Incidence of high-frequency hearing loss after microvascular decompression for hemifacial spasm

Clinical article

Tingting Ying, M.D.,1 Parthasarathy Thirumala, M.D.,2,5 Aalap Shah, M.D.,6 Tara Niknow, B.S.,7 Kelley Wichman, B.S.,2 Maura Holmes, B.S.,2 Barry Hirsch, M.D.,2,3 Yuefang Chang, Ph.D.,2 Paul Gardner, M.D.,2 Miguel Habeych, M.D., M.P.H.,2 Donald J. Crammond, Ph.D.,2 Lois Burkhart, R.N.,2 Michael Horowitz, M.D.,2,4 and Jeffrey Balzer, Ph.D.2

1Department of Neurological Surgery, Xinhua Hospital, Shanghai Jiaotong University School of Medicine, Shanghai, China; Departments of 2Neurological Surgery, 3Otolaryngology, 4Radiology, and 5Neurology, University of Pittsburgh, Pennsylvania; 6Department of Anesthesiology, University of Washington, Seattle, Washington; and 7School of Medicine, Drexel University, Philadelphia, Pennsylvania

Object. The primary aim of this study was to evaluate the incidence and discuss the pathogenesis of high-frequency hearing loss (HFHL) after microvascular decompression (MVD) for hemifacial spasm (HFS).

Methods. Preoperative and postoperative audiogram data and brainstem auditory evoked potentials (BAEPs) from 94 patients who underwent MVD for HFS were analyzed. Pure tone audiometry at 0.25–2 kHz, 4 kHz, and 8 kHz was calculated for all individuals pre- and postoperatively ipsilateral and contralaterally. Intraoperative neurophysiological data were reviewed independently. An HFHL was defined as a change in pure tone audiometry of more than 10 dB at frequencies of 4 and 8 kHz.

Results. The incidence of HFHL was 50.00% and 25.53% ipsilaterally and contralaterally to the side of surgery, respectively. The incidence of HFHL adjusted for conductive and nonserviceable hearing loss was 26.6% ipsilaterally. The incidence of HFHL at 4 and 8 kHz on the ipsilateral side was 37.23% and 45.74%, respectively, and it was 10.64% and 25.53%, respectively, on the contralateral side. Maximal change in interpeak latency Waves I–V compared with baseline was the only variable significantly different between groups (p < 0.05). Sex, age, and side did not increase the risk of HFHL. Stepwise logistic regression analysis did not find any changes in intraoperative BAEPs to increase the risk of HFHL.

Conclusions. High-frequency hearing loss occurs in a significant number of patients following MVD surgery for HFS. Drill-induced noise and transient loss of CSF during surgery may impair hearing in the high-frequency ranges on both the ipsilateral and contralateral sides, with the ipsilateral side being more affected. Changes in intraoperative BAEPs during MVD for HFS were not useful in predicting HFHL. Follow-up studies and repeat audiological examinations may be helpful in evaluating the time course and prognosis of HFHL. Prospective studies focusing on decreasing intraoperative noise exposure, as well as auditory shielding devices, will establish causation and allow the team to intervene appropriately to decrease the risk of HFHL.

(http://thejns.org/doi/abs/10.3171/2013.1.JNS121153)

Key Words • microvascular decompression • hemifacial spasm • high-frequency hearing loss • pure tone audiometry • skull base • auditory brainstem response • functional neurosurgery

The auditory nerve is at risk during MVD for HFS. Although hearing loss has been reported following MVD,14,19,23,26,30 there are no studies focused on HFHL in patients who underwent MVD for HFS. Patients with significant HFHL often may have deficiencies in high-frequency components of speech, such as consonant sounds, and can have difficulties understanding speech in the presence of background noise. In addition to normal age-related presbycusis, it has been proposed that higher frequencies may be more sensitive than lower frequencies to noise, acoustic trauma, or ototoxic substances, and thus early diagnosis of hearing loss in these frequencies may predict hearing in lower frequencies.20 For the purposes of this article, we defined high frequencies as 4 kHz and 8 kHz and an increase greater than 10 dB in the pure tone audiometry as constituting HFHL.5,28 The primary aim of the study was to evaluate the incidence of HFHL and to discuss its pathogenesis after MVD for HFS.

