Lifetime noise exposure affects human auditory brainstem responses

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Motivation

The effects of noise exposure on the auditory system are currently well characterized in **animal studies**: (a) the synaptic connections of the auditory nerve fibers, particularly those with low spontaneous rate (LSR), are the most vulnerable elements; (b) synaptic disconnections accelerate the loss of spiral ganglion cells; (c) hair cells remain intact and audiometric pure tone thresholds unaltered.

In **humans**, these findings have motivated investigation of a specific type of auditory neuropathy believed to contribute to communication deficits in people with a normal audiometry, presently known as 'hidden hearing loss' (HHL). However, the results reported to date in human studies are inconclusive. The **objective** of this study was to evaluate the effects of lifetime noise exposure on the auditory brainstem response.

Methods

68 **subjects** (aged 29-55, mean = 44.29 years, SD = 6.39 years, 33 females) participated in the study. **Lifetime noise exposure** was calculated (in $\log_{10}(Pa^2h)$) through an online questionnaire. **Auditory brainstem responses (ABRs)** were elicited with 12,500 rarefaction clicks presented on the right ear at 39.1 Hz at 108.5 dB peak-to-peak equivalent sound pressure level, with an electrode setup Fz-TIPtrode (wave I) and Fz-M2 (wave V and interpeak values). **Statistical analysis** consisted of a linear regression model for each evaluated parameter, considering age, gender, lifetime noise exposure, and averaged audiometric threshold in the high frequencies (3-6 kHz) as predictor variables.

Results & Discussion

Figures A and B show the raw data, adjusted values, and adjusted values excluding outliers (values outside 2 standard deviations from the mean) of the amplitudes and latencies of waves I and V, as well as the interpeak latency and ratio of amplitudes. These figures show that, despite the large inter-subject variability, **there is a statistically significant effect of lifetime noise exposure on the amplitudes of both waves (full dataset), and on the latency of wave I (reduced dataset)**. These results are consistent with the HHL theory. Overall, the loss of fibers would result in lower amplitudes, and since LSR fibers present a longer latency, the selective loss of these fibers would lead to shorter wave I latencies. Figure C shows the ABR grand average (Fz-TIPtrode) from the five most (in purple) and least (in green) noise-exposed subjects.

Figure D shows the waves I and V amplitude distributions for the group of subjects with larger (orange) and smaller (blue) wave I amplitudes. This figure shows that **the wave V amplitudes from the subjects with low-amplitude wave I do not differ from the subjects with normal wave I**. These results are consistent with the Central Gain Model, in which a neural amplification occurs to compensate for a reduced input from the cochlea. Figure E shows the grand-average ABR of the two groups.

Conclusions

- ✓ Lifetime noise exposure affects the morphology of human ABRs.
- ✓ Our data show evidence of central gain mechanisms.
- ✓ Large inter-subject variability is a challenge for the diagnosis of HHL.