Intraoperative monitoring of brain-stem auditory evoked potentials

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Intraoperative brain-stem auditory evoked potentials (BAEP's) were monitored in 46 patients undergoing intracranial surgery for a variety of pathological conditions to determine whether this technique was capable of providing useful information to the operating surgeon. Intraoperative BAEP's were unchanged throughout surgery in 34 patients (74%), and these individuals had no postoperative hearing deficits. Four patients (9%) developed an abrupt ipsilateral loss of all waveform components beyond Wave I and had postoperative evidence of a pronounced hearing loss in the affected ear. An additional patient demonstrated BAEP loss contralateral to the side of surgery, and this was associated with subsequent signs of severe brain-stem dysfunction. Seven patients (15%) developed intraoperative delays of BAEP waveform latency values, but maintained recognizable waveforms beyond Wave I. Postoperatively, their hearing was either normal or mildly impaired, and there were no indications of other brain-stem abnormalities. This group represents the individuals who may have been benefited by evoked potential monitoring, since corrective surgical measures were taken when latency delays were observed. Intraoperative BAEP's can be reliably and routinely recorded in an operating room environment. They provide a good predictor of postoperative auditory status, and may have prevented permanent neurological deficits in a small segment of patients by alerting the surgeon to potentially reversible abnormalities.

KEY WORDS  •  auditory evoked potentials  •  acoustic nerve  •  brain stem  •  Intraoperative monitoring

An objective means of continuously monitoring neurophysiological function in the brain stem and cochlear nerve may be of considerable assistance to surgeons operating within the posterior and middle fossa. Some postoperative neurological deficits may be secondary to excess mechanical traction or vascular insufficiency rather than physical transection of neural structures. These might be avoidable if deterioration could be recognized and corrected promptly before irreversible changes occurred. Since their initial description by Jewett and Williston in 1971, brain-stem auditory evoked potentials (BAEP's) have been intensively investigated and have come to play an increasingly important role in diagnostic neurology. It therefore seemed logical to apply these techniques to an operating room setting to determine whether evoked potential monitoring could be used to identify or prevent impending neurological dysfunction. This paper describes our initial experience with intraoperative BAEP monitoring in 46 patients undergoing neurosurgical procedures in which the brain stem or cochlear nerve was at risk.

The normal human BAEP consists of seven vertex positive submicrovolt waves occurring within 10 msec of an acoustic stimulus. This signal reflects the sequential firing of putative neural generators as the acoustic response ascends up the brain-stem auditory relay nuclei. The first five components of the BAEP are the ones most consistently identified, and these are designated Waves I through V as illustrated in Fig. 2. It has been suggested, but not verified, that these individual waves arise from the eighth nerve, cochlear nucleus, superior olivary complex, nucleus of the lateral lemniscus, and the inferior colliculus, respectively. Good clinical correlations between changes in BAEP latency and amplitude values, and a variety of brain-stem abnormalities have been established by several investigators. These BAEP component changes are independent of the subject’s level of arousal and are an objective measure of auditory or brain-stem function.

Several features make BAEP’s particularly attractive for intraoperative monitoring. The waveforms are stable and reproducible, and can be recorded nonin-


<table>
<thead>
<tr>
<th>Surgical Procedure</th>
<th>No. of Cases</th>
<th>BAEP Changes</th>
<th>Postop Deficit</th>
</tr>
</thead>
<tbody>
<tr>
<td>posterior fossa exploration for neoplastic or</td>
<td>27</td>
<td>2 latency delay</td>
<td>1 mild hearing loss</td>
</tr>
<tr>
<td>vascular lesions</td>
<td></td>
<td>1 ipsilateral loss</td>
<td>1 deaf</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 contralateral loss</td>
<td>1 brain-stem dysfunction</td>
</tr>
<tr>
<td>microvascular decompression of 5th nerve for</td>
<td>8</td>
<td>1 latency delay</td>
<td>1 mild hearing loss</td>
</tr>
<tr>
<td>trigeminal neuralgia</td>
<td></td>
<td>1 ipsilateral loss</td>
<td>1 severe hearing loss</td>
</tr>
<tr>
<td>microvascular decompression of 7th nerve for</td>
<td>6</td>
<td>4 latency delay</td>
<td>1 mild hearing loss</td>
</tr>
<tr>
<td>hemifacial spasm</td>
<td></td>
<td>1 ipsilateral loss</td>
<td>1 severe hearing loss</td>
</tr>
<tr>
<td>vestibular nerve section</td>
<td>5</td>
<td>1 ipsilateral loss</td>
<td>1 deaf</td>
</tr>
<tr>
<td>total</td>
<td>46</td>
<td>12</td>
<td>8</td>
</tr>
</tbody>
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**INTRAOPERATIVE BRAINSTEM POTENTIALS**

