The Incidence of Early Postoperative Conductive Hearing Loss after Microvascular Decompression of Hemifacial Spasm

Tingting Ying¹ Parthasarathy Thirumala^{2,3} Paul Gardner² Miguel Habeych² Donald Crammond² Jeffrey Balzer²

¹ Department of Neurological Surgery, Xinhua Hospital, Shanghai Jiaotong University School of Medicine, Shanghai, China

² Department of Neurological Surgery, University of Pittsburgh, Pittsburgh, Pennsylvania, United States

³ Department of Neurology, University of Pittsburgh, Pittsburgh, Pennsylvania, United States

Address for correspondence Parthasarathy D. Thirumala, MD, MS, Department of Neurological Surgery, UPMC Presbyterian-Suite B-400, 200 Lothrop Street, Pittsburgh, PA 15213, United States (e-mail: thirumalapd@upmc.edu).

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Abstract

Objectives To evaluate the incidence and discuss the pathogenesis of early postoperative conductive hearing loss (CHL) after microvascular decompression (MVD) for hemifacial spasm (HFS).

Design Pre- and postoperative audiogram data and brainstem auditory evoked potentials (BAEPs) from patients who had undergone MVD for HFS were analyzed.

Setting The study was conducted at the University of Pittsburgh Medical Center.

Participants MVD for HFS patients who had pre- and postoperative audiogram data, BAEP data, and normal structure of the external and middle ear were included in the study.

Main Outcome Measures CHL was diagnosed if there was an air-bone gap in pure tone audiometry of at least 10 dB at 0.5, 1, 2, or 4 kHz.

Results The incidence of early postoperative CHL in the ipsilateral ear was 18.7% postoperatively. No CHL was observed in the contralateral side. No significant relationship between CHL and intraoperative BAEP changes was found. Demographic parameters were not significantly different between groups with and without CHL.

Conclusions Early postoperative CHL is a significant complication after MVD. Fluid entering the mastoid air cells and/or bone-dust deposition during craniotomy may result in CHL. Long-term audiograms will be needed to evaluate the risk factors that lead to permanent CHL.

Keywords

- ▶ air-bone gap
- ▶ brainstem auditory evoked potentials
- ▶ conductive hearing loss
- ▶ hemifacial spasm
- ▶ microvascular decompression

Introduction

Hearing loss (HL) is a well-recognized complication of microvascular decompression (MVD) for hemifacial spasm (HFS).^{1–5} Sensorineural hearing loss may result from stretching of the eighth nerve, manipulation of the artery, or direct trauma to the nerve. The incidence or pathogenesis of early

postoperative conductive hearing loss (CHL) after MVD has not been documented previously. CHL might not be a major complication after MVD for HFS, but it is a situation often encountered in practice that can make patients and caregivers anxious. For the CHL patients, sounds are not only softer but may seem muffled or garbled, and they may find it harder to separate one sound from another and interpret the

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meaning of speech and sounds. Although most cases resolve, permanent CHL can be treated with either surgery or hearing aids with good audiometric results.⁶ Currently it is unclear which subset of patients have permanent CHL. CHL is diagnosed if there is an increased air-bone gap (ABG) on pure tone audiometry (PTA) of at least 10 dB at any octave frequency from 0.5 to 4 kHz.⁷ The primary aim of this study was to evaluate the incidence and discuss the pathogenesis of early CHL after MVD for HFS. To our knowledge no study to date has documented this change after MVD for HFS.

Materials and Methods

Study Design

In this retrospective study, 151 consecutive cases of patients with HFS who underwent retromastoid MVD at the University of Pittsburgh Medical Center (UPMC) between 2000 and 2007 were reviewed. Inclusion criteria included (1) patients who had MVD for HFS, diagnosed based on preoperative electromyography (EMG) testing, (2) documented pre- and postoperative evaluation of hearing function, (3) availability of intraoperative neurophysiologic brainstem auditory evoked potential (BAEP) responses for analysis, and (4) normal structure of the external and middle ear. The study was approved by the institutional review board for a retrospective review of data on human participants at the University of Pittsburgh (OD08120394-04/PRO08120394).

Microvascular Decompression Surgery

After induction of anesthesia using a short-acting neuromuscular junction blocking medication, no additional paralytic agent was administered during EMG monitoring. MVD was performed in the lateral decubitus position through a retromastoid craniotomy. When the offending vessel was moved away from the seventh cranial nerve, soft shredded Teflon was pledged at the site of vascular compression.

