Intraoperative monitoring of brain-stem auditory evoked potentials

BETTY L. GRUNDY, M.D., PETER J. JANETTA, M.D., PHYLLIS T. PROCOPIO, B.A., AGNES LINA, M.D., J. ROBERT BOSTON, PH.D., AND EARL DOYLE

Departments of Anesthesiology/Critical Care Medicine and Neurological Surgery, University of Pittsburgh, Pittsburgh, Pennsylvania

Brain-stem auditory evoked potentials (BAEP) were monitored during 54 neurosurgical operations in the cerebellopontine angle. The BAEP were irreversibly obliterated in five patients who required deliberate section of the auditory nerve. Technical difficulties interfered with monitoring in three cases, and three patients had deafness and absent BAEP preoperatively.

Reversible alterations in BAEP were seen during 32 operations, with recovery after as long as 177 minutes of virtually complete obliteration. Changes in BAEP were associated with surgical retraction, operative manipulation, positioning of the head and neck for retromastoid craniectomy, and the combination of hypocarbia and moderate hypotension. In 19 cases, waveforms improved after specific interventions made by the surgeon or anesthesiologist because of deteriorating BAEP. In 13 other cases, BAEP recovered after maneuvers not specifically related to the electrophysiological monitoring, most often completion of operative manipulation. Whenever BAEP returned toward normal by the end of anesthesia, even after transient obliteration, hearing was preserved. Irreversible loss of BAEP occurred only when the auditory nerve was deliberately sacrificed. The authors conclude that monitoring of BAEP may help prevent injury to the auditory nerve and brain stem during operations in the cerebellopontine angle.

KEY WORDS • auditory evoked potentials • posterior cranial fossa • brain stem • cranial nerve disease • intraoperative complications
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FIG. 1. Normal brain-stem auditory evoked potentials recorded in the operating room prior to induction of anesthesia for retromastoid craniectomy and microvascular decompression of the fifth cranial nerve. Recording was from the vertex to the ear ipsilateral to click stimulation (CZ-A1). Peaks are labeled to show customary designations. Purported generators of the individual peaks are listed in Table 1. Stimulation and recording parameters are shown in Table 2.

Clinical Material and Methods

Patient Population

Brain-stem auditory evoked potentials were monitored during 54 operations in 50 patients (Table 3). There were 20 males and 30 females, ranging in age from 22 to 69 years. Physical status according to the classification of the American Society of Anesthesiologists ranged from I to IV. Hearing was clinically assessed at the bedside before and after operation by members of the neurosurgical team. Pure-tone audiograms, tympanograms, and recordings of the middle ear acoustic reflex response were obtained preoperatively in 44 patients and postoperatively in 32. The BAEP were recorded in the audiology laboratory before operation in 37 patients, and after operation in 29.

Anesthesia

Most patients received intramuscular premedication with meperidine, hydroxyzine, and either atropine or glycopyrrolate. Many were additionally premedicated in the operating room with intravenous fentanyl. Anesthesia was induced with thiopental, then maintained with fentanyl, thiopental, and nitrogen oxide in oxygen. Pancuronium provided muscle relaxation. Cardiovascular responses to surgical stimulation were controlled using vasoactive agents, including propranolol, trimethaphan, and nitropussride. Halothane was used in eight cases, enflurane in one, and isoflurane in three. Monitoring included electrocardiogram, intra-arterial blood pressure, and nasopharyngeal temperature, as well as BAEP. Arterial blood was sampled at intervals for determination of blood gas tensions, hematocrit, and serum potassium and osmolarity. Central venous pressure was measured in 45 cases and end-tidal pCO₂ in eight.

Evoked Potentials

The BAEP were recorded by the method described previously.** The parameters used for stimulation and recording are shown in Table 2. Three averaged

* Bilateral ear insert transducers manufactured by Madsen Electronics, Inc., Buffalo, New York. BAEP recorded on a MED-80 Biomedical Data System manufactured by Nicolet Biomedical, Inc., Madison, Wisconsin.
BAEP in response to monaural stimulation of each ear and one averaged BAEP without stimulation were recorded before induction of anesthesia. Then BAEP were recorded continually throughout anesthesia and operation. Baseline records were related to preoperative clinical findings, and subsequent recordings were compared to the preanesthetic baseline BAEP. Averaged evoked potentials simultaneously recorded from the vertex to the ipsilateral earlobe and from the vertex to the contralateral earlobe were plotted on graph paper, so that sequential records could be compared intraoperatively. To minimize the delay between completed BAEP averages, only the latency of Peak V was measured on the averaging computer in the operating room. Waveforms were stored on disc for subsequent measurement of latencies and peak-to-following-trough amplitudes of all identifiable peaks in both ipsilateral and contralateral recordings.

