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Authors

Ward, Bryan K
Agrawal, Yuri
Nguyen, Elena
[et al.](#)

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Hearing Outcomes Following Surgical Plugging of the Superior Semicircular Canal by a Middle Cranial Fossa Approach

Bryan K Ward, MD, Yuri Agrawal, MD, Elena Nguyen, BA, Charles C Della Santina, MD, PhD, Charles J. Limb, MD, Howard W Francis, MD, Lloyd B Minor, MD, and John P Carey, MD

Johns Hopkins School of Medicine – Department of Otolaryngology-Head and Neck Surgery

Abstract

Objective—To determine post-operative hearing outcomes following surgical plugging via middle cranial fossa approach for superior semicircular canal dehiscence syndrome (SCDS).

Study Design—Clinical Review

Setting—Tertiary Care Medical Center

Patients—43 cases of SCDS based on history, physical examination, vestibular function testing, and computed tomography imaging confirming the presence of a dehiscence. All patients underwent surgical plugging of the superior semicircular canal via middle cranial fossa approach.

Intervention—Pure tone audiometry was performed pre-operatively and at 7 days and at least 1 month post-operatively.

Main Outcome Measures—Change in air-bone gap (ABG) and pure tone average (PTA).

Results—Pre-operative average ABG across 0.25, 0.5, 1, and 2 kHz was 16.0 dB (SD 7.5). At 7 days post-operatively, average ABG was 16.5 dB (SD 11.1, $p=0.42$), and at >1 month was 8.1 dB (SD 8.4, $p<0.001$). 53% (95% CI, 33–69) of affected ears had greater than 10 dB increase in their four frequency (0.5, 1, 2, 4 kHz) PTA measured by bone-conduction (BC) threshold 7 days postoperatively and 25% (95% CI 8–39) at 3–15 months post-operatively. Mean BC PTA of affected ears was 8.4 dB (SD 10.4) hearing loss (HL) pre-operatively. Compared to baseline, this declined to 19.2 dB HL (SD 12.6, $p<0.001$) at 7 days post-operatively and 16.4 dB HL (SD 18.8, $p=0.01$) at >1 month. No significant differences in speech discrimination score were noted ($F=0.17$).

Conclusions—Low-frequency air-bone gap decreases following surgical plugging, and appears to be due to both increased BC thresholds and decreased AC thresholds. Surgical plugging via a middle cranial fossa approach in SCDS is associated with mild high-frequency sensorineural hearing loss that persists in 25%, but no change in speech discrimination.

INTRODUCTION

Superior canal dehiscence syndrome (SCDS) is a clinical syndrome of dehiscence in the bone overlying the superior semicircular canal causing a spectrum of symptoms including bone-conduction hyperacusis and vertigo with loud sounds or changes in pressure (1,2). A phenomenon consistent with this syndrome is the presence of a low-frequency air-bone gap (ABG) on audiogram (3). The current theory describing this air-bone gap in SCDS is the

‘third mobile window’ hypothesis, which suggests that acoustic energy transmitted to the oval window is partially shunted through this abnormally compliant “window” caused by a dehiscence of the superior semicircular canal (4,5). A few prior studies have documented correction of this air-bone gap in several patients post-operatively, but these studies have focused on early results in small numbers of patients (3,6,7).

The dehiscence in SCDS can be surgically repaired with resolution of symptoms by a middle cranial fossa approach and plugging of the superior semicircular canal (8,9). Variations on this technique include resurfacing (rather than plugging) the canal or plugging it via the mastoid cavity (10–12). While patients appear to improve regardless of operative technique, limited sample sizes have restricted the ability to draw conclusions regarding differences in postoperative outcomes.

Canal plugging via the middle cranial fossa approach has been the most studied technique. Patients undergoing this procedure experience improved autophony and Dizziness Handicap Inventory scores post-operatively (8,9). In addition, though patients may experience temporary global vestibular hypofunction post-operatively, data show good long-term vestibular function (apart from the expected permanent deficit in the plugged canal) after unilateral surgical repair (13,14). Prior studies of post-surgical hearing outcomes have demonstrated stable hearing, but have been limited by small sample sizes, heterogeneous samples, and post-operative duration (6,7). The purpose of this study was to investigate longer-term post-operative hearing outcomes in a large series of patients with SCDS who have undergone superior canal plugging via the middle cranial fossa approach.

