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Air- and Bone-Conducted Sources of Feedback with an Active Middle Ear Implant

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Abstract

Objectives: Active middle ear implants (AMEI) have been used to treat hearing loss in patients for whom conventional hearing aids are unsuccessful for varied biologic or personal reasons. Several studies have discussed feedback as a potential complication of AMEI usage, though the feedback pathway is not well understood. While reverse propagation of an acoustic signal through the ossicular chain and tympanic membrane constitutes an air-conducted source of feedback, the implanted nature of the device microphone near the mastoid cortex suggests that bone conduction pathways may potentially be another significant factor. This study examines the relative contributions of potential sources of feedback during stimulation with an AMEI.

Design: Four fresh-frozen, hemi-sectioned, human cadaver specimens were prepared with a mastoid antrostomy and atticotomy to visualize the posterior incus body. A Carina™ active middle ear implant actuator (Cochlear Ltd., Boulder, CO, U.S.A.) was coupled to the incus by two means: (1) a stereotactic arm mounted independently of the specimen and (2) a fixation bracket anchored directly to the mastoid cortical bone. The actuator was driven with pure-tone frequencies in 1/4 octave steps from 500–6000 Hz. Acoustic sound intensity in the ear canal was measured with a probe-tube microphone (Brüel & Kjær, Naerum, Denmark). Bone-conducted vibration was quantified with a single-axis laser Doppler vibrometer (Polytec Inc., Irvine, CA, U.S.A.) from both a piece of reflective tape placed on the skin overlying the mastoid and a bone-anchored titanium screw and pedestal (Cochlear Ltd., Centennial, CO, U.S.A.) implanted in the cortical mastoid bone.

Results: Microphone measurements revealed ear canal pressures of 60–89 dB SPL, peaking in the frequency range below 2 kHz. Peak LDV measurements were greatest on the mastoid bone (0.32–0.79 mm/s with mounting bracket and 0.21–0.36 mm/s with the stereotactic suspension); peak measurements on the skin ranged from 0.05–0.15 mm/s with the bracket and 0.03–0.13 mm/s with stereotactic suspension.

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Conclusion: AMEI produce both air- and bone-conducted signals of adequate strength to be detected by the implanted device microphone, potentially resulting in re-amplification. Understanding the relative contribution of these sources may play an important role in the development of targeted mitigation algorithms, as well as surgical techniques emphasizing acoustic isolation.

Keywords

bone conduction

Introduction:

Active middle ear implants (AMEI) have successfully treated patients with both sensory and conductive hearing losses and are potentially useful for patients in whom conventional hearing aids are not successful (Boeheim et al. 2010; Wagner et al. 2010; Zenner & Rodriguez Jorge 2010; Butler et al. 2013; Beleites et al. 2014; Carlson et al. 2014; Bruschini et al. 2016; Zwartenkot et al. 2016). Though direct comparison between conventional hearing aids and AMEI is difficult due to lack of both objective fitting parameters and a strong common metric, some research suggests that patients perform at least equally as well (Sava et al. 2016), if not better (Boeheim et al. 2010), with these surgical devices.

Feedback is described as a “whistling” noise that is generated by an acoustic amplification system in which the output from a speaker is picked up by the microphone and undergoes repeated re-amplification. Several conditions are required for feedback to be generated, including an additive phase relationship between the input and output. Additionally, the amplification provided by the system must be larger than the attenuation of the amplified signal from its point of output to the microphone input. Like conventional hearing aids, AMEI are susceptible to leakage of stimulus back to the microphone, resulting in feedback (Pulcherio et al. 2014). Several studies have discussed feedback as a complication of this type of device for some patients (Jenkins et al. 2007a; Bruschini et al. 2009, 2010, 2016; Zenner & Rodriguez Jorge 2010; Kam et al. 2012; Pulcherio et al. 2014; Lefebvre et al. 2016), though the pathway for feedback is not completely understood. While reverse propagation of an acoustic signal through the ossicular chain and tympanic membrane can create an air-conducted source of feedback, the implanted AMEI microphone position near the mastoid cortex suggests that the bone conduction pathway might also contribute to the generation of audible feedback.

