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Intracochlear pressures in simulated otitis media with effusion: a Temporal bone study

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Abstract

Hypothesis—Simulated otitis media with effusion reduces intracochlear pressures comparable to umbo velocity.

Background—Otitis media with effusion is a common cause of temporary hearing loss, particularly in children, that can produce conductive deficits of 30–40 dB. Previous studies have measured the effects of simulated effusion on ossicular mechanics; however, no studies have measured cochlear stimulation directly during effusion. Here, we compare pressures in the scala vestibuli and tympani to umbo velocity, before and after induction of simulated effusion in cadaveric human specimens.

Methods—Eight cadaveric, hemi-cephalic human heads were prepared with complete mastoidectomies. Intracochlear pressures were measured with fiber optic pressure probes, and umbo velocity measured via laser Doppler vibrometry (LDV). Stimuli were pure tones (0.1 – 14 kHz) presented in the ear canal via a custom speculum modified to accept speaker and microphone tubing, and sealed with a glass cover slip. Effusion was simulated by filling the mastoid cavity and middle ear space with water.

Results—Acoustic stimulation with middle ear effusion resulted in decreased umbo velocity up to ~26 dB, whereas differential pressure at the base of the cochlea, the driving force to the basilar membrane, decreased by only ~16 db.

Conclusion—Simulating effusion leads to a frequency-dependent reduction in intracochlear sound pressure levels consistent with audiological presentation and prior reports. Results reveal that intracochlear pressure measurements (P_{SV} & P_{ST}) decrease less than expected, and less than the decrease observed in the differential pressure (P_{Diff}). The observed decrease in umbo velocity is greater than in the differential intracochlear pressures, suggesting that umbo velocity

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overestimates the induced conductive hearing loss. These results suggest that an alternate sound conduction pathway transmits sound to the inner ear during effusion.

Keywords

intracochlear pressure; otitis media with effusion; middle ear mechanics

Introduction

Otitis media with effusion (OME) is characterized by the accumulation of serous or mucoserous fluid within the middle ear cleft and is considered the most common cause of fluctuating conductive hearing loss (CHL). Otitis media occurs with a peak incidence at the age of 2–3 years (1,2), with a point prevalence on screening tests of about 20%, and experienced by 90% of children at least once by the age of three, but is not considered uncommon in adults (3,4). Several factors contribute to the incidence of OME including recurrent acute otitis media, allergies, adenotonsillar disease, poor Eustachian tube function and inadequate middle ear ventilation (5). OME results in a mild to moderate conductive hearing loss of 15 to 50 dB between 500–4000 Hz (6). Notably, the impact of such conductive hearing loss can have long-lasting effects after resolution of the acute fluid accumulation: chronic OME in children has been associated with long-term auditory processing disorders, and can interfere with both speech and language development in affected children (7–9).

Recently, several studies have investigated the effects of middle ear effusion on the sound transmission through the ossicular chain. In particular, studies have investigated changes to umbo velocity (using laser Doppler vibrometry; LDV) in cadaveric human temporal bones while gradually filling of the middle ear cavity with fluid of different viscosities. Maximum reduction in umbo velocity was observed at high frequencies (>500 Hz) when the middle ear cavity was completely filled with fluid, and interestingly the effect size was not affected by the fluid viscosity (10,11). Similarly, measurements of cochlear microphonic in chinchillas revealed that thresholds increased by 20–40 dB across the range of frequencies tested, umbo velocities decreased by 15–35 dB, and once again the increases in threshold were directly proportional to the amount of fluid in the middle ear and not affected by the viscosity of the fluid (12,13). Similar experiments in guinea pigs measuring basilar membrane motion with LDV revealed a reduction of 25 dB at all tested frequencies (14). In sum, previous studies have described conductive losses associated with middle ear effusion, but with conflicting reports of the frequency-dependence and magnitude of the induced conductive loss. This is in part because no study has been able to comprehensively study ossicular chain movement and intracochlear pressures in a human model. In this study, we seek to bridge this gap by measuring cochlear stimulation directly, simultaneously measuring ossicular chain motion with intracochlear pressures in the scala vestibuli (SV) and scala tympani (ST), with and without effusion. Measurements are made in a cadaveric human model to provide a coherent description of mechanical effects of effusion on middle ear transduction.

Methods

Temporal Bone Preparation

Eight fresh-frozen hemi-cephalic cadaveric human heads, with no history of ear disease or previous ear surgery, were used for this study (Lone Tree Medical, Littleton, Co, USA). The use cadaveric tissues was conducted under the oversight of the University of Colorado Anschutz Medical Campus Biosafety Committee, and the Colorado Multiple Institutional Review Board (COMIRB Exempt #14-1464).

The methods for preparation and examination are described in detail in previous studies from our group, and are only briefly described here (15–20). First, a microscopic examination of the tympanic membrane was completed in order to screen subject for perforation or evidence of previous middle ear disease. A complete cortical mastoidectomy was then performed, exposing surgical landmarks including the attic, the body of the incus, lateral canal, sinus plate, tegmen plate, and the digastric region. An extended facial recess approach then exposed the stapes, the cochlear promontory, and round window niche. The round window was examined to ensure integrity, and the false membrane removed if present. The Eustachian tube was occluded with cyanoacrylate adhesive at its nasopharyngeal opening to prevent water from leaking out of the middle ear cavity during experimental procedures. A 0.5 mm diamond burr was used to blue-line the SV and ST near the oval and round windows.

The specimen was fastened to a stainless-steel baseplate with locking screws to maintain position. Cochleostomies were made in each scala with a fine pick, under water to prevent air leakage into the cochlea. Pressure sensors (FOP-M260-ENCAP; FISO Inc., Québec, Canada) were inserted into the cochlea (P_{SV} and P_{ST}) under water (to prevent air infiltration), and mounted to the specimen with stainless steel guide tubes attached to the skull with hydroxyapatite dental cement. The cochleostomies were sealed using alginate dental impression material (Jeltrate; Dentsply International Inc., York, PA, U.S.A.). Figure 1 shows an illustration (A) and two photographs (B–C) demonstrating the modified speculum and the specimen preparation, with the cochlea, round window, and blue lining for both the scala tympani and vestibuli visible through the facial recess. In particular, Figures 1A–B demonstrate the approximate location of the stainless steel cannula relative to the ear canal, mastoidectomy, and the acrylic cover plate, which was used to fill the middle ear with water to simulate effusion. Care was taken when creating cochleostomies to ensure that the openings were as small as possible while still allowing insertion of pressure probes, and during alginate application to ensure that the minimum amount necessary to seal the cochlea was applied (see small white dome of alginate on the cochlear promontory surrounding the base of each pressure in Figure 1C) to ensure the alginate did not interfere with middle ear function. Pressure probes were inserted until the probe tip was just within the scala wall (~100 μ m), and stapes and round window velocities compared before and after insertion to ensure that cochlear input impedance was not altered (21). At the end of each experiment, the cochlea was dissected to ensure proper placement of the cochlear pressure probes within each scala.

Stimulus Presentation and Data Acquisition

All experiments were performed in a double-walled sound attenuating chamber (IAC Inc., Bronx, NY, U.S.A.). Acoustic stimuli were presented using a stainless-steel speculum (Fig. 1) customized with two sections of tubing: 1) a horizontal 3 mm probe tube for the speaker and 2) a right angle 2.5 mm probe tubing for the microphone that extends 5 mm beyond the speculum edge for optimal placement (~1 mm from the tympanic membrane). A round microscope cover glass was attached at an angle inside the large end of the speculum, and held in place with a bead of epoxy resin around the circumference to acoustically seal the ear canal. The speculum was inserted into the external ear canal and fixed in place with cyanoacrylate adhesive.

A loudspeaker (Tucker Davis Technology (TDT), Alachua, FL, USA) was paired to the speculum via silastic tubing. Tone stimuli were generated by custom written MATLAB software (MathWorks Inc., Natick, MA, USA) from 100 Hz to 14 kHz in 1/4th octave steps. A probe tube microphone (type 4182; Bruel & Kjaer, Nærum, Denmark) was similarly attached to the speculum to calibrate the ear canal stimulus intensity close to the tympanic membrane. Stimuli were generated, and responses recorded, with an external sound card (Hammerfall Multiface II, RME, Haimhausen, Germany).

