Use of intraoperative auditory evoked potentials to preserve hearing in unilateral acoustic neuroma removal

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Twenty-two patients with unilateral acoustic neuromas and preoperative speech discrimination scores of 35% or more had intraoperative monitoring of the electrocochleogram (ECoG) using a transtympanic electrode, and of the brain-stem auditory evoked potentials (BAEP's) using scalp electrodes. Rapid feedback was provided about the status of the cochlear microphonics from the hair cells of the inner ear (CM of the ECoG), the compound action potential of the auditory nerve (N-1 of the ECoG or Wave I of the BAEP's) and the potentials from the lower brain stem (Wave V of the BAEP's). All patients had total removal of the tumor. In 21, the cochlear nerve was anatomically preserved, and 20 had good postoperative facial nerve function. Correlation of tumor size with postoperative hearing was as follows: discrimination scores of more than 35% in three of four patients with 1-cm tumors, two of eight with 1.5-cm tumors, two of six with 2- to 2.5-cm tumors, and one of four with tumors of 3 cm or more. Two other patients with 1.5-cm tumors had discrimination scores of less than 35%, and one patient with a 2-cm tumor had only sound perception. In two patients, the discrimination scores improved. At the end of the operation, all patients with hearing had a detectable N-1, and, when recorded, CM. All but one patient with no hearing had lost N-1, and CM was absent or reduced. Unless Wave V was unchanged, it was a poor predictor of postoperative hearing, and its absence did not preclude preservation of good hearing.

The electrophysiological changes during each stage of the operation were analyzed and correlated with events during surgery. Areas in which there was an increased risk of loss of the potentials were determined. In some patients monitoring was unnecessary, because either there were no significant changes or the changes were abrupt and no recovery occurred. However, in other patients, monitoring alerted the surgeon to a possible problem and the method of dissection was altered. Possible mechanisms of hearing loss were suggested from the changes in the recordings.

KEY WORDS • acoustic tumor • auditory evoked potentials • intraoperative monitoring • preservation of hearