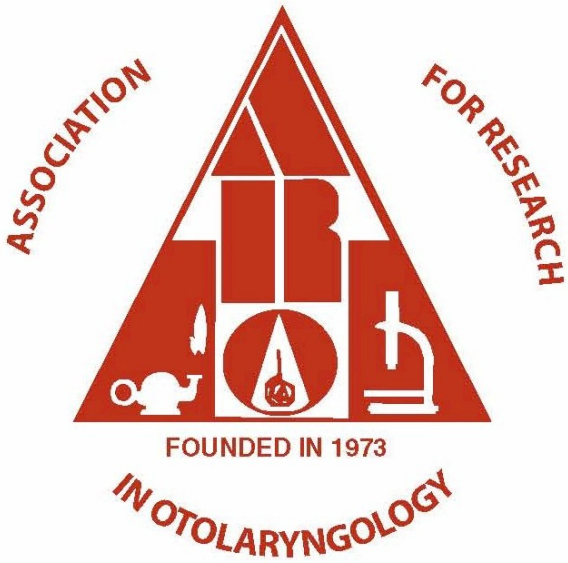
ABSTRACTS OF THE TWENTY-NINTH ANNUAL
MIDWINTER RESEARCH MEETING

ASSOCIATION FOR RESEARCH IN OTOLARYNGOLOGY



February 5-9, 2006

Baltimore Marriott Waterfront
Baltimore, Maryland, USA

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Editor

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was less than or equal to 10 days; 2) The exposure level was less than or equal to 108 dB SPL; & 3) Focal lesions were less than 1.5 mm in size. The data sets included a variety of exposures ranging from those that were high-level, short duration to those that were moderate-level, moderate duration. The % location of the center of each focal lesion was determined. Means, SDs & medians were calculated for lesion size for each OBN. Histograms were then constructed from the % location data using 2.0% bins & the counts were graphed relative to total number of lesions. For the 4-kHz OBN, 94% of the lesions were in the basal half of the OC & 6% were in the apical half. For the 0.5-kHz OBN, 29% of the lesions were in the apical half of the OC & 71% were in the basal half. The mean lesion size was 1.48% & 0.68% for the 4-kHz & 0.5-kHz OBN, respectively, with medians of 1.10% & 0.50%. The mean lesion size (in mm) for the 0.5-kHz OBN was less than half that for the 4-kHz OBN. For the 4-kHz OBN, a histogram of the % location of lesions showed that most occurred in the 5-7-kHz region, at & just above the upper edge of the OBN. Clusters of lesions were also found around 8 & 12 kHz. A cluster was present at & just below the lower edge of the OBN, as well as in the 1.5-kHz region. For the 0.5-kHz OBN, a histogram of the % location of lesions showed clusters at 0.25, 0.75 & 1.5 kHz in the apical half. In the basal half, the pattern was very similar (Pearson's r=0.69) to that seen with the 4-kHz OBN. The distribution of basal-turn lesions suggests that the 4-kHz & 0.5-kHz OBN are damaging that region of the cochlea in the same way.

33 Histopathological Changes in the Cochlea Following Exposure to Low-Frequency Noise

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Thirteen chinchillas were exposed for 24 h to a 0.5-kHz OBN at 95 dB SPL. The cochleae of 8 animals were fixed at 0-d post-exposure; 5 were fixed after 1-2 wks of recovery. To keep cellular debris from washing away, all cochleae were plastic-embedded before being dissected into flat preparations. By phase-contrast microscopy, hair-cell losses were determined from apex to base. Damage consisted of scattered loss of OHCs in the apical half of the organ of Corti (OC) & small focal lesions (i.e., > 50% hair-cell loss over at least 0.03 mm) in the basal half. These specific patterns of loss suggest that noise damaged the apex & base by different mechanisms. In order to estimate the timing of cell loss, differential counts of missing & severely injured cells were performed. The presence of immature phalangeal scars & necrotic, oncotic & apoptotic hair cells indicates a recent loss while mature phalangeal scars indicate a long-standing loss. In the apical half of the OC in both groups of animals, many of the phalangeal scars replacing the missing OHCs were immature. Cellular debris was seen in the OC fluid spaces beneath these scars. By TEM, the debris was found dispersed in the Nuel spaces & consisted of vesicles of various sizes, small granules, shrunken & swollen mitochondria &, rarely, fragments of plasma membrane. TEM also revealed the presence of cellular debris in the endolymphatic space near the reticular lamina. This latter finding indicates that the barrier function of the reticular lamina broke down temporarily when the hair cells degenerated, before phalangeal scars formed. Debris in the Nuel spaces was not surrounded by plasma membrane as would be the case if the hair cells had been apoptotic before they died & then formed apoptotic bodies. In the basal half of the OC, focal lesions were found in 2 of eight 0-day & 3 of five 1-2 wk animals. In the 0-day ears, oncotic OHCs were found at the edges of the basal-turn lesions. In the 1-2 wk ears, the basal-turn lesions primarily consisted of immature phalangeal scars. The appearance of debris in the apex & base suggests that many of the OHCs were oncotic rather than apoptotic before they disappeared.

34 Intense Noise Causes Damage to the OHC Lateral Wall Leading to Hearing Loss

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The cochlear active process results in a 40-60 dB cochlear amplification. Outer hair cells (OHCs) constitute an important part of cochlear micro-mechanics and are believed to be the driving force of the cochlear active process by way of their electromotility. The OHC membrane skeleton, consisting of F-actin and spectrin, maintains the unique OHC cylindrical shape and provides stiffness to the cell. In addition to its well known basic functions, the OHC plasma membrane is a main contributor to OHC axial stiffness. Prestin, a membrane protein, has been recently recognized as the OHC motor protein. Changes in the OHC lateral wall may affect OHC electromotility and/or cochlear micromechanics and, subsequently cochlear sensitivity. In this report OHC membrane fluidity (by laser bleach approach), gene expression of beta-actin, beta-spectrin, and prestin were determined after noise exposure. The noise exposure caused a reduction of OHC membrane fluidity and a time-dependent gene expression of the proteins in the OHC lateral wall. The noise-induced changes were associated with permanent threshold shifts. The motor protein appeared to be the most sensitive to noise trauma among the three proteins. The data suggest that non-lethal injuries in the OHC lateral wall may cause loss of the OHC electromotility or the cochlear micromechanics leading to a reduction of cochlear amplification and then cochlear sensitivity.

35 Mechanisms of Oxidative Stress in the Potentiation of Noise-Induced Hearing Loss by Acrylonitrile

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Acrylonitrile (ACN), one of the top 50 chemicals produced in the world, is a very powerful pro-oxidant compound whose metabolism leads to a profound glutathione (GSH) depletion and to a production of cyanide (CN) which, in turn, can inhibit superoxide dismutase (SOD). ACN, by itself, is not ototoxic, but we have shown that it can strongly promote noise-induced hearing loss (NIHL), even at noise levels that do not produce auditory impairment. The mechanism by which ACN renders the cochlea more