Abbreviations used in this paper: BAEP = brainstem auditory evoked potential; HFHL = high-frequency hearing loss; HFS = hemifacial spasm; IPL = interpeak latency; MVD = microvascular decompression; SPL = sound pressure level; w = Wave.

This article contains some figures that are displayed in color online but in black-and-white in the print edition.
Study Design

We retrospectively reviewed data obtained in 152 consecutive patients with HFS who underwent retromastoid MVD at the University of Pittsburgh Medical Center between 2000 and 2007. Inclusion criteria included: 1) patients who had MVD for HFS, diagnosed based on preoperative electromyography testing; 2) documented pre- and postoperative evaluation of hearing function; and 3) availability of intraoperative neurophysiological BAEP responses for analysis. Patients whose native language was English and who did not have a history of noise exposure or disequilibrium were included in the study. According to these criteria, 94 patients were enrolled. The study was approved by the institutional review board for retrospective review of data in human subjects at the University of Pittsburgh.

Microvascular Decompression

Short-acting neuromuscular junction blocking medications were used for intubation. No additional paralytic agent was administered during electromyography monitoring. Microvascular decompression, through a retromastoid craniotomy, was performed with the patient in the lateral decubitus position. At the site of vascular compression caused by arterial loop(s) and/or veins, we decompressed cranial nerve VII using a Teflon pledget.

Preoperative Auditory Investigation

Pure tone audiometry was considered to be an objective indicator of auditory function. It was determined for each ear at frequencies of 0.25, 0.5, 1, 2, 4, and 8 kHz and was performed in all patients before and after surgery. Pure tone audiometry was conducted in a double-walled, sound-treated booth with standard procedures and equipment (GSI 61 audiometer with TDH-BOP headphones [calibrated to American National Standards Institute S36–1989], Grason Stadler). Hearing thresholds were obtained for octave frequencies from 0.25 to 8 kHz, and masking was used at any frequency if the difference between the left and right not-masked air conduction thresholds was 40 dB or more. The pure tone average was defined as the mean of the thresholds measured at 0.5, 1, and 2 kHz. For the purposes of this article, we defined high frequencies as 4 kHz and 8 kHz, and an increase in the threshold of greater than 10 dB constituted hearing loss. Nonserviceable hearing loss was defined by a change in pure tone audiometry of greater than 50 dB and speech discrimination scores of less than 50%, according to the American Academy of Otolaryngology–Head and Neck Surgery classification system. Nonserviceable hearing loss does not derive a perceived benefit from hearing aids when hearing in the contralateral ear is normal. An air-bone conduction gap identified in the speech range of frequencies determined postoperative conductive hearing loss status.

We analyzed BAEP responses to evaluate the effect of nonserviceable and conductive hearing loss on HFHL. An audiological examination was performed in all patients before (median 1 day, range 1–16 days) and after (median 7 days, range 2–16 days) surgery.

Methods

Intraoperative BAEP Monitoring

Stimulation and Recording. In all patients undergoing MVD, preoperative baseline BAEPs and continuous intraoperative BAEP monitoring were performed. The ear ipsilateral to symptoms was stimulated, using alternating rarefaction and condensation clicks with at least an 85–db-SPL intensity. A stimulus rate of 17.5 Hz was used. White noise was applied to the contralateral ear at 65 db-SPL. The observation interval was 12 msec. Five hundred twelve stimuli were delivered for each average. The recording electrodes were positioned as follows: Channel 1, vertex to left ear mastoid (Cz/A1); Channel 2, vertex to right mastoid (Cz/A2); and Channel 3, vertex to cervical C2 (Cz/Cv2). The amplifier band pass was 100–1000 Hz for all channels. Baseline responses were obtained after anesthesia induction and patient positioning.