![Diagram of BAEP monitoring results](image)

**Fig. 1.** Summary of brain-stem auditory evoked potential (BAEP) monitoring results obtained in this series of 46 patients.

Changes be used to prevent auditory or other neurological deficits?

**Clinical Material and Methods**

Intraoperative BAEP's were recorded in 46 patients undergoing surgery for various posterior fossa vascular and neoplastic lesions, microvascular decompression of cranial nerves V and VII, and vestibular nerve section for intractable vertigo.

All patients were tested preoperatively to obtain a baseline recording of the BAEP. Hearing threshold was measured, and a 60-dB click was delivered monaurally at 11.3/sec with a 40-dB masking noise presented to the other ear. The clicks were generated by passing 0.1-msec square-wave pulses through shielded headphones to produce rarefaction-condensation waves. In the operating room, molded ear inserts were fitted to an acoustically matched transformer, and the click stimulus intensity was increased to 85 dB. The bipolar electroencephalographic (EEG) activity recorded from vertex Cz to ipsilateral earlobe AI in response to acoustic stimulation was filtered from 150 to 1500 Hz and amplified × 10^5. The electrical activity following the stimulus for 10.24 msec was sampled 2000 to 4000 times to generate an averaged waveform. This averaging process required approximately 3 minutes for each individual BAEP. Peak latencies and peak-to-peak amplitudes were measured with a digital cursor. Inter-electrode impedance was carefully reduced to values less than 3000 ohms. All averaged responses were superimposed on one another to demonstrate reproducibility from one 3-minute interval to the next.

The earpieces and recording electrodes were draped out of the surgical field. Brain-stem recording did not interfere with or delay the surgical procedure. Averaging was suspended during use of electrocautery without loss of signal acquisition. The studies were performed by an EEG technician under the direct supervision of the attending neuroanesthesiologist. Alterations in evoked potentials were reported immediately to the operating surgeon for possible modification of surgical dissection or retractor positioning.
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Fig. 2. These normal brain-stem auditory evoked potentials (BAEP’s) recorded during vestibular nerve section through a middle fossa approach demonstrate the remarkable stability of the waveforms over several hours of surgical anesthesia. The initial upward deflection on the tracings represents stimulus artifact. Wave V is the most readily identifiable waveform during surgery, and its baseline latency is indicated here and in subsequent illustrations by a vertical line. Tracings 4 to 6 (left) demonstrate the temporary latency and amplitude changes seen when drilling is performed around the cochlear nerve and internal auditory canal. The BAEP’s promptly returned to normal when drilling was discontinued, and the patient had no postoperative auditory or brain-stem deficits.

Results

The types of surgical procedures and the BAEP changes recorded are listed in Table 1 and summarized in Fig. 1.

Technically satisfactory BAEP’s were recorded in all 46 cases. Analysis was limited primarily to waveform latency values because of the variability of waveform amplitude and morphology. No intraoperative BAEP changes were seen in 34 patients (74%). This group included six patients who had preoperative BAEP abnormalities that were not altered during surgery. An example of a normal intraoperative BAEP recording is seen in Fig. 2.

There was no tendency for BAEP’s to fatigue or deteriorate with repetitive testing or prolonged anesthesia, and our longest continuous recording time was 16 hours. No patients with stable BAEP’s during surgery were found to have postoperative changes in auditory function. A single individual, who underwent removal of a meningioma within the right lateral ventricle, had stable BAEP’s intraoperatively but failed to regain consciousness after surgery. She remained unresponsive to verbal stimuli until her death 1 month later, despite serial postoperative BAEP’s showing a normal response.

