Beyond the audiogram: Characterizing auditory neurodegeneration in humans

Lay Summary:

It is common knowledge that loud noise can damage hearing. If the noise is loud enough, or the noise exposure long enough, a permanent hearing loss can result which renders soft sounds inaudible. This hearing loss can be shown using a clinical chart called an *audiogram* which graphs the intensity of just-detectable tones as a function of their frequency ('pitch'). When hearing loss is present, these tones have to be more intense than normal just to be heard.

Loud sounds can also lead to a temporary dullness of hearing which recovers over time. If the ability to hear soft sounds eventually recovers (that is, if the audiogram returns to normal), it used to be thought that no permanent damage to the hearing system had actually occurred. Recent scientific studies, however, have shown that permanent damage to the inner ear and hearing nerve can still happen even when the hearing loss appears to be only temporary. This means that even though the audiogram returns to normal after loud sounds, the hearing nerve remains permanently damaged. Because this damage is not apparent from the audiogram, it has become known as 'hidden hearing loss.'

Although the concept of 'hidden hearing loss' associated with a damaged hearing nerve is reasonable, the evidence for it in humans is actually lacking. The purpose of this study was to address this omission by trying to identify signs of 'hidden hearing loss' in humans whose noise exposure history made them likely candidates for this condition. Specifically, the study looked at young adults who frequently attended very loud music events but whose audiograms remained clinically 'normal'. As a control, young adults who avoided loud music events were also recruited. A wide variety of hearing tests were administered to gauge hearing abilities that likely would be affected by a damaged hearing nerve. The sounds in these tests were all very audible, and some included additional challenges like listening in background noise. Behavioral tests included speech perception, the ability to follow dynamic changes in sound, and the ability to perceive small timing differences in sound between the two ears. Objective tests included distortion product otoacoustic emissions (DPOAEs), the auditory brainstem response (ABR), and other sound-evoked brain wave measures.

Despite the extensive variety of tests, the results showed only one pattern that was compatible with an expectation of 'hidden hearing loss', namely a modestly abnormal ABR finding in the presence of normal DPOAEs and a normal audiogram. None of the other tests showed any specific effects of loud music exposure. This general absence of effects suggests that, even if 'hidden hearing loss' associated with a damaged hearing nerve is a valid condition in humans, its consequences for hearing are either too diffuse or too subtle to permit a simple diagnosis.

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Project Title: Beyond the audiogram: Characterizing auditory neurodegeneration in humans

Primary Project Goal: The purpose of this study was to test the hypothesis that listeners with frequent exposure to loud music exhibit deficits in supra-threshold auditory performance consistent with cochlear synaptopathy ('hidden hearing loss').

Knowledge Translation: Animal studies have indicated that one cause of 'hidden hearing loss' is cochlear synaptopathy – a form of noise-induced injury that leads to permanent damage to the auditory nerve. There is wide-spread interest in determining whether this form of 'hidden hearing loss' exists in humans and, if so, how it can best be diagnosed and clinically addressed. The general motivation of this project, therefore, was to probe for its existence in humans in a systematic study.

Results: The results demonstrated that a history of loud music exposure can lead to a profile of peripheral auditory function that is consistent with an interpretation of cochlear synaptopathy in humans, viz. modestly abnormal auditory brainstem response (ABR) Wave I/Wave V ratios in the presence of normal DPOAEs and normal audiometric thresholds. However, there were no other electrophysiological, psychophysical, or speech perception effects.

Methods: Young adults with normal audiograms were recruited who either did (n=31) or did not (n=30) have a history of frequent attendance at loud music venues where the typical sound levels could be expected to result in temporary threshold shifts. A test battery was administered that comprised three sets of procedures: (1) electrophysiological tests including distortion product otoacoustic emissions, auditory brainstem responses, envelope following responses, and the acoustic change complex evoked by an interaural phase inversion; (2) psychoacoustic tests including temporal modulation detection, spectral modulation detection, and sensitivity to interaural phase; (3) speech tests including filtered phoneme recognition and speech-in-noise recognition.

Background information about your research group: This project was undertaken in the Psychophysics & Electrophysiology Auditory Research Laboratory (PEARL) directed by Dr. John H. Grose. This laboratory – along with its sister laboratory SPARC (Speech Perception & Auditory Research @ Carolina) directed by Dr. Emily Buss – investigates human hearing across the lifespan. Studies include both normal-hearing listeners and listeners with hearing loss. Effects of age on hearing are examined using parallel evoked potential and psychoacoustic paradigms, with a particular focus on temporal processing. The development of auditory function – including speech and binaural hearing – is studied in school-aged children using 'child-friendly' psychophysical tasks. Another line of research investigates speech perception in monolingual and bilingual speakers of English and Spanish.

References: The results of this study are scheduled to be published in the journal Trends in Hearing under the title '*Loud music exposure and cochlear synaptopathy in young adults: Isolated ABR effects but no perceptual consequences*'.