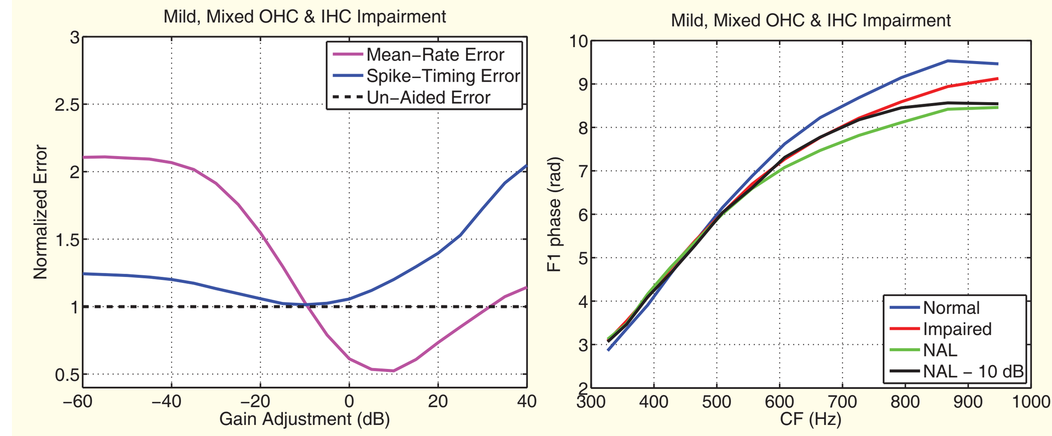

Figure 7: *Left panel: Normalized neurogram error versus gain adjustment for mild, mixed OHC/IHC impairment and NAL prescription in response to the synthesized vowel /ε/. Right panel: Effects of mild mixed OHC/IHC impairment and amplification on phase of synchronization to first formant of the vowel /ε/ versus fiber CF.*

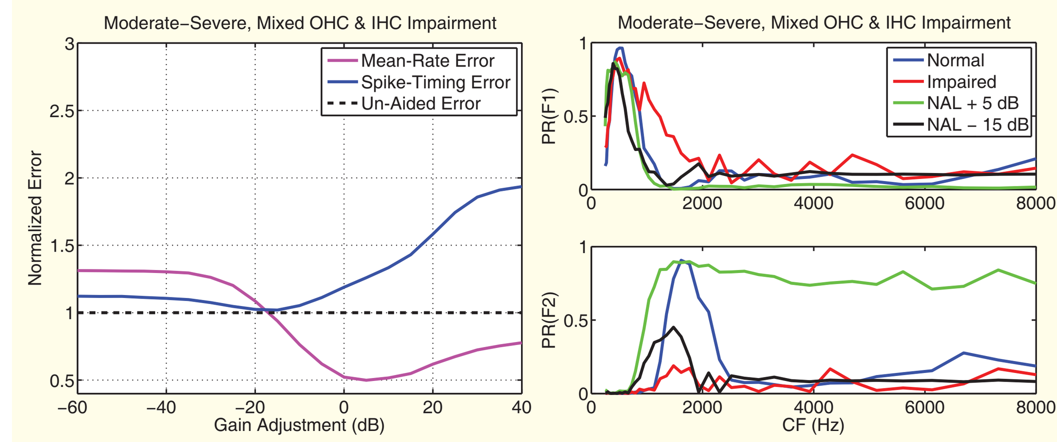

Figure 8: *Left panel: Normalized neurogram error versus gain adjustment for moderate-severe, mixed OHC/IHC impairment and NAL prescription in response to the synthesized vowel /ε/. Right panels: Effects of moderate-severe mixed OHC/IHC impairment and amplification on power of synchronization to first (top) and second (bottom) formants of the vowel /ε/ versus fiber CF.*

B. Effects of OHC Impairment Alone

- For mild OHC impairment alone, NAL is optimal for restoring the mean-rate response to the vowel, and the spread and phase of synchrony to the vowel formants are only slightly disrupted by the amplification.

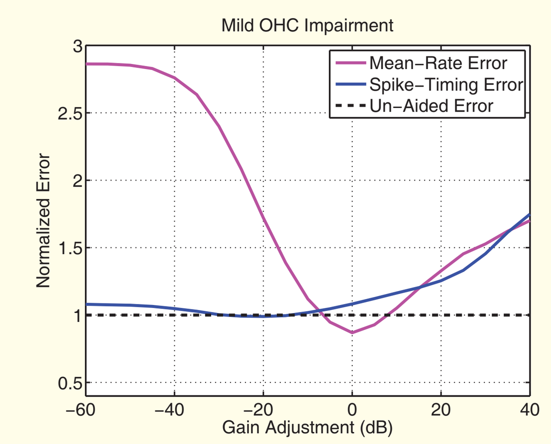

Figure 9: *Normalized neurogram error versus gain adjustment for mild OHC impairment alone and NAL prescription in response to the synthesized vowel /ε/.*

C. Effects of IHC Impairment Alone

- For mild IHC impairment alone, large errors in the phase of synchrony are produced if the gain is optimized to restore the mean-rate response.