MATERIALS AND METHODS

Study Population

Clinical data from patients undergoing surgical repair of superior canal dehiscence via the middle cranial fossa approach from June 2009 until July 2011 were reviewed retrospectively. Identifying patient information was removed prior to analysis. Patients were diagnosed with SCDS based on history and physical examination and at least one abnormal clinical physiologic test consistent with superior canal dehiscence syndrome, including vestibular evoked myogenic potentials (VEMP) or sound and/or pressure induced eye movements. Patients must also have had evidence from high-resolution computed tomography (CT) of a dehiscence of the superior semicircular canal on reformation in the plane of the affected canal and orthogonal to that plane (15,16). All patients involved in this study had agreed to undergo surgical repair of the dehiscence of the superior semicircular canal. This study was a review of existing clinical data with patient identifiers removed. It qualified for exemption from an institutional review board protocol on the basis of United States Department of Health and Human Services criteria 45 CFR 46.101(b4). This exemption was approved by the Johns Hopkins Institutional Review Board.

Surgical Technique

The middle cranial fossa surgical approach was used in all patients. Image guidance was used for all subjects to localize the dehiscence intraoperatively. Plugging of the canal was performed using fascia strips, bone dust and bone chips, which are gently but securely placed inside the dehiscence of the canal to obliterate the canal lumen for 2–3 mm beyond either end of the dehiscence opening. Careful avoidance of unnecessary force on or suction near the membranous labyrinth was observed throughout the procedure. The repair was then covered with hydroxyapatite cement, followed by a layer of fascia and fibrin glue. Intraoperative neural monitoring of the facial nerve, auditory brainstem response, electrocochleography, and somatosensory evoked potentials was performed. All patients received dexamethasone 6

to 8 mg three times per day for three doses. If post-operative clinical examination revealed no evidence of either sensorineural hearing loss by Weber tuning fork examination or labyrinthine hypofunction by head thrust examination, then steroids were tapered over five days. Patients with abnormal findings suggesting sensorineural hearing loss or pan-labyrinthine hypofunction on examination had steroids tapered over a 10–14 day period.

Audiometry

All patients underwent audiometric testing prior to surgery and at 7–10 days post-operatively. A follow-up audiogram was routinely performed at least 1 month post-operatively at our institution. Pure-tone hearing thresholds were obtained using both air conduction (AC) at 250 Hz, 500 Hz, 1000 Hz, 2000 Hz, 4000 Hz, and 8000 Hz and bone conduction (BC) at 250 Hz, 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz. Speech discrimination was also tested for all patients using the Northwestern University Auditory Test No. 6 (N.U. No 6) word list. Presentation levels were 40 dB sensation level (SL) above speech reception threshold (SRT). All testing was performed in a sound attenuated booth with regularly calibrated equipment (ANSI S3.6–1996). To ensure consistency, only audiometry performed at our institution was included in the analysis.

The air-bone gap was calculated by subtracting the BC threshold from the AC threshold at each frequency. Average low-frequency air-bone gap (ABG) was defined as the average for each individual frequency air-bone gap over the lower frequencies including 250 Hz, 500 Hz, 1000 Hz, and 2000 Hz. A 4-tone pure tone average (PTA) was calculated across 500 Hz, 1000 Hz, 2000 Hz, and 4000 Hz (17). Due to the high-prevalence of hemotympanum and middle ear effusion in the immediate post-operative period, BC thresholds were used to calculate PTA for all audiograms.

Data Analysis

Summary statistics were performed for demographics, pre-operative and post-operative hearing thresholds at each frequency and for average low-frequency ABG and four-frequency BC PTA. ANOVA was used to assess between-group differences for ABG, PTA, and speech discrimination scores over the three audiograms. If significant, pairwise comparisons were performed. Paired *t* tests were used to compare differences in means over time and unpaired *t* test was used to compare means between groups. Associations were considered statistically significant for 2-sided statistics with a *p*-value < 0.05. Stata 12.0 (StataCorp, College Station, TX, USA) was used for all analyses.