Transient changes in otherwise normal BAEP’s were seen in some instances. A variability of latency values up to 0.5 to 1.0 msec was frequently observed and might have been due to changes in temperature, the effects of irrigating solutions, mechanical distortion, or ischemic effects on the cochlear nerve or brainstem. Additionally, a temporary increase in wave latency and a decrease in amplitude were routinely noted when drilling of bone was performed around the internal auditory canal. This is illustrated in Fig. 2. We consider this degree of variability normal if
TABLE 2

Wave latency values for brain-stem auditory evoked potentials (BAEP's) at our institution

<table>
<thead>
<tr>
<th>BAEP Values</th>
<th>Absolute Latencies (msec)</th>
<th>Interwave Latencies (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Wave I</td>
<td>Wave III</td>
</tr>
<tr>
<td>induction of anesthesia</td>
<td>2.47 ± 0.62</td>
<td>4.92 ± 0.5</td>
</tr>
<tr>
<td>wound closing</td>
<td>2.24 ± 0.47</td>
<td>4.68 ± 0.49</td>
</tr>
<tr>
<td>normal</td>
<td>1.90 ± 0.40</td>
<td>3.96 ± 0.40</td>
</tr>
</tbody>
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Persistent intraoperative changes in BAEP's were identified in 12 patients (26%). These fell into three distinct categories. The first type of change was a delayed latency of waveform components III and V beyond 1.0 msec. This pattern was recorded in seven of the 46 cases. Of these, four patients were undergoing microvascular decompression of the seventh cranial nerve for hemifacial spasm, one patient had microvascular decompression of the fifth cranial nerve for trigeminal neuralgia, and two patients had posterior fossa explorations for a meningioma and a trigeminal schwannoma.

The usual sequence of change was a failure of two consecutive averages to superimpose, followed by a progressive latency lengthening and amplitude attenuation of Wave V. Alterations of this type could be

Fig. 3. Brain-stem auditory evoked potentials recorded during microvascular decompression for left hemifacial spasm. Evoked potentials during initial surgical exposure showed a normal Wave V latency. Waves IV and V sometimes blend into a single waveform as was the case here. During cerebellar retraction, there was a sudden prolongation of Wave V latency and a failure of two consecutive tracings to superimpose. This patient was monitored early in our experience, and the significance of this abnormality was not immediately appreciated. There was further attenuation of Wave V with manipulation about the seventh cranial nerve, but by the time of closure there were still identifiable waves beyond Wave I. Following surgery, a mild hearing deficit was present in the left ear, and the evoked response was abnormal compared to the right. Both these abnormalities cleared within the 1st postoperative month.

Fig. 4. These brain-stem auditory evoked potentials (BAEP's) demonstrate changes occurring during removal of a meningioma in the vicinity of the eighth cranial nerve. Initial recordings were normal, but Tracings 6 to 8 show progressive deterioration of Wave V latency and amplitude over a 10-minute interval. Retraction on the cerebellum was removed and the BAEP recovered to near normal over the next 10 to 15 minutes. There were no postoperative auditory or brain-stem deficits.
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detected within 3 minutes (the time to average 2000 trials at a stimulus rate of 11.3/sec) of surgical manipulation or retraction in the vicinity of the eighth cranial nerve. An example of this phenomenon is illustrated in Fig. 3. In most instances, the operating surgeon attempted to modify his dissection or change the degree of cerebellar retraction when latency delays were reported. This resulted in improved latency and amplitude values on several occasions, although in most situations the BAEP did not completely return to its preoperative status prior to wound closure. Figure 4 demonstrates a progressive prolongation in Wave V latency occurring over a 10-minute interval. This seemed clearly related to retraction pressure, and latency values promptly reverted to near baseline levels with removal of the cerebellar retractor.

Postoperatively, patients with delayed intraoperative latency values but some recognizable waveforms beyond Wave I had either normal brain-stem and auditory function or a mild sensorineural hearing loss. Both auditory function and the BAEP's tended to return to normal within 30 to 60 days following surgery. The most pronounced persisting deficit in this group of patients was a 52% reduction in speech discrimination 6 months postoperatively, although pure tone audiometry had recovered to its preoperative level.

