Infrasound transmission in the human ear: Implications for acoustic and vestibular responses of the normal and dehiscent inner ear

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The transmission of infrasound within the human ear is not well understood. To investigate infrasound propagation through the middle and inner ear, velocities of the stapes and round window membrane were measured to very low frequencies (down to 0.9 Hz from 2000 Hz) in fresh cadaveric human specimens. Results from ear-canal sound stimulation responses show that below 200 Hz, the middle ear impedance is dominated by its stiffness term, limiting sound transmission to the inner ear. During air-conduction, normal ears have approximately equal volume velocities at the oval (stapes) and round windows, known as a two-window system. However, perturbing the impedance of the inner ear with a superior canal dehiscence (SCD), a pathological opening of the bone surrounding the semicircular canal, breaks down this simple two-window system. SCD changes the volume velocity flow in the inner ear, particularly at low frequencies. The experimental findings and model predictions in this study demonstrate that low-frequency auditory and vestibular sound transmission can be affected by a change in the inner-ear impedance due to a SCD.

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NOMENCLATURE

SVTF stapes velocity transfer function
RWM round window membrane
SCD superior canal dehiscence

I. INTRODUCTION

It is widely believed that human hearing is insensitive to infrasound, defined as frequencies below 20 Hz. However, there is evidence that infrasound can alter the processing of sound in the cochlea. For example, animal studies show that exposure to infrasound can modulate the endocochlear potential, leading to a change in the electrochemical voltage that drives the receptor current through the transduction channels of the auditory hair cells (Salt *et al.*, 2013; Salt and DeMott, 1999). Moreover, high-intensity, low-frequency bias tones can alter distortion product otoacoustic emissions and shift the frequency and level of spontaneous otoacoustic emissions, indicating that cochlear processing is affected by infrasound in humans (Hensel *et al.*, 2007; Kugler *et al.*, 2014; Marquardt *et al.*, 2007). The long-term consequences of exposure to infrasound are not clear, but subjective reports claim that exposure to infrasound affects sleep habits, disrupts work performance, and compromises the well-being of the population (e.g., review Baliatsas *et al.*, 2016). Given these observations, it is worthwhile to objectively investigate

how infrasound propagates through the human middle and inner ear, which we address here.

In addition to studies of hearing thresholds, it has been shown that the vestibular system, equipped with specialized low-frequency sensing organs, is also sensitive to acoustic stimulation (Møller and Pedersen, 2004; Young *et al.*, 1977). The experiments in the mentioned studies rely on infrasound entering the inner ear via the middle ear. However, the mechanical constraints that the middle ear and inner ear impose on the transmission of infrasound are unknown.

To understand how infrasound is transmitted, this study describes low-frequency middle ear and inner ear transfer functions in fresh cadaveric normal ears. This study also determines how these transfer functions change due to perturbation of inner-ear mechanics. We will specifically focus on superior canal dehiscence (SCD), a disorder characterized by an abnormal opening of the bone surrounding the superior semicircular canal, which was shown to change the transmission of sound especially at low frequencies (Chien *et al.*, 2007; Niesten *et al.*, 2015; Pisano *et al.*, 2012). With a prevalence of 0.7%–1.9% in the U.S. population, SCD is being recognized as a relatively common otologic pathology (Carey *et al.*, 2000; Minor *et al.*, 1998). Vestibular and auditory symptoms, many of which are induced by low-frequency sounds, including vertigo and autophony, are debilitating for the patients (Minor, 2005).

In the normal ear, air-conducted sound is transmitted through a “two-window” system, consisting of the oval window and the round window membrane (RWM). In the two-window system, the volume velocity of the oval window is

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equal to the volume velocity of the round window—no appreciable volume velocity is lost to another sound-conducting path in the inner ear (Stenfelt *et al.*, 2004).¹ In patients with SCD, an additional sound-conducting path, known as a “third window,” is added within the inner ear, changing the distribution of volume velocities. Figures 1(a) and 1(b) show a circuit diagram of a normal ear and an ear with SCD, respectively. In the normal ear, the volume velocity (U) at each node is identical ($U_{\text{Stapes}}^{\text{Normal}} = U_{\text{Diff}}^{\text{Normal}} = U_{\text{RWM}}^{\text{Normal}}$). However, in the SCD-affected ear, the volume velocity is divided in the vestibule and can flow through (1) the series impedances of the cochlear partition (Z_{Diff}) and RWM (Z_{RWM}), or (2) the SCD impedance (Z_{SCD}). Z_{Diff} , the “differential impedance” across the cochlear partition is defined as the impedance at the base of the cochlea (near the oval and round windows), and is also influenced by the helicotrema (Nakajima *et al.*, 2009). $Z_{\text{Diff}} = P_{\text{Diff}} / U_{\text{Diff}}$, where the differential pressure across the partition (P_{Diff}) is defined as $P_{\text{Diff}} = P_{\text{SV}} - P_{\text{ST}}$ (P_{SV} is the pressure in scala vestibuli and P_{ST} the pressure in scala tympani near the oval and round windows; Nakajima *et al.*, 2009). Experiments on fresh cadaveric human specimens show that SCD causes decreases in RWM velocities, P_{SV} , P_{ST} , and P_{Diff} at frequencies below 1000 Hz, resulting in decreased low-frequency, air-conducted sound transmission across the partition, consistent with low-

frequency hearing loss in SCD patients (Pisano *et al.*, 2012; Niesten *et al.*, 2015; Minor, 2005). Additionally, SCD results in shunting of volume velocity from the oval window to the SCD, resulting in sound-induced stimulation of the ampulla of the vestibular system and vertigo known as Tullio phenomenon (Minor *et al.*, 1998).

A SCD functions as an acoustic volume velocity leak between the inner ear vestibule and the middle cranial fossa. Because of the low-pass characteristics of a small opening, a SCD predictably influences auditory and vestibular thresholds at low frequencies (Songer and Rosowski, 2006). These changes have been observed in clinical studies and temporal bone experiments, but because previous measurements were made only for frequencies above 100 Hz, the effects of a SCD on intracochlear pressures, RWM velocities, audiometric thresholds, and vestibular thresholds were small (0–15 dB; Chien *et al.*, 2007; McEvoy *et al.*, 2013; Milojevic *et al.*, 2013; Niesten *et al.*, 2015; Pisano *et al.*, 2012). Without lower frequency information, we lack an understanding of the acoustic effects of SCD.