Results

A total of 43 consecutive cases of superior canal dehiscence plugging via middle cranial fossa approach (40 patients) was performed between June 2009 and June 2011, and all cases were included in this study. During this period a consistent technique of SCDS plugging was used based on 10 years of previous experience. Three subjects underwent a second surgery on the contralateral ear during the time period captured by this study. Subjects were 43.9 (SD 10.2) years old and 24 (57%) were female (Table 1). Seven subjects (16%) had undergone prior surgery on the affected ear, including 3 prior middle ear explorations at outside institutions and 4 prior surgical plugging procedures for SCDS (two middle cranial fossa approaches at our institution and one trans-mastoid and one middle cranial fossa approach at outside institutions). All subjects had pre-operative and 7–10 day post-operative audiograms performed at our institution. 32 of 43 affected ears had a follow-up audiogram at our institution at least 1 month post-operatively (range 1–15 months, median 3 months). Because many patients traveled from a long distance, long-term audiograms were not uniformly available. However, with respect to possible bias that might be created by these

missing audiograms, there was no difference in BC PTA or average low-frequency ABG at 7–10 days between patients missing long-term follow-up audiometry (n=11) and those who had an audiogram at least 1 month post-operatively.

The four-frequency BC PTA of all pre-operative affected ears was 8.4 dB hearing level (HL; SD 10.5). At 7–10 days post-plugging, the average BC PTA was 19.3 dB HL (SD 12.6, $p<0.001$). 53% (n=23) of subjects had at least a 10 dB increase or functional decline in BC PTA at 7–10 days compared to pre-operative audiometry. A repeat audiogram at >1 month demonstrated average BC PTA of 16.4 dB HL (SD 18.8, $p=0.01$), with 25% (n=8) having at least a 10 dB increase in 4-frequency BC PTA compared to baseline. There were no between-group differences for speech discrimination score across all subjects (Table 2). Figure 1 demonstrates BC and AC thresholds across all frequencies for each audiogram. BC thresholds at each tested frequency were increased at >1 month post-operatively compared to pre-operative values ($p<0.05$). This remained significant after removing the two subjects with the largest post-operative increase in thresholds (range of mean change in db HL from 5.6 db HL, 95% CI 1.5, 9.8 to 8.9 db HL, 95% CI 5.6 to 12.2, $p<0.05$). For AC thresholds for all subjects, there was no long-term change at 250 Hz, 500 Hz, and 1000 Hz, but there was a significant increase in mean thresholds at 2000 Hz (7.5 dB, 95% CI 0.2 to 14), 4000 Hz (10.4 dB, 95% CI 2.6 to 18 dB), and 8000 Hz (12.9 dB, 95% CI 4.8 to 21 dB, $p<0.05$). After removing the two patients with the largest increase, there remained a significant increase in AC thresholds at 4000 Hz and 8000 Hz and a decrease or functional improvement in mean thresholds at 250 Hz (–6 dB, 95% CI 2.0 to 10, $p<0.05$).

Pre-operative average low-frequency ABG for affected ears was 16.0 (SD 7.6) dB. This was significantly different from the contralateral ear (6.4 dB, SD 7.1, $p<0.001$). 78% (n=33) of patients had a pre-operative average low-frequency ABG of at least 10 dB in the affected ear. At 7–10 days post-operatively, there was no change in the ABG (mean 16.4 dB, SD 11.1, $p=0.83$). At the 7–10 day audiogram, a majority of patients (84%, n=37) had evidence of either hemotympanum or middle ear effusion on otoscopic examination, and at this visit these patients had a significantly increased average low-frequency ABG than those without effusions (18.1 dB, SD 11.4 vs. 8.8 dB, SD 3.9, $p<0.05$). At >1 month, however, the average low-frequency ABG was significantly reduced (mean 8.1 dB, SD 8.4, $p<0.001$). Of patients who had a pre-operative average low-frequency ABG of at least 10 dB, 63% (n=14 out of 22) had a reduction to less than 10 dB at >1 month. Figure 2 demonstrates average low-frequency AC and BC thresholds over time. Average low-frequency BC thresholds on both post-operative audiograms differed from the pre-operative value ($p<0.01$); however, average AC thresholds significantly increased at 7–10 days ($p<0.01$), before returning to baseline at >1 month.