We hypothesize that AMEI generate both air and bone-conducted signals of a sufficient level to generate feedback (see Figure 1 for an illustration). Air-conducted feedback (Fig 1A) is generated either by: (1) reverse transmission of signal through the ossicular chain and eardrum, contributing to the movement of the column of the air in the ear canal or (2) osseotympanic bone-conducted movement of the column of air in the ear canal and inertial bone-conductive movement of the ossicular chain with propagation via air conduction from the external ear canal. Bone-conducted feedback (Fig 1B) is generated through two mechanisms: (1) direct stimulation of the mastoid bone from AMEI transducer stimulation of the ossicular chain (resulting in energy transmission to the bone via soft tissues, such as

eardrum, middle ear suspensory tissues, and stapedial annular ligament) and (2) indirect stimulation of the mastoid bone via energy radiated from the mounting bracket coupled to the AMEI transducer. Bone-conducted signals create feedback in these mechanisms by direct microphone oscillation. This study investigates the relative contributions of these potential sources of feedback.

Materials and Methods:

Data was collected and analyzed from four ears in fresh-frozen, hemi-sectioned human cadaveric heads. All specimens were reported to have no known history of middle ear disease or surgery and confirmed to have intact temporal bones prior to investigation (Lone Tree Medical, Littleton, CO, U.S.A.). The use of cadaveric human tissue was in compliance with the University of Colorado Anschutz Medical Campus Institutional Biosafety Committee and Review Board (COMIRB Exempt #14–1464).

Temporal Bone Preparation

Cadaveric specimens were prepared in a manner similar to those previously described by our laboratory (Mattingly et al. 2015; Banakis Hartl et al. 2016). Prior to surgical preparation, frozen cadaveric specimens were thawed and inspected to rule out injury or disease of the temporal bone and the external and middle ear. A mastoid atticostomy was performed to allow access to the posterior incus body. A 4-mm titanium implant and abutment (Cochlear Ltd., Centennial, CO, U.S.A.) was situated 50 mm posterior-superior from the external auditory canal to serve as a reflective point for laser Doppler measurements of mastoid cortical bone vibration.

Stimulus Generation and Presentation

Stimulus generation and response recordings were performed as described previously (Greene et al. 2015; Mattingly et al. 2015; Banakis Hartl et al. 2016). Specimens were stimulated with one-second tone pips, ramped on and off with 1/2 (5 ms) of a Hanning window. Driven by an external sound card (Hammerfall Multiface II, RME, Haimhausen, Germany), stimuli were digitally generated to drive the active middle ear actuator (Carina™, Cochlear Ltd., Boulder, CO, U.S.A.). Pure-tone frequencies from 500–6000 Hz were presented in 1/4 octave steps at two intensity levels, 1.0 V and 0.1 V. The two intensity levels were chosen to reflect an output close to the maximum voltage drive (1.0 V) and a level close to commonly used voltage drive (0.1 V). Sound intensity in the ear canal was measured with a probe microphone (type 4182; Brüel & Kjær, Naerum, Denmark) which was independently suspended from the specimens and coupled to a 5-cm flexible probe tube that was placed in the ear canal.

The AMEI (Carina™, Cochlear Ltd., Boulder, CO, U.S.A.) transducer was held in place by one of two methods: suspended from a rigid stereotactic arm (to avoid actuator contact with the mastoid cortical bone), or via the standard mounting bracket. The active middle ear device was coupled to the incus body according to methods previously described (Jenkins et al. 2007c). Briefly, the transducer was advanced slowly while continuously monitoring the inductance and impedance of the device linear actuator utilizing the transducer loading

assistant (TLA) software (Cochlear Ltd., Boulder, CO, U.S.A.). Initial contact was verified by a decrease in impedance by 100–200 ohms. Advancement of the transducer was continued until the inductance was in the range of 14.2 to 12.9 milliHenries (mH) and the impedance was within 300–400 ohms, values shown by Tringali et al. (2011) to optimize the performance of the transducer. Data were collected for suspended and mastoid anchor conditions for statistical comparison of the two paradigms.

