Analysis of interpeak latencies of brainstem auditory evoked potential waveforms during microvascular decompression of cranial nerve VII for hemifacial spasm

Parthasarathy D. Thirumala, M.D., M.S.,1,3 Preethi Ilangovan, M.B.B.S.,1 Miguel Habeych, M.D., M.P.H.,1 Donald J. Crammond, Ph.D.,1 and Jeffrey Balzer, Ph.D.1,2

Departments of 1Neurological Surgery, 2Neuroscience, and 3Neurology, University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania

Object. Microvascular decompression (MVD) of the facial nerve is an effective treatment for patients with hemifacial spasm. Intraoperative monitoring of brainstem auditory evoked potentials (BAEPs) during MVD can reduce the incidence of hearing loss. In this study the authors’ goal was to evaluate changes in interpeak latencies (IPLs) of Waves I–V, Waves III–V, and Waves I–III of BAEP Waveforms I, III, and V during MVD and correlate them with postoperative hearing loss. To date, no such study has been performed. Hearing loss is defined as nonuseful hearing (Class C/D), which is a pure tone average of more than 50 dB and/or speech discrimination score of less than 50%.

Methods. The authors performed a retrospective analysis of IPLs of BAEPs in 93 patients who underwent intraoperative BAEP monitoring during MVD. Patients who did not have hearing loss were in Class A/B and those who had hearing loss were in Class C/D.

Results. Binary logistic regression analysis of independent IPL variables was performed. A maximum change in IPLs of Waves I–III and Waves I–V and on-skin change in IPLs of Waves I–V increases the odds of hearing loss. However, on adjusting the same variables for loss of response, change in IPLs did not increase the odds of hearing loss.

Conclusions. Changes in IPL measurements did not increase the odds of postoperative hearing loss. This information might be helpful in evaluating the value of IPLs as alarm criteria during MVD to prevent hearing loss.

Key Words • brainstem auditory evoked potential • hemifacial spasm • microvascular decompression • interpeak latency • intraoperative neurophysiological monitoring

Hemifacial spasm is due to neurovascular compression at the root exit zone of the facial nerve (CN VII). This condition can be effectively treated by MVD of CN VII.1,5,8,17 An infrequent but significant risk of MVD is hearing loss caused by damage to the vestibulocochlear nerve (CN VIII).1,11,17 Intraoperative neurophysiological monitoring of BAEPs during MVD has reduced the incidence of this complication to around 2.3%–12.3% by alerting the surgeons to potentially reversible situations.1,13,15,19,21 Mechanisms of damage to CN VIII include retraction of the cerebellum resulting in traction on CN VIII, manipulation of its blood supply, direct mechanical/thermal trauma, or neocompression caused by the iatrogenic placement of prosthetic material that compresses the CN VII/VIII complex.16,19 Although there are multiple references to the loss of Wave V and hearing loss, no correlation has been described regarding the value of IPL evaluation during MVD. Interpeak latency is a relatively consistent measurement when compared with absolute latency, which is susceptible to a host of factors (such as age, sex, and intensity of stimulation).2,10 Interpeak latency also eliminates individual variation in the magnitude of the latency of Wave I and the latency of Wave V at baseline and at the point of maximal change. The primary aim of this paper is to analyze the changes in IPL during
MVD and to evaluate if these changes can predict postoperative hearing loss using data collected at University of Pittsburgh Medical Center.

Methods

Study Design

We performed a retrospective analysis of consecutive patients who underwent an MVD procedure for HFS between January 2000 and December 2007 at the University of Pittsburgh Medical Center. Patients with clinical HFS confirmed by electromyographic studies and for whom documented pre- and postoperative hearing function and viable intraoperative neurophysiological BAEP data available were selected for the study. Patients with HFS who had poor preoperative hearing function were excluded from the study. The study was approved by the institutional review board for retrospective review of data on human subjects at the University of Pittsburgh Medical Center.