Subjects who underwent prior surgery on the affected ear had increased BC PTA at >1 month post-operatively compared to those without prior ear surgery (32.8 dB HL vs. 13.6 dB HL, $p=0.01$). Mean >1-month post-operative speech discrimination scores were also lower among those with prior surgery (74.7%, SD 16.2 vs. 97.4%, SD 8.0, $p<0.01$). In addition, of the four subjects who had undergone prior surgery and had available long-term audiometry, only one had a reduction of the low-frequency ABG to less than 10 dB. Table 3 shows hearing results for patients with and without prior affected ear surgery. For subjects with no prior affected ear surgery, similar to the overall group, the four-frequency BC PTA was increased from baseline at 7–10 days (17.9 dB, SD 10.7 vs. 5.8 dB, SD 6.6, $p<0.0001$) and at least 1 month post-operatively (13.6 dB, SD 13.6, $p=0.001$). At both time points, there was a significant increase compared to the four-frequency BC PTA of the contralateral ear ($p<0.05$). Also, for those without prior affected ear surgery, the average low-frequency ABG was not different 7–10 days postoperatively, but was significantly lower at least 1

month after surgery compared to baseline (6.3 dB, SD 4.4 vs. 16.2 dB, SD 6.6, $p < 0.0001$). There was no difference in speech discrimination score over time in either group.

Based on post-operative day 1 clinical head thrust examination, 14 subjects (32.5%) had pan-labyrinthine hypofunction in the operated ear and received a 10–14 day course of steroids instead of the usual 5-day taper. The presence of pan-labyrinthine hypofunction on postoperative day 1 was associated with increased BC PTA at 7 days compared to those without hypofunction ($p = 0.02$), but not long-term ($p = 0.08$). Thus, there was no long-term difference in hearing outcome based on the presence of post-operative labyrinthine hypofunction in patients who received a prolonged steroid taper. Patients with pan-labyrinthine hypofunction additionally had larger ABG pre-operatively and at >1 month compared to those without pan-labyrinthine hypofunction, but there were no significant differences in speech discrimination scores between those with and without labyrinthine hypofunction. Table 4 shows postoperative hearing outcomes by patients with the presence of pan-labyrinthine hypofunction.

Discussion

This study demonstrates that the air-bone gap present in SCDS reduces after surgical plugging of the affected canal, and that plugging of the canal is associated with both reduction of bone conduction hyperacusis and a mild sensorineural hearing loss. Prior studies have identified correction of the low-frequency ABG in some patients undergoing SCDS repair, as well as sensorineural hearing loss in some patients (1,3). Larger series, however, found minimal changes in hearing in patients who had not previously undergone otological surgery on the affected ear (6). Benefits of this study include a larger sample size of subjects all undergoing the same surgical repair and the majority of who had long-term follow-up audiometry including expanded low-frequency threshold testing.

This series of patients demonstrates that the ABG closes after surgery, but that these results are usually not identifiable on the initial post-operative audiogram. While the ABG remains stable one week after surgical plugging, both the low-frequency bone conduction and the low-frequency air conduction lines increase or functionally decline. The bone conduction line remains stable over several months, at a level above the pre-operative value, indicating this is likely a stable effect of the initial surgery. Since this increase in bone conduction threshold of approximately 5 to 10 dB is present across all frequencies, it may be the result of the same phenomenon. The air conduction line also increases one week post-operatively, but decreases towards baseline values long-term. The majority of patients had fluid or hemotympanum on examination at the time of the first post-operative audiogram, and we believe that this expected conductive hearing loss explains the lack of immediate ABG closure. While the high-frequency AC thresholds tended to increase along with the BC thresholds long-term, the lower frequency thresholds tended to decrease or improve. This suggests there may be two phenomena: a diffuse mild increase in BC thresholds that is present immediately post-operatively across all frequencies, and a long-term improvement in low-frequency AC thresholds.