Out-of-plane cortical mastoid velocity (V_{Mast}) was measured with a single-axis LDV (OFV-534 & OFV-5000; Polytec Inc., Irvine, CA, U.S.A.) mounted to a dissecting microscope (Carl Zeiss AG, Oberkochen, Germany). In order to estimate the relative damping effect of the soft tissue overlying the cortical bone, LDV measurements were made from two locations: (1) reflective tape placed on the skin overlying the mastoid and (2) the titanium bone-anchored implant (BAI) and abutment surgically implanted into the mastoid bone.

Data Acquisition and Data Analysis

Recordings from the probe-tube microphone and LDV were simultaneously captured via the sound card analog inputs. Acquired signals were band-pass filtered between 15–15000 Hz with a second order Butterworth filter.

Statistical analyses were completed using functions in the Statistics and Machine Learning toolbox in Matlab. Differences in sound pressure level recorded for each stimulus frequency from the probe tube microphone were compared between the bracket and suspended testing conditions utilizing a Wilcoxon rank sum test (*ranksum* function). Differences in LDV measurements across manner of coupling (bracket or suspended) and measurement locations (BAI post or skin) were assessed with an n-way analysis of variance (ANOVA – *anovan* function), and post hoc testing was performed with a Tukey–Kramer HSD (honest significant difference) test (the *multcompare* function). Statistical comparisons are assessed at the $\alpha = 0.05$ level unless otherwise specified.

Results:

Air-Conduction

Air-conducted signal measured in the ear canal is shown for the lower stimulus intensity (0.1 V) in Figure 2, for the bracket in place (blue line) and suspended actuator (red line). Sound pressure ranged from 40–80 dB SPL, generally peaking around 2 kHz. While presence of the bracket added on average 5–10 dB to the sound pressure recorded in the EAC below 2 kHz, it was only significant at the lowest frequency tested (500 Hz).

Air-conducted signal for higher stimulus intensity (1.0 V) is shown in Figure 3, for the bracket in place (purple line) and suspended actuator (green line). Sound pressure ranged from 40–85 dB SPL, with two large peaks at 700 and 2000 Hz. Presence of the bracket added on average 5–10 dB to the signal recorded in the EAC below 2 kHz. A Wilcoxon rank sum test revealed this difference at this higher intensity to be significant at four frequencies (594.6, 707.1, 840.9, and 1189 Hz), indicated by asterisks in the figure.

Bone-Conduction

Bone-conducted signal resulting from stimulation with the AMEI is illustrated in Figure 4A and 5A with a solid line for recordings made from the BAI post and a dashed line for those recorded from skin reflective tape. Figure 4A illustrates LDV data collected during lower intensity stimulation (0.1 V) with the actuator coupled to the incus with the bracket (blue line) and suspended actuator (red line). Figure 5A illustrates data recorded with higher intensity stimulation (1.0 V), with the purple and green lines indicating bracket and suspended coupling, respectively.

LDV measurements were grouped into three bands of relatively low ($f < 1$ kHz), middle ($1 \text{ kHz} < f < 2.5 \text{ kHz}$), and high ($f > 2.5 \text{ kHz}$) frequencies, with approximately the same number of frequencies (5) within each band. A two-way ANOVA was performed for both intensity levels with velocity as the dependent variable, and manner of coupling (bracket or suspended) and recording location (BAI or skin) as independent variables for each frequency band. For 0.1V stimuli, results revealed a main effect of recording location in the low and mid frequency range, but not manner of coupling. For 1.0V stimuli, a main effect of recording location in all frequencies and an effect of manner of coupling in the high frequencies was present. Post-hoc Tukey honest significant difference pairwise comparisons revealed significant differences between the recording conditions shown in the bar graphs in Figure 4B and 5B. Table 1 shows the results of the above statistical analysis.

Discussion:

Results from this study confirm that AMEI generate both air- and bone-conducted signals that may be picked up by the device microphone and undergo re-amplification.