In this present study, we characterize stapes and RWM velocities for the first time down to very low frequencies of 0.9 Hz. We then determine the effects of a mechanical perturbation to the inner ear (in the form of a SCD) on stapes and RWM velocities at low frequencies and infrasound. To characterize and understand the mechano-acoustic mechanisms behind SCD, we apply a lumped element network model.

II. MATERIALS AND METHODS

A. Temporal bones

Five fresh human temporal bones (without the use of fixative) were used for this study. Upon death of the donor, the temporal bones were removed from the skull base using a Schuknecht plug cutter (Nadol, 1996) at Massachusetts General Hospital. Minutes after removal, the specimens were stored in 250 ml of 0.9% saline with two drops of betadine solution and stored in a refrigerator. The saline solution was renewed every 24 h between the bone extraction and the experiment. Three of the temporal bones were kept in the refrigerator before the experiment (1–6 days) and two temporal bones were frozen shortly after extraction and then defrosted for experiments. To defrost the frozen temporal bone specimens, we placed it in room-temperature saline for 30 min. Experiments were conducted at room temperature. The age range of the donors was between 28 and 85 yr.

To enable access to the middle-ear ossicles and the RWM, we exposed the middle ear cavity and epitympanic space by drilling out the bone surrounding the posterior-lateral aspect of the temporal bone. We severed the stapedial tendon to access the area close to the stapes and to have an unobstructed view of the posterior crus of the stapes. Initial measurements of stapes and RWM velocities were performed to confirm that no air entered the cochlea during the preparation process. A phase difference of one-half cycle between stapes and RWM velocities at frequencies between 50 and 300 Hz was a good indicator that no air was enclosed in the inner ear (Nakajima *et al.*, 2009). After baseline

FIG. 1. Schematic and impedances of the inner ear. (a) The circuit of the normal inner ear consists of the differential impedance of the cochlear partition (Z_{Diff}) and the impedance of the RWM (Z_{RWM}). Z_{Diff} is not a local impedance of the basilar membrane, but defined as the differential pressure across the partition at the base, over the volume velocity of the stapes (U_{Stapes}), i.e., $Z_{\text{Diff}} = (P_{\text{SV}} - P_{\text{ST}}) / U_{\text{Stapes}}$, where P_{SV} and P_{ST} are pressures in scala vestibuli and scala tympani at the base. Z_{Diff} is also influenced by the helicotrema. The volume velocities of the stapes (U_{Stapes}), across the partition (U_{Diff}), and the RWM (U_{RWM}) are shown with arrows and are equivalent during air conduction in normal ears. (b) Circuit model for the SCD case includes an impedance and volume velocity for the semicircular canal (Z_{SCD} , U_{SCD}). [Figure adapted from Stieger *et al.* (2013).]

measurements for the normal ear, a dehiscence was introduced at the lateral aspect of the semicircular canal near the arcuate eminence with varying size between 0.21 mm^2 and 0.7 mm^2 across specimens. We used a micro-measurement device that allowed us to measure the SCD under the operating microscope to within a certainty of $50\text{--}100\text{ }\mu\text{m}$. It is rather difficult to control the SCD size during an experiment, which is why the sizes differed, but the sizes of the SCDs we introduced were comparable to other studies and within the range of what is observed in patients (Niesten *et al.*, 2015; Pisano *et al.*, 2012). The lateral aspect of the canal was covered by a fluid column of about 0.5 mm to prevent air from entering the inner ear (see Pisano *et al.*, 2012; Niesten *et al.*, 2015, for details). The time between baseline measurements and SCD measurements were as much as 30 min. Similarly, patching the SCD to restore the baseline measurements took up to 30 min. Thus, the time elapsed between initial and final measurements could have been up to 1 h. The middle ear was kept moist during that time to prevent it from drying.

We used normal specimens without known middle- and inner-ear pathologies. In this series of experiments, we included five out of eight experiments (three experiments were abandoned early, because the RWM was damaged, air was enclosed in the cochlea, or the noise floor was too high). The five experiments that were completed had baseline stapes velocities that were within or near the range of Rosowski *et al.* (2007) standards and had a signal-to-noise ratio $> 10\text{ dB}$. We did include an experiment with a stapes velocity resonant frequency near 350 Hz (this low resonance might be due to a compliant middle-ear system). Because this study focuses on low frequencies, we included this experimental result (below 200 Hz , the data were within the standard, we plotted the data with a dashed line, distinguishing it from the other experiments that are plotted with solid lines). Fresh human cadaveric temporal bone specimens, including those previously frozen, have been shown to have very similar middle and inner ear macro-mechanical properties to that of the living human, which is why a temporal bone model is appropriate for this work (Nakajima *et al.*, 2005; Chien *et al.*, 2009; Rosowski *et al.*, 1990). Because of the high consistency of our data, agreement with previous studies (Chien *et al.*, 2007; Niesten *et al.*, 2015; Pisano *et al.*, 2012, to 100 Hz), and our ability to reverse the effects of our manipulation (a strong experimental control), five temporal bones seemed sufficient for this technically challenging study.

B. Sound stimulation

Acoustic pure tones between 0.9 Hz and 2 kHz (3 points per octave in the frequency range $0.9\text{--}6\text{ Hz}$; 5 points per octave between 5 and 100 Hz ; and 10 points per octave between 50 and 2000 Hz) were generated by a Beyer dynamics DT 770 M headphone driver (Heilbronn, Germany) and delivered to the ear canal via a flexible polyethylene tube. The time to measure all frequencies for each condition was 3–4 min. The sound levels employed were between 75 and 110 dB sound pressure level (SPL), i.e., in the linear operating range of the middle ear (Greene *et al.*,

2017). Details regarding the generation of high-intensity, low-frequency pure tones with commercial headphone drivers can be found in the Appendix, and have also been presented elsewhere (Hensel *et al.*, 2007). All data acquisition in response to sound (from microphone and vibrometry) were recorded with a PXI interface, using a standard sampling frequency of 500 kS/s .

C. Microphone

The SPL in the sealed ear canal was monitored with a Brüel and Kjær (BK, Nærum, Denmark) infrasound microphone (type 4964 pre-polarized cartridge, type 2671-W-001 preamplifier, and 1704-A-001 signal conditioner). A polyethylene tube with an inner radius of 1.5 mm and 2 in. in length was used to connect the microphone to the ear canal. Mounting putty was used to seal the microphone tube, as well as the speaker tube, to the ear canal. The infrasound microphone was calibrated in a standard manner (see the Appendix for details).