Experiments by Attias et al. in sand rats have shown that canal fenestration is associated predominantly with a decline in air-conduction thresholds rather than bone-conduction thresholds and propose that this is due to shunting of acoustic energy to the third mobile window (18,19). This study suggests that in humans there may be two separate processes contributing to the air-bone gap: an initial decline in low-frequency BC and a long-term stabilization or improvement in low-frequency AC. One explanation for this difference may be interspecies variations in the sound-conductive environment of the inner ear. The exposure of the membranous labyrinth after fenestration to dura and cerebrospinal fluid in

humans rather than air in sand rats and chinchilla may account for interspecies differences in the effect of a labyrinthine dehiscence (20). Exposure of the dehiscent canal to a fluid environment may contribute to bone conduction hyperacusis. Future studies could explore the influence of differing sound-conductive environments in SCDS to explain the presence of and variation in ABG findings in humans with SCDS.

Additionally, this study identified mild sensorineural hearing loss after plugging in greater than 50% of subjects, a finding that persists in 25%. This mild loss, however, does not affect speech discrimination. A theory for sensorineural loss in these and similar cases is loss of perilymph, a loss that may also explain vestibular hypofunction in the immediate post-operative period (13,21). Work by Parnes et al. demonstrated that hearing could be preserved in guinea pigs after canal plugging of the posterior semicircular canal (22). Subsequent human studies indicate a prevalent immediate post-operative mixed hearing loss that resolves long-term except in a minority of subjects (23). While data is unavailable to directly compare outcomes between these procedures, our study indicates similar general findings.

The relationship between prior affected ear surgery and post-surgical hearing outcomes in this study indicates worse long-term BC PTA and speech discrimination. Other studies have reported cases of patients with prior ear surgery having worse post-operative hearing loss in SCDS (6). An additional finding in this series is a difference in the ability to correct the ABG in patients who had undergone prior ear surgery; however, this was largely the case in patients who had undergone prior middle ear exploration, and not for patients with prior canal plugging procedures. Due to the small sample size of available audiometry for patients with prior affected ear surgery (n=4), however, additional study is required to confirm these findings.

Pan-labyrinthine hypofunction as measured by horizontal semicircular canal head thrust testing post-operatively has been shown to resolve over several months and to be related to the size of the dehiscence (13,14). This hypofunction has been suspected to be a result of intraoperative perilymph loss. This study found that subjects with this clinical finding did have worse hearing outcomes shortly after surgery, but that these effects resolved over time. All subjects with pan-labyrinthine hypofunction, however, received a prolonged course of steroids (10–14 days), which may have affected their long-term hearing results for the better. These patients also had evidence of a larger air-bone gap both pre-operatively and long-term post-operatively. While the cause of this difference is unknown, patients with post-operative labyrinthine hypofunction tend to have a larger dehiscence and some studies have shown a larger dehiscence to correlate with a larger low-frequency air-bone gap (13,24).

Limitations of this study include being a retrospective chart review. Several long-term post-surgical audiograms were not performed at our institution and therefore not included in the analysis. Though we did not find a between-group difference in hearing outcomes for those with and without a long-term follow-up audiogram, there may be unrecognized biases in this series. In addition, all subjects with evidence of pan-labyrinthine hypofunction received a prolonged oral steroid course, which may have biased hearing outcomes. Additionally, this study evaluated surgical plugging via the middle cranial fossa approach alone. While this provides a more homogeneous sample for analysis, future prospective studies are needed to identify differences in post-surgical outcomes for SCDS symptoms and in post-surgical complications among multiple surgical approaches.

