LIMITATION OF BRAINSTEM AUDITORY EVOKED POTENTIAL MONITORING FOR FACIAL SPASM SURGERY

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SUMMARY

Objective. Postoperative hearing loss is the most serious complication of microvascular decompression for facial spasm and trigeminal neuralgia. To test the usefulness of brainstem auditory evoked potential (BAEP) as an intraoperative monitoring, in relation to hearing loss we compared BAEP records and postoperative hearing. We also compared BAEP data during microvascular decompression to that obtained during acoustic tumor surgery.

Methods. BAEP was monitored every 5 to 10 minutes with a loudness of 100dB and 75Hz per second of frequency. 15 cases of trigeminal neuralgia and 10 cases of facial spasm were monitored during microvascular decompression. 8 cases of acoustic tumor were monitored intraoperatively with BAEP using the same method used with microvascular decompression and the results were compared.

Results. Mean delay of the Vth wave latency was 1.16 msec for facial spasm and 1.15 msec for trigeminal neuralgia. Both were significantly longer than the delay found in acoustic tumor removal, in which hearing was preserved postoperatively. However, hearing disturbance was found in two cases of facial spasm, one severe case of hearing loss and another mild hearing impairment. No hearing impairment was seen in trigeminal neuralgia cases.

Conclusions. Delay of Vth wave latency occurred equally in surgery for facial spasm and trigeminal neuralgia, yet hearing disturbance was found only in surgery for facial spasm. Delay of BAEP may be caused by the retraction of the cochlear nerve, but hearing disturbance might be caused by direct manipulation injury including inflammation and microcirculatory impairment to the cochlear nerve which may occur in surgery for facial spasm and may not be detected by intraoperative BAEP monitoring.

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INTRODUCTION

One of the most serious complications of microvascular decompression is hearing loss. It occurs in a relatively high percentage of operations for hemifacial spasm, and is occasionally seen in trigeminal neuralgia surgery (1, 2, 3, 4). Samii et al (2002) (3) reported 145 consecutive cases of microvascular decompression for hemifacial spasm and found complete loss of hearing in 8.3% and partial hearing impairment in 7.6% of cases postoperatively. The total complication rate of 15.9% for hearing is rather striking. These data indicate that hearing loss remains a major complication of microvascular decompression for hemifacial spasm.

Several clinical studies have indicated that hearing loss can be prevented by monitoring brainstem auditory evoked potential (BAEP) (5, 6, 7, 8, 9). However, Dannenbaum and Lega (2008) (10) indicated that the complication rate of hearing loss was as low as 1.8% among their 114 cases of microvascular decompression for hemifacial spasm, and they proposed that BAEP had no value for intraoperative monitoring of hearing. Therefore, questions still remain about the usefulness of intraoperative BAEP for the prevention of hearing loss.

In recent years, we have been routinely monitoring BAEP during microvascular decompression for facial spasm and trigeminal neuralgia. We have also monitored BAEP during removal of small acoustic neurinoma. To assess the effectiveness of BAEP, we have compiled these BAEP data and compared the results. On the basis of our analyses, we propose that BAEP has limitation as a intraoperative monitoring tool for facial spasm, because mild direct injury to the facial nerve including inflammation and microcirculatory impairment may not be detected by BAEP monitoring.

CLINICAL MATERIALS AND METHODS

Selection of patients: During the last 3 years (2005-2008), we have experienced microvascular decompression for trigeminal neuralgia in 15 cases and hemifacial spasm in 10 cases. All cases were treated through the lateral suboccipital approach, and BAEP was monitored in all cases. Hearing was evaluated preoperatively and postoperatively. During this period, we also experienced 8 cases of small acoustic neurinomas removed through the lateral suboccipital approach, with hearing preserved postoperatively (Table 1). During removal of acoustic neurinoma, BAEP was monitored using the same method used with microvascular decompression. BAEP records for both surgical procedures were compared.

Method for intraoperative BAEP monitoring: We have been using Nicolet Viking Select apparatus (Nicolet, Madison, Wisconsin, USA) as a monitoring instrument. BAEP was monitored every 5 to 10 minutes during surgery with the conditions of stimulation being 100 dB loudness and 75 Hz per second of frequency. Records of BAEP during microvascular decompression were compared to the BAEP taken during the surgery for acoustic neurinoma, in which hearing was preserved. Microvascu-
lar decompression and acoustic neurinoma were all operated on using the same surgical approach. The sole difference was that for trigeminal neuralgia we retracted the cerebellum from the upper lateral aspect to the lower medial direction, for facial spasm and acoustic neurinoma we elevated the cerebellum from the lower lateral aspect to the upper medial direction.