Preoperative Auditory Investigation

PTA was considered an objective indicator of auditory function. It was performed both in air conduction and bone conduction on all patients before and after surgery. PTA was conducted in a double-walled sound-treated booth with standard procedures and equipment (Grason Stadler Milford [Milford, New Hampshire, United States], NHIGSI 16 audiometer with TDH-BOP headphones, calibrated to American National Standards Institute [ANSI] S36-1989). Hearing thresholds were obtained for octave frequencies from 0.25 kHz to 8 kHz. Bone-conduction thresholds were measured using masked or unmasked conditions. When the unmasked ABG exceeded 10 dB in a given participant, effective masking levels of narrowband noise were applied to the nontest ear during bone-conduction testing to ensure against participation of that ear in the determination of threshold. CHL was diagnosed via observation of an ABG on audiometry, at any octave frequency from 0.5 to 4 kHz. The ABG should be at least 10 dB.⁷ A speech discrimination score (SDS) was also used to analyze hearing status. A decrease in SDS $\geq$ 15% by itself (SDS score $>$ 15% only), within the speech range of

frequencies, is considered HL.² For the purposes of this article, we defined high-frequency hearing loss (HFHL) as an increase in the threshold $>$ 10 dB at 4 kHz and 8 kHz.^{4,8} Nonserviceable HL (class C/D) was defined as PTA $>$ 50 dB and/or SDS $<$ 50% using the American Academy of Otolaryngology-Head and Neck Surgery (AAO-HNS) classification system.⁹ The audiological examination was performed on all patients before surgery (median: 1 day; range: 1–16 days) and after surgery (median: 7 days; range: 2–16 days).

Intraoperative Brainstem Auditory Evoked Potential Monitoring

Stimulation and Recording

All patients undergoing MVD underwent preoperative baseline BAEPs and continuous intraoperative BAEP monitoring. The ipsilateral ears were stimulated, using alternating rarefaction and condensation clicks with at least an 85 dB sound pressure level (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 ms. At least 512 stimuli were delivered for each average. The recording electrodes were positioned as follows: channel (Ch) 1: vertex to left ear mastoid (Cz/A1); Ch 2: vertex to right mastoid (Cz/A2); and Ch 3: vertex to cervical C2 (Cz/Cv2). Amplifier band pass was 100 to 1000 Hz for all channels. Baseline responses were obtained after anesthesia induction and patient positioning.

Monitoring and Alarm Criteria

Persistent decreases in amplitude $>$ 50% of wave V are considered clinically significant.¹⁰ Additionally, a significant clinical change is also defined as a persistent absolute latency, increase of the peak of wave V $\geq$ 0.5 ms.³ "Persistent changes" represent changes in amplitude or latency of the BAEPs when compared with baseline responses in more than two averaged trials. The primary reason for using more than two trials is to eliminate technical issues like noise. Henceforth these threshold signal changes are referred to as "significant." Physician oversight and interpretation was performed using a combined on-site and remote model utilized by UPMC.¹¹

Analysis of Neurophysiologic Parameters

Wave I, III, and V latencies (LwI, LwIII, and LwV), interpeak latencies (IPLs) between waveforms I–III, III–V, I–V, amplitudes of wIII and wV (AwIII and AwV), were measured. These parameters were noted separately for analysis at the following times during the procedure: (1) baseline: after intubation and positioning of the patient; (2) dura open: the period when the opening of the dura was documented; (3) change MAX: the maximum change in latency or amplitude recorded as compared with baseline waveform average including the period when the responses were completely lost; (4) on skin: the last period recorded.

Statistical Analysis

The prognostic value of intraoperative BAEPs was estimated by correlating BAEP changes during surgery with pre- and

Table 1 Patient characteristics

Characteristics	CHL (<i>n</i> = 29)	NCHL (<i>n</i> = 122)
Gender, M/F	8/21	46/76
Age, y	50.34 ± 10.07	51.26 ± 12.95
Side, L/R	17/12	66/56

Abbreviations: CHL, conductive hearing loss; NCHL, no conductive hearing loss.

postoperative audiometry. Preoperative and postoperative hearing results were compared in each group using the Wilcoxon signed rank test and chi-square test. Analyses were performed with SAS v.9.1.3 (SAS Institute, Cary, North Carolina, United States) and were two sided at $\alpha = 0.05$.