Umbo, Stapes and RW Velocity Measurements

Microscopic retro-reflective glass beads (P-RETRO 45–63 μm dia., Polytec Inc., Irvine, CA) were placed on the stapes capitulum just above the insertion of the stapedius tendon (white mass visible in Figure 1C), on the TM at the umbo, and on the center of the RW membrane (white dot visible in Figure 1C) to enhance the reflected signal of the LDV laser (Model No. HLV-1000 with CLV-700 head; Polytec Inc., Waldbronn, Germany). The LDV controller sensitivity was typically set to 10 mm/s/V, and the input was band-pass filtered between 100 Hz and 30 kHz. The LDV beam was positioned via a joystick-controlled aiming prism (HLVMM2; Polytec Inc.); the beam was visualized using a Zeiss microscope onto which the LDV head was attached.

Creation of Stimulated Effusion Model

A transparent acrylic plate was fashioned with a semicircular edge, and fixed in place over the mastoidectomy using alginate dental impression material (Jeltrate; Dentsply International Inc., York, PA, U.S.A.) to completely close the mastoid cavity. At least two milliliters (22) of water was inserted into the middle ear through a stainless-steel cannula (Figure 1A–B) fixed in place with dental cement, to ensure the complete filling of the middle ear cavity and absence of any entrapped air. Pressure probe guide tubes were likewise sealed with dental impression material. A sealed effusion model was created by occluding any remaining mastoid air cells with hydroxyapatite bone cement (the majority were removed with the surgical drill), and completely filling the mastoid cavity with water. The middle ear was inspected visually to ensure there were no air bubbles present prior to making measurements in the effusion condition.

Data Processing and Analysis

All acquired signals were band-pass filtered between 15 Hz and 15 kHz with a second-order Butterworth filter for data analysis. Responses were calculated from the average of at least 3 repetitions, and analysis was completed only for recordings with a signal-to-noise ratio greater than 3 dB to ensure adequate signal was present in each signal (signal-to-noise ratios were typically greater than 10 dB).

Data collected from stapes and umbo velocities ($V_{\text{Stapes/Umbo}}$) and intracochlear pressures ($P_{\text{SV/ST}}$) are presented as acoustic transfer functions ($H_{\text{Stapes/Umbo/SV/ST}}$), which quantify the relationship between a given sound stimulus in the ear canal (i.e. P_{EAC}), and the recorded output. Briefly, transfer functions (H) were computed from the RMS amplitude of LDV measurements (V) for stapes ($H_{\text{Stapes}} = V_{\text{Stapes}}/P_{\text{EAC}}$) and umbo ($H_{\text{Umbo}} = V_{\text{Umbo}}/P_{\text{EAC}}$) velocities, and sound pressure levels (P) for scala vestibuli ($H_{\text{SV}} = P_{\text{SV}}/P_{\text{EAC}}$) and scala tympani ($H_{\text{ST}} = P_{\text{ST}}/P_{\text{EAC}}$), normalized to the sound pressure level in the External Auditory Canal (P_{EAC}). Differential pressure, which is correlated with auditory nerve function and is considered to be the driving force to the basilar membrane (21,23), is calculated as the complex difference between the pressures recorded in scala vestibuli and tympani ($P_{\text{Diff}} = P_{\text{SV}} - P_{\text{ST}}$), and the differential pressure transfer function computed as above ($H_{\text{Diff}} = P_{\text{Diff}}/P_{\text{EAC}}$). In summary figures below, responses are shown as the mean $\pm$ the standard error of the mean (SEM) of the samples (i.e. specimens) recorded, in order to illustrate the range within which we expect the actual mean of the population exists. The SEM is exactly one half the 95% confidence interval (CI_{95}), thus the spread of responses within the samples recorded may be readily estimated by the reader.

The change in umbo velocity (V_{Umbo}) and intracochlear pressures ($P_{\text{SV/ST}}$) from the experimentally induced effusion was grouped into 3 frequency bands for statistical analysis: low (< 0.4 kHz), mid (0.4–2 kHz), and high (>2 kHz.). Statistical analyses were completed using functions in the Statistics and Machine Learning toolbox in MATLAB (R2014b; The MathWorks, Inc., Natick, MA, U.S.A.). A 2-way repeated measures analysis of variance (ANOVA) was used to compare the response across the four measurements, and three frequency bands, where a p-value less than 0.05 is considered significant. The change in response with effusion relative to baseline was assessed with a paired samples Student's t-test, with a Bonferroni correction for (12) multiple comparisons ($\alpha = 0.0042$).

