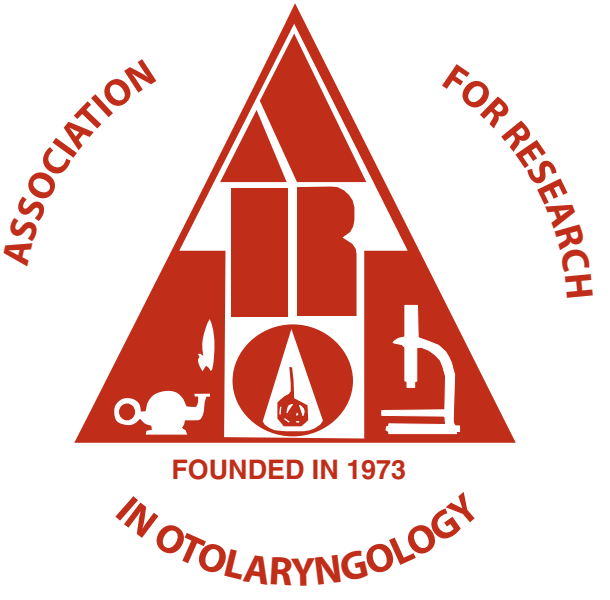
ABSTRACTS OF THE TWENTY-EIGHTH ANNUAL
MIDWINTER RESEARCH MEETING

ASSOCIATION FOR RESEARCH IN OTOLARYNGOLOGY



February 19-24, 2005

The Fairmont New Orleans
New Orleans, Louisiana

ABSTRACTS OF THE TWENTY-EIGHTH ANNUAL MIDWINTER RESEARCH MEETING OF THE

Association for Research in Otolaryngology

February 19 – 24, 2005

New Orleans, Louisiana, USA

Peter A. Santi, Ph.D.
Editor

Association for Research in Otolaryngology
19 Mantua Road, Mt. Royal, NJ 08061 USA

574 Correlation of Cochlear Pathology with ABR Threshold Shift and DPOAE Level Shift Following Exposure to Low-Frequency Noise

Devon Snow[1], Gary W Harding[1], **Barbara A Bohne[1]**

[1]Washington University School of Medicine

ABR thresholds & DPOAE levels were determined before & 1-3 times after a 24-h exposure to a 0.5-kHz OBN at 95 dB SPL. At termination, the EP was recorded in the 1st turn & carbon tracer injected into the endolymphatic space (ES). After 45 min, the cochlea was fixed with OsO_4, plastic-embedded & dissected as a flat preparation. Quantitative data on hair-cell losses were collected. Cytocochleograms were prepared with ABR threshold shifts (TS) & DPOAE level shifts (LS) overlaid. Based on the functional results, organ-of-Corti (OC) segments in the 2nd & 1st turns were sectioned at a radial angle for light & TEM study. Four chinchillas were terminated by 3 h post-exposure (0-d); two were terminated at 1 wk. At 0 d, the animals had a TTS of 20-60 dB over 0.5-10 kHz & a TLS of 10-50 dB over 2-10 kHz. EP averaged 84 mV, similar to controls. OHC loss ranged from 2.2-10.5% & 0.8-2.8% in the apical & basal halves of the OC, respectively. IHC loss was negligible throughout the OC. Viewed by phase contrast microscopy, most missing OHCs were found to have been replaced by immature phalangeal scars & cellular debris was seen in the ES & Nuel spaces. Most remaining hair cells had normal shapes & stereocilia arrays. Hair-cell loss & damage were insufficient to account for the TTS & TLS. Over the extent of the TTS & TLS, the nerve fibers & endings below the IHCs were not damaged, while the outer pillar bodies were either non-parallel or buckled. By 1 wk, the TS & LS had improved considerably; EP averaged 77 mV. The pillars were buckled over a smaller linear distance in the OC that was aligned with the frequency range of the residual TS & LS. Immature scars were still visible in the reticular lamina but the amount of cellular debris was decreased. We conclude that in the chinchilla, the most consistent pathological correlates of noise-induced TTS & TLS are an accumulation of cellular debris in cochlear fluids & injury to supporting cells that uncouples the stereocilia from the tectorial membrane.