D. Velocity measurements

A commercial laser Doppler vibrometry system (Polytec CLV 700, Waldbronn, Germany) was used to measure velocities of the posterior crus of the stapes and the RWM. Three to six auto reflective, metal coated beads ($50\text{ }\mu\text{m}$ in diameter and $\sim 0.07\text{ }\mu\text{g}$ each) were placed on the posterior crus of the stapes and the center of the RWM to enhance the signal-to-noise ratio for stapes and RWM measurements. Keeping the stapes footplate dry for measurement is difficult because the SCD region needs to be under fluid. We find that the posterior crus and footplate center have similar measurements at low frequencies ($< 2\text{ kHz}$) if the laser is as close to perpendicular to the plane of the footplate and if the laser direction is parallel to the plane defined by the posterior and anterior crus of the stapes (to prevent measuring superior-inferior rocking of the stapes). Stapes measurements were made at an angle of ~ 30 degrees with respect to the axis of the piston-type motion; a cosine-correction was not taken into account because it would change the magnitude only by about 1 dB . The measurement location and laser-beam angle before and after SCD did not change. For the RWM measurements, we tried to be consistent across specimens with the placement of the beads near the center of the RWM. The RWM is not a perfectly circular membrane, but has been described as a hyperbolic paraboloid with almost a flat area near the center. The noise floor of the laser measurement was determined by measuring the velocity of the cochlear promontory during acoustic stimulation.

To allow for low-frequency velocity measurements, the noise floor of the system needed to be reduced considerably. Four interventions enabled measuring stapes and RWM responses down to 0.9 Hz . First, the temporal bone holder was placed on an air table inside a double-walled booth with an elevated floor to isolate it from vibratory building noise. Second, the microscope, where the laser system was attached, was mounted on the same air-floating table, reducing the differential modes between specimen and laser system. Third, the position of the laser was enforced by

supporting the headpiece of the microscope with rigid rods and clamps to further minimize relative motion between the specimen and laser system. Fourth, relatively high SPLs (between 90 and 110 dB SPL at low frequencies, checked for linear operation detailed below) were used to record stapes and RWM velocities.

E. Harmonic distortions and noise sources

Figure 2(a) shows an example of the sound pressure in the ear canal (continuous solid line) and the spectrum during the presentation of a 6 Hz pure tone, indicated as f_0 . The odd-order harmonic distortion products are indicated. The nonlinearities produced by the loudspeaker were 30–40 dB ($\sim 1\%$) below the level of the probe frequency for all stimulus frequencies used. The acoustic noise floor in our sound-attenuating chamber is considerably higher at low frequencies—the slope of the noise floor below 200 Hz is approximately -40 dB/decade. Figure 2(b) shows an example of a raw stapes velocity measurement (thick solid line) next to the velocity of the cochlear bone in response to the same acoustic input at the ear canal. At around 3 Hz and 12 Hz, the laser Doppler vibrometer (LDV) picked up considerable vibration of the specimen, possibly due to vibrational building noise in the environment. For our measurements in normal ears, a signal-to-noise ratio of >10 dB was maintained at all frequencies.

III. RESULTS

A. Stapes and RWM velocities in normal ears

Figure 3(a) shows the magnitude and phase response of stapes velocity transfer functions (SVTFs; stapes velocity referenced to ear-canal pressure) for five fresh human specimens. We show reference data (95% confidence interval across 13 studies) from Rosowski *et al.* (2007) as a gray-shaded area.

For individual specimen [Fig. 3(a), colored lines], the magnitudes of the SVTFs between 0.9 and 300 Hz are increasing proportionally by about 20 dB/decade (dashed black reference line indicates an increase of 20 dB/decade). The frequency corresponding to the peak magnitude varies from 350 Hz to 1 kHz across specimens, and the absolute values of the maximum vary from 1×10^{-4} to 2×10^{-4} m/s

FIG. 2. Distortion and noise of the acoustic stimuli. (a) Sound pressure measured in the ear canal (continuous line) with a typical noise floor (dotted line) during the presentation of a 6 Hz pure tone. Harmonic distortions of the loudspeaker were 30–40 dB below the primary level for all frequencies. (b) Stapes velocity measurements compared to the velocity of the cochlear promontory during sound stimulation (raw measurements, not referenced to ear canal pressure).

Pa^{-1} . For higher frequencies, SVTFs generally decrease with increasing frequency. However, this magnitude decrease was not necessarily monotonic and the phase varied with frequency. Complex motions of the stapes can lead to distinct anti-resonances at frequencies above 2 kHz (Rosowski *et al.*, 2007). Such complex behavior is not observed at frequencies below 300 Hz, where the magnitudes of the SVTFs increase monotonically with frequency. Below 300 Hz, the velocity of the stapes leads the ear canal pressure by a quarter-cycle, as can be observed in the phase response. The monotonically increasing magnitude and quarter-cycle phase relative to the ear canal pressure imply a stiffness-dominated middle ear and is consistent with previous studies (Greene *et al.*, 2017; Rosowski *et al.*, 2007).

An interesting new observation is that below 10 Hz, the phase of the SVTF starts deviating from 0.25 cycles toward 0 cycles, consistent with the SVTF becoming more resistive toward lower frequencies. In the same frequency range, the increase in magnitude is less than 20 dB/octave. Below 10 Hz, the middle ear may have additional resistive components (frictional losses) that are not accounted for in current middle ear models, which predict a perfectly horizontal phase at low frequencies (Kringelbotn, 1988; Rosowski and Merchant, 1995).

Figure 3(b) shows the velocity of the RWM referenced to the sound pressure in the ear canal. Similarly to the stapes, the velocity of the RWM increases by about 20 dB/decade for frequencies between 1 and 200 Hz. RWM velocity magnitudes, measured at the center of the RWM, are generally higher than the velocity of the posterior crus of the stapes (as a reference, the horizontal grid lines in each graph represent the same magnitudes). This observation is consistent with previous measurements of RWM motion, where it was shown that the center of the RWM moves considerably more than the stapes footplate (Stenfelt *et al.*, 2004). The phase of the RWM is lagging the ear canal pressure by a quarter-

FIG. 3. (Color online) Normal data for five ears. (a) Individual stapes velocities (v_{stapes}) referenced to ear canal pressure (P_{EC}). Gray-shaded area is normative data from Rosowski *et al.* (2007). One specimen had a low resonance (at 350 Hz, blue dashed line) with similar response to other ears at lower frequencies. (b) Individual RWM velocities (v_{RWM}) from the center of the RWM referenced to P_{EC} . Black dashed lines in (a) and (b) indicate an increase by 20 dB/decade. The artifact was determined by measuring the vibration of the cochlear promontory during sound stimulation.

cycle at frequencies below 200 Hz, leading to a phase difference between stapes and RWM of 0.5 cycles (the RWM bulges out when the stapes pushes in).