Results

Baseline Acoustic Stapes Velocity Transfer Functions

Baseline stapes velocity transfer functions were collected prior to making cochleostomies for pressure probe insertion (Figure 2), and compared to the range of responses reported for normal, healthy, human temporal bones (24). Temporal bones showing H_{Stapes} magnitudes with a majority of responses outside the reported 95% CI were excluded from further analysis. After pressure probes were inserted and sealed into both scalae, stapes velocity transfer functions were repeated and again confirmed to fall within the range of normal responses. Note, absolute transfer function magnitudes could vary somewhat between measurements due to differences in the orientation of the LDV laser, but this change was not

used as an exclusion criteria so long as the responses remained within the 95% CI range observed previously (24). Likewise, H_{Stap} magnitudes are somewhat higher than the 95% CI range for high frequencies (> 2 kHz), indicating higher than expected stapes velocities; this difference may be due to making measurements on the stapes head rather than the stapes footplate (which is not readily visible in our preparation), thus may include the effects of alternate vibrational modes (i.e. rocking as well as piston-like motion) of the stapes, and was not used as the basis for exclusion. Finally, round window and stapes velocities were compared to ensure anti-phasic motion at low frequencies, both before and after intracochlear pressure probe insertion (not shown). Eight (out of 12) specimens met all criteria and were included in the study.

Following pressure probe insertion, baseline umbo velocity and intracochlear pressure measurements were obtained. Figure 3 shows baseline transfer function magnitudes for V_{Umbo} , P_{SV} , P_{ST} , and P_{Diff} , superimposed onto the range of responses reported for normal, healthy, human temporal bone measurements of umbo velocity (10), and each of the intracochlear pressures (21). Note, V_{Umbo} transfer function magnitudes (H_{Umbo}) appear somewhat more variable in the current dataset than predicted based on the literature values (10), potentially due to imprecise aiming of the LDV laser, as measurements made on the TM off of the umbo may show substantially higher or lower amplitude vibrations in response to acoustic stimulation than expected. For this study, we were not concerned about precise placement on the umbo, instead focusing on the change in amplitude due to induction of the simulated effusion. For this reason the laser placement and orientation were held constant between baseline and effusion measurement conditions.

Effect of Simulated Effusion on Umbo Velocities and Intracochlear Pressures

All measurements were performed both before and after water was introduced to fill the middle ear cleft, simulating an effusion. Figure 4 (top) shows mean ($\pm$ SEM) transfer function magnitudes for both baseline (i.e. data presented in Fig. 3), and simulated effusion conditions. Responses of H_{Umbo} , H_{SV} , H_{ST} , and H_{Diff} , are once again shown superimposed onto the range of normal responses reported in the literature (10,21). In each measure, responses are substantially lower in the effusion than baseline measurements, except at the lowest frequencies (< 200 Hz). The effect of middle ear effusion is particularly notable for moderate frequencies (~ 400 Hz – ~ 3 kHz), and generally peaks at ~ 2 kHz.

The change in transfer function magnitudes are shown in the bottom row, with shows the mean ($\pm$ SEM) transfer function magnitude gain of inducing the effusion (in dB re: baseline). As indicated above, the change in transfer function magnitude is largest between ~ 400 Hz and ~ 3 kHz, and generally showed a large (> 10 dB) decrease in transfer function magnitude. H_{Umbo} showed the largest decline, showing a decrease of ~ 25 dB relative to baseline, whereas the intracochlear pressures showed more moderate decreases (approximately -10 to -15 dB re: baseline).

Pressure and Velocity Changes with Simulated Effusion Relative to Baseline Measures

To compare responses across measures, we calculated the average (mean $\pm$ standard deviation) change in transfer function magnitude for each measure within specific frequency

bands (Table 1). Results reveal small decreases in each measure for low frequencies (< 400 Hz), larger decreases for mid frequencies (> 400 Hz & < 2 kHz), and intermediate decreases for high frequency stimuli (> 2 kHz). This attenuation was largest in umbo velocity, and greater in scala vestibuli than in the scala tympani. Responses were compared in two ways.