Monitoring and Alarm Criteria. Persistent decreases in amplitude greater than 50% of wV were considered to be significant. Additionally, a significant change was also defined as a persistent absolute latency increase of the peak of wV, which equals or exceeds 0.5 msec. Persistent changes” represent differences in amplitude or latency of the BAEPs compared with baseline responses in more than 2 consecutive averaged trials. The primary reason for using more than 2 trials was to eliminate technical issues such as noise. Henceforth, these threshold signal changes will be referred to as “significant.” Physician oversight and interpretation was performed using a combined on-site and remote model employed by University of Pittsburgh Medical Center.

Analysis of Neurophysiological Parameters. Wave I, III, and V latencies, IPL (wI–III, wIII–V, wI–V), and amplitudes of wIII and wV were measured. The aforementioned parameters were noted separately for analysis at the following times during the procedure: 1) baseline (after intubation and positioning of the patient); 2) dura opening (the epoch when the opening of the dura mater was documented); 3) change maximum (the maximum change in latency or amplitude recorded compared with average baseline waveform, including epochs when the responses were completely lost); and 4) on skin (the last epoch recorded). The changes in amplitude of response were reported to the surgeon as a percentage of baseline (for example, if the baseline value was 1 and decreased by 0.2, then it was reported as 80% of the baseline).

Statistical Analysis

The prognostic value of intraoperative BAEPs was estimated by correlating BAEP changes intraoperatively with pre- and postoperative audiometry. Patients were grouped into those with HFHL and those without HFHL. Preoperative and postoperative hearing results were compared in each group using the Wilcoxon signed rank test. Intergroup comparisons (HFHL vs no HFHL) of hearing results were evaluated using Kruskal-Wallis tests. Finally, a logistic regression analysis with stepwise selection procedure was used to identify significant factors that were associated with hearing loss while adjusting for covariates. Analyses were performed with SAS version 9.1.3 (SAS Institute) and were two sided at alpha of 0.05.
High-frequency hearing loss after MVD for HFS

Results

Demographic Variables

Ninety-four patients were included in the study. Sex, age, and side of surgery were not significantly different between the HFHL and no-HFHL groups (Table 1).

Incidence Rates of Hearing Loss

Forty-seven patients (50%) had HFHL on the ipsilateral side of surgery, and 24 (25.5%) patients had HFHL on the contralateral side postoperatively (Table 2). Of the 47 patients with ipsilateral HFHL, 9 had nonserviceable hearing loss and 13 patients had conductive hearing loss. In contrast, only 2 patients without HFHL had conductive hearing loss and none had Class C/D hearing (p = 0.0036 and p = 0.0026, respectively). The incidence of HFHL alone was 26.6% (25 patients) (Table 3).

Of patients in whom there was a change in threshold at 4 kHz, 91% and 100% had changes at 8 kHz on the ipsilateral and contralateral side, respectively. Of those who had hearing loss at 0.5–2 kHz, 88.9% and 87.5% had changes at 4 kHz and/or 8 kHz in the ipsilateral and contralateral ear, respectively. With increasing tone frequencies, an increasing proportion of patients demonstrated hearing loss at postoperative audiometry; half the study group had significantly increased thresholds at 4 kHz and/or 8 kHz. The mean postoperative changes in pure tone audiometry were greater in the ipsilateral ear compared with the contralateral ear (Tables 2 and 4, Fig. 1).

Three patients (3.19%) exhibited contralateral HFHL but without ipsilateral HFHL.

Intraoperative BAEP Changes and Correlation to Postoperative Hearing Loss

Maximal change in IPL wI–V, when compared with baseline values, was significantly different between groups with and without HFHL (p < 0.05) (Table 5). No significant difference between groups was present at any time during MVD for the latency of wI, wIII, wV, and amplitudes of wIII and wV. No significant difference in the IPL wI–III and wIII–V was observed except in IPL wI–V. Stepwise logistic regression analysis did not identify a significant intraoperative variable to increase the odds of HFHL (p = not significant).

