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Measurements of Human Middle- and Inner-Ear Mechanics With Dehiscence of the Superior Semicircular Canal

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Abstract

Objectives—(1) To develop a cadaveric temporal-bone preparation to study the mechanism of hearing loss resulting from superior semicircular canal dehiscence (SCD) and (2) to assess the potential usefulness of clinical measurements of umbo velocity for the diagnosis of SCD.

Background—The syndrome of dehiscence of the superior semicircular canal is a clinical condition encompassing a variety of vestibular and auditory symptoms, including an air-bone gap at low frequencies. It has been hypothesized that the dehiscence acts as a “third window” into the inner ear that shunts acoustic energy away from the cochlea at low frequencies, causing hearing loss.

Methods—Sound-induced stapes, umbo, and round-window velocities were measured in prepared temporal bones (n = 8) using laser-Doppler vibrometry (1) with the superior semicircular canal intact, (2) after creation of a dehiscence in the superior canal, and (3) with the dehiscence patched. Clinical measurements of umbo velocity in live SCD ears (n = 29) were compared with similar data from our cadaveric temporal-bone preparations.

Results—An SCD caused a significant reduction in sound-induced round-window velocity at low frequencies, small but significant increases in sound-induced stapes and umbo velocities, and a measurable fluid velocity inside the dehiscence. The increase in sound-induced umbo velocity in temporal bones was also found to be similar to that measured in the 29 live ears with SCD.

Conclusion—Findings from the cadaveric temporal-bone preparation were consistent with the third-window hypothesis. In addition, measurement of umbo velocity in live ears is helpful in distinguishing SCD from other otologic pathologies presenting with an air-bone gap (e.g., otosclerosis).

Keywords

Air-bone gap; Conductive hearing loss; Semicircular canal dehiscence; Superior semicircular canal dehiscence

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Superior semicircular canal dehiscence (SCD) syndrome is a recently recognized entity where patients often present with vestibular symptoms typically provoked by sound and/or static pressure (1,2). As clinical experience with SCD accrues, it has become apparent that some patients with SCD present with unexplained air-bone gaps of 5 to 50 dB, with or without vestibular symptoms (3–6). Our investigations and those of others have shown that the clinical presentation in such cases can so closely mimic that observed in otosclerosis that surgical exploration and unsuccessful stapedectomy procedures have been performed mistakenly in some instances (3–5).

It is somewhat puzzling that an air-bone gap on audiometry can be produced by a pathologic condition affecting the inner ear rather than the middle ear. Definitive proof that the air-bone gap in these patients is indeed the result of the SCD is the closure of the gap when the dehiscence is repaired (3,7). Figure 1A shows a preoperative audiogram from a 35-year-old patient with SCD who presented with both vertigo and an air-bone gap. The SCD was repaired via a middle-fossa approach, wherein the SCD was plugged with bone wax and temporalis fascia. Figure 1B is an audiogram performed 3 months postoperatively showing closure of the air-bone gap.

Our research group and others have proposed a mechanism to explain the air-bone gap in SCD (1,2,5,8). The SCD is hypothesized to introduce a mobile “third window” into the inner ear (in addition to the oval and round windows). In the normal ear, air-conducted sound sets the stapes into back-and-forth motion as a result of the motion of the tympanic membrane, malleus, and incus (Fig. 2A). Because the normal vestibular system has no window, no sound-induced fluid motion is produced within the semicircular canals. In the presence of an SCD (Fig. 2B), it is hypothesized that a part of the fluid volume displaced by the oscillating stapes is shunted through the opened superior canal, resulting in measurable fluid velocity within the dehiscence. Inasmuch as the inner-ear fluid volume is displaced away from the cochlea, this results in a decrease in the sound activating the cochlea and an elevation of air-conducted hearing thresholds, which should manifest as a decrease in round-window velocity. The third-window hypothesis also predicts that the shunt path through the SCD will lower the inner-ear input impedance, which should result in increased stapes and umbo velocities.