Measurements made with probe-tube microphones in the ear canals of cadaveric specimens peaked near 90 dB SPL at 2 kHz, confirming that air-conducted energy is transmitted from the AMEI. We hypothesized two mechanisms contributing to the air-conducted signal, i.e., (1) reverse-transmission and (2) mastoid vibration resulting in osseotympanic oscillation of the column of air within the ear canal and inertial motion of the ossicular chain. The loss of 5–10 dB below 2 kHz from decoupling the bracket suggests generation via the latter. Targeted damping of the mounting bracket may serve to mitigate this source of feedback.

Laser Doppler measurements confirmed that attachment of the mounting bracket increased velocity in higher frequencies above 2 kHz. Damping of bracket coupling to the cortical mastoid may decrease bone-conducted signals in the high frequencies, as well as mitigate air-conducted signals in the lower frequencies, as noted above. Vibratory signals between the LDV measurement sites (titanium abutment and skin surface) significantly dropped by 1/2 to 1/3. These data suggest bone-conducted feedback can potentially be reduced by avoiding direct contact of the microphone with the cortical mastoid bone.

Impact of Feedback on Hearing Aid Compliance:

Acoustic feedback has a long history of interfering with patients' abilities to optimally utilize hearing aids (Kates 1991; Agnew 1996; Chung 2004; Madsen & Moore 2014). The MarkeTrak survey of the hearing-impaired population and hearing aid market, sent every 3–

4 years to 80,000 households, consistently identified feedback as an important concern of hearing aid users that was often cited as a reason for device non-utilization (Kochkin 1993; Abrams & Kihm 2015).

Few studies of active middle ear implants contain an in-depth discussion regarding generation of feedback. While excessive acoustic feedback with traditional hearing aids is a problem often cited by patients prior to implantation, only a small number of investigations discuss device feedback as a post-surgical complication for AMEI (Jenkins et al. 2007a; Bruschini et al. 2010, 2016; Lefebvre et al. 2016). Suboptimal device compliance due to feedback has been reported (Jenkins et al. 2007a; Lefebvre et al. 2016), with report of one severe case necessitating return to the operating room (Bruschini et al. 2010). Less frequently discussed is possible mechanisms for generation of feedback, generally attributed to a feedback loop similar to that seen in conventional amplification and related primarily to the leakage of air-conducted signal back to the device microphone (Lefebvre et al. 2016). Given the mechanical nature of AMEI, it is important to consider that bone-conducted signals may also be contributing to feedback in these devices. Feedback via vibration transmission to device microphones is not new or unique to AMEI; mechanical feedback in conventional aids is known to result from the transmission of vibratory energy from the device receiver to the microphone via the hearing aid faceplate (Agnew 1996; Chung 2004).

While new digital technologies, including feedback mitigation algorithms, have improved patient satisfaction with hearing devices (Abrams & Kihm 2015), these advances in digital programming come at a cost, namely power use and battery life. Power consumption may be even more important in the case of AMEI, where actuator motion requires more energy than a microphone in a traditional device. As such, other techniques for feedback mitigation may be of even more importance for active implanted devices.

Microphone Placement:

Surgical placement of the microphone has received the most attention as a potential target for feedback mitigation and reduction of internally-generated noise interference (Jenkins et al. 2007a; Bruschini et al. 2009, 2010, 2016; Kam et al. 2012; Jenkins & Uhler 2014; Pulcherio et al. 2014; Lefebvre et al. 2016), e.g. muscle motion noise. Excessive feedback due to microphone location has necessitated return to the operating room in at least one case (Bruschini et al. 2010). Three locations are generally considered convenient for AMEI: temporalis region (anterior and superior to the ear canal), retroauricular region (posterior to the ear canal), and the region overlaying the mastoid tip (posterior and inferior to the ear canal) (Pulcherio et al. 2014). The ideal placement of the implantable microphone continues to be clinically debated (Martin et al. 2009), though most authors suggest the retroauricular position as most optimal with regards to surgical ease of access and reducing device feedback (Martin et al. 2009; Bruschini et al. 2010; Lefebvre et al. 2016).