Results

Demographic Variables

A total of 151 participants were included in the study. Twenty-nine patients (18.7%) were diagnosed with a postoperative CHL due to the presence of an ABG in the ipsilateral ear, and among them 13 had a mixed HL. No CHL were observed in the contralateral side. Gender, age, and side were not significantly different between groups with and without CHL (►Table 1).

Incidence Rates of Conductive Hearing Loss

►Table 2 lists the average ABGs in each frequency and the air-conductive thresholds. Of the 151 patients, the incidence of CHL at 0.5 kHz and 1 kHz was 17.22%. And 11.26% and 15.23% of patients had an ABG at a frequency of 2 kHz and 4 kHz, respectively (►Table 3).

Of the 29 patients with CHL, 3 were classified as having nonserviceable hearing (class C/D) by the AAO-HNS classification system, which also combined with sensorineural HL. Four of 29 patients who had CHL were diagnosed with HL based on changes in SDS (►Table 4).

Of the 29 patients who had CHL, 24 patients also had HFHL. The patients with CHL were significantly associated with HFHL status ($p < 0.01$) (►Table 3). Only one patient with CHL had no other HL. No CHL was reported in the contralateral side.

Table 2 Air-conductive thresholds of conductive hearing loss patients before and after microvascular decompression ipsilaterally

Frequency	Preoperative, dB, mean ± SD	Postoperative, dB, mean ± SD
0.25 kHz	16.38 ± 10.77	32.41 ± 18.74
0.5 kHz	15.70 ± 11.30	34.00 ± 18.70
1 kHz	15.00 ± 11.00	35.00 ± 16.00
2 kHz	16.00 ± 8.70	37.00 ± 18.00
4 kHz	23.80 ± 17.80	52.80 ± 24.80
8 kHz	26.40 ± 21.00	54.70 ± 20.90

Abbreviation: SD = standard deviation.

Table 3 Air-bone gaps of conductive hearing loss patients in different frequencies after microvascular decompression ipsilaterally

Frequency	<i>N</i> (%)	Air-bone gap, dB, mean ± SD
0.5 kHz	26 (17.22)	19.0 ± 10.0
1 kHz	26 (17.22)	19.7 ± 10.7
2 kHz	17 (11.26)	10.2 ± 8.2
4 kHz	23 (15.23)	17.1 ± 12.2
All frequencies	12 (7.95)	20.5 ± 7.58

Abbreviation: SD, standard deviation.

Intraoperative BAEP Changes and Correlation to Postoperative Hearing Loss

No significant difference between groups was present at any time during MVD for the latency of waves I, III, V, IPL, and amplitudes of waves I, III, and V. No significant differences were noted in IPL I–III, I–V, and III–V (►Table 5). Stepwise logistic regression analysis did not identify a significant intraoperative variable to increase the odds of CHL.

Discussion

CHL occurs when sound is not conducted efficiently due to a mechanical problem in the outer or middle ear. Three tiny bones of the ear (ossicles) may not conduct sound properly, the eardrum may not vibrate in response to sound, or fluid in the middle ear can cause this type of HL. It was reported that craniotomy might also result in CHL, due to the fluid deposition and/or bone dust in the middle ear.^{12–14}

During craniotomy the use of a high-speed drill results in production of large quantities of fine bone dust. Intermittent suction irrigation with a saline solution can be used to remove it. In some cases, the mastoid cells are opened due to the surgical exposure, letting the irrigating solution escape the suction and enter the middle ear through the aditus of the mastoid. The acoustic pathophysiology involved in this type of HL is similar to that encountered in an effusion in

Table 4 Postoperative hearing status of conductive hearing loss patients after microvascular decompression

Criteria	CHL (total <i>n</i> = 29)	NCHL (total <i>n</i> = 122)
AAO-HNS class C/D	3	13
SDS	4	15
HFHL	24	53
Pure CHL	1	0
Subjective HL	5	14

Abbreviations: AAO-HNS, hearing loss status assessed by 1995 American Academy of Otolaryngology-Head and Neck Surgery classification system; CHL, conductive hearing loss; HFHL, high-frequency hearing loss; HL, hearing loss; NCHL, no conductive hearing loss; SDS, speech discrimination score.