Conclusions

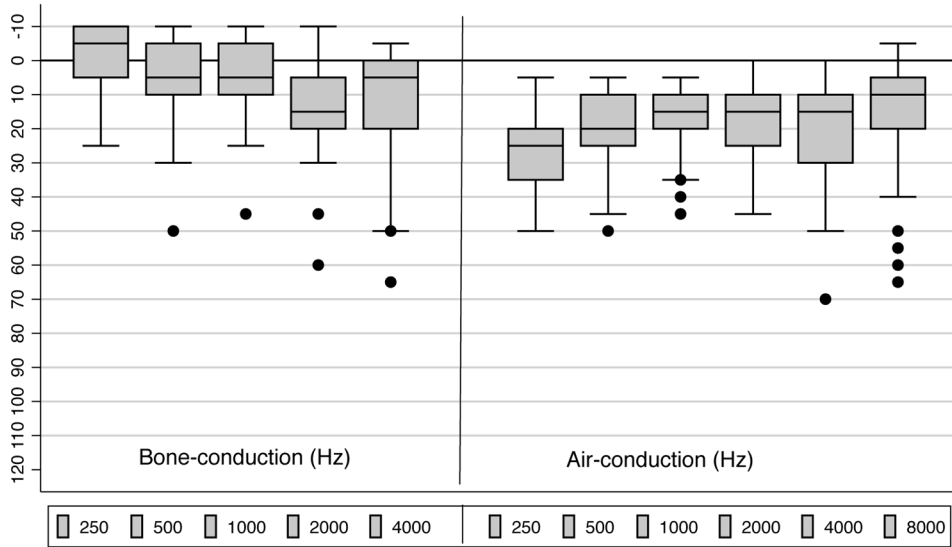
The air-bone gap associated with superior canal dehiscence corrects following surgical plugging in the long-term post-operative period, and appears to be due to both an increase in bone-conduction thresholds and decrease in air-conduction thresholds. Plugging via the middle cranial fossa approach is also associated with a mild sensorineural hearing loss post-operatively that persists in 25% of subjects.

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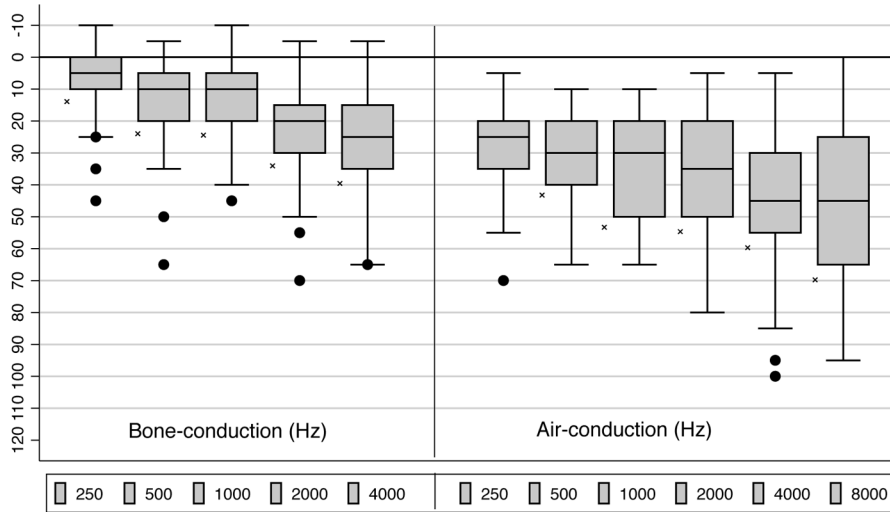
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Pre-operative Air and Bone-conduction Thresholds