For analysis of BAEP records, we focused on the delay of Vth wave latency and maximum delay was recorded in each patient, since it is always detectable and in the most sensitive marker for acoustic nerve injury (11, 12, 13, 14). After general anesthesia with patients placed in the lateral park-bench position, BAEP was taken and this initial record was used as a standard recording. During decompression procedures the Vth wave latency was compared to the initial recording, and delay of latency was measured and expressed as milliseconds. Statistical analysis was done with Mann-Whitney’s U-test.

RESULTS

During microvascular decompression for trigeminal neuralgia, maximum prolongation of the Vth wave latency was 1.15 msec in mean value. The delay of Vth wave latency for facial spasm was 1.16 msec as a mean value. For acoustic neurinoma, maximum delay of the Vth wave latency was 0.78 msec in mean value (Figure 1). Acoustic neurinoma showed significantly shorter delay of Vth wave latency as compared to trigeminal neuralgia and facial spasm (p < 0.01). Delay of latency for facial spasm and trigeminal neuralgia was about the same and no statistical significance was found (Figure 1).

A typical recording of BAEP for trigeminal neuralgia is shown in Figure 2. This case showed 1.64 msec as the maximal delay of Vth wave latency. The maximal delay occurred during the decompression procedure while retracting the cerebellum. We stopped the procedure for a while, and the

<table>
<thead>
<tr>
<th>Surgery for</th>
<th>cases</th>
<th>age (mean ± S.D.)</th>
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</thead>
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<tr>
<td>trigeminal neuralgia</td>
<td>15</td>
<td>61 ± 12</td>
</tr>
<tr>
<td>hemifacial spasm</td>
<td>10</td>
<td>45 ± 15</td>
</tr>
<tr>
<td>acoustic neurinoma</td>
<td>8</td>
<td>33 ± 9</td>
</tr>
<tr>
<td>total</td>
<td>33</td>
<td>49 ± 17</td>
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Table 1. Patients with intraoperative monitoring of BAEP
Vth wave disappeared (Figure 2B2). The Vth wave reappeared shortly after stopping the procedure and reappeared fully after completion of the procedure, but delay of the Vth wave latency remained at 0.57 msec (Figure 2C). This patient showed no hearing disturbance postoperatively. Surgery for trigeminal neuralgia in 15 cases did not cause any hearing loss or decreased hearing postoperatively.

Typical recording of BAEP for facial spasm is shown in Figure 3. This case showed 1.52 msec as the maximum delay of Vth wave latency, which occurred during the decompression procedure (Figure 3B2). By completion of the procedure, delay of the Vth wave latency recovered gradually, but remained at 0.57 msec at the complication of the procedure (Figure 3C). This patient had no hearing disturbance postoperatively.

Among the 10 cases of facial spasm surgery, we experienced one case with rather severe hearing loss and one case of mild hearing disturbance. The BAEP recording in the case of a severe hearing loss is shown in Figure 4. This case is 59-year-old female with a 5-year history of the left facial spasm. The surgery was performed uneventfully, and Vth wave latency was delayed 1.28 msec at most during the decompression procedure (Figure 4B2). Delay of Vth wave latency shortened to 0.51 msec at the end of the procedure (Figure 4C). The patient had no symptoms for 2 days but complained of rather strong left hearing loss 3 days after surgery (Figure 5). Another case of mild hearing loss occurred in a 60-year-old female with right facial spasm. This case had a neurovascular
FIG. 2. Intraoperative monitoring of a case with trigeminal neuralgia. A: precraniotomy recording, B1: microsurgical procedure started, B2: Retraction of the cerebellum was maximal, B3: microsurgical procedures completed. C: end of surgery. Maximum delay was 1.64 msec. Two vertical lines indicate Vth wave peak time at precraniotomy recording (left) and at maximally delayed time point (right).
Fig. 3. Intraoperative monitoring of a case with facial spasm and hearing preserved. A: precraniotomy recording, B1: initiation of microsurgical procedure, B2: maximal retraction of cerebellum, B3: completion of microsurgical procedures. C: end of surgery. Two vertical lines indicate Vth wave peak time at precraniotomy recording (left) and at maximally delayed time point (right).