575 Noise-induced Loss of Spiral Ligament Fibrocytes in Mice

Joe Adams[1]

[1]Mass. Eye & Ear Infirmary

The locations and specializations of type I and type II fibrocytes have strongly implicated them in K+ ion recycling from perilymph to the stria vascularis. In contrast, the localizations and specializations of type III and type IV fibrocytes have thus far provided few clues regarding their functional roles. Based upon their finding that type IV fibrocyte loss precedes sensory cell loss in C57BL/6 mice Hequembourg and Liberman suggested that loss of these fibrocytes may result in other cochlear cells being more vulnerable. Noise exposure of CBA/CaJ mice induces degeneration of type IV fibrocytes in ways that are very different from hair cell degeneration patterns. For example, following 92 dB 8-16 kHz noise exposures type IV fibrocytes in the upper basal turn degenerate. This exposure level is 18 dB less intense than that required to produce loss of hair cells at the place of maximal basilar membrane motion. Furthermore, the site of type IV cell loss following 4-8 kHz noise exposures is the same as that produced by higher frequency noise exposures. Noise-induced loss of fibrocytes at this location may be associated with damage to sensory cells located in the lower basal turn. Degeneration of type IV cells can occur rapidly and the region of cell loss expands to include type III fibrocytes when high level noise exposures are utilized. If loss of type III and type IV fibrocytes creates vulnerabilities in sensory cells as Hequembourg and Liberman suggested, and if noise-induced losses of type III and type IV cells are associated with damage to basal turn sensory cells, then vulnerabilities associated with type III and type IV losses should be detectable by assaying the status of sensory cells in the extreme basal turn.

Supported by grants DC03929 and P30 DC 005209.

576 The Spiral Ligament Participates in Cochlear Inflammation Following Acoustic Trauma

Kunihiro Sato[1], Peter Billings[1], Elizabeth M. Keithley[1,2]

[1]University of California, San Diego, [2]VA Medical Center, San Diego

Hearing loss following acoustic trauma results from cochlear damage. This damage is associated with an inflammatory-like response. We investigated the morphology and location of cells expressing CD45 (leukocyte common antigen) and F4/80 (a marker of activated macrophages), as well as ICAM-1 (intercellular adhesion molecule) expression following acoustic trauma. Swiss-mice were exposed to an octave-band noise (8-16 kHz) at 118 dB for 2 hours and sacrificed 0.5, 1, 2, 4, 7, and 14 days later. All animals had severe hearing loss, 90 dB or more, at every survival time. Non-exposed cochleae showed only a few immunopositive cells with anti-CD45 or anti-F4/80 antibody and constitutive expression of ICAM-1 by venules and fibrocytes in the lower part of the spiral ligament. Following acoustic trauma, the number of CD45(+) cells was increased dramatically, reaching its maximum at 2 days, decreasing slightly at 4 and 7 days. By 14 days most CD45(+) cell were gone. CD45(+) cells were most predominant within the spiral ligament, but detected also in the stria vascularis, scala tympani, and scala vestibuli in lower numbers. Most CD45(+) cells also expressed F4/80, but some F4/80(+) cells did not express CD45. Morphologically, CD45 and/or F4/80 immunopositive cells comprised two types of cells: round/oval cells throughout the cochlea, which are likely to be recruited macrophages, and irregularly-shaped cells with a few processes in the spiral ligament, which are likely to be activated microglia. Following acoustic trauma ICAM-1 expression was detected in an increased number of spiral ligament fibrocytes. These results imply that both leukocyte recruitment and activation of resident cells within the spiral ligament are important in cochlear responses to acoustic trauma.