B. The effect of SCD on stapes and RWM velocities

Figure 4 follows the same format as Fig. 3, but the measurements were recorded after a dehiscence was introduced at the lateral aspect of the semicircular canal near the arcuate eminence. The size of the dehiscence varied between 0.21 mm^2 and 0.7 mm^2 across bones.

After SCD, stapes vibrations [Fig. 4(a)] are similar to measurements in normal ears [before SCD, Fig. 3(a)]. For frequencies between 0.9 and 200 Hz, the magnitude of SVTFs increase by 20 dB/decade and the velocity of the stapes is leading the ear canal pressure by 0.25 cycles. Thus, in SCD-affected ears, the volume velocity entering the inner ear is not notably different from the normal ear for low frequencies.

The response of the RWM [Fig. 4(b)], unlike the stapes, is greatly affected by the dehiscence in the otic capsule. Compared to the reference measurements in Fig. 3(b), the slope of the magnitude is more variable and almost twice as large, i.e., 35–40 dB/decade with SCD [a black dashed line of 40 dB/decade is plotted in Fig. 3(b) for reference]. At frequencies below 300 Hz, the phase response of the SCD-affected ears is more positive compared to the normal ears in Fig. 3(b).

Another way to quantify the effects of SCD on stapes and RWM velocities is by plotting the ratio (difference in log-domain) between the SCD and normal condition. In Fig. 5(a), the SCD effect on stapes velocities is plotted for the individual tested specimens. Four out of the five tested specimens show a slight increase in stapes velocity after a SCD was introduced. The increase in stapes velocity is $\leq 5 \text{ dB}$ and similar across frequencies. One of the tested specimens shows a slight decrease in stapes velocity after the dehiscence was created, but the effect is $< 3 \text{ dB}$ across frequencies. On average, stapes velocities increased by 1.89 dB. The phase is unchanged for frequencies below 300 Hz. Above 300 Hz, we

FIG. 4. (Color online) SCD data for five ears. (a) Individual stapes velocities (v_{Stapes}) referenced to ear canal pressure (P_{EC}) with SCD. (b) Individual RWM velocities (v_{RWM}) referenced to P_{EC} . The black dashed line in (a) indicates an increase by 20 dB/decade; the black dashed line in (b) indicates an increase by 40 dB/decade. The data represented by the purple line are in the noise below 20 Hz and are not reliable in this frequency range.

observe slight differences of 0.05–0.1 cycles in the individual experiments, but no notable changes on average.

Figure 5(b) shows that with SCD, RWM velocities exhibit a profound decrease at low frequencies compared to the reference measurements: the changes in RWM velocity magnitude due to SCD show decreases of 10–35 dB per decade from high to low frequencies (100 Hz to 1 Hz). This is accompanied by a change in phase by +0.2 to +0.25 cycles [Fig. 5(b)]. The frequency range at which the SCD causes a systematic decrease in RWM velocity magnitude with decreasing frequency varies greatly across ears, leading to a much larger variance after SCD as compared to our reference measurements. In one specimen the RWM exhibits a decrease in velocity for frequencies below 1000 Hz (green line), whereas another specimen exhibits a decrease in RWM velocity only below 200 Hz (red line). Similar observations were made in Chien *et al.* (2007) although their low-frequency limit was 100 Hz (gray-shaded areas in Fig. 5) and thus, the effect of a SCD they observed was much smaller.

The decrease in RWM velocity and change in stapes velocity was reversible in all specimens when the SCD was patched, as indicated by thin black lines in Fig. 5. The reversal demonstrates an important experimental control.

C. RWM velocities referenced to stapes velocities

A useful way by which stapes and RWM velocities can be analyzed is by determining the ratio between RWM velocity and stapes velocity. This provides insights into losses occurring between the oval window and round window.

Figure 6(a) plots RWM velocities referenced to stapes velocities for the normal ear. The positive values of the magnitude imply that the velocity of the RWM center is higher than that of the stapes. Although it is known that the volume velocities of the oval window and round window are

FIG. 5. (Color online) Effect of SCD. (a) Changes in magnitude and phase of stapes velocity (v_{Stapes}) due to SCD. On average (bold black line), the stapes velocity increased by 1.89 dB after SCD. The SCD effect was reversible (plotted with thin black lines) within 1 dB in four specimens and to within 2 dB in one specimen. Gray-shaded area represents 95% confidence interval from Chien *et al.* (2007). (b) Change in RWM velocity (v_{RWM}) due to SCD.

generally equal in air conduction, it is not surprising that the center of the RWM moves more than the posterior crus of the stapes (Stenfelt *et al.*, 2004). While the stapes is rigid and the excursion of the posterior crus is similar to the piston-type excursion of the whole oval window at these low frequencies, the RWM is very compliant and the excursion near the center of the membrane (our measurement location) is largest compared to other locations on the RWM (Stenfelt *et al.*, 2004; Voss *et al.*, 1996). In our experiments, the velocity of the center of the RWM is approximately 5–15 dB larger than the velocity of the posterior crus of the stapes. For frequencies below 300 Hz, the difference between stapes and RWM velocities is independent of frequency.

In the normal ear [Fig. 6(a)], the phase response of the stapes and RWM velocities is generally 0.5 cycles out of phase for frequencies between 10 and 200 Hz, which indicates that the cochlear impedance is normal and the fluid in the cochlea is incompressible and free of air bubbles, and that there are no unusual fluid leaks or dehiscences of the otic capsule. In two out of five specimens, a perfect half-cycle relationship is observed for frequencies as low as 0.9 Hz (green and blue lines); four out of five bones show a perfect half-cycle difference down to 10 Hz (all but the purple line). The purple line deviates from the half-cycle below 10 Hz, which could be explained by experimental error (too much time elapsed during measurement of stapes and RWM velocities resulting in the accumulation of fluid on the RWM from residual saline in the surrounding middle ear space), small amounts of air in the cochlea, or an unusually low-impedance leak (for example, through the aqueducts).