7-10 day Post-operative Air and Bone-conduction Thresholds



Long-term Post-operative Air and Bone-conduction Thresholds

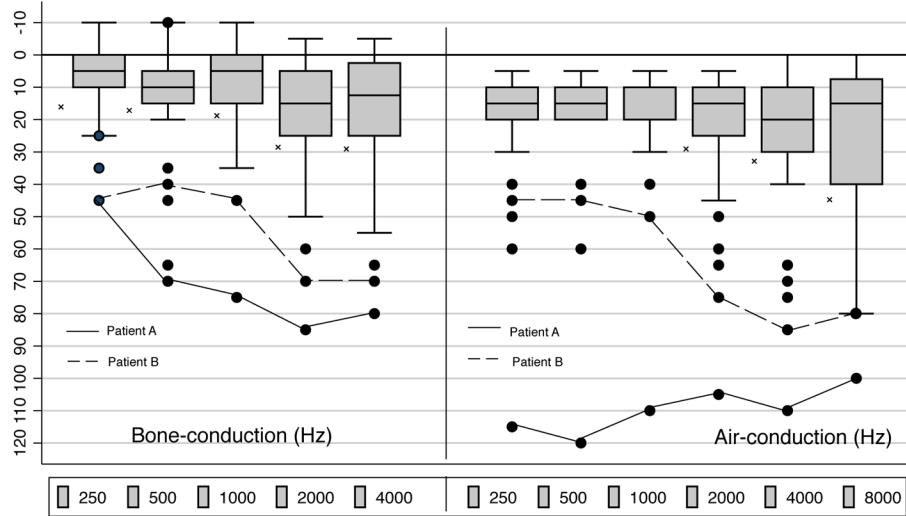


Figure 1. Box plot of bone-conduction and air-conduction thresholds at all tested frequencies for: a) pre-operative, b) 7–10 days post-operative and c) >1 month post-operative audiometry. Box parameters represent 25–75% interquartile range, with median represented by solid horizontal line. Whiskers represent the highest and lowest points within 1.5 times the interquartile range. Dots represent outliers. The two subjects with greatest hearing loss are shown as outliers in plot c. (x indicates significant difference from pre-operative values)

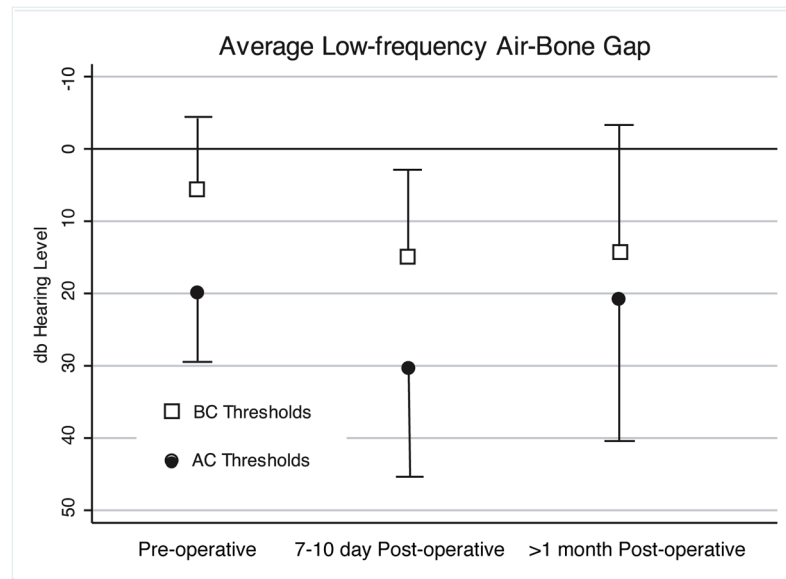


Figure 2. Air-bone gap: Average low-frequency air-conduction and bone-conduction pure tone thresholds (0.25, 0.5, 1, 2 kHz) pre-operatively, 7–10 days post-operatively, and >1 month postoperatively. Whiskers represent standard deviation from the group mean.

Table 1

Characteristics of the Study Participants

| <i>Characteristics</i> | Total n = 43 ears |
|---|--------------------------|
| Age, y (SD) | 43.9 (10.2) |
| Gender, female, n, (%) | 24 (55.8) |
| Affected ears, right, n, (%) | 21 (48.8) |
| Prior ear surgery, n, (%) [*] | 7 (16.3) |
| Post-operative vestibular hypofunction, n, (%) ^φ | 14 (32.5) |

^{*}Prior ear surgery includes superior canal plugging [n=4], and middle ear exploration [n=3]

^φVestibular hypofunction by abnormal head thrust testing (all affected ear planes) on postoperative day 1