Table 5 Significant changes in brainstem auditory evoked potential responses between conductive hearing loss and no conductive loss groups

Variables		CHL, mean ± SD	NCHL, mean ± SD	p value
Latency wV, ms	Baseline	6.51 ± 0.61	6.51 ± 0.40	0.925
	Change MAX ^a	9.32 ± 1.77	9.24 ± 1.762	0.874
	Change MAX vs baseline	144.76 ± 28.85	141.89 ± 26.74	0.709
Amplitude wV, uV	Baseline	0.32 ± 0.16	0.34 ± 0.12	0.594
	Change MAX	0.15 ± 0.12	0.16 ± 0.13	0.887
	Change MAX vs baseline	36.29 ± 28.92	50.30 ± 42.37	0.226
IPL wI–V, ms	Baseline	4.50 ± 0.53	4.45 ± 0.39	0.637
	Change MAX	7.06 ± 1.64	7.03 ± 1.95	0.955
	Change MAX vs baseline	162.12 ± 37.56	158.12 ± 46.21	0.762

Abbreviations: CHL, conductive hearing loss; IPL wI–V, interpeak latency between wave I and V; NCHL, no conductive hearing loss; SD, standard deviation; wV, wave V.
^aThe percentage change of change MAX (a) versus baseline (b) is obtained by this equation: 100*(a/b).

the middle ear. Furthermore, in a partially fluid-filled ear, patients often report improvement or worsening of hearing simply by changing head position, without any insufflation of air. The latter observation of improved hearing with change in head position can be explained by fluid moving away from the tympanic membrane (TM) into other parts of the middle ear or mastoid.¹⁵ Although fluid drainage can occur resulting in resolution of the CHL, it is unclear which subset of patients will eventually have permanent CHL.

The ABG changes were observed at all octave frequencies from 0.5 to 4 kHz. Alternative forms of CHL may affect sound transmission for various frequencies differently and may thereby affect the recorded responses in a dissimilar way.¹⁶ It was reported that the primary mechanism responsible for the reduction in umbo velocity at medium and high frequencies by middle ear fluid is the increase in effective TM mass by fluid entrained to TM motion. The reduction in middle ear compliance by a reduction in middle ear air volume is the primary mechanism responsible for the reduction in umbo velocity at low frequencies by middle ear fluid.¹⁵

It was agreed that significant delays were seen for wave V as well as for the I–V and III–V interpeak intervals in patients with CHL.¹⁷ The increase in the latency of the BAEPs correlated with the CHL, and the decrease in latency correlated with the improvement in hearing.¹⁸ However, no significant difference between groups was present at any time during MVD for the BAEP test in this study. It was probable that both the patients with or without CHL had other kinds of HL.

Devesa et al¹⁴ reported five cases of ossicular fixation due to bone-dust deposition causing CHL after procedures that involved drilling the mastoid bone without the intention of entering the middle ear. Bone dust can cause a postoperative conductive hearing deficit that becomes apparent 6 months following surgery, and progressive deterioration can occur up to 18 months.^{13,14,19,20} However, the incidence of CHLs produced by ossicular fixation, resulting from deposition of bone-dust particles into the middle ear during MVD for HFS,

is not known. It is also unclear whether there are clinical or audiometric signs that can identify this subset of patients. Because our hearing data reflect early postoperative results, the CHL in this study could be mainly secondary to fluid entering the mastoid air cells. Generally, CHL can be treated with good audiometric results with either surgery or hearing aids.⁶ Although the results of long-term follow-up audiograms in this study are not yet known, the results clearly underscore a very significant finding after MVD. To prevent CHL postoperatively, early closure of the mastoid antrum with a piece of Gelfoam or bone wax has been recommended during the course of microvascular surgery. This prevents liquid from entering the mastoid air cell and accumulation in the attic and middle ear.^{20,21} Although our study was not designed to address the exact etiology of CHL, this condition clearly needs to be recognized to further evaluate if this is a complication that resolves spontaneously or leads to permanent HL.

Our study was limited because we did not have long-term follow-up audiograms. It was a retrospective analysis of the data limiting the amount of information collected. To overcome these limitations, additional well-designed prospective studies with long-term follow-up and more extensive data about the patients are needed in the future.

Conclusions

Early postoperative CHL is a significant complication after MVD. Fluid entering the mastoid air cells and or bone-dust deposition during a craniotomy may result in CHL. Because other HL patterns exist after MVD with CHL, the diagnosis might be missed without an air-bone pure tone test. Changes in intraoperative BAEPs during MVD were not useful in predicting the incidence of CHL. Long-term audiograms will be needed to evaluate the incidence and the risk factors that lead to permanent CHL. Formal documentation of such cases may allow the development of techniques to reduce its occurrence during surgery.

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