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ABSTRACTS

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velope-following responses (EFRs), the sensitivity of these subcortical EEG metrics in diagnosing synaptopathy in humans is unclear. Confounding factors include head size, gender and other forms of peripheral hearing loss (e.g., outer-hair-cell (OHC) loss) that may co-exist and affect subcortical EEG amplitudes as well. Although OHC loss can be quantified using otoacoustic emissions, it is worthwhile focusing on a single EEG metric that can quantify both aspects of peripheral hearing loss. EEG measurement accuracy can be improved by designing stimulation paradigms on the basis of model simulations that can simulate and disentangle how different forms of peripheral hearing loss impact EEG metrics.

In this work, we optimize subcortical EEG stimulation paradigms for synaptopathy using a computational model of the auditory periphery that simulates the broadband and level-dependent features of human ABR and EFRs. First, we focused on rendering EFR methods more frequency-specific by masking off-CF, and enhancing on-CF, contributions to the EFR. The optimized stimulation paradigms allow for an interpretation of EFR strength in terms of localized AN synapse differences. Secondly, we simulated how OHC loss or dysfunction impacts the sensitivity of ABR and EFR metrics of synaptopathy and show that: (i) a combination of the slope between the EFR fundamental and 1st harmonic together with the ABR/EFR amplitude ratio, or (ii) the ABR growth ratio that takes into account both the amplitude and latency growth of ABRs, can map listeners onto a OHC loss vs synaptopathy degree scale. We compared the simulations to recordings from listeners with normal and sloping high-frequency audiograms to evaluate the practical use of the proposed relative subcortical EEG metrics.

SYMP 06

Noise Exposure, Cochlear Synaptopathy and Listening Difficulties: EEG and Behavioural Measures

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We investigated the relationship between noise exposure, cochlear synaptopathy, and listening difficulties in 122 adults aged 29 to 57 with normal or near-normal hearing. Participants undertook a large test battery that included audiometric testing, tasks that assessed attention and memory, speech-in noise tasks, and other measures of auditory perception. A subset of participants (n=74) completed additional electrophysiological testing which included responses from the auditory brainstem and cortex. When auditory brainstem responses (ABR) were plotted against lifetime noise exposure, there was considerable variability across the sample. Nevertheless, we observed a significant (but weak) association between noise exposure and wave I ABR amplitudes to clicks presented at 75 dB nHL (-0.038 µV/log10Pa2h, p = 0.026). The significant trend revealed that those with more noise exposure produced smaller wave I ABR amplitudes than those with less noise exposure, although the result appeared to be driven by a small number of subjects whose data were consistent with the hypothesised pattern of results. Analysis of cortical auditory evoked potentials (in response to /da/ presented at (a) +10 dB, (b) +5dB and (c) 0dB SNR; and 'iterated ripple noise' obtained at (a) SNR=+60dB; (b) SNR=+60dB; (c) SNR=+15dB; (d) SNR=+5dB) revealed no association between noise exposure and any of the cortical responses. This suggests that any effects of noise exposure either dissipate at higher levels of the auditory pathway or are unable to be observed using our chosen measures. In the larger cohort, we found no relationship between noise exposure and performance on speech-in-noise or other auditory tasks. However, regression analysis showed that attention, working memory and extended high frequency hearing thresholds were significant factors that affected listening in noise. When taken together, our results suggest that listening difficulties arise from a constellation of factors, and although noise-related cochlear synaptopathy may be one of these, we need better methods for measuring it if we are to fully understand its role relative to other cognitive and auditory factors.

SYMP 07

Reduced ABR wave I amplitude in Veterans and firearm users suggests synaptopathy

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ABR wave I amplitude reductions are associated with animal models of cochlear synaptopathy. As a proxy for synaptopathy in humans, wave I amplitude was measured in young people with normal pure tone thresholds. Veterans with high levels of military noise exposure and non-Veterans who reported using firearms demonstrated smaller wave I amplitudes at suprathreshold levels than participants with less noise exposure. Tinnitus was associated with the Veteran high exposure group and with reduced ABR wave I amplitude. These data suggest that synaptopathy may occur in response to noise exposure in humans and be associated with tinnitus.