Microvascular Decompression for HFS

Patients were given short-acting muscle relaxants for intubation; no additional muscle relaxants were adminis-
tered for the purpose of intraoperative electromyographic monitoring of CN VII in order to evaluate the disappearance of lateral spread as a sign of adequate decompression.6,12 Microvascular decompression was performed using a retromastoid craniotomy with the patient in a park bench position, and the patient was maintained on a balance of inhalational and intravenous anesthetic agents. The conflicting vessels were identified, and CN VII was freed by placement of Teflon pledgets.5

Perioperative Audiological Investigations and Hearing Loss Criteria

We considered tone audiometry consisting of PTA and SDSs to be the indicators of auditory function.14,19 An otoneurological examination was performed in all patients before surgery (median 1 day, range 1–49 days) and after surgery (median 7 days, range 1–90 days), consisting of an audiogram with measurement of pure tone thresholds (air and bone conduction for octave frequencies 250–8000 Hz). Values obtained at 500, 1000, and 2000 Hz were used to calculate the PTA in decibels. The SDSs were obtained by monitored live voice using phonemically balanced word lists at 40 dB above the calculated PTA. To avoid misinterpretation of audiometry, postoperative otoscopy and tympanometry were performed to detect conductive hearing loss due to middle ear dysfunction in relation to opening of mastoid cells during craniotomy.

Postoperative hearing loss status was assessed using the 1995 American Academy of Otolaryngology–Head and Neck Surgery classification system.2 Class B refers to useful or serviceable hearing loss, and Class C/D refers to nonserviceable hearing loss that is not amenable to hearing aids (Table 1).3

Intraoperative Monitoring Recordings

All patients who received MVD underwent preop-

<table>
<thead>
<tr>
<th>Hearing Class</th>
<th>PTA (dB)</th>
<th>SDS %</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>&lt;30</td>
<td>&gt;70</td>
</tr>
<tr>
<td>B</td>
<td>30–50</td>
<td>&gt;50</td>
</tr>
<tr>
<td>C</td>
<td>&gt;50</td>
<td>&gt;50</td>
</tr>
<tr>
<td>D</td>
<td>any level</td>
<td>&lt;50</td>
</tr>
</tbody>
</table>

P. D. Thirumala et al.
was estimated by correlating changes during surgery with pre- and postoperative audiometry and SDSs for any hearing loss. With hearing loss so classified (Table 1), we compared IPL variables between Class A/B and Class C/D. Due to skewed distribution, the statistical significance of differences in the delay of waves was determined using the Kruskal-Wallis test, and the Levene test was used to determine if individual group variances were similar. The chi-square test and Fisher exact test for categorical variables were used to examine hearing outcome for changes in the IPL variables. A binary logistic regression analysis was performed on independent IPL variables of Waves I–III and Waves I–V at Change Max and On-Skin. Further analysis of the same variables was performed adjusting for loss of response (waveform loss). Statistical analysis was calculated using SPSS (version 20, IBM SPSS, Inc.).

Results

Incidence of Hearing Loss

Figure 1 displays the outcomes of MVD in our study group. Of 93 patients, 25 (26.88%) had a change in hearing class. Sixteen (17.20%) of these patients were classified in Class B and 9 (9.68%) were classified in Class C/D (nonserviceable hearing loss). A loss of response/waveforms during surgery was observed in 23 patients, of whom 11 recovered during the course of the surgery.

Analysis of IPL Variables in Class A/B Versus Class C/D

Logistic regression analysis of independent IPL variables demonstrated that maximum changes in IPLs of Waves I–III and IPLs of Waves I–V and On-Skin changes IPLs of Waves I–V increases the odds of hearing loss (Table 2). However, on adjusting the same variables for loss of response, IPL variables did not increase the odds of hearing loss (Table 3).

Relationship Between the Latency of Wave V and IPL

Figure 2 shows the relationship between the latency of Wave V and IPLs of Waves I–III, I–V, and III–V for all hearing classes combined at Change Max (no hearing loss and hearing loss). The IPLs of Waves I–V had a strong positive correlation with the latency of Wave V, and the IPLs of Waves I–III and of Waves III–V had a weakly positive and poor correlations, respectively, with the latency of Wave V. Figure 3 shows the comparison of the latency of Wave V and IPLs of Waves I–V for age and sex. Results of the Student t-test analysis of the latency of Wave V and the IPLs of Waves I–V were not significantly different for age and sex.