Table 2

Post-operative hearing outcomes

| All patients, n = 43 | Pre-operative | 7–10 days | >1 month | Contralateral Ear |
|---|----------------------|------------------|--------------------|--------------------------|
| Average Low-frequency ABG, dB, (SD) | 16.0 (7.5) | 16.4 (11.1) | 8.1 (8.4) * | 6.3 (7.1) * |
| Average four-frequency PTA, dB HL, (SD) | 8.4 (10.4) | 19.3 (10.4) * | 16.4 (18.8) * | 11.1 (9.6) |
| Speech Discrimination Score, %, (SD) | 98.6 (4.1) | 94.4 (14.5) | 93.1 (19.6) | 99.2 (4.3) |

ABG = Air-Bone Gap (0.25, 0.5, 1, 2 kHz), PTA = Pure Tone Average (0.5, 1, 2, 4 kHz)

* indicates statistical significant difference from pre-operative audiometry of affected ear at $p < 0.05$

Table 3

Post-operative hearing outcomes with prior affected ear surgery

| | Pre-operative | 7–10 days | >1 month | Contralateral Ear |
|---|---------------|-------------|---------------|-------------------|
| <i>Prior Affected Ear Surgery, n=7</i> | | | | |
| Average Low-frequency ABG, dB, (SD) | 15.0 (12.5) | 20.8 (17.9) | 17.2 (19.5) * | 7.5 (12.4) |
| Average four-frequency PTA, dB HL, (SD) | 14.8 (16.6) | 26.3 (19.1) | 32.8 (33.9) * | 16.6 (16.4) |
| Speech Discrimination Score, %, (SD) | 96.8 (14.8) | 85.6 (21.7) | 74.7 (39.6) * | 99.2 (1.7) |
| <i>No Prior Ear Surgery, n=36</i> | | | | |
| Average Low-frequency ABG, dB, (SD) | 16.2 (6.6) | 15.3 (9.7) | 6.3 (4.4) * | 6.1 (5.6) |
| Average four-frequency PTA, dB HL, (SD) | 5.8 (6.6) | 17.7 (10.8) | 13.6 (13.9) * | 7.9 (11.2) |
| Speech Discrimination Score, %, (SD) | 99.0 (3.7) | 95.6 (13.1) | 97.4 (8.0) * | 99.7 (1.6) |

ABG = Air-Bone Gap (0.25, 0.5, 1, 2 kHz), PTA = Pure Tone Average (0.5, 1, 2, 4 kHz)

* indicates statistical significant difference between groups at $p < 0.05$

Table 4

Post-operative hearing outcomes with pan-labyrinthine hypofunction

| | Pre-operative | 7–10 days | >1 month | Contralateral Ear |
|---|---------------|---------------|---------------|-------------------|
| <i>Pan-labyrinthine Hypofunction[†], n=14</i> | | | | |
| Average Low-frequency ABG, dB, (SD) | 19.5 (4.7) * | 18.1 (12.6) | 12.2 (11.0) * | 9.5 (10.3) |
| Average four-frequency PTA, dB HL, (SD) | 8.9 (7.9) | 25 (22.4) * | 23.0 (22.4) | 11.7 (8.2) |
| Speech Discrimination Score, %, (SD) | 99.1 (2.3) | 96.3 (4.5) | 86.3 (29.4) | 99.4 (1.4) |
| <i>No Labyrinthine Hypofunction, n=29</i> | | | | |
| Average Low-frequency ABG, dB, (SD) | 14.2 (8.2) * | 15.7 (10.4) | 5.8 (5.5) * | 4.9 (4.6) |
| Average four-frequency PTA, dB HL, (SD) | 8.1 (11.6) | 16.7 (11.8) * | 13.0 (16.3) | 10.8 (10.3) |
| Speech Discrimination Score, %, (SD) | 98.4 (4.8) | 93.7 (16.8) | 97.2 (9.0) | 98.8 (5.2) |

ABG = Air-Bone Gap (0.25, 0.5, 1, 2 kHz), PTA = Pure Tone Average (0.5, 1, 2, 4 kHz)

* indicates statistical significant difference between groups at $p < 0.05$