Discussion

Hearing loss after MVD for HFS may occur for the following reasons: 1) stretching of cranial nerve VIII when retracting the cerebellum, 2) manipulation of the labyrinthine artery and/or the anterior inferior cerebellar artery, 3) direct trauma to the nerve by instruments or nearby coagulation, and 4) neurocompression of the nerve by the prosthesis interposed between the offending vessel and the cranial nerve VII complex at the end of surgery.26 However, drill-generated noise, by a mechanism of acoustic trauma, can also cause or aggravate sensory-neural hearing loss and deteriorate bone-conduction thresholds.2,10,13,25 Noise levels ranging from 120 to 122 dB-SPL during drilling in cortical bone and from 117 to 121 dB-SPL during drilling in the mastoid cavity have been reported.33 These sound levels are equivalent to an amplified rock concert in front of speakers, or a nearby airplane engine. The exposure to high-intensity noise damages the hair cells of the cochlear structures. At the subcellular level, the tip links of a hair cell, which are thought to gate mechanoelectrical transduction channels, are broken secondary to high-intensity noise. Broken tip links were detected after acoustic overstimulation, which resulted in reduced hair bundle stiffness, disrupted mechanoelectric transduction

### Table 1: Characteristics of the patients*

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Ipsilateral</th>
<th>Contralateral</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HFHL (n = 47)</td>
<td>NHFHL (n = 47)</td>
</tr>
<tr>
<td>sex (M/F)</td>
<td>17:30</td>
<td>16:31</td>
</tr>
<tr>
<td>age (yrs)†</td>
<td>50.85 ± 14.11</td>
<td>52.40 ± 10.94</td>
</tr>
<tr>
<td>side (lt/rt)</td>
<td>29:18</td>
<td>22:25</td>
</tr>
</tbody>
</table>

* NHFHL = no HFHL.
† Values presented as the mean ± SD.

### Table 2: Incidence of hearing loss at various frequencies after MVD*

<table>
<thead>
<tr>
<th>Audiometric Frequency (kHz)</th>
<th>Ipsilateral</th>
<th>Contralateral</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HL†</td>
<td>NHL†</td>
</tr>
<tr>
<td>0.25</td>
<td>23</td>
<td>71</td>
</tr>
<tr>
<td>0.5–2</td>
<td>27</td>
<td>67</td>
</tr>
<tr>
<td>4</td>
<td>35</td>
<td>59</td>
</tr>
<tr>
<td>8</td>
<td>43</td>
<td>51</td>
</tr>
<tr>
<td>4 &amp;/or 8</td>
<td>47</td>
<td>47</td>
</tr>
</tbody>
</table>

* HL = hearing loss; NHL = no hearing loss.
† Values represent the number of patients.
Reduced blood flow and free radical formation in the cochlea were also regarded as essential mechanisms in noise-induced hearing loss. Although conductive hearing loss and non-serviceable hearing loss can affect postoperative audiometric testing, we found a significant number of patients with exclusive HFHL, suggesting that mechanisms independent of direct iatrogenic auditory nerve trauma are at play. Farzanegan et al. prospectively evaluated 39 consecutive patients who underwent elective craniotomy for supratentorial brain tumors with different locations and compared their auditory status by pure tone audiometry and speech recognition thresholds before and after craniotomy. It was found that drill-generated noise during craniotomy significantly affected hearing function.

Audiometric testing in our study showed that most patients had normal hearing levels at lower and middle frequencies (0.5–2 kHz) after surgery. However, an increasing percentage of patients experienced significant postoperative pure tone threshold changes characteristic of hearing loss at higher tone frequencies (4 kHz and/or 8 kHz) consistent with other reported studies. It has been shown that the short outer hair cells of the high-frequency region were more vulnerable to sound stimulation than the taller receptor cells in the low-frequency area. It has been proposed that higher frequencies may be more sensitive than lower frequencies to noise, acoustic trauma, or ototoxic substances. High-frequency hearing loss may not be detected without an audiometric evaluation, and, importantly, early diagnosis of HFHL may predict hearing loss in lower frequencies, even though it is still not completely resolved.

