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Using auditory steady-state responses to evaluate auditory nerve integrity in normal-hearing and mild hearing-impaired listeners

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Introduction

Hearing impairment (HI) has traditionally been defined according to an increase in pure-tone threshold as measured by an audiogram. There is, however, emerging evidence that just evaluating threshold sensitivity does not fully characterize functional deficits in auditory processing. Recent animal studies have shown that noise over-exposure can cause loss of auditory nerve fibers (ANF) synapses — known as deafferentation (see Kujawa and Liberman (2015) for a review) — without causing hair cell loss. Furman et al., (2013) reported that deafferentation occurs predominantly to low-sensory-rate (low-SR) fibers, which have higher thresholds and therefore respond to higher acoustic intensities. Consequently, these ANF synapse losses do not alter detection of thresholds, but do degrade the encoding of supra-threshold sounds. The loss of ANF synapses might be a primary neural degeneration that precedes both hair cell and ANF cell body loss (Kujawa and Liberman, 2015).

ASSR are gross electroencephalography (EEG) potentials that follow the envelope of periodic acoustic stimuli.

• ASSR magnitudes grow monotonically and compressively (slopes lower than 1) with increasing stimulation level (Encina-Llamas et al., in press).

• It is suggested in the present study that ASSR growth-level functions may be a potential tool to evaluate ANF integrity.

Hypothesis

• ASSR growth-level functions with shallow modulation (m=25%) have similar slopes as ASSR functions with high modulation (m=85%) but with lower magnitudes.

• For preferential low-SR deafferentation: ASSR magnitudes for shallow modulation depths are reduced for higher stimulation levels, diverging from typically non-deafferentated level-curves.

Methods

• Subjects: 9 young NH listeners (3 females), 4 mild HI listeners (1 female) impaired at CF0-4 kHz.

• Equipment: Biosemi ActiveTwo EEG system. Sound presented via ER-3 earphones mounted on an ER-10B+ OAE probe.

Results

A mixed model post-hoc analysis revealed that:

• ASSR magnitudes are statistically different for each modulation depth, both in the whole dataset and within each of the three arbitrary subject groups.

• Slopes estimates for modulation high and shallow modulations in the mild-HI group are statistically different, but not in the other groups.

Discussion

• For shallow modulation and levels above 60 dB SPL, it appears that ASSR magnitudes are reduced for the mild-HI group when compared with the NH group. For the NH group, ASSR growth-function levels seem to reduce initially but fully recover at very high stimulus levels, i.e. non-monotonic.

• The effects of off-frequency contributions to the ASSR at high stimulus intensities and the test-retest variability intrinsic to physiological signals need to be investigated. It is inappropriate to speculate on the potential effect size of deafferentation until more data is available, so it is vital to minimize other factors that could confound the interpretation of ASSR growth-level curves.

• A phenomenological model can be used to disentangle the possible contributing factors to the ASSR growth-level functions. The relative loss of the different groups of SR fibers, the degree of deafferentation, and the off-frequency contributions at a cochlear level will be investigated.

Future work

• ASSR growth-level functions obtained at shallow modulation depths show different slopes in non-impaired frequencies in NH and mild-HI listeners.

Conclusion

• ASSR growth-function levels show a potential tool to evaluate the integrity of the peripheral hearing system at supra-threshold levels.

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Bharadwaj et al. (2014). Cochlear neuropathy and the coding of suprathreshold sounds. Front Sleep Med. 9:26