In Fig. 6(b), RWM velocities referenced to stapes velocities are shown for the SCD-affected specimens. Compared to the flat magnitude ratio results of normal ears in Fig. 6(a), the SCD ratio magnitudes increase by about 20 dB/octave with increasing frequencies. The phase relationship of one-half cycle between stapes and RWM velocities is completely diminished at frequencies below 300 Hz, where it is converging to -0.3 to -0.25 cycles. The measurements in Fig. 6(b) imply that low-frequency volume velocity is directed away from the RWM, toward the dehiscence.

FIG. 6. (Color online) RWM velocities (v_{RWM}) referenced to stapes velocities (v_{Stapes}). (a) Individual normal data and (b) after SCD. The black dashed line in (b) indicates a change of 20 dB/decade.

D. A circuit model for the inner ear with SCD

1. Estimating the SCD effect on P_{Diff} with RWM velocity measurements

To better understand and quantify the mechano-acoustic mechanisms involved, we use the lumped-element circuit model shown in Fig. 1. The impedances, volume velocities, and pressures are complex numbers (magnitude and phase). The circuit in Fig. 1 can be used to establish a relationship between the pressures in the inner ear and the RWM velocities. The circuit of the *normal* inner ear consists of a volume velocity source ($U_{\text{Stapes}}^{\text{Normal}}$, at the stapes footplate), an impedance of the cochlear partition (Z_{Diff} , which includes the helicotrema), and the impedance of the RWM (Z_{RWM}). For the pressures in the inner ear, the cochlea input drive is defined as the differential pressure P_{Diff} across the partition, i.e., $P_{\text{Diff}} = P_{\text{SV}} - P_{\text{ST}}$, where P_{SV} and P_{ST} are the pressures in scala vestibuli and scala tympani, measured in the cochlear base far from the partition. Note that the cochlear impedance Z_C (as used in other studies) is not equal to Z_{Diff} , but defined as $Z_C = P_{\text{SV}}^{\text{Normal}} / U_{\text{Stapes}}^{\text{Normal}} = Z_{\text{Diff}} + Z_{\text{RWM}}$ (Nakajima *et al.*, 2009; Puria and Allen, 1991; Shera, 2007).

P_{Diff} is an important measure as it estimates the input drive to the cochlea and has frequency responses near identical to neurophysiological measures, such as cochlear microphonic (assuming that the neurosensory mechanism is intact; Dancer and Franke, 1980; Nakajima *et al.*, 2009). As shown in Fig. 1, the differential pressure across the partition can be written as

$$P_{\text{Diff}}^{\text{Normal}} = U_{\text{RWM}}^{\text{Normal}} Z_{\text{Diff}} \quad (1)$$

$$P_{\text{Diff}}^{\text{SCD}} = U_{\text{RWM}}^{\text{SCD}} Z_{\text{Diff}}. \quad (2)$$

By combining Eqs. (1) and (2), the effect (ratio) of SCD on the differential pressures, ΔP_{Diff} , can be expressed by means of the change in RWM velocities, i.e.,

$$\Delta P_{\text{Diff}} = \frac{P_{\text{Diff}}^{\text{SCD}}}{P_{\text{Diff}}^{\text{Normal}}} = \frac{U_{\text{RWM}}^{\text{SCD}}}{U_{\text{RWM}}^{\text{Normal}}} \approx \frac{v_{\text{RWM}}^{\text{SCD}}}{v_{\text{RWM}}^{\text{Normal}}}. \quad (3)$$

Equation (3) states that measuring a change in RWM velocity due to SCD is an estimate of a change in P_{Diff} , ΔP_{Diff} . Because ΔP_{Diff} closely resembles a change in hearing sensitivity due to a macro-mechanical (not sensorineural) change, the change in RWM velocity as seen in Fig. 5(b) can also be used to predict SCD-related hearing loss. Following the results in Fig. 5(b), we predict a low-frequency hearing loss due to SCD, which is consistent with clinical findings (Minor *et al.*, 1998). Furthermore, because volume velocity is shunted away from the cochlea toward the dehiscence, SCD patients can experience sound-induced dizziness (Tullio phenomenon) due to the shunted volume velocity stimulating the vestibular system (Minor *et al.*, 1998).

2. Determining the acoustic properties of a SCD

We can use our experimental results in conjunction with a computational model to determine the acoustic properties of a SCD. In Figs. 7(a) and 7(b), the values for Z_{Diff} and

FIG. 7. (Color online) Modeling results. (a) Impedance model employed with (b) the respective lumped elements. (c) Measured (black): experimental results from this study [same as average in Fig. 5(b)]. Model predictions from Eq. (7) using the parameter elements in (b). Measured ΔP_{Diff} (gray): actual measured change in P_{Diff} from Pisano *et al.* (2012); Niesten *et al.* (2015) ($n = 5$). $\Delta P_{\text{Diff}} = P_{\text{Diff}}^{\text{SCD}} / P_{\text{Diff}}^{\text{Normal}}$.

Z_{RWM} are similar to what has been used previously (Frear *et al.*, 2018; Nakajima *et al.*, 2009). Notably, the most updated estimate of Z_{Diff} consists of a resistor-inductor (RL) circuit element in parallel (instead of a resistor alone as used in older models), with $R = 3.04 \times 10^{10} \text{ Nsm}^{-5}$ and $L = 6.46 \times 10^7 \text{ Ns}^2\text{m}^{-5}$ [Fig. 7(b)]. The round window impedance Z_{RWM} is an resistor-inductor-capacitor (RLC) circuit element in series with $R = 1.47 \times 10^{10} \text{ Nsm}^{-5}$, $L = 7.76 \times 10^5 \text{ Ns}^2\text{m}^{-5}$, and $C = 3.59 \times 10^{-14} \text{ N}^{-1}\text{m}^5$. The elements used for the impedances are presented in Fig. 7(b).