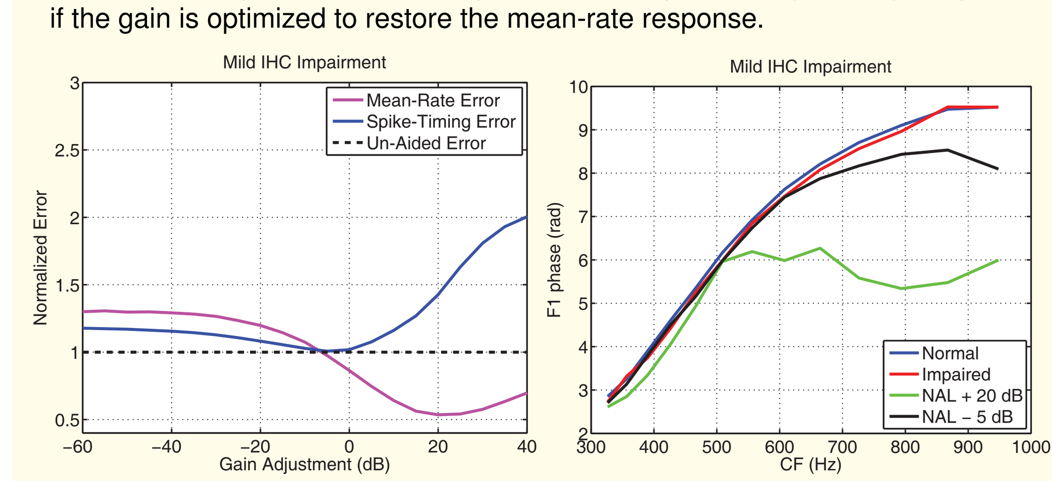

Figure 10: *Left panel: Normalized neurogram error versus gain adjustment for mild IHC impairment alone and NAL prescription in response to the synthesized vowel /ε/. Right panel: Effects of mild IHC impairment and amplification on phase of synchronization to first formant of the vowel /ε/ versus fiber CF.*

- For moderate-severe IHC impairment alone, substantial spread of synchrony is produced if the gain is optimized to restore the mean-rate response.

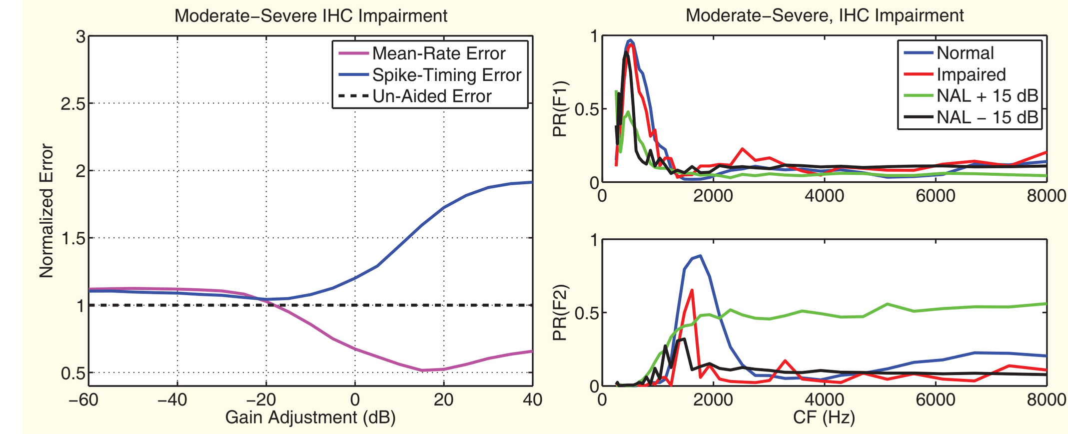

Figure 11: *Left panel: Normalized neurogram error versus gain adjustment for moderate-severe IHC impairment alone and NAL prescription in response to the synthesized vowel /ε/. Right panels: Effects of moderate-severe IHC impairment alone and amplification on power of synchronization to first (top) and second (bottom) formants of the vowel /ε/ versus fiber CF.*

IV. CONCLUSIONS

- Both linear and nonlinear amplification prescriptions find a balance between restoring the spike-timing and mean-rate information in AN responses.
- Gains for DSL pediatric prescriptions are weighted more towards mean-rate restoration than the adult prescriptions.
- Ideal single-band phonemic WDRC provides optimal gain adjustments for the AN on average.
- Errors in response phase are more dominant for mild loss, while errors in synchronization frequency dominate for more severe losses.
- Any IHC impairment is a big problem for linear amplification and WDRC, because IHC impairment: i) widens the gap between the optimal gains for restoring the mean-rate and spiking-timing neural representations and ii) exacerbates the errors in the spike-timing information at the gain that optimally restores the mean-rate representation.
- Optimal gain adjustment can prevent spike-timing errors from getting worse but cannot undo the effects of impairment.

V. ACKNOWLEDGMENTS

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