Intraoperative interpretation of BAEP thus depended on four factors: 1) overall patterns of waveforms; 2) definition of individual peaks; 3) cursor measurements of Peak V latencies; and 4) estimates of additional latencies and amplitudes derived from hand measurements and superimposition of paper records. Although additional measurements were subsequently made on the stored waveforms, all the intraoperative BAEP alterations described here were detected and reported in the operating room. To eliminate observer variability, all intraoperative interpretation of BAEP was performed by a single individual (B.L.G.).

Because BAEP were abnormal in several patients preoperatively, and because the acceptable degree of intraoperative variability in BAEP had not been established, the tolerance limits for peak latencies employed in the diagnostic laboratory could not be applied directly to waveforms recorded during anesthesia and operation. Rather, each patient served as his own control, and departures from baseline values were considered more important than the absolute values of peak latencies. Quantitative criteria for warning of intraoperative alteration in BAEP were developed during this study by modeling the warnings given in 20 operations performed early in the series. In the more recent cases, warnings were given for either an increase in the latency of Peak V exceeding 0.07 msec/min (as calculated for successively recorded waveforms) or an absolute increase in Peak V latency of more than 1.5 msec.

Results

The BAEP were stable throughout the operation in only 11 cases, whereas alterations in evoked potentials thought to be clinically important were seen in 37 cases. These intraoperative changes in BAEP were reversible in 32 cases, and irreversible in five. Six studies early in the series were excluded from these tabulations because of technical difficulties or because patients were deaf in the affected ear before surgery.

Feasibility

Preparation for surgery was lengthened approximately 30 minutes by the recording of preanesthetic baseline BAEP, but careful planning prevented delays in the operating room schedule. No intraoperative delays were necessary for BAEP monitoring. The click stimulators and recording electrodes (Fig. 2) did not interfere with access to the operative site for either suboccipital or retromastoid craniectomy. Electrodes remained electrically and mechanically stable over many hours. Equipment was expensive and bulky, but easily portable. Adequate quality control, although somewhat cumbersome, was regularly attainable. The full attention of an experienced technician was required throughout each monitoring session.

Technical problems interfered with monitoring during three operations early in the series, twice when compression of pliable earpieces impeded sound transmission and once when an inaccessible electrode was dislodged. We subsequently avoided these difficulties by using rigid molded earpieces and improved techniques for protecting the electrodes.

The BAEP were remarkably stable under anesthesia prior to operative manipulation, except in those patients described below who had waveform changes related to positioning or to the combination of hypocarbia and modest hypotension. The variability of Peak V latency was usually less during anesthesia than prior to induction of anesthesia, perhaps because the artifact from muscle activity was reduced. Standard deviations for repeated measurements in individual patients with "stable" BAEP prior to operative manipulation ranged from 0.002 to 0.902 msec before induction of anesthesia and from 0.016 to 0.380 msec during anesthesia but prior to opening of the dura mater. The mean post-stimulus latencies for Peak V...
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Events Causing BAEP Alterations

Events associated with clinically notable BAEP changes were identified during 37 operations. The BAEP were totally and irreversibly lost in the five patients whose eighth cranial nerves were deliberately sacrificed. In three of these cases, BAEP were lost prior to section of the nerve.

Retraction of the eighth nerve, cerebellum, or brainstem was associated with BAEP deterioration during 22 operations (Fig. 3). Typically, Peak V latency increased continually after retractors were placed. In six cases, the BAEP alteration progressed to virtual obliteration of the waveform. The BAEP changes related to retraction invariably returned toward normal after repositioning or removal of retractors, and hearing was preserved in each case. One case has been reported previously.9

Operative manipulation of the eighth nerve was associated with BAEP alteration in three cases. The most dramatic reversible BAEP changes with operative manipulation were seen in a patient who underwent microvascular decompression of the left fifth and ninth cranial nerves (Fig. 4). Evoked potentials were obliterated soon after the dura was opened, and failed to recover on removal of the retractors. With complete cessation of the operation for 12 minutes, the BAEP reappeared and were used to guide the surgeon’s subsequent manipulations.

In two patients undergoing microvascular decompression of the cranial nerves, BAEP alterations seen prior to incision were temporally related to the combination of hypocarbia and a degree of arterial hypotension that we would have previously considered acceptable (PaCO2 22 to 25 mm Hg, mean arterial blood pressure 60 to 87 mm Hg). The sequence of events is depicted in Fig. 5.