Discussion

The vestibulocochlear nerve is susceptible to iatrogenic injury during procedures performed in and around the cerebellopontine angle. Hearing loss in MVD is much more preventable compared with other procedures involving the cerebellopontine angle as the manipulation can be stopped, and persistent hearing deficits can be prevented. Conventionally, the latency and amplitude of Wave V were the variables used to monitor CN VIII. However, IPL is a relatively consistent variable when compared with the absolute latency of waveforms, which is susceptible to a host of factors (such as stimulus intensity, age, and sex). In the present study, the changes in the IPLs of Waves I–V seemed to strongly reflect absolute latency of Wave V, making them reliable alternative intraoperative measurement and valid alarm criteria.
The IPLs of Waves I–III represent impulse transmission from the distal portion of CN VIII up to the cochlear nucleus. As MVD procedures primarily involve manipulation of the CN VIII proper or where it exits, any potential insult to CN VIII should affect impulse conduction across this segment of auditory pathway, which would be reflected as prolongation of the IPLs of Waves I–III. Measurement of Wave III is difficult and unreliable during MVD due to a poor signal-to-noise ratio. The changes in IPLs of Waves I–III can indicate very focal changes in the auditory pathways but due to recording difficulty they are typically unreliable in predicting hearing loss during MVD. The apparent increase in the odds of hearing loss by the IPLs of Waves I–III at Change Max was nullified on adjusting it for loss of response. No significant changes in the IPLs of Waves III–V were noted during MVD procedures in this series. The potentials of IPLs of Waves III–V represent the conduction of auditory impulses in the brainstem. In addition, changes in the IPLs of Waves III–V did not correlate well with changes in the latency of Wave V, which makes these IPLs even less valuable during MVD.

The IPLs of Waves I–V reflect total central auditory conduction from the distal part of CN VIII to the inferior colliculus and include components of the IPLs of Waves I–III and of Waves III–V. The latencies of Waves I and V can be measured consistently during MVD with a good signal-to-noise ratio. Traction on CN VIII can increase the latency and decrease the amplitude of these waves. Acutely the increase in latency of a waveform is directly proportional to the magnitude of the stress. In nerve stretch studies, a reversible increase in latency and a decrease in amplitude were not associated with ischemic changes in the nerve. The loss of latency and amplitude for prolonged periods of time is associated with poor recovery, similar to our findings.

Brainstem auditory evoked potential variables to be used as alarm criteria should reliably predict hearing loss during MVD. Although Change Max and On-Skin IPL of Wave I–V changes independently seemed to increase the odds of hearing loss, the results were not consistent after adjusting for loss of response. It is possible that latency changes were present during early retraction of CN VIII and these changes are reversible without hearing loss. However, our current analysis was limited and did not characterize the temporal changes in the latency and amplitude of the BAEP waveforms during MVD. This was a retrospective review of the data, which also limits the amount of information collected and analyzed.

Conclusions

Changes in the IPLs of Waves I–V correlate with
Interpeak latency evaluation and microvascular decompression

changes in the latency of Wave V during MVD. Changes in the IPLs of Waves I–III and of Waves I–V did not increase the odds of hearing loss after MVD after adjusting for the loss of BAEP waveforms. Further analysis with a combination of variables including changes in latency and amplitude of the BAEPs needs to be conducted before an alarm criterion is adopted.

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: Thirumala. Analysis and interpretation of data: Thirumala, Illogovan. Drafting the article: Thirumala, Illogovan. Critically revising the article: Thirumala, Illogovan. Reviewed submitted version of manuscript: Thirumala. Approved the final version of the manuscript on behalf of all authors: Thirumala. Statistical analysis: Thirumala. Administrative/technical/material support: Thirumala, Balzer, Crommond, Habeych. Study supervision: Thirumala.

References


Manuscript submitted November 19, 2012. Accepted December 6, 2012. Portions of the abstract were presented as a poster at the 59th Annual Meeting of the American Association of Neuromuscular & Electrodiagnostic Medicine, October 3–6, 2012, in Orlando, Florida. Please include this information when citing this paper: DOI: 10.3171/2012.12.FOCUS12393.

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

Neurosurg Focus / Volume 34 / March 2013