The goals of the present study were (1) to develop a cadaveric temporal-bone preparation of SCD to allow us to investigate the pathways of air-conducted sound transmission in SCD and test the predictions of the third-window hypothesis and (2) to assess the potential clinical usefulness of measurements of umbo velocity using laser-Doppler vibrometry as a diagnostic test for patients with SCD who present with an air-bone gap.

MATERIALS AND METHODS

The present study was approved by the institutional review board of the Massachusetts Eye & Ear Infirmary.

Temporal-Bone Preparation

Eight fresh cadaveric temporal bones from donors aged 52 to 83 years were used. We used fresh cadaveric human temporal bones in this study because fresh bones have been shown to have similar mechanical properties to live ears (9–11). Donors had no evidence of otologic disease. The specimens were removed within 24 hours after death with a circular saw following the procedure described by Schuknecht (12). The bones were stored in normal saline at 5°C between measurements. The temporal-bone preparation is shown schematically in Figure 3. A canal wall-up mastoidectomy was performed. The facial recess was opened, and the oval and round windows were exposed via a posterior tympanotomy. The stapedius tendon was sectioned, and the mastoid segment of the facial nerve was removed. The external auditory canal was shortened to ~1 cm. A 5-mm-diameter opening was made in the anterior wall of the

external auditory canal to expose the umbo. This opening was tightly sealed with a transparent plastic cover slip and Vaseline during measurements. The superior semicircular canal was carefully “blue-lined” by drilling in the mastoidectomy cavity, leaving a thin piece of bone covering the superior semicircular canal. The mastoid cavity was left open to the atmosphere during all measurements. The temporal bones were periodically moistened by soaking in normal saline.

Measurement Setup

Sound-induced velocities of the stapes (V_s), umbo (V_u), round window (V_{rw}), and the fluid inside the SCD (V_{scd}) were measured with a laser-Doppler interferometer (Polytec, Inc., Model No. HLV-1000, CA, U.S.A.). Both magnitude and phase components of the velocity were measured. Small pieces of reflective tape (with dimensions of ~ 0.25 mm by 0.25 mm) were placed on the posterior crus of the stapes, the center of the round-window membrane, the umbo, and the surface of the fluid inside the SCD during velocity measurements to increase the signal strength of the reflected laser light. In cases where the natural reflection was strong enough, the velocity measurements were made directly without reflective tape. A logarithmic chirp stimulus (containing harmonic frequency components of 48 Hz to 12.5 kHz with a frequency^{-1/2} preemphasis) was presented to an earphone (Etymotic Research, Inc., Model No. ER-4, IL, U.S.A.) coupled to the residual ear canal using a standard otologic speculum. Ear-canal sound pressure (P_{ec}) was measured with a probe tube microphone (Etymotic Research, Inc., Model No. ER-7A) within 5 mm of the center of the tympanic membrane. All velocity measurements were normalized by P_{ec} .

We confirmed the fluid-filled integrity of the inner ear in each cadaveric temporal bone by demonstrating a half-cycle phase difference between V_{rw} and V_s below ~ 1 kHz in the predehiscence condition (13); all eight bones met this criterion. The measurement noise and artifact were also estimated (14). Data contaminated by noise and data above the frequencies where we expect a substantial variation in sound pressure between the microphone location and the tympanic membrane were omitted. The high-frequency limit of each measurement was set at the frequency where the distance between the probe tip and the center of the tympanic membrane was greater than 0.1 times the wavelength. The velocities and the ear-canal sound pressure measurements were averaged and stored using SYSid software (SYSid Industries, CA, U.S.A.). Post-measurement analysis was done using MATLAB software (The MathWorks, Inc., MA, U.S.A.).

Simulation of SCD

After making baseline measurements of V_s , V_u , and V_{rw} , a dehiscence was created near the anterior end of the superior semicircular canal, approximately 1 mm away from the ampulla. Drilling was performed while a layer of saline was present over the superior canal. Care was taken to ensure that the SCD was created in a manner that prevented introduction of air bubbles into the inner ear. The dehiscences varied in size among bones, with lengths ranging from 2 to 4.5 mm. The width of the dehiscence was limited by the 0.5-mm diameter of the canal. Measurements of V_s , V_u , and V_{rw} were repeated after creating the dehiscence. Fluid velocity in the SCD (V_{scd}) was also measured. After these measurements, the SCD was patched with dental or cyanoacrylic cement. Measurements of V_s , V_u , and V_{rw} were then repeated; and the velocity on the patch (at the same location where V_{scd} was initially obtained) was also measured. We also estimated noise and artifact levels (where artifact is sound-induced motion of the bone) by measuring the bony motion adjacent to the superior canal.

RESULTS

Fluid Velocity Inside the SCD

Figure 4 shows measurements of the sound-induced fluid velocity inside the dehiscence (V_{scd}) after introduction of an SCD. In all eight bones, when SCD was present, V_{scd} magnitude was 20 to 50 dB higher than the mean of the eight noise-artifact estimates over the frequency range of valid measurement (100–5,000 Hz). The magnitude of V_{scd} increased with frequency from 100 to 800 Hz and decreased at higher frequencies. When the SCD was patched, the velocity of the patch was similar in magnitude to the noise floor. The phase of V_{scd} showed a 0.1- to 0.4-period lag relative to sound pressure at frequencies below 500 Hz and a 1- to 1.5-period lag at frequencies above 3 kHz.

Round-Window Velocity

Figure 5A shows round-window velocity (V_{rw}) measurements in a representative bone at baseline, after creating an SCD, and with the SCD patched. The baseline V_{rw} magnitude increased with frequency up to ~1.2 kHz and decreased at frequencies above 1.2 kHz. The SCD induced a factor of 2 to 3 decrease in the magnitude of V_{rw} and an increase in V_{rw} phase of approximately 0.1 periods below 2 kHz, and these changes were reversed when the SCD was patched. Similar changes were observed in all eight bones. The SCD-induced change in V_{rw} (ΔV_{rw} , where the magnitude of ΔV_{rw} equals $20 \cdot \log_{10}[V_{rw_{SCD}}/V_{rw_{Normal}}]$, and the phase angle of ΔV_{rw} equals the angle with SCD minus the normal angle) in eight bones, as well as the mean and 95% confidence interval (CI) around the mean, are shown in Figure 5B. The magnitude of ΔV_{rw} in individual bones was reduced by as much as 22 dB and the phase tended toward +0.25 periods below 2 kHz. The mean magnitude of ΔV_{rw} was –5 dB between 0.5 and 2 kHz, decreasing below 500 Hz to –16 dB near 100 Hz. These changes are statistically significant ($p < 0.05$) below 2 kHz; the 95% CI does not include the zero line. ΔV_{rw} exhibits a statistically significant positive phase angle over the same frequency range. The low-frequency decrease in the magnitude of V_{rw} has similar features to the low-frequency hearing loss observed in patients with SCD (Fig. 1A).

Stapes Velocity

Figure 6A shows stapes velocity (V_s) measurements in a representative bone at baseline, after creating an SCD, and with the SCD patched. The baseline V_s magnitude increased with frequency up to ~1.2 kHz, and decreased at frequencies above 1.2 kHz. An SCD resulted in increased V_s magnitude below 4 kHz (with the largest changes at frequencies above 500 Hz) and caused small changes in V_s phase between 600 and 4,000 Hz. These changes were reversed when the SCD was patched. Similar changes were observed in all eight bones (Fig. 6B). The magnitude of ΔV_s in individual bones was generally between 0 and 10 dB below 4 kHz. The mean magnitude of ΔV_s was 3 to 5 dB below 4 kHz. The changes in V_s phase were not statistically significant. The increase in V_s magnitude is consistent with a decrease in inner-ear input impedance produced by the SCD shunt path.

Umbo Velocity

Figure 7A shows umbo velocity (V_u) measurements in a representative bone at baseline, after creating an SCD, and with the SCD patched. The baseline V_u magnitude increased with frequency up to ~1.2 kHz, was flat between 1.2 and 3 kHz, and decreased with frequency above 3 kHz. The SCD increased V_u magnitude below 1.5 kHz and decreased V_u magnitude from 1.5 to 3 kHz. The phase of V_u decreased slightly between 700 Hz and 2 kHz. These changes were reversed when the SCD was patched. Similar changes were observed in all eight bones (Fig. 7B). The mean magnitude of ΔV_u was +3 dB near 500 Hz and –2 dB near 2 kHz. The magnitude of ΔV_u was significantly different from zero between 500 Hz and 1 kHz.

Comparison of Our Results With Measurements in Patients

We have previously described measurement of umbo velocity (V_u) in patients with conductive hearing loss using laser-Doppler vibrometry (15–17). To date, we have made V_u measurements in 29 live ears (23 patients) with SCD and no previous middle-ear surgery. We have also measured V_u in 56 normal subjects (16). In Figure 8, we show the mean ΔV_u measured in the 29 live ears with SCD, which was computed by normalizing the V_u data in SCD patients by the mean V_u from 56 normal subjects, as well as the mean ΔV_u and its 95% CI in our cadaveric ears (from Fig. 7B). The mean ΔV_u in patients with SCD (solid curve with circles) was similar in magnitude to the SCD-induced change in V_u in cadaveric temporal-bone measurements; both increased with frequency below 700 Hz, peaked at 700 Hz, and decreased at frequencies between 700 and 2,000 Hz. The ΔV_u in SCD patients was within the 95% CI of the cadaveric temporal-bone measurements, except at 3 kHz. The phase change in SCD patients was also similar at low frequencies (<2 kHz) compared with the cadaveric temporal-bone measurements. At higher frequencies (2–4 kHz), the phase change in SCD patients was greater than that measured in cadaveric temporal bones.

DISCUSSION

Using the Cadaveric Human Temporal-Bone Preparation to Investigate Mechanics of Hearing Loss in SCD

Our temporal-bone preparation enabled us to make measurements of sound transmission through the middle and inner ears before and after creation of a controlled SCD. We used the round-window velocity measurements (V_{rw}) as an index of sound delivery to the cochlear partition; a decrease in V_{rw} after creating an SCD is analogous to the air-conduction hearing loss seen in an audiogram (13). The creation of an SCD resulted in a decrease in V_{rw} at low frequencies by as much as 22 dB, which is similar to the hearing loss observed in patients with an SCD (e.g., Fig. 1A). The observation that the low-frequency decrease in V_{rw} was reversible when the SCD was patched is also consistent with the improved hearing observed in patients after surgical repair of an SCD (Fig. 1B).

The dehiscences that we created in our temporal bones were all relatively similar in size (1 to 2.25 mm²) and were at similar locations (the anterior limb of the superior canal). It would be possible in future studies to use the temporal-bone preparation to systemically vary the size and location of the SCD in an attempt to determine if these parameters affect the resulting hearing loss. Similarly, the temporal-bone preparation could be used to explore the influence that other structural factors (such as the baseline input impedance of the inner ear, middle-ear impedance, etc.) may have on the SCD-induced hearing loss. Such future studies may shed light on the structural determinants of the variability in air-bone gaps that is observed clinically in ears with SCD.

There are some limitations to our temporal-bone preparation with respect to investigating the mechanics of hearing loss in SCD. We were unable to test for changes in the bone-conducted response to sound due to an SCD. Such investigations would need to be made on an intact skull using a mastoid stimulator for bone conduction stimuli, similar to what is done clinically in patients. Such measurements of bone conduction in a cadaveric setting pose practical difficulties that are difficult to overcome. Another potential limitation is the absence of brain and cerebrospinal fluid against the SCD. Despite these limitations, there are several similarities, described in the following sections, between our cadaveric temporal-bone measurements and findings in live SCD ears.

Comparison of Measurements in Cadaveric Temporal-Bone preparation to Predictions of the Third-Window Hypothesis

There Was Significant Sound-Induced Fluid Velocity in the SCD (V_{scd})—The SCD caused a measurable fluid velocity within the dehiscence (Fig. 4). The fact that V_{scd} was measurable indicates that volume velocity (hence, acoustic energy) was being shunted away from the cochlea and through the dehiscence, which is consistent with the third-window hypothesis. This sound-induced fluid movement within the superior semicircular canal provides an explanation of how an acoustic stimulus can elicit vestibular symptoms: the SCD-induced fluid movement within the superior semicircular canal stimulates the vestibular sensory end-organs in the ampulla, resulting in vestibular symptoms. Such activation is consistent with the characteristic vertical-torsional nystagmus reported in patients with SCD (1,2).

SCD Produced a Decrease in Round-Window Velocity (V_{rw})—In all eight bones, V_{rw} magnitude was decreased below 500 Hz in response to an SCD. This is consistent with the low-frequency hearing loss observed in patients with an SCD as shown in Figure 1A (3,5). The reduction in V_{rw} magnitude by an SCD is also consistent with the reduction in cochlear potential in response to an air-conducted stimulus by an SCD observed in chinchilla (18).

SCD Produced an Increase in Stapes Velocity (V_s) and an Increase in Umbo Velocity (V_u)—We observed a significant 3- to 5-dB increase in the mean ΔV_s below 3 kHz, and a significant 1- to 3-dB increase in mean ΔV_u at ~500 to 900 Hz in response to an SCD. The fact that ΔV_s is larger in magnitude than and of a somewhat different frequency dependence from ΔV_u may be a consequence of flexibility in the ossicular joints (19).

The ΔV_u in the present study was similar in magnitude and phase at low frequencies to the ΔV_u measured in patients with an SCD (Fig. 8). Some of the small differences at higher frequencies may arise from the differences in size and location of SCD between patients and temporal bones or from the differences in inner-ear input impedance between live and cadaveric ears. Nevertheless, the similarity of ΔV_u in the live and cadaveric measurements suggests that the cadaveric temporal-bone preparation can simulate SCD in patients and supports the notion that the cadaveric temporal bone is a valid model for studying the middle- and inner-ear mechanics in SCD.

Potential Clinical Usefulness of Measurements of Umbo Velocity as a Diagnostic Tool for SCD

We believe that V_u measurements can be helpful for the diagnosis of SCD in patients who present with an unexplained air-bone gap, an intact tympanic membrane, and an aerated middle ear. In particular, it is useful for differentiating otosclerosis from SCD. As previously noted, the clinical presentation of SCD can mimic that of otosclerosis; and SCD patients have, in some instances, undergone stapedectomy without improvement in their hearing (3–5). In otosclerosis, the V_u magnitude is decreased compared with the mean normal (15) because of an increase in inner-ear input impedance by fixation of the stapes footplate, whereas in SCD, the V_u magnitude is increased because of a decrease in inner-ear input impedance. Therefore, V_u measurements can be helpful in distinguishing between these two clinical conditions.

Although V_u measurements can help discriminate whether an air-bone gap results from stapes fixation or SCD, it alone cannot be used to diagnose SCD. This is because the variation in V_u measurements among normal subjects is larger than the 4-dB increase in V_u (compared with the mean normal) observed in patients with SCD. Thus, one cannot reliably distinguish between V_u in normal versus SCD ears in the absence of any additional diagnostic tests, such as an audiogram, acoustic reflex testing, vestibular-evoked myogenic potentials, or high-resolution

computed tomographic scans. However, in the presence of an air-bone gap, Vu measurement is an easy and effective way of differentiating between SCD and otosclerosis.

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A

B

FIG. 1.

(A), Preoperative audiogram of a 35-year-old woman with vertigo and left-sided conductive hearing loss due to an SCD. There is a low-frequency air-bone gap. (B), Postoperative audiogram of the SCD patient shown in A 3 months after a middle-fossa procedure wherein the SCD was repaired with bone wax and temporalis fascia. Note the resolution of the air-bone gap (courtesy of Dr. Dennis Poe).