Equation (3) states that the change in P_{Diff} after a SCD (ΔP_{Diff}) can be estimated by the change in RWM velocity $v_{\text{RWM}}^{\text{SCD}} / v_{\text{RWM}}^{\text{Normal}}$ [plotted in Fig. 5(b)]. Furthermore, using the model in Figs. 1(a) and 1(b), we can derive equations

$$U_{\text{RWM}}^{\text{Normal}} (Z_{\text{Diff}} + Z_{\text{RWM}}) = U_{\text{Stapes}}^{\text{Normal}} (Z_{\text{Diff}} + Z_{\text{RWM}}), \quad (4)$$

$$U_{\text{RWM}}^{\text{SCD}} (Z_{\text{Diff}} + Z_{\text{RWM}}) = U_{\text{Stapes}}^{\text{SCD}} (Z_{\text{Diff}} + Z_{\text{RWM}}) || (Z_{\text{SCD}}), \quad (5)$$

where the symbol “||” indicates the element is in parallel, as shown in Fig. 1(b). By using Eq. (3) and taking the ratio of Eqs. (4) and (5), we obtain the ratio of the RWM velocities before and after SCD

$$\begin{aligned} \Delta P_{\text{Diff}} &\approx \frac{v_{\text{RWM}}^{\text{SCD}}}{v_{\text{RWM}}^{\text{Normal}}} \approx \frac{U_{\text{RWM}}^{\text{SCD}}}{U_{\text{RWM}}^{\text{Normal}}} \\ &= \frac{U_{\text{Stapes}}^{\text{SCD}} (Z_{\text{Diff}} + Z_{\text{RWM}}) || (Z_{\text{SCD}})}{U_{\text{Stapes}}^{\text{Normal}} (Z_{\text{Diff}} + Z_{\text{RWM}})}. \end{aligned} \quad (6)$$

Including our observation that stapes velocities increase by 1.89 dB (24%) after SCD [Fig. 5(a)], Eq. (6) simplifies to

$$\Delta P_{\text{Diff}} \approx 1.24 \left(1 + \frac{Z_{\text{RWM}} + Z_{\text{Diff}}}{Z_{\text{SCD}}} \right)^{-1}. \quad (7)$$

The SCD impedance Z_{SCD} is found by fitting Eq. (7) with the known Z_{RWM} and Z_{Diff} to our experimentally measured changes in RWM velocities, enabling us to model ΔP_{Diff}

[Fig. 7(c)]. The above derived relationships assume that (1) the measurement locations on the stapes and RWM are the same for the normal and SCD ears, (2) the vibration modes of the stapes and RWM are unchanged by a SCD, and (3) the proportionality of the volume velocity to the measured point velocity of the stapes and RWM is the same for the normal and SCD ears, so that volume velocities are estimated by point velocities.

In Fig. 7(c), we plot the average of our experimental data ($v_{\text{RWM}}^{\text{SCD}} / v_{\text{RWM}}^{\text{Normal}}$, with black lines), the model calculations using Eq. (7) (green lines), and the average ΔP_{Diff} obtained from experimental intracochlear pressure measurements (from Pisano *et al.*, 2012, and Niesten *et al.*, 2015, with gray lines). The values for the SCD impedance Z_{SCD} that result in the model fit in Fig. 7(c) are: $R_{\text{SCD}} = 2.5 \times 10^{10} \text{ Nsm}^{-5}$ and $L_{\text{SCD}} = 1.0 \times 10^7 \text{ Ns}^2\text{m}^{-5}$. The computational model results closely follow our experimental data from velocity measurements. Furthermore, the model and our experimental data closely follow the experimental intracochlear pressure data as predicted by Eq. (3). Thus, we can refer to the change in RWM velocities as a change in the cochlear input drive, or ΔP_{Diff} .

In Fig. 7(c), we observe that the SCD affects ΔP_{Diff} predominantly at low frequencies. At frequencies above 1 kHz, the SCD has only minor effects on the input drive. This is observed in the individual experimental results [Fig. 5(b), colored lines], as well as in the model fit [Fig. 7(c), green line], where the curves converge to magnitude changes of almost 0 dB for frequencies greater than 1 kHz. At lower frequencies, the SCD leads to a substantial loss in the cochlear input drive (large negative ΔP_{Diff}). The magnitude decreases systematically as the frequency decreases, and the phase is converging to 0.25 cycles at low frequencies. For example, at 1 Hz the loss is as large as 40 dB. The SCD is acting like a first-order low-pass shunt, allowing low-frequency volume velocities to pass more easily through the SCD impedance. As a result, the cochlear input drive is decreasing with decreasing frequency. Because the cochlear input drive is decreasing at low frequencies and the volume velocity passes through the semicircular canal, the vestibular input drive (across the ampulla of the semicircular canal) increases at low frequencies with SCD.

E. Sensitivity of model parameters

To investigate whether the variability observed in Fig. 7(c) is due to the variability of the SCD impedance or other elements in the circuit, we investigate the sensitivity of the parameters in our model. In Figs. 8(a) and 8(b), the SCD impedance Z_{SCD} and the resistive component (R_{SCD}) of Z_{SCD} are multiplied with scalars. Changing the SCD impedance in this manner changes ΔP_{Diff} (the extent to which the cochlear input drive is affected by SCD). Differences in the SCD impedance may explain the variation across ears observed in Fig. 5(b). The resistive component (R_{SCD}) of the SCD impedance alone can explain most of the low-frequency variation in SCD effect [Fig. 8(b)], suggesting that the acoustic mass of the SCD plays a minor role at low frequencies compared to its resistive losses.

Figures 8(c) and 8(d) explore the consequences of altering the RWM impedance (and compliance, C_{RW}) on SCD's effect

FIG. 8. Perturbation of model parameters. The gray line in each panel represents the average experimental data (Fig. 5), and the black continuous lines represent the best model fit to the experimental data. (a) The SCD impedance and (b) SCD resistance are multiplied with scalars ranging from 0.3 to 3. (c) The RWM impedance and (d) compliance are multiplied by scalars from 0.3 to 3. $\Delta P_{\text{Diff}} = P_{\text{Diff}}^{\text{SCD}} / P_{\text{Diff}}^{\text{Normal}}$.

on cochlear input drive, ΔP_{Diff} . For our model, we use an average value for the RWM impedance (Z_{RWM}) across many experiments; however, Z_{RWM} varies by at least an order of magnitude across specimens (Frear *et al.*, 2018; Nakajima *et al.*, 2009). Changing the impedance or compliance of the RWM has a similar effect on ΔP_{Diff} as varying the SCD impedance, and can account for the variance we observe in the experimental measurements. The less compliant the RWM, the greater impact a SCD has on the cochlear and vestibular input drives. In Pisano *et al.* (2012) and Niesten *et al.* (2015), we showed that despite keeping the SCD size and location the same across ears, ΔP_{Diff} differed. These observations are consistent with our current modeling results in Figs. 8(c) and 8(d), where we show that the compliance of the RWM plays a critical role in how severely a SCD changes the pressures within the inner ear. The almost identical appearance of Figs. 8(c) and 8(d) suggests that variations in the RWM compliance, as opposed to variations in the acoustic mass or resistive losses, play a more direct role in how ΔP_{Diff} is affected.