Maximum delay of Vth wave was 1.52 msec.
Fig. 4. A case of the left facial spasm surgery with hearing severely impaired postoperatively. A: precraniotomy recording, B1: initiation of microsurgical procedure, B2: maximal retraction of cerebellum, B3: completion of microsurgical procedures. C: end of surgery. Two vertical lines indicate Vth wave peak time at precraniotomy recording (left) and at maximally delayed time point (right). Maximum delay of Vth wave was 1.28 msec but hearing was impaired postoperatively (Figure 5).
Fig. 5. Pre-operative (left) and post-operative (right) audio-grams in a case of the left facial spasm with hearing severely impaired postoperatively (Figure 4). Preop: preoperative recording, Postop: postoperative recording. —○—right hearing, ---X---left hearing.

Fig. 6. Postoperative audiogram in a case of facial spasm with hearing mildly impaired in the right side. —○—right hearing, ---X---left hearing.
compression site slightly at the medial side of the ponto-medillary junction, and we had to retract the
cerebellum more than usual. Postoperative audiogram indicated 30-50dB hearing loss at 125dB to 500
dB (Figure 6).

DISCUSSION

We selected the delay of latency period for the Vth wave of the BAEP as a marker for monitoring, because this is the most easily recordable and stable marker (2, 4, 7). Some reports have indicated the amplitude of the Vth wave as a useful marker of the monitoring (13), but it may depend on monitoring conditions. Therefore, a majority of reports have chosen latency of the Vth wave as a marker of hearing.

The most striking finding of this study is that BAEP was delayed equally among cases of hemifacial spasm and trigeminal neuralgia, yet hearing impairment occurred only in cases of hemifacial spasm. Because the trigeminal nerve is 10-15 mm apart from the cochlear nerve, we originally speculated that BAEP might not alter during trigeminal neuralgia surgery. However, BAEP latency during surgery of the trigeminal neuralgia delayed as equally as that of the facial spasm. Previous reports describe a delay of latency of the Vth wave in surgery for trigeminal neuralgia (2, 6, 15), but no report has found a delay equal to that in surgery for facial spasm. This suggests that delay of Vth wave latency is caused by traction of the cochlear nerve as a result of cerebellar retraction. In considering the delay for trigeminal neuralgia and facial spasm, traction of the cerebellum is needed equally in surgery for both. Although surgery for facial spasm requires retraction from the lower lateral to the upper medial direction and trigeminal neuralgia needs upper lateral to lower medial retraction, this difference did not affect the BAEP monitoring. As the Vth wave delay was significantly shorter during acoustic tumor surgery than surgery for hemifacial spasm and trigeminal neuralgia, the tumor itself may act as an anchor of the cochlear nerve (Figure 1).

As for trigeminal neuralgia is concerned, majority of the previous report indicated less incidence of postoperative hearing disturbance (2, 6) as compared to the facial spasm (1, 3, 4). In the present report, we have no hearing loss among 15 cases of trigeminal neuralgia. We also experienced no hearing loss among 115 cases of trigeminal neuralgia operated on without BAEP monitoring (personal records of one (KY) of the authors). Therefore, we are not sure whether BAEP is a useful monitoring tool for hearing during trigeminal neuralgia surgery.

If traction to the cochlear nerve is equal in facial spasm and trigeminal neuralgia, some other factor must affect postoperative hearing loss in facial spasm. It is most likely due to direct cochlear nerve manipulation, which causes slowly progressing nerve injury. As the cochlear nerve runs parallel to the facial nerve, surgical manipulation of the facial nerve may easily affect the cochlear nerve. As for small acoustic tumor surgery, BAEP often disappears suddenly while dissecting the tumor from the cochlear nerve, suggesting direct injury to the cochlear nerve. However, in our case of hearing loss
Figure 4), BAEP did not disappear, and delay of Vth wave latency was 1.28 msec, similar to the mean value of 1.16 msec. This case complained of hearing loss not immediately after surgery but 3 days after surgery. Therefore slowly progressing nerve injury may be the cause of hearing loss as has been reported previously (15). The reported case started hearing loss 3 days postoperatively and progressed over two weeks. The case was the second surgery for trigeminal neuralgia. Therefore, the authors suggested scar formation as a possible cause of slowly developing hearing loss. This postoperative course was similar to our case as depicted in Figure 4 and Figure 5, and similar mechanism might be the cause of hearing loss in our case.

Another possibility is microcirculation injury. We often find small perforators arising from compressing vessel loop and running to the brain stem and cochlear nerve. These perforating vessels may be stretched by microvascular decompression procedure and gradual functional impairment may develop as a result of microcirculatory impairment.

In summary, BAEP monitoring for facial spasm surgery may have limitation on its reliability and we must be careful to avoid slowly progressing inflammation and scar formation of the cochlear nerve or to avoid microcirculation injury to the brain stem and cochlear nerve.

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