Two authors have attempted to objectively assess the importance of microphone placement with implantable hearing devices. In an investigation of subcutaneous microphone placement in a porcine model, Deddens et al. (1990) noted that sound attenuation was directly proportional to skin thickness between 0.5 mm and 3.0 mm, with a slope of -9.1 dB/mm. Authors suggested that minimal skin thickness (0.5 mm) is ideal for optimal sound

transmission in an implantable microphone, though this study did not take feedback into account with these measurements. Jenkins et al. (2007b) examined patient-generated noise in the mastoid tip location relative to a retroauricular position. Soft tissue vibrations measured during subject phonation with laser Doppler vibrometry demonstrated 10–25 dB greater vibration at the mastoid tip, suggesting this location may be more prone to feedback from subject-generated noise.

Device Damping:

Another important mitigation strategy to consider may be physical damping of the device to reduce transmission of mechanical vibration to the device microphone. To date per our review of the literature, this strategy has not been discussed as a method for targeting feedback in AMEI. Historically, the use of damping has been suggested as a manner of reducing mechanical feedback in conventional hearing aids (Agnew 1996; Chung 2004). The addition of rubber padding to the receiver of a conventional hearing aid has been suggested to reduce mechanical feedback (Chung 2004). In the case of AMEI, coupling to the ossicular chain is important to device performance (Jenkins et al. 2007c), and dampening should not impact this coupling. Our data suggest that the mounting bracket contributes to both air- and bone-conducted AMEI feedback, and potentially accounts for up to 5–10 dB of osseotympanically- and inertially-generated air-conduction signal, as well as ~10 dB of mechanical feedback. This suggests that utilization of a dampening material between the mounting bracket and the cortical bone of the mastoid may significantly reduce this feedback.

One concern that may arise with interposition of any damping material is the potential undesired reduction of stimulus intensity, or reduction of the effective device stroke. Damping the bracket may be viewed as interposing a spring and damper between the actuator and its anchoring point. The actuator effective stroke will be reduced by the ratio of the compliance (inverse of spring rate) with damper to the compliance without damper. For most practical dampers, the spring rate is higher than the actuator internal spring rate; also the damping factor at acoustically important frequencies will also result in a high mechanical impedance. For these reasons, a damper would reduce the effective stroke of the actuator by only a small amount.

Limitations

One important limitation of this study is that we did not utilize the AMEI microphone to quantify objective feedback levels, but rather measured the sources that may potentially contribute to the generation of feedback. With this data, we cannot verify that the measured signals would cause feedback for all patients, but the relative comparisons remain helpful in discussing device modifications, surgical techniques, or programmatic changes as helpful targets for feedback mitigation. Additionally, the use of single-axis LDV does not allow us to account for the three-dimensional motion of the head resulting from bone conduction stimulation.

Another limitation is the use of a cadaveric model. Though hemicephali provide a more complete model than isolated temporal bones, we recognize that cadaveric specimens may

be subject to post-mortem degradation. Additionally, cadaveric hemicephali lack intracranial structures and fluid that potentially affect bone-conduction. While this study may have underestimated this component of bone-conducted energy, it has been previously demonstrated to contribute relatively little to the total signal transmitted to the inner ear via bone conduction (Stenfelt & Goode 2005a, 2005b). Only four specimens were used for this study; inclusion of additional specimen could have strengthened significance data, particularly when looking at differences across frequency. Finally, though the microphone was independently suspended, the probe tube was placed in the ear canal and may potentially have contacted the canal wall. This may have provided an unintended source of coupling, thus potentially overestimating the air-conducted measurements.

Conclusions:

AMEI produce both air- and bone-conducted signals of adequate strength to be detected by the device microphone and undergo re-amplification. Understanding the relative contribution of these sources may play an important role in the development of specific, targeted mitigation algorithms as well as surgical techniques emphasizing acoustic isolation. Air-conducted sound pressure measured in the ear canal during stimulation with AMEI ranged between 40–89 dB SPL. Air-conducted signal was greatest at 2 kHz and below. Use of a mounting bracket added 5–10 dB, suggesting this difference may be due to an osseotympanic or inertial component of bone-conducted energy. Damping of the bracket may help to reduce a component of this feedback.