IV. DISCUSSION

A. Ossicular motion at low frequencies and third-window effects

In Fig. 9, we present a summary of our main findings. In normal ears (continuous lines), both stapes and RWM

FIG. 9. Summary of normal and SCD data. (a) Average stapes velocity (v_{Stapes}) referenced to ear canal pressure (P_{EC}). (b) Average RWM velocity (v_{RWM}) referenced to P_{EC} . Shaded areas indicate ± 1 standard deviation.

velocities are monotonically increasing with a slope of 20 dB/decade for frequencies between 0.9 and 200 Hz. The phase at these low frequencies is close to ± 0.25 cycles, although we observe a slight decrease in phase below 10 Hz, possibly due to resistive losses causing the phase to become more real (or closer to 0 cycles). The velocity of the center of the compliant RWM is about 10 dB higher than the velocity of the posterior crus of the stapes. Although the area ratio between the stapes footplate and RWM (2.39/3.85) would only predict a difference of 4 dB (if we assume that the volume velocities of stapes and RWM are equal; see Stenfelt *et al.*, 2004), this assumption rests on a piston-like motion of both the stapes and RWM. Whereas the piston-like motion is a reasonable assumption for the stapes at low frequencies, the RWM behaves like a drum, clamped at its edges. As a result, the velocity of the center of the RWM is higher compared to other locations on the membrane and approximately 10 dB above the velocity of the stapes crus. Below 300 Hz, the half-cycle relationship between stapes and RWM is consistent with a simple relative motion between the two measurement locations (the RWM bulges out when the stapes pushes in).

Surprisingly, in normal ears (Fig. 9), RWM motion appears to be more consistent (smaller standard deviation) across ears than the stapes motion at frequencies below 300 Hz. In the tested frequency range, SVTFs are barely affected by a SCD [dashed lines in Fig. 9(a)]. On the other hand, velocities of the RWM are changed by a SCD in a predictable manner [dashed lines in 9(b)], making it a promising tool to investigate impedance changes in the inner ear, such as SCD.

An important finding from this study is that the SCD induced a decrease in RWM velocities that is frequency dependent. In some bones, a SCD affects RWM velocities at frequencies below 2 kHz, whereas other specimens are affected only at frequencies lower than 200 Hz. Thus, inner-ear dehiscences can be left unnoticed when the frequency range of the measurements is not sufficiently low. By extending the frequency range to lower frequencies, inner-

ear dehiscences can be reliably detected by measuring RWM velocities [Fig. 9(b)].

B. The effect of the size of the SCD

We investigated the effect of SCD size on stapes and RWM velocities in one of the five specimens (blue line in Figs. 3–6). Figure 10(a) shows stapes velocities in the normal case (black continuous line) and its response under varying SCD sizes (colored lines). Consistent to Fig. 5, stapes velocities are generally unaffected by a SCD. The size of the SCD does not change this conclusion.

Interestingly for RWM velocities [Fig. 10(b)], we observe that an increase in SCD size from the size of a pin-hole ($\sim 50 \mu\text{m}$ diameter) to a $0.2 \times 0.2 \text{ mm}$ dehiscence, increases the effect on RWM velocities. However, further increasing the size of the dehiscence by another millimeter or so did not produce much effect on RWM velocity.

Our current observations with RWM velocities are consistent with our previous findings from intracochlear pressure measurements (Pisano *et al.*, 2012; Niesten *et al.*, 2015)—an increase in SCD size decreases the sound transmission across the cochlear partition at low frequencies. However, once the SCD size exceeds certain dimensions, SCD effects on P_{Diff} and RWM velocities are independent of the size of the SCD. It was also shown that the saturation size varied across ears (Niesten *et al.*, 2015; Pisano *et al.*, 2012). Predictions were made by Songer and Rosowski (2006) and Kim *et al.* (2013) that the SCD size effect saturated when the area of the SCD equals the cross-sectional area of the canal. However, our experimental results from Pisano *et al.* (2012) and Niesten *et al.* (2015) contradicted this prediction. The cross-sectional diameter of the canal is approximately 1 mm. We found that the low-frequency decrease in sound transmission continued up to a length of 2 mm in some specimens (Niesten *et al.*, 2015; Pisano *et al.*, 2012). In the present study, our SCD sizes (between 0.21 and 0.7 mm^2) were similar or below the cross-sectional area of the canal ($\sim 0.7 \text{ mm}^2$). We used these sizes because it is

technically challenging to repair larger SCD sizes to completely reverse the effects of SCD (thus losing our experimental control). Our model is able to explain the consequences of SCD on the cochlear input drive below this saturation region. Figure 8(b) suggests that resistive losses (likely along the walls of the semicircular canal, as well as from the opening of the dehiscence itself) rather than an increase of the acoustic mass, could be responsible for the size-specific decrease in the cochlear input drive at low frequencies.

In this present study (as well as in our earlier intracochlear pressure studies) where SCD size was similar across ears, the variance of our results in Figs. 4(b) is likely attributed to anatomical variations across bones. Our present modeling results in Fig. 8 show that changes in RWM impedance and compliance, which can vary by an order of magnitude across ears (Frear *et al.*, 2018; Nakajima *et al.*, 2009), can change how a SCD affects the cochlear input drive, ΔP_{Diff} . An increase in Z_{RWM} (through decreasing the compliance of the RWM) results in a larger decrease of the cochlear input drive due to SCD. Therefore, our study may have implications for round window reinforcement surgery, where tissue and/or glue is used to stiffen the RWM (decrease its compliance) in hopes of reducing symptoms associated with SCD (Silverstein *et al.*, 2014). Our present findings are *inconsistent* with this surgical notion. In fact, round window reinforcement may possibly worsen the auditory and/or vestibular symptoms associated with SCD at low frequencies.