First, responses were compared with a 2-way ANOVA with measurement and frequency band as independent variables, and change in transfer function magnitudes as dependent variables. Results reveal significant main effects of both measurement ($F_{3,612} = 21.37$; $p = 3.56 \times 10^{-13}$) and frequency ($F_{2,612} = 65.85$; $p = 1.24 \times 10^{-26}$), as well as a significant interaction ($F_{6,612} = 8.26$; $p = 1.30 \times 10^{-8}$). Post-hoc multiple comparisons testing with a Tukey Honest Significant Difference (HSD) test reveals significant differences between V_{Umbo} and each of the three intracochlear pressures (but not between each other), and between responses in each of the three frequency bands.

Second, responses of each individual measurement condition are assessed for a significant change between baseline and effusion conditions with a paired Student's t-test, accounting for multiple comparisons with a Bonferroni correction ($\alpha = 0.0042$). None of the four measures showed a significant change from baseline in the low-, all four measures showed a significant change in the mid-, and all measures except the P_{Diff} showed a significant change from baseline in the high-frequency band (indicated in Figure 4 and Table 1 with *).

Discussion

Otitis media with effusion occurs due to accumulation of fluid in the middle ear in the absence of bacterial or viral infection, and is the most common cause of CHL in school aged children worldwide (6). The principal cause of this CHL in OME is impairment of the middle ear transduction mechanism (13). The purpose of this investigation was to elucidate the effect of middle ear effusion on the transmission of sound energy to the inner ear by measuring the difference in sound pressure level across the cochlear partition.

Temporal bone model of experimental effusion

In this investigation, we used water to approximate an effusion, similar to previous studies performed in our lab and others (10–12). We filled the entire mastoid cavity with fluid in order to achieve the maximum conductive loss observed in those studies, across all frequencies. A similar set of experiments in human cadaveric temporal bones showed that umbo velocities are reduced to the greatest degree at high frequencies (>1 kHz) when the ear is completely filled with fluid (10), and that this reduction may be reduced by re-introduction of air into the middle ear cavity (25). These reports suggest that changes in umbo velocity are directly related to the proportion of the middle ear filled with fluid. Consistent with this report, we observed no significant reduction in umbo velocities at the lowest frequencies (below ~400 Hz).

At higher frequencies, the change in umbo velocity is positively correlated with the degree by which the fluid contacts the TM (10). In our data, the effect of effusion on umbo velocity is large at mid to high frequencies (with a peak of 26 dB between 0.4–2 kHz), similar to the peak reduction reported previously in human cadavers, where the most significant reduction

was above 800 Hz (10), as well as the attenuation observed in animal studies (12–14). However, intracochlear responses showed lower changes in response magnitude, and were relatively unaffected by effusion at high frequencies. Umbo velocity thus appears to overestimate both the magnitude and range of frequencies affected by effusion.

Changes in cochlear input relative to hearing changes

In general, our results are consistent with studies examining changes in behavioral hearing thresholds associated with OME, suggesting that our model faithfully recreates the CHL expected from OME. In particular, a large study of pure tone thresholds in school children showed an average conductive hearing loss of 25 dB, occurring mainly in the mid frequency range (26). Similarly, another study reports CHL values between 15 to 50 dB at frequencies between 500 Hz - 4 kHz (6). The current results somewhat underestimate the magnitude of changes reported during effusion, which may be a result of incomplete filling of the middle ear cavity with water, but otherwise show qualitatively similar results, including recapitulating the frequency dependence of hearing loss due to OME.

Effects of effusion on intracochlear pressures

Results reveal a somewhat larger reduction in sound pressure level in scala vestibuli than scala tympani, where effusion reduces mean values by ~5–15 dB and ~5–10 dB, respectively, for frequencies over 800 Hz (peaking at ~12 dB and ~10 dB, respectively, at ~2 kHz). Interestingly, although sound pressure levels decreased in both scalae, a somewhat larger reduction is noted in the differential pressure than either scala individually, where effusion reduced sound pressure levels between ~10–15 dB at the same frequencies (peaking at ~16 dB at ~2 kHz). Differential intracochlear pressure correlates well with ossicular chain motion in normal hearing ear, and is considered a direct measure of the drive to the basilar membrane (21,23), thus provides a convenient metric by which to assess conductive hearing loss (20,27,28). Effusion resulted in a decrease in umbo velocity of up to ~35 dB, whereas the differential intracochlear pressure revealed a decrease of only ~15 dB, suggesting changes in umbo velocity overestimates the CHL induced by middle-ear effusion. This is consistent with a previous study from our laboratory that revealed stapes velocities are reduced by only 4–10 dB during effusion (29). Similarly, another study using a 3D finite element model of ossicular chain motion also showed less reduction of stapes than umbo velocities (especially at frequencies >5 kHz), which may be attributed to the low compressibility, and thus introduction of an alternate sound conduction pathway through water (30).