The BAEP elicited by stimulation of the ear ipsilateral to the projected site of operation were altered to the point of virtual obliteration on positioning of patients for retromastoid craniectomy in six cases. Waveforms evoked by stimulation of the contralateral ear were not affected. Our first observation of this phenomenon was in a patient with a large acoustic neurinoma who had diminished hearing and a markedly abnormal BAEP before surgery. Loss of his evoked potential was initially attributed to local ischemia produced by moving the patient to the sitting position. The eighth nerve was extensively involved by tumor and had to be sacrificed. The BAEP never reappeared, and hearing was, of course, lost.

Changes in BAEP associated with manipulation of the head and neck prior to retromastoid craniectomy

Fig. 3. Brain-stem auditory evoked potential (BAEP) changes related to retraction of the eighth cranial nerve (CN VIII) during microvascular decompression of the seventh cranial nerve for hemifacial spasm. The BAEP's were virtually obliterated past Peak I but returned toward normal after the retractor was adjusted.

Fig. 4. Changes in brain-stem auditory evoked potentials with operative manipulation during microvascular decompression of the fifth and ninth cranial nerves (CN) for trigeminal and glossopharyngeal neuralgia. Waveforms were restored when manipulation was interrupted, then served to guide the surgeon as he completed the operation.
were also seen when patients were operated on in the lateral position. We demonstrated the reproducibility of BAEP changes with positioning in one patient who required reexploration of the seventh nerve 10 days after her initial microvascular decompression for hemifacial spasm. Except for the two patients who had abnormal BAEP preoperatively and required deliberate section of the eighth nerve intraoperatively, all the patients who had BAEP changes on positioning for retromastoid craniectomy showed return of waveforms toward normal when the head and neck were returned to a neutral and relaxed position (Fig. 6).

Alterations were seen in BAEP during closure of the dura in two patients. In one, the dura was reopened and a small clot was removed from the eighth nerve. In the other, BAEP alterations were transient and no interventions were made. The cause of BAEP change in this last patient was not apparent.

Interventions Related to BAEP Changes

Reversible BAEP changes during anesthesia and operation were seen in 32 cases, irreversible loss of waveforms in five. Interventions were made by the surgeon and/or anesthesiologist in specific response to BAEP alterations during 22 operations. In 19 of these 22 cases, the interventions were followed by improvement in the monitored waveforms, and hearing was preserved. Interventions included repositioning or removal of retractors (11 cases), modification of operative manipulation (six cases), volume expansion with colloid and/or crystalloid (five cases), alteration of ventilatory rate or volume (five cases), and increase in the inspired concentration of oxygen (one case). Two or more interventions were made during eight operations.

During 13 operations, BAEP changes resolved after maneuvers that were performed as a usual part of the procedure rather than in response to BAEP changes. For example, the surgeon might reposition a retractor independently of a specific warning, or BAEP might recover after retractors were removed at the end of the procedure. We saw recovery of BAEP after periods of virtual obliteration ranging from 5 to 177 minutes.

BAEP Prediction of Postoperative Auditory Function

The 54 monitored operations were categorized according to intraoperative BAEP findings (Table 3) as follows:

- **Category I:** Minimal change (presumably due to anesthesia and temperature changes only)
- **Category II:** Transient latency increase, with return toward normal before end of anesthesia
- **Category III:** BAEP obliteration, with return toward normal before end of anesthesia
- **Category IV:** BAEP obliteration, with no recovery before end of anesthesia (all had deliberate section of the eighth cranial nerve)
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**TABLE 3**

<table>
<thead>
<tr>
<th>Operative &amp; Auditory Function</th>
<th>Category</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>I</td>
<td>II</td>
</tr>
<tr>
<td>no. of cases operative procedure</td>
<td>11</td>
<td>24</td>
</tr>
<tr>
<td>microvascular decompression of cranial nerves</td>
<td>11</td>
<td>20</td>
</tr>
<tr>
<td>resection of acoustic neuroma</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>resection of meningioma in posterior fossa</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>clipping of basilar artery aneurysm</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>section of CN VIII for intractable tinnitus</td>
<td>preserved</td>
<td>preserved</td>
</tr>
<tr>
<td>postop auditory function</td>
<td>preserved</td>
<td>preserved</td>
</tr>
</tbody>
</table>

* BAEP = brain-stem auditory evoked potentials; CN VIII = eighth cranial nerve. For a description of the categories see text.

**Discussion**

Deafness is a recognized complication of operations in the cerebellopontine angle. It is the most common complication seen after microvascular decompression of the seventh cranial nerve. Injury of the brain stem is rare but may be devastating. In some of our patients, interventions made in response to intraoperative BAEP changes may have prevented new deficits in auditory or brain-stem function. No patient in this series suffered injury to the brain stem, and only those five who required deliberate section of the auditory nerve suffered loss of hearing.