We found a high incidence of HFHL in the ear contralateral to the affected side. Hearing function in either the operated or contralateral ear has been shown to be affected temporarily by the mastoid drilling. In one study, da Cruz et al. evaluated hearing loss in the nonoperated ear in 12 patients after ear surgery. Drill-related outer hair cell dysfunction was seen in 2 of 12 cases. The authors suggested that this hearing loss was caused by drill noise conducted to the ear by vibration of the intact cranium. Interestingly, there were fewer patients who had a change in pure tone audiometry for 4 kHz compared with 8 kHz in both the ipsilateral and contralateral ear. This could be due to the fact that higher-frequency hair cells are damaged first followed by the lower-frequency hair cells. Additionally, we found that 3 patients had contralateral HFHL without ipsilateral HFHL, implying other mechanisms for HFHL such as transient loss of CSF and so on.

Most studies have suggested that a latency prolongation or a decrease in amplitude of wV of the BAEP is a predictor of hearing loss. However, no correlation between intraoperative BAEP changes and HFHL has been reported. In our study, maximal change in IPL w–V, compared with baseline, was the only variable significantly different for patients with and without HFHL, implying other mechanisms for HFHL such as transient loss of CSF and so on.

Most studies have suggested that a latency prolongation or a decrease in amplitude of wV of the BAEP is a predictor of hearing loss. However, no correlation between intraoperative BAEP changes and HFHL has been reported. In our study, maximal change in IPL w–V, compared with baseline, was the only variable significantly different for patients with and without HFHL. The change in IPL w–V represents the stretching of the auditory nerve due to retraction of the cerebellum. Significant changes in the IPL may be secondary to stretch injury in the retrocochlear segment of the auditory nerve. Additionally, there was no significant difference between the groups with and without HFHL.

Fig. 1. Mean pure tone threshold of patients before and after MVD for HFS in the ipsilateral (IPSI) and contralateral (CONTRA) ear. With increasing tone frequencies, an increasing proportion of patients exhibited hearing loss during postoperative audiometry. The mean postoperative changes in pure tone audiometry were greater in the ipsilateral ear than in the contralateral ear.

TABLE 3: Incidence of other hearing loss in addition to HFHL in the ipsilateral ear after MVD

<table>
<thead>
<tr>
<th>Criteria</th>
<th>HFHL (n = 47)</th>
<th>NHFHL (n = 47)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>conductive hearing loss</td>
<td>13</td>
<td>2</td>
<td>0.0036</td>
</tr>
<tr>
<td>AAO-HNS Class C/D</td>
<td>9</td>
<td>0</td>
<td>0.0026</td>
</tr>
</tbody>
</table>

High-frequency hearing loss after MVD for HFS

TABLE 4: Mean pure tone threshold in patients before and after MVD

<table>
<thead>
<tr>
<th>Audimetric Frequency (kHz)</th>
<th>Preop (dB)</th>
<th>Postop (dB)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ipsilateral</td>
<td>Contralateral</td>
</tr>
<tr>
<td>0.25</td>
<td>16.51 ± 6.93</td>
<td>15.2 ± 6.07</td>
</tr>
<tr>
<td>0.5–2</td>
<td>15.16 ± 8.60</td>
<td>16.06 ± 9.01</td>
</tr>
<tr>
<td>4</td>
<td>24.31 ± 17.07</td>
<td>24.26 ± 16.74</td>
</tr>
<tr>
<td>8</td>
<td>23.72 ± 18.96</td>
<td>22.77 ± 18.86</td>
</tr>
</tbody>
</table>

without HFHL during any stage of MVD for any other BAEP waveform latency or amplitude measurements including wI, wIII, and wV and IPLs. Moreover, logistic regression analysis did not identify a significant intraoperative variable that would increase the odds of HFHL. Thus, HFHL probably was not secondary to stretching of the auditory nerve during MVD but likely induced by high-intensity drilling noise during craniotomy early on in the procedure.