Bone-conducted vibratory motion was measurable on the skin surface and on a titanium abutment coupled to the cortical mastoid during stimulation with AMEI. Bone-conducted signals were greatest with the mounting bracket in place, especially in mid to high frequencies. Damping of the bracket may help to reduce feedback due to bone-conducted energy in the higher frequencies. A significant drop in bone-conducted signal was noted when measuring from the skin surface compared with the titanium abutment. Strategic microphone placement remains important, as our data suggests bone-conducted feedback can potentially be reduced by avoiding microphone contact with the cortical mastoid bone.

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Figure 1:

Schematic of hypothesized pathways for generation of feedback in active middle ear implants (AMEI). Air-conducted feedback may arise from reverse transmission through ossicular chain and tympanic membrane or mounting bracket osseotympanic or inertial vibrations oscillating of the column of air within the ear canal or the ossicular chain, respectively. This acoustic energy subsequently leaks out the ear canal to the device microphone, where it can undergo re-amplification (A). Bone-conducted energy may result direct transmission to the ossicular chain, or indirect transmission to the mastoid cortex via the mounting bracket (B).

Figure 2:

Sound pressure in the ear canal during AMEI stimulation with low intensity drive (0.1 V). Blue and red lines illustrate data with bracket in place and suspended actuator, respectively. Colored bands show the standard error of the mean. Asterisks (*) indicate significant differences ($p < 0.05$) at presented frequencies determined with a Wilcoxon rank sum test.

Figure 3:

Sound pressure in the ear canal during high intensity drive (1.0 V) AMEI stimulation. Purple and green lines indicate the bracket and suspended conditions, respectively, with bands indicating standard error of the mean. Asterisks (*) indicate significant differences ($p < 0.05$) at presented frequencies determined with a Wilcoxon rank sum test.

Figure 4:

Bone-conducted mastoid velocity measured during low intensity (0.1 V) stimulation with an AMEI. The upper panel (A) shows the mean $\pm$ SEM mastoid velocities collected in both the bracket (blue) and suspended (red) conditions. Data collected from the BAI and abutment is depicted with a solid line, while data collected from reflective tape on the skin is shown with a dashed line. Divisions for the low ($f < 1$ kHz), middle ($1 \text{ kHz} < f < 2.5 \text{ kHz}$), and high ($f > 2.5 \text{ kHz}$) frequency bands are overlain on the figure, with roughly the same number of frequencies (5) within each band. Bar graphs in the lower panel (B) show the mean

mastoid velocity within each frequency band for each recording condition. Significant differences between recording conditions were determined with an ANOVA and are indicated by asterisks (* $p < 0.05$; ** $p < 0.01$).

Figure 5:

Bone-conducted mastoid velocity measured during high intensity (1.0 V) stimulation with an AMEI. The upper panel (A) shows the mean $\pm$ SEM mastoid velocities collected in both the bracket (purple) and suspended (green). Data collected from the BAI and abutment is depicted with a solid line, while data collected from reflective tape on the skin is shown with a dashed line. Divisions for the low ($f < 1$ kHz), middle ($1\text{ kHz} < f < 2.5\text{ kHz}$), and high ($f > 2.5\text{ kHz}$) frequency bands are overlain on the figure, with roughly the same number of frequencies (5) within each band. Bar graphs in the lower panel (B) show the mean mastoid

velocity within each frequency band for each recording condition. Significant differences between recording conditions were determined with an ANOVA and are indicated by asterisks (* $p < 0.05$; ** $p < 0.01$).

Table 1:

Posthoc Tukey honest-significant difference (hsd) pairwise comparisons for mastoid velocity measurements

Stimulus Intensity	Frequency Band	Manner of coupling (Bracket or suspend)		Recording location (BAI or skin)	
		F	p value	F	p value
0.1 V	Low (<1 kHz)	0.8	0.3758	11.42	0.0015 *
0.1 V	Mid (1–2.5 kHz)	0.79	0.3769	24.25	<0.0001 **
0.1 V	High (>2.5 kHz)	2.17	0.1472	3.61	0.0634
1.0 V	Low (<1 kHz)	0.78	0.3813	12.25	0.0009 **
1.0 V	Mid (1–2.5 kHz)	0.09	0.7687	41.62	0.0001 **
1.0 V	High (>2.5 kHz)	5.94	0.0175 *	6.75	0.0116 *

*
p<0.05

**
p<0.01