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Comment on "When an air-bone gap is not a sign of a middle-ear conductive loss" By Sohmer et al

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I'd like to thank Dr. Sohmer and his colleagues who, in their letter to *Ear and Hearing* (Sohmer, Sichel, Perez & Adelman, in press), cited our work on conductive hearing loss produced by inner-ear pathology. They have summarized some of the results my coworkers and I have gathered while investigating what we and others (Minor 2000; Mikulec et al. 2004; Rosowski et al. 2004; Songer, Brinsko & Rosowski 2004; Songer and Rosowski 2005, [2006], 2007; Chien, Ravicz, Rosowski & Merchant 2007; Merchant, Nakajima, Halpin et al. 2007; Merchant & Rosowski 2008) refer to as conductive hearing loss due to the presence of an abnormal third inner-ear window. However, Dr. Sohmer and his coworkers feel that there are methodological issues with the experiments we performed and question our interpretation.

Sohmer et al.'s first criticism of our work revolves around the difference in the *characteristic impedance* (which depends on the density and compressibility of a fluid or gas) between air and body fluid. Sohmer and coworkers point out that our experimentally produced canal dehiscences in the chinchilla (Songer & Rosowski 2005, 2006, 2007) and temporal bone (Chien et al. 2007) models of superior canal dehiscence were between the inner-ear lymph of the semicircular canal and the air in the middle-ear cavity, while the human pathology couples inner-ear lymphs with cranial fluids and tissues. Sohmer et al. argue that the large mismatch between the characteristic impedances of inner-ear lymph and air seen in the dehiscences we introduced is fundamentally different from the impedance 'match' between inner-ear and cranial fluids in pathological superior-canal dehiscence.

What Sohmer et al. ignore in their argument is that simple comparisons of the characteristic impedance of fluids or gases are only relevant in the ideal case when the two fluids (a gas, like air, can be considered a fluid) are separated by a near infinite interface. Under those conditions each acoustic particle (Kinsler et al. Chapter 6, 1982) of each of the fluids need only interact with other like particles of fluid, or, when near the interface, with particles of the other fluid. In the real world, with restricted dimensions, the fluid particles also interact with the walls of the containers that surround them. In these real conditions, the *acoustic impedance* is relevant; this impedance is determined not only by the physical characteristics of the fluids but also the dimensions and characteristics of the fluid containers (Kinsler et al. Chapters 9 & 10, 1982). In the case of the dehiscent semicircular canal in chinchillas or humans, the container is a rigid-walled tube-like bony canal with a diameter of about 0.1 mm and length of 3–5 mm. The frequency-dependent acoustic impedance of such a small fluid-filled structure is very large in the range of sound frequencies over which we hear. Intuition and experience support this notion: It takes a much larger pressure head to move the same flow rate through a narrow hose than through a wide hose. A similar dependence on the dimensions of the container affects the

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acoustic impedance looking into the fluid-filled cranium from the dehiscence. Our calculations (Rosowski et al., 2004; Songer 2006; Songer & Rosowski 2007) suggest that over the 200 to 20000 Hz range, the acoustic impedance related to sound flow through the dehiscent human canal is 3 to 3,000 times larger than the impedance of the fluid in the cranium. This difference in impedance is large enough that our model calculations would be essentially unchanged if the cranium were full of air rather than body fluids. The basic conclusion here is yes, Sohmer and colleagues are correct in noting that there is a mismatch between the impedance associated with sound flow into the middle-ear cavity from the dehiscence in our experiments and the much larger impedance associated with fluid motion in the semicircular canal. However, the impedance associated with the flow of sound into the fluid- and tissue-filled cranium from a dehiscence is also very small compared to the impedance of the canal. Indeed, over the audible range, the impedance of both an air-filled and fluid-filled human cranium are so small compared to the canal impedance that neither are matched to the canal impedance, and neither represents a significant load on the canal. Support for this conclusion comes from the similarity of our measurements of the effects of canal dehiscence on ossicular and round-window velocity in temporal bones with measurements of ossicular velocity and hearing thresholds in live humans with superior-canal dehiscence (Chien et al. 2007). Further support comes from reports of changes in bone-conducted thresholds in humans with dehiscences between the lateral (or horizontal) canal and middle-ear air space that are similar to the hearing changes observed in superior canal dehiscence (Merchant & Rosowski 2008).