The second type of intraoperative BAEP change recorded was an abrupt loss of all waveform components beyond Wave I ipsilateral to the side being operated on. This was observed in four patients. There was no tendency for the BAEP to improve or recover in this situation regardless of corrective efforts made by the operating surgeon. In three cases, the cochlear nerve was anatomically intact at the time of BAEP loss. These included microvascular decompressions for the fifth and seventh cranial nerves, respectively, and a middle fossa vestibular nerve section for intractable vertigo. An example of this situation is illustrated in Fig. 5. The fourth case (Fig. 6) involved an intra-

Fig. 5. Evoked potentials recorded during microvascular decompression of the left fifth cranial nerve for trigeminal neuralgia. A well defined Wave V with normal latency was identifiable during initial stages of the procedure. As cerebellar retraction was deepened to expose the trigeminal nerve, the evoked response was abruptly lost on the left side although the cochlear nerve was physically intact. There was no return of the waveforms with readjustment of the retractor. Postoperatively, the patient had a severe sensorineural hearing loss. Evoked potentials from the unoperated right side remained normal, but there was no improvement in left-sided evoked potentials or auditory status at follow-up review 6 months later.

Fig. 6. Evoked potentials recorded during removal of an intracanicular left acoustic neuroma, with diminished but preserved hearing in the affected ear. Initially, an attenuated response was present from the left ear compared to a normal Wave V latency on the right. As nerve decompression and tumor resection progressed, waveform amplitudes increased and a clearly recognizable Wave V became apparent on the left. It was eventually not possible to preserve the nerve and obtain complete tumor removal, and the left cochlear nerve was sectioned with loss of all evoked waveforms beyond Wave I. Wave V latency on the right remained unchanged. Postoperatively, the patient was deaf in the left ear and had no signs of brain-stem dysfunction.
canalicular acoustic schwannoma with reduced but preserved hearing on the affected side. The cochlear nerve could not be preserved during the course of tumor removal, and coincident with its section all components of the BAEP beyond Wave I were lost. Postoperatively, patients with a sudden intraoperative BAEP loss of this type were either deaf or had a profound sensorineural hearing loss. None of the patients had signs or symptoms of more generalized brain-stem dysfunction. Hearing did not improve over a 3- to 6-month follow-up interval despite a physically intact cochlear nerve in three of the four patients.

The third type of intraoperative BAEP change observed was loss of all waveform components beyond Wave I contralateral to the side of surgery. This was seen in a single case (Fig. 7) and involved an acoustic neuroma patient who had absent preoperative BAEP's from the affected ear but a normal study on the opposite side. During tumor removal, marked cerebellar edema developed, followed by hypertension, bradycardia, and deterioration in the previously normal contralateral BAEP. Corrective measures, including cerebellar resection, were not effective in reversing this process, and all BAEP waveforms beyond Wave I were lost. Postoperatively, the patient had signs of severe brain-stem dysfunction and died without regaining consciousness or demonstrating any improvement on subsequent BAEP studies.

Discussion

We believe that our experience with intraoperative BAEP monitoring in an initial 46 patients establishes this test as one that can be routinely and reliably performed in an operating room environment. No patient was subjected to added risk by monitoring, and inconvenience to the surgeon was minimal. The BAEP's were not substantially affected by conventional doses of anesthetics or by long periods of continuous auditory stimulation, as shown in Table 2. Interwave latencies during surgery more closely approximate preoperative values than absolute latencies because of the delayed Wave I latency produced by the plastic ear inserts used in the operating room instead of the usual earphones.

Fluctuations in latency values up to 1.0 msec were frequently seen, but did not interfere with data interpretation since they either reverted to baseline levels or showed no tendency toward progressive deterioration. These changes are presumably attributable to multiple factors, including fluctuations in stimulus intensity, temperature changes produced by irrigating solutions, mechanical distortion of the cochlear nerve or brain stem, or alterations in regional blood flow. The temporary deterioration in waveform latency and amplitude consistently observed when drilling was performed around the internal auditory canal may occur because random noise produced by this procedure approaches or exceeds the discrete auditory stimulus provided through the earpieces.