To the best of our knowledge, this report represents the first measurements of intracochlear pressures in the presence of middle ear fluid; however, several prior studies have been conducted in animal models to determine the effect of such effusions on mechanical and physiological responses. One study measured basilar membrane mobility at both the apical and basal turns of the cochlea in guinea pig (14). A mucopolysaccharide was injected into the middle ear of the animals, effusion was confirmed by both tympanometry and otoscopic examination, and measurements were carried out at 3 and 14 days to resemble the acute and chronic state, respectively. Results revealed a reduction of basilar membrane mobility of up to 16 dB in the apical turn of the cochlea in the region associated with sensitivity to 200–600

Hz, and a reduction of 10 dB in the basal turn in regions associated with 5–9 kHz and 11–13 kHz. This change in the basilar membrane mobility is notably lower than the observed reductions of up to 20 dB in umbo velocities described in the same report (10,14). The results of this study closely resemble the ~15 dB reduction in differential pressure and ~25 dB attenuation of umbo velocities we observed, thus supporting our proposition that umbo velocity overestimates the effects of effusion.

In contrast, results from animal studies measuring auditory neurophysiological measures of sound transmission are equivocal on the effects of effusion on sound transmission. A study on chinchillas assessing the effects of experimentally-induced effusion on tympanic membrane mobility showed a greater decrease in cochlear microphonic amplitudes (20–40 dB) than in umbo velocities (15–35 dB), particularly for mid and high frequencies (12). Similarly, two studies showed a greater reduction of auditory brainstem response (ABR) amplitudes than umbo velocities, particularly for high frequency stimuli; however, a separate investigation on mice showed opposite results: greater reductions in umbo velocities than in ABR amplitudes (31–33). The correlation between umbo velocity, intracochlear pressures, basilar membrane motion, and auditory transduction (either cochlear microphonic or ABR) thus remains unclear, and additional studies investigating the relationships between these measures are necessary.

Limitations of the study

Although alterations in ossicular chain motion play a major role in driving auditory transduction during normal, air-conducted sound transmission, other sound transmission pathways, such as bone-conduction (34), as well as the effects of middle ear nonlinearities (15), must be taken into account. While these pathways are normally dominated by the air-conduction pathway, our current results suggest that the presence of an effusion may create new transmission pathways (e.g. by direct coupling of the fluid to the round window) that significantly complicate the patterns of stimulation in the cochlea. Additionally, OME is a complex pathophysiologic process that is not limited to the presence of fluid in the ear but may also include negative middle ear pressure, and anatomical changes involving the ossicles, middle ear cleft, the tympanic membrane, and/or the round window membrane. All of those may alter material properties and in turn middle ear transmission. The current study clearly cannot address changes in material properties of the middle ear, particularly those resulting from chronic OME, thus interpretation of the results should be limited to an acute effusion.

One potential limitation in the methods used in this study is the use of water rather than saline to simulate the effusion. Since water is hypotonic, prolonged contact between the water and the middle ear may alter the material characteristics of the soft tissue; however, we believe this is a minimal risk due to the details of our temporal bone preparation. Historically, temporal bone preparations involved removal of the majority of the skull and skin leaving the bone susceptible to drying out (especially under hot surgical lights), thus it is critical to periodically moisten the bone with saline to maintain normal physiological conditions (35). Here, we use a hemi- or full-cephalic head preparation in which the skull and soft tissue is left intact (except for the pinna and skin immediately posterior, which is

removed in order to access the mastoid surface). This preparation is much less susceptible to drying out, since the soft tissue holds moisture and the skin forms a hermetic seal with the skull, and while we have not made a systematic investigation, we have not noted any substantial changes in response characteristics over the duration of a typical experiment (8–12 hours).