Sohmer et al. (in press) also raise the issue of sound level, and point out all of the measurements of cochlear potential, dehiscence velocity, stapes velocity and middle-ear input impedance made in the chinchillas and temporal bones (Rosowski et al. 2004; Songer, Brinsko & Rosowski 2004; Songer and Rosowski 2005, 2006, 2007; Chien et al. 2007) used sound stimuli of 70 to 110 dB SPL. Considering, the large amount of data on the linearity of middle-ear input and output characteristics over a broad stimulus range (Guinan and Peake 1967; Nedzelnitsky 1980; Goode, Killion, Nakamura and Nishihara 1994; Dalhoff, Turcanu, Zenner & Gummer 2008), as well as the linearity of the cochlear microphonic over sound levels of 0 to 80 dB (Wever & Lawrence, Chapter 9, 1954; Dallos 1970) it is unclear to me why Sohmer et al. suggest that our results would differ if we were able to measure mechanical or sensory responses near perceptual threshold levels. Furthermore, as noted above, our measurements of the effects of canal dehiscence on ossicular and round-window velocity in temporal bones compare well with measurements of ossicular velocity and hearing thresholds in live humans with dehiscent canals (Chien et al. 2008).

Since Sohmer et al. decided to introduce our work into a discussion of their (Freeman, Sichel & Sohmer 2000; Sohmer, Freeman, Geal-Dor, et al. 2000; Sohmer & Freeman 2004) and others' (Watanabe, Bertoli & Probst in press) demonstrations of the sensitivity of the ear to vibration sources placed on non-bony parts of the head, I will take the liberty to comment on this issue. I think the works referenced at the start of this paragraph contain repeated demonstrations that the term 'bone-conducted sound' is misleading. Clearly, the head is made of bone, fluid and soft tissues and the bony cranium is filled with fluid and fluid-like soft tissues. It should not be surprising that compression and translation of the cranium also compresses and translates the fluid and soft-tissues within the cranium. Nor should it be surprising that compression and translation of the fluids and soft-tissues within the cranium act to compress and translate the bone that surrounds them. While I believe the Sohmer group's and others' demonstrations that vibration sources placed on the eye or open cranium produce a significant auditory response is a demonstration of the coupling of the vibration of the cranial contents to the ear, some part (large or small) of that coupling must arise because of compressions and translation of the bone around the ear secondary to compression and translation of the fluid and tissues within the cranium. Simply put: If you vibrate the nearly incompressible contents of a container, the container walls also vibrate. Therefore, while most of us don't appreciate

the role of the cranial fluids and soft-tissues in producing the response of the ear to vibration of the cranial bones, theories that suggest that inner-ear fluid channels are important to the conduction of soft-tissue vibrations to the ear don't always account for the effect of such fluid vibrations on the bones surrounding the ear.

While I find Dr. Sohmer's hypothesis of fluid-coupling of head vibration to the ear worth consideration, given that vibration of the cranial contents must be linked to vibration of the surrounding bone, I think demonstrations that closing some of the potential fluid pathways reduce the ears responses to fluid-borne stimuli are part of the 'existence proof' needed for the demonstration of such paths. I realize that the Sohmer group has argued that their observations of an increase in response to bone-conduction stimuli after a superior canal dehiscence is opened is an argument in favor of their fluid-channel hypothesis (Sohmer, Freeman and Perez 2004). However, Rosowski et al. (2004) and Songer (2006) have been able to predict just such an enhancement based on dehiscence-induced changes in compressional bone conduction without the need to invoke the direct fluid coupling of cranial and inner-ear contents. Furthermore, our group (Songer, Brinsko and Rosowski 2004; Rosowski et al. 2004; Songer 2006) has repeatedly produced increases in bone conduction sensitivity in the chinchilla model with dehiscences placed between the superior canal and the middle ear, a result that is difficult to explain in terms of improved fluid coupling from brain to the inner ear. Similar increases in bone conduction seen in clinical cases of dehiscence or fenestration of the lateral canal are also not easily explained in terms of improved fluid coupling between the inner ear and cranial contents, but can be explained by an alteration in compressional bone conduction (Merchant and Rosowski 2008).