The criteria we used for warning the surgeon of intraoperative BAEP change may have been excessively sensitive. Warnings of BAEP change were given with a far greater frequency than we would have predicted from the historical incidence of deafness after similar operations at our institution. In all likelihood, many of the patients with BAEP alterations that we considered clinically important would have done well even if no warnings had been given. Yet, because the complications we sought to avoid were potentially devastating, we wanted early indication of deteriorating function so that we could intervene to prevent permanent injury. We therefore selected an approach similar to that used for intraoperative monitoring of other physiological parameters such as heart rate or arterial blood pressure, attempting to correct undesirable trends as soon as these could be identified with certainty.

Other workers have recorded BAEP intraoperatively. Allen, et al., obtained technically satisfactory BAEP in nine of the 10 patients they monitored. Five had transient BAEP changes during surgery, and none of these suffered new neurological deficits. Three patients had persistent intraoperative deterioration of BAEP waveforms, with decreased amplitudes and increased latencies or complete loss of peaks past Peak...
Two of these patients had new neurological deficits after the operation, but, in one, auditory function and BAEP were better after surgery than before. These authors proposed no explanation for the irreversible intraoperative loss of BAEP seen in the patient who postoperatively had preservation of hearing and recovery of BAEP. In our experience, BAEP have often begun to recover within minutes after the removal of factors associated with transient obliteration. A more detailed description of associated clinical events might provide an explanation for Starr's observation.

Raudzens monitored BAEP in 66 patients during operations in the cerebellopontine angle. Ten patients developed delays greater than 1.5 msec in "BAEP latencies" (peak or peaks not specified); these patients had decreased hearing postoperatively that cleared within 30 days. In six patients, all peaks past Peak I were lost intraoperatively despite an apparently intact eighth nerve. All six patients were deaf in the affected ear after surgery. In contrast, none of our patients lost hearing when the auditory nerve was intact. Differences in surgical technique or clinical management, if they could be defined, might help explain this discrepancy in findings.

Injuries to the brain stem and to the cerebral cortex were seen in Raudzens' series. One patient developed uncontrollable cerebellar edema intraoperatively and lost BAEP past Peak II. He died 1 month later, never having awakened. Another patient suffered extensive damage to the cerebral cortex during resection of a meningioma from the lateral ventricle. He had normal BAEP throughout anesthesia and operation. This observation is important because it reemphasizes the lack of information about cortical function in recordings of BAEP and other short-latency subcortical evoked potentials. Although subcortical potentials offer some advantages for intraoperative monitoring because they are less affected by anesthetics than are cortical potentials, these short-latency waveforms are also less sensitive to generalized insults, such as ischemia and hypoxia, which could damage the brain intraoperatively.

Hashimoto and his colleagues monitored BAEP during 12 neurosurgical operations. One patient showed the increased latencies and decreased amplitudes with hypothermia (32° to 34°C) that were previously described by Stockard, et al. Another had transient latency increases related to operative manipulations. Each of these patients recovered with no new neurological deficit, as did one patient whose Peak V latency decreased intraoperatively. Two patients in this series had BAEP changes during operation that did not resolve. One was stuporous and had respiratory difficulties for several hours postoperatively. The other was sleepy and had signs of pyramidal dysfunction. Final outcome was not reported for these two patients. Waveforms were stable throughout the operation in seven of Hashimoto's patients. One of these seven had a choroid plexus papilloma resected from the fourth ventricle, and failed to awaken from anesthesia. She lost BAEP past Peak I postoperatively and died on the 3rd day after surgery; unfortunately, permission for autopsy was refused. This report suggests that electrophysiological monitoring may have some value after high-risk operations, at least until patients are responsive and neurological function can be assessed clinically.

Our experience indicates that intraoperative monitoring of sensory evoked potentials may be useful when the following five conditions are met: 1) a sensory pathway amenable to monitoring must be at risk; 2) sites for stimulation and recording should be accessible during the operation; 3) adequate equipment must be available; 4) experienced personnel must be present in the operating room to record technically satisfactory waveforms and interpret them accurately; and 5) in the event that intraoperative alterations in evoked potentials are seen, there should be some possibility of intervention to minimize the risk of permanent neurological injury. We found that all these conditions could be satisfied for monitoring of BAEP during neurosurgical operations in the cerebellopontine angle.

We conclude that intraoperative BAEP monitoring is a valuable technique for minimizing injury to auditory pathways and adjacent structures during neurosurgical operations in the posterior fossa. The value of this monitoring will increase with continuing improvements in technology and with the development of quantitative tolerance limits for intraoperative BAEP alterations. Additional clinical observations and controlled experiments in animals are needed, not only to define more precisely the effects of operative manipulations on BAEP, but also to further elucidate the relationships between intraoperative alterations in BAEP and neurological outcome.

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