645 Something Old and Something New in NIHL

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With the wide-ranging response and exquisite sensitivity of our hearing organ comes an unfortunate sensitivity to loud sound. This may not have been an evolutionary oversight since aside from an occasional thunderclap, nature does not inundate us with high levels of noise. Noise-induced hearing loss seems to be a modern affliction in the wake of gunpowder and the Industrial Revolution. Today 12-15% of all employed people in developed countries are exposed to potentially damaging noise levels of 85 dB or more, expanding on a tradition that in the past had been limited primarily to blacksmiths, millers and artillerymen.

Sir Francis Bacon (1561-1626) was one of the first to associate excessive sound with deafness but systematic investigations of noise-induced hearing loss did not begin in earnest until early in the 20th century. Today we understand well the audiological, pathophysiological and micro-anatomical effects of noise trauma, and we are beginning to explore the underlying cellular mechanisms. Furthermore, recent studies in experimental animals give hope that therapeutic prevention of noise-induced hearing loss is within reach.

646 The Relationship Between Noise-Induced Hearing Loss and Hair Cell Loss in Rats

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The relationship between noise-induced hearing loss (NIHL) and hair cell loss is not consistent. Interestingly, hair cells may still survive after a complete loss of their auditory function. The present report compares hair cell loss at different cochlear locations with CAP (compound action potential) threshold elevation at related frequencies in rats. CAP threshold elevation and hair cell loss were determined 4 weeks after noise exposure. No hair cell loss was observed in the low-frequency region (<8 kHz) even when CAP threshold elevation exceeded 60 dB. In the middle turn, significant hair cell loss was not observed until NIHL exceeded about 40-50 dB. In the basal turn, while inner hair cell (IHC) loss was not observed until NIHL exceeded about 50 dB, outer hair cell (OHC) loss was observed in almost all of the noise-exposed rats, even in some cases without detectable NIHL. OHC-loss increased gradually with NIHL and in the region of the highest frequencies tested in this study (30-40 kHz), a linear NIHL/OHC-loss relationship was observed. The data indicate that the NIHL/hair cell loss relationship is cochlear location dependent and many hair cells survive under a severe NIHL.

647 Noise-Induced Hair-Cell Loss Versus Total Energy: Analysis of a Large Data Set

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The relation between total noise-exposure energy, recovery time, or rest & % hair-cell (HC) loss was examined in 378 chinchillas. The continuous exposures were either a 4-kHz octave band of noise (OBN) at 47-108 dB SPL (N=169) for 0.5 h to 36 d, or a 0.5-kHz OBN at 65-128 dB SPL for 3.5 h to 432 d (N=131). Recovery times varied from 0-365 d. With both OBNs, other animals were exposed on interrupted schedules [6 h/d, /2d or /wk for 9-365 d (N=78)]. HC loss as a function of age in 117 non-exposed animals was used to correct for loss due to aging. For the 4- & 0.5-kHz OBN, the noise-exposed cochleas (N=607) were separated into 3 groups: 1) Acute (≤ 9 d exposure, 0 d recovery; N=90 & 56) to characterize the primary effects of the noise; 2) Chronic (> 9 d exposure, 0-730 d recovery; N=184 & 144) to determine secondary effects & 3) Interrupted (N=46 & 87) to show the effect of rest during the exposure. Cluster & regression analyses were performed in the basal & apical halves of the cochlea to determine the specific rates (relative to doubling of total energy, recovery & rest) at which primary & secondary effects produced HC loss & rest prevented loss. The effect of recovery time was isolated by subtracting the primary effect determined in the Acute group from the loss in the Chronic group. The effect of rest was isolated by subtracting the primary effect from the Acute group & the secondary effect determined from the Chronic group from the loss in the Interrupted group. It was found that: 1) When the OBN was above a critical level, there was no relation between total energy or recovery time & HC loss; 2) Below a critical level, there were highly significant log-linear relations, but at a low rate; 3) Except for the highest exposure levels, the majority of HC loss from the 4-kHz OBN occurred after the exposure had terminated, while that from the 0.5-kHz OBN occurred during the exposure & 4) Rest periods during either OBN exposure significantly reduced HC loss.

648 Can Infrasound Protect the Cochlea from a Damaging Level of Noise?

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Infrasounds (ie, < 20 Hz for humans; < 100 Hz for chinchillas) are not audible, but they produce large movements of cochlear fluids (Salt & DeMott, 1999). It was speculated that infrasound might bias the basilar membrane & perhaps minimize noise-induced hearing loss (NIHL). Chinchillas were simultaneously exposed to a 30 Hz tone at 100 dB SPL & a 4-kHz OBN at either 108 dB SPL for 1.75 h or 86 dB SPL for 24 h. One tympanic membrane (TM) was perforated prior to exposure to attenuate infrasound transmission to that cochlea. Controls were exposed to infrasound or the 4-kHz OBN only. ABR threshold shifts (TS) & DPOAE level shifts (LS) were determined post-TM-puncture & immediately post-exposure, just before cochlear fixation. The cochleae were dehydrated, embedded in plastic, dissected as flat preparations & evaluated for hair-cell (HC) losses. For each animal, the magnitude & pattern of functional & HC losses were compared between their right & left cochleae. The infrasound exposure alone resulted in a 10-20 dB TS below 1 kHz, no LS & no HC damage/loss. Exposure to the 4-kHz OBN alone at 108 dB produced a 50-60 dB TS for 1-16 kHz, a 40-50 dB LS for 2-12 kHz & severe OHC loss in the middle of the first turn. When infrasound was added, the functional and HC losses extended much farther apically & basally than for the 4-kHz OBN alone. Exposure to the 4-kHz OBN alone at 86 dB produced a 40 dB TS for 3-12 kHz & 30 dB LS for 3-8 kHz, but no HC loss in the middle of the first turn. When infrasound was added, no differences in the functional and HC losses were found compared to the 4-kHz OBN alone. We hypothesize that exposure to infrasound & an intense 4-kHz OBN increases cochlear damage because the large fluid movements from infrasound cause more intermixing of cochlear fluids through the damaged reticular lamina. Simultaneous infrasound & a moderate 4-kHz OBN did not increase cochlear damage because the reticular lamina rarely breaks down during this exposure.

649 The Influence of Varying Degrees of Permanent Hearing Loss on a Polynomial Model of Mechano-Electric Transduction

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Excessive noise exposure damages cochlear structures and alters mechano-electric transduction (MET). Assessment of the pathophysiology associated with the disruption of cochlear MET can provide a better understanding of sensory hearing loss, and may invite new aural rehabilitative options. In our previous work, we characterized MET with a third-order polynomial equation and showed that physiologic indices derived from the equation are sensitive to different cochlear pathologies. Here we explore the influence of various degrees of permanent noise-induced hearing loss on the polynomial model of MET.

Mongolian gerbils (N=43) were exposed to an 8 kHz narrow band noise at 117 dB SPL. Exposure duration ranged from 1 to 128 hours.

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After attending the Scientific Meeting participants should be better able to:

1. To understand current concepts of function of normal and diseased ears and other head and neck structures.
2. To understand current controversies in research methods and findings that bear on this understanding.
3. To understand what are considered to be the key research questions and promising areas of research in otolaryngology.

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