References

Chien W, Ravicz ME, Rosowski JJ, Merchant SN. Measurements of human middle- and inner-ear mechanics with dehiscence of the superior semicircular canal. Otol Neurotol 2007;28:250–257. [PubMed: 17255894]

Dalhoff E, Turcanu D, Zenner HP, Gummer AW. Distortion product otoacoustic emissions measured as vibration on the eardrum of human subjects. PNAS 2007;104:1546–1551. [PubMed: 17242353]

Dallos P. Low frequency auditory characteristics: species dependence. J. Acoust. Soc. Am 1970;48:489–499. [PubMed: 5470495]

Freeman S, Sichel JY, Sohmer H. Bone conduction experiments in animals - evidence for a non-osseous mechanism. Hear. Res 2000;146:72–80. [PubMed: 10913885]

Goode RL, Killion M, Nakamura K, Nishihara S. New knowledge about the function of the human middle ear: Development of an improved analog model. Am. J. Otol 1994;15:145–154. [PubMed: 8172293]

Guinan JJ Jr, Peake WT. Middle-ear characteristics of anesthetized cats. J. Acoust. Soc. Am 1967;41:1237–1261. [PubMed: 6074788]

Kinsler, LE.; Frey, AR.; Coppens, AB.; Sanders, JV. Fundamentals of Acoustics. New York: Wiley and Sons; 1982.

Merchant SN, Nakajima HH, Halpin C, Nadol JB Jr, Lee DJ, Innis WP, Curtin H, Rosowski JJ. Clinical investigation and mechanism of air-bone gaps in large vestibular aquaeduct syndrome. Ann. Oto.l Rhinol. Laryngol 2007;116:532–541.

Merchant SN, Rosowski JJ. Conductive hearing loss caused by third-window lesions of the inner ear. Otol. Neurotol 2008;29:282–289. [PubMed: 18223508]

Mikulec AA, McKenna MJ, Ramsey MJ, Rosowski JJ, Herrmann BS, Rauch SD, Curtin HD, Merchant SN. Superior semicircular canal dehiscence presenting as conductive hearing loss without vertigo. Otol Neurotol 2004;25:121–129. [PubMed: 15021770]

Minor LB. Superior canal dehiscence syndrome. Am. J. Otol 2000;21:9–19. [PubMed: 10651428]

Nedzelnitsky V. Sound pressures in the basal turn of the cat cochlea. J. Acoust. Soc. Am 1980;68:1676–1689. [PubMed: 7462467]

Rosowski JJ, Nakajima HH, Merchant SN. Clinical utility of laser-doppler vibrometer measurements in live normal and pathologic human ears. Ear & Hearing 2008;29:3–19. [PubMed: 18091103]

Rosowski JJ, Songer JE, Nakajima HH, Brinsko KM, Merchant SN. Clinical, Experimental and Theoretical investigations of the effect of superior semicircular canal dehiscence on hearing mechanisms. Otol. Neurotol 2004;25:323–332. [PubMed: 15129113]

Sohmer H, Freeman S, Geal-Dor M, et al. Bone conduction experiments in humans - a fluid pathway from bone to ear. Hear. Res 2000;146:81–88. [PubMed: 10913886]

Sohmer H, Freeman S. Further evidence for a fluid pathway during bone conduction auditory stimulation. Hear. Res 2004;193:105–110. [PubMed: 15219325]

Sohmer H, Freeman S, Perez R. Semicircular canal fenestration - improvement of bone- but not air-conducted auditory thresholds. Hear. Res 2004;187:105–100. [PubMed: 14698091]

Sohmer H, Sichel J-Y, Perez R, Adelman C. When an air-bone gap (ABG) is not a sign of middle ear conductive loss. Ear and Hear. (in press).

Songer, JE. PhD Thesis. Massachusetts: Institute of Technology; 2006. Superior Canal Dehiscence: Auditory Mechanisms.

Songer, JE.; Brinsko, KM.; Rosowski, JJ. Superior semicircular canal dehiscenece and bone conduction in chinchilla. In: Gyo, K.; Wada, H.; Hato, N.; Koike, T., editors. Middle-ear mechanics in research and otology. Singapore: World Scientific; 2004. p. 234-241.

Songer JE, Rosowski JJ. The effect of superior canal dehiscence on cochlear potential in response to air-conducted stimuli in chinchilla. Hear. Res 2005;210:53–62. [PubMed: 16150562]

Songer JE, Rosowski JJ. The effect of superior-canal opening on middle-ear input admittance and air-conducted stapes velocity in chinchilla. J Acoust. Soc Am 2006;120:258–269. [PubMed: 16875223]

Songer JE, Rosowski JJ. A mechano-acoustic model of the effect of superior canal dehiscence on hearing in chinchilla. J. Acoust. Soc. Am 2007;122:943–951. [PubMed: 17672643]

Watanabe T, Bertoli S, Probst R. Transmission pathways of vibratory stimulation as measured by subjective thresholds and DPOAEs. Ear Hear. (in press).

Wever, EG.; Lawrence, M. Physiological Acoustics. Princeton: Princeton University Press; 1954.