Studies performed on hair cells in animal models have reported evidence of reestablished signal transduction upon regeneration of the tip links of hair cells in cultured explants 6 hours after the stimulus or in vivo after 24–120 hours. The time course of hair cell tip link regeneration suggests that this process may underlie recovery from temporary threshold shift induced by noise exposure. The recovery may take place over a 2- to 4-week period. We did not have long-term follow-up audiograms to evaluate hearing improvement. Karatas et al. noted the temporary influence of the drill noise on the contralateral ear with the deteriorated amplitudes on Day 0 and progressive improvement leading to preoperative normal values within 72–96 hours. Although our study was limited because of its retrospective nature and lacked long-term follow-up audiograms, we highlight a very important clinical change in hearing function at high frequencies immediately after MVD.

TABLE 5: Significant changes in BAEP responses between HFHL and no-HFHL groups at high frequency*

<table>
<thead>
<tr>
<th>Variable</th>
<th>HFHL†</th>
<th>NHFHL†</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>latency wV (msec)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>baseline</td>
<td>6.52 ± 0.51</td>
<td>6.51 ± 0.38</td>
<td>0.817</td>
</tr>
<tr>
<td>change maximum</td>
<td>9.53 ± 1.90</td>
<td>8.87 ± 1.52</td>
<td>0.14</td>
</tr>
<tr>
<td>change maximum vs baseline</td>
<td>146.53 ± 30.39</td>
<td>136.52 ± 21.44</td>
<td>0.25</td>
</tr>
<tr>
<td>amplitude wV (µV)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>baseline</td>
<td>0.32 ± 0.14</td>
<td>0.34 ± 0.10</td>
<td>0.466</td>
</tr>
<tr>
<td>change maximum</td>
<td>0.14 ± 0.13</td>
<td>0.17 ± 0.11</td>
<td>0.155</td>
</tr>
<tr>
<td>change maximum vs baseline</td>
<td>43.96 ± 43.78</td>
<td>54.13 ± 36.43</td>
<td>0.104</td>
</tr>
<tr>
<td>IPL wI–V (msec)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>baseline</td>
<td>4.44 ± 0.44</td>
<td>4.49 ± 0.39</td>
<td>0.445</td>
</tr>
<tr>
<td>change maximum</td>
<td>7.41 ± 2.12</td>
<td>6.57 ± 1.52</td>
<td>0.096</td>
</tr>
<tr>
<td>change maximum vs baseline</td>
<td>168.85 ± 50.83</td>
<td>147.37 ± 33.46</td>
<td>0.047‡</td>
</tr>
</tbody>
</table>

* The percentage change of change maximum (a) versus baseline (b) is determined by the following formula: 100 × (a/b).
† Values are presented as the mean ± SD.
‡ Statistically significant.

Conclusions

High-frequency hearing loss occurs in a significant number of patients following MVD for HFS. Drill-induced noise and transient loss of CSF during the surgery may impair hearing in the high-frequency ranges on both the ipsilateral and contralateral sides, with the ipsilateral side being more affected. Changes in intraoperative BAEPs during MVD for HFS were not useful for predicting HFHL. Follow-up studies and repeat audiological examinations may be helpful in evaluating the time course and prognosis of HFHL. Prospective studies focusing on decreasing intraoperative noise exposure, as well as auditory shielding devices, will establish causation and allow the team to intervene appropriately to decrease the risk of HFHL.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: Thirumala. Acquisition of data: Nikonow, Wichman, Holmes. Analysis and interpretation of data: Ying, Chang. Drafting the article: Ying. Critically revising the article: all authors. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Thirumala. Statistical analysis: Ying. Administrative/technical/material support: Shah, Nikonow, Hirsch.
References


Manuscript submitted June 10, 2012. Accepted January 8, 2013.

Please include this information when citing this paper: published online February 8, 2013; DOI: 10.3171/2013.1.JNS121153.

Address correspondence to: Parthasarathy D. Thirumala, M.D., Center for Clinical Neurophysiology, Department of Neurological Surgery, UPMC Presbyterian, Suite B-400, 200 Lothrop Street, Pittsburgh, Pennsylvania 15213, email: thirumalapd@upmc.edu.