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An animal model of hearing loss in superior-canal dehiscence syndrome

J. Songer, M. Wood, J. Rosowski, Boston; USA

Purpose: Superior semicircular canal dehiscences (SCD) have been identified as a non-middle-ear cause of conductive hearing loss (e.g. Mikulec et al. Otol Neurotol, 2004; 25:121-129). The purpose of this study is to understand the mechanisms through which SCDs induce changes in auditory sensitivity to both air-conducted (AC) and bone-conducted (BC) stimuli.

Materials & Methods: We surgically introduced holes (dehiscences) into the superior semicircular canal (SC) of 32 chinchillas while monitoring cochlear potential (CP) in response to BC stimuli, and also monitoring CP, middle-ear input admittance (Yme), and stapes velocity (Vs) in response to AC stimuli. Also, an anatomically-realistic, lumped-element, mathematical model used to predict the effect of SCD on auditory responses to AC stimuli.

Results: The introduction of an SCD increased the sensitivity to BC stimuli by 8 dB (near 500Hz) and decreased sensitivity to AC stimuli by 8dB (near 500Hz), resulting in an air-bone gap of 16dB. Additionally, both Yme and Vs increased after SCD in response to AC sound. The AC results are consistent with the 'third-window' hypothesis, in which the SCD shunts stapes volume velocity away from the cochlea through the dehiscent canal resulting in decreased auditory sensitivity to AC stimuli and increases in Yme and Us. Our mathematical model quantitatively supports this hypothesis.

Conclusions: Superior canal dehiscence produces an air-bone-gap in an animal model by inducing both increased sensitivity of the ear to bone-conducted sound and decreased sensitivity to air-conducted sound. Dehiscence induced changes in middle-ear mechanics are consistent with the SCD shunt reducing the cochlear input impedance. [Work supported by NSF and NIDCD]