A total of 33 patients showed no permanent change in their BAEP's during surgery and had no postoperative alteration in their auditory status. We have not yet seen a false-positive BAEP with respect to auditory function, and a stable intraoperative BAEP provides the surgeon with a high degree of confidence that hearing will be unaffected postoperatively. A single
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patient with normal BAEP's throughout surgery failed to awaken following anesthesia and remained comatose until her death. This indicates that the BAEP may not be a reliable indicator of level of consciousness in all instances. Postmortem examination was not available in this patient, but the clinical circumstances suggest she sustained damage to bilateral cerebral or thalamic sites rather than to lower brain-stem areas.

Twelve of 46 patients demonstrated a persistent change in their intraoperative BAEP, for a test yield of 26%. As shown in Table 1, this varied considerably with the type of surgery performed. Patients undergoing microvascular decompression of the seventh cranial nerve, which inevitably involved some dissection around the cochlear nerve, had an 83% incidence of BAEP changes.

Four patients lost all BAEP waveforms beyond Wave I on the side being operated on, and this was correlated with either unilateral deafness or a severe unilateral sensorineural hearing loss in all instances. The cochlear nerve was anatomically intact in three of these patients despite the pronounced physiological dysfunction. In view of the abrupt nature of the BAEP change, the absence of waveforms beyond Wave I, and the persistence of normal BAEP's from the opposite ear, we speculate that the mechanism of injury was ischemic damage to either the cochlear nerve or the ipsilateral brain-stem cochlear nucleus. Although there was good correlation between the intraoperative BAEP and postoperative neurological function in this group of patients, they were not benefited by monitoring since the changes occurred rapidly and were not reversible with any alteration in surgical technique.

Seven patients had persistent prolongation of BAEP wave latencies during surgery but not complete obliteration of the evoked response. Postoperative follow-up review in these cases suggests that subsequent auditory or brain-stem deficits will be mild or absent if some recognizable waveforms beyond Wave I are preserved on the side of surgery and if the contralateral BAEP is unchanged. This group (15%) represents the patients who may have had permanent hearing loss or brain-stem dysfunction prevented by intraoperative monitoring. Surgical dissection or the degree of cerebellar retraction was altered when waveform latency delays were observed, and in these instances the BAEP either improved or failed to deteriorate further. The pathophysiological mechanisms responsible for these potentially reversible latency delays are not completely clear, but probably involve both mechanical distortion and vascular insufficiency of the cochlear nerve or brain stem. The value of BAEP monitoring in preventing permanent neurological deficits in patients with intraoperative latency prolongations must remain speculative, however, since the surgeons involved felt compelled to modify their dissection when such changes were observed. It could therefore be argued that the eventual clinical outcome in these patients would have been the same if BAEP monitoring had not been available or if the information from monitoring had been ignored. We believe data such as that presented in Fig. 4 suggest that BAEP monitoring may help prevent hearing deficits in some patients, but further experience will be required before this question can be answered definitively.

Complete loss of the BAEP beyond Wave I contralateral to the side of surgery was seen in one patient and was associated with profound coma and absent brain-stem reflexes in the immediate postoperative period. A similar case has been reported by Hashimoto, et al. Although the onset of contralateral or bilateral BAEP deterioration seems to be a grave prognostic sign, it occurred in this instance simultaneously with other indicators of brain-stem dysfunction, such as cerebellar swelling, bradycardia, and hypertension, and did not provide any advance notice of impending difficulty. Our impression thus far has been that intraoperative BAEP monitoring is a much more sensitive and useful indicator of auditory function than of brain-stem viability. However, only two of our 46 patients demonstrated postoperative worsening in their level of consciousness or general brain-stem status, and it is possible that these conclusions may need to be altered as further data are accumulated.1,16

We presently believe that intraoperative BAEP monitoring provides a reliable, objective assessment of neurophysiological function within the auditory system of the anesthetized patient. It is particularly useful during procedures where hearing is at risk, such as tumor removal in the vicinity of a functioning cochlear nerve, microvascular decompression of the fifth and seventh cranial nerves, or vestibular nerve section. Postoperative auditory status seems to correlate well with intraoperative BAEP findings.

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References


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