C. Effective dimensions of the semicircular canal with dehiscence

The anatomical dimensions of the superior semicircular canal can be used to calculate the acoustic mass and resistance within the canal associated with a SCD, based on equations for narrow tubes (Beranek, 1986). The acoustic impedance of a narrow tube is $Z = R + j\omega L_A$, where $R = 8\eta l / (\pi a^4)$ and $L_A = 4\rho l / (3\pi a^2)$.²

We extracted average values for l and a from Muren *et al.* (1986). The length l refers to the distance between the vestibule and the opening of the canal near the arcuate eminence (about one-third of the full revolution of the superior semicircular canal) and the radius a refers to the inner radius of the bony part of the semicircular canal interfacing the inner-ear fluid. Using $l = 1.44 \text{ mm} \times 120^\circ/360^\circ = 0.48 \text{ mm}$ and $a = 1.06 \text{ mm}/2 = 0.53 \text{ mm}$, we obtain values of $R_A = 1.4 \times 10^7 \Omega$ and $L_A = 7.3 \times 10^5 \Omega$ [our modeling results in Fig. 7(c) yielded $R_{\text{SCD}} = 2.5 \times 10^{10} \text{ Nsm}^{-5}$ and $L_{\text{SCD}} = 1.0 \times 10^7 \text{ Ns}^2\text{m}^{-5}$]. Compared to the experimental results, the values of the analytical solution are 2–3 orders of magnitude lower. We can obtain similar results to our model predictions when we reduce the diameter of the semicircular canal to an effective diameter of 20% of the anatomical measurements, yielding $R_A = 8.6 \times 10^9 \Omega$ and $L_A = 1.8 \times 10^7 \Omega$. Much of the area of the semicircular canal is taken up by the membranous labyrinth (especially near the crista), which we hypothesize will change the effective resistive losses and mass of the perilymph considerably.

FIG. 10. (Color online) Effect of SCD size on (a) stapes velocity (v_{Stapes}) and (b) RWM velocity (v_{RWM}) referenced to ear canal pressure (P_{EC}). The data are presented from one specimen with different SCD sizes.

V. CONCLUSIONS

At low frequencies and infrasound, the middle ear considerably limits the sound energy transmitted to the inner ear. In normal ears, SVTFs and RWM velocities increase by 20 dB/decade for frequencies between 1 and 200 Hz. At low frequencies, stapes and RWM velocities are remarkably similar across specimens (within 10 dB). When a dehiscence is introduced in the superior semicircular canal, stapes velocity is relatively unaffected, whereas RWM velocity decreases in a predictable, frequency-dependent manner, similar to the effect of SCD on P_{Diff} . With SCD, volume velocity is shunted away from the cochlea and toward the vestibular system to the opening of the semicircular canal when stimulated with sound at the ear canal. Thus, our results are consistent with classic SCD symptoms such as low-frequency hearing loss and/or sound-induced dizziness, also known as Tullio phenomenon. The effect of a SCD on RWM velocity is larger for lower frequencies, indicating that an opening in the bony labyrinth affects auditory and vestibular thresholds especially at infrasonic frequencies.

Our findings show that changing impedances in the inner ear can have a considerable effect on (infra)sound transmission through the auditory and vestibular system. Pathological mechanical changes can result in impedance changes that greatly affect sound transmission. These findings suggest that perhaps some individuals may have mechanical pathologies resulting in unusual susceptibility to infrasound sound transmission.

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APPENDIX: DETAILS REGARDING SOUND GENERATION AND MICROPHONE CALIBRATION

The loudspeaker (driver of a Beyer Dynamics DT 770 M) was removed from the headphone housing and mounted on a flat wooden board to limit the volume in front of the loudspeaker membrane to about 2 cm^3 (see Fig. 11). At the center of the wooden board, a hole was drilled to tightly attach a flexible polyethylene tube to deliver sound to the ear canal. The inner diameter of the polyethylene tube was 4 mm with a length of 2 in. In the experiment, the polyethylene tube that is attached to the loudspeaker on one end was sealed to the ear canal on the other end with mounting putty. During microphone calibration (Fig. 11), we simulated the ear canal volume with a body of a plastic syringe. The total volume of

FIG. 11. (Color online) Schematic of the setup to calibrate the probe tube microphone. In order to generate high-intensity low-frequency sound pressures with a conventional loudspeaker, the acoustic field has to be closed and the volume minimized. The total volume of the acoustic field consists of the volume in front of the loudspeaker membrane, the polyethylene (PE) tube connecting the loudspeaker to the ear canal, the ear canal volume (simulated by the body of a syringe), and the PE tube with adapter for the probe tube microphone.

the acoustic field (colored blue in Fig. 11) consisted of the ear canal volume, the volume in front of the loudspeaker membrane, the volume of the polyethylene tube connecting the loudspeaker to the ear canal, and the volume of another polyethylene tube used to connect the microphone to the ear canal.

The microphone was calibrated in a standard manner using a 2 cm^3 uniform tube (the body of a 6 ml Monoject plastic syringe, Medtronic, Minneapolis, MN) that simulated the ear canal (see Fig. 11). The polyethylene tubing coming from the loudspeaker was attached to one end of the syringe body and held in place with a perforated foam ear plug. The $1/4$ in. BK microphone was placed at the other end of the syringe body (reference location) where the tympanic membrane would be, leaving a total volume of 2 cm^3 between the microphone cartridge and the end of the polyethylene loudspeaker tubing. We perforated the wall of the syringe body near the microphone to attach another polyethylene tube with an inner radius of 1.5 mm and 2 in. in length (used in the actual experiment) at which end the probe microphone was located. We exchanged the position of the BK microphone from the reference location to the probe location but had another microphone cartridge at the other location to keep the acoustic field unchanged. Not surprising for the long wavelengths, we observed small differences (within 1 dB) between the two measurement locations for frequencies between 0.9 Hz and 2 kHz, which we accounted for by correcting the response of the probe-tube microphone by the calibration file.

¹Micro openings, such as the cochlear and vestibular aqueducts, can be neglected in AC stimulation as their impedance magnitude is much higher than the impedance of the oval window and round window.

² η = viscosity of water = $8.90 \times 10^{-4} \text{ Pa}$; l is the length of the narrow tube, a is the radius of the narrow tube, and ρ is the density of water = 1000 kg/m^3 .

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