FIG. 2.

(A), Schematic representation of air-conducted sound transmission in the normal ear. In the normal ear, air-conducted sound via the ear canal produces motion of the tympanic membrane and ossicles that sets the stapes into back-and-forth motion. The resulting pressure difference between the oval and round windows leads to motion of the cochlear partition, resulting in perception of air-conducted sound. Because the bony walls of the inner ear and the cochlear fluid are essentially incompressible, the net inward motion of the stapes is balanced by an equal outward motion of the round window. The endolymphatic compartment is shaded in *light gray*. The perilymphatic compartment is in *white*. (B), Schematic representation of the mechanism of hearing loss for air-conducted sounds due to SCD. In the presence of an SCD,

it is hypothesized that some fraction of the fluid volume displaced by the oscillating stapes is shunted through the dehiscence canal away from the cochlea, resulting in a decrease in the sound activating the cochlea and an elevation of air-conducted hearing thresholds. Thus, a decrease in round-window motion is predicted. The SCD is also predicted to lower the input impedance of the inner ear, which should result in an increased motion of the stapes and, in turn, an increased motion of the umbo.

FIG. 3.

Schematic drawing showing the temporal-bone preparation to explore the effects of SCD. A sound source was placed in the external auditory canal. An SCD was created in the anterior limb of the superior canal. Umbo velocity (V_u) was measured on the tympanic membrane via an opening in the anterior wall of the external auditory canal. Stapes velocity (V_s) and round-window velocity (V_{rw}) were measured through a posterior tympanotomy/facial recess approach. The velocity of fluid within the exposed dehiscence (V_{scd}) was also measured.

FIG. 4. Sound-induced fluid velocity within the SCD (V_{scd}), normalized by ear canal sound pressure in eight bones (*solid curves*): magnitude (top) and phase (bottom). The *dashed curve* is the mean velocity measured at the same location after the SCD was patched in the eight bones, and the *dotted curve* represents the mean noise level determined by measuring the sound-induced motion of the temporal bones.

FIG. 5.

(A), Round-window velocity (V_{rw}) normalized by ear-canal sound pressure in a typical bone in the baseline condition (solid curve), after creating a 2-mm² SCD (dot-dashed curve), and after the SCD was patched (dotted curve). Top: magnitude; bottom: phase. (B), Ratio of V_{rw} with SCD to baseline (ΔV_{rw}) in eight bones (dotted curves). Top: magnitude in dB; bottom: phase. The solid curve and shaded area are the mean and the 95% CI.

FIG. 6.

(A), Stapes velocity (V_s) normalized by sound pressure in a typical bone in the baseline condition (*solid curve*), after creating a 2-mm² SCD (*dot-dashed curve*), and after the SCD was patched (*dotted curve*). Top: magnitude; bottom: phase. (B), Ratio of V_s with SCD to baseline (ΔV_s) in eight bones (*dotted curves*). Top: magnitude in dB; bottom: phase. The *solid curve* and *shaded area* are the mean and the 95% CI.

A

B

FIG. 7.

(A), Umbo velocity (V_u) normalized by sound pressure in a typical bone in the baseline condition (*solid curve*), after creating a 2-mm² SCD (*dot-dashed curve*), and after the SCD was patched (*dotted curve*). Top: magnitude; bottom: phase. (B), Ratio of V_u with SCD to baseline (ΔV_u) in eight bones (*dotted curves*). Top: magnitude in dB; bottom: phase. The *solid curve* and *shaded area* are the mean and the 95% CI.

FIG. 8.

Ratio of mean Vu in 29 live ears with SCD normalized by the mean of 56 normal subjects (*solid curve with circles*), as well as the mean ΔVu and its 95% CI of cadaveric ears with SCD obtained from Figure 7B. Top: magnitude in dB; bottom: phase.