A second methodological limitation lies in our inability to ensure a complete evacuation of air in the middle ear. The issue of complete filling the middle ear with fluid is complicated, and was addressed in detail previously (10). In particular, the mastoid air cells represent a significant air-filled cavity, that are very difficult to replace with fluid due to the high surface area of the air cells. We took several measures to minimize these effects. First, the surgical approach removes the majority of air cells, thus reducing the potential volume of air trapped in the middle ear cavity. Second, the middle ear was visually inspected for air bubbles prior to making measurements (although we acknowledge some areas of the middle ear are not visible). Finally, prior measurements in our lab suggest that it is the proportion of the TM in contact with fluid that is the primary determinant of the signal attenuation (Thornton et al. 2013). These measures produced reductions in umbo velocity that were similar, though somewhat lower than in previous reports (10); however, this difference does not change the conclusion that umbo velocity overestimates the conductive loss provided by a complete middle ear effusion.

Conclusions

The current study represents the first description of the change in the driving force to the cochlea during middle ear effusion.

- Differential intracochlear pressure displayed somewhat lower attenuation than umbo velocity (~15 dB vs ~25 dB, respectively), suggesting umbo velocity overestimates the expected conductive hearing loss.
- The ~15 dB change in differential intracochlear pressure is consistent with the change in basilar membrane motion reported in guinea pigs (14), and the ~25 dB change in umbo velocity is consistent with a prior report in human cadaver (10).
- The decrease in differential pressure is substantially larger than the decreases in either scala vestibuli or scala tympani pressures, suggesting a complex stimulation pattern is introduced during middle ear effusion.
- Our results are consistent with previous results in animal models, and with clinical data characterizing hearing loss due to otitis media with effusion with maximum peak conductive hearing loss between 0.5–2 kHz (10,14).

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Figure 1.

A) Illustration of the experimental setup and measurement procedures. Each ear was prepared with a mastoidectomy, atticotomy, and extended facial recess to expose the cochlear promontory and ossicular chain. Stainless steel guide tubes for P_{SV} and P_{ST} fiber optic pressure sensors, and a stainless -steel cannula for fluid insertion, were implanted in dental cement in appropriate orientations to access appropriate middle ear structures. A stainless steel aural speculum was modified to accept speaker and microphone probe tubing, and the opening covered with a microscope cover glass to provide a sealed acoustical environment. Umbo velocity was measured with an LDV directed through the cover glass. B) photograph of the speculum, microphone, speaker, and cannula tubing, relative to the ear canal and mastoidectomy. C) photomicrograph showing the intracochlear pressure probe (P_{SV} & P_{ST}) locations, relative to the stapes and round window. Retroreflective glass beads are visible on the stapes, and alginate dental impression material is visible at the base of each pressure probe.

Figure 2.

Baseline stapes velocity acoustic functions ($H_{\text{Stapes}} = V_{\text{Stapes}}/P_{\text{EAC}}$) for each specimen. Gray bands represent the range of responses reported previously (24).

Figure 3.

Baseline acoustic function magnitudes for individual specimen, calculated for umbo velocity, scala vestibuli pressure, scala tympani pressure, and the differential intracochlear pressure. Gray bands represent the range of responses reported previously (H_{Umbo} (10); $H_{SV/ST/Diff}$ (21)).

Figure 4.

Top: Mean ($\pm$ standard error of the mean, SEM) transfer function magnitudes (i.e. response magnitude normalized to sound pressure level in the ear canal) recorded before (Baseline) and after insertion of water into the middle ear (Effusion), as a function of stimulation frequency. Responses are superimposed onto the range of responses observed in prior literature (same as in Fig. 3). Bottom: the change in response magnitude following insertion of water into the middle ear (in dB re: Baseline). Frequency bands assessed in statistical comparisons are indicated at the top of the each axis, and significant changes during effusion relative to baseline (assessed with Student's t-tests) are indicated with an asterisk.

Table 1

Summary of mean ($\pm$ standard deviation) change in transfer function magnitude for each 469 measurement, across frequency bands.

Result (dB re: Baseline)	Frequency Band		
	Low (0.1–0.4kHz)	Mid (0.4–2kHz)	High (>2kHz)
Umbo velocity	-1.0 ± 8.1	-18.8 ± 11.1 *	-13.5 ± 12.3 *
Scala vestibuli pressures	-1.4 ± 6.2	-9.8 ± 6.3 *	-6.1 ± 9.0 *
Scala tympani pressures	-1.2 ± 7.9	-5.5 ± 8.4 *	-4.8 ± 6.7 *
Differential pressure	-2.4 ± 6.6	-11.6 ± 11.3 *	-0.7 ± 14.3

Student's t-test: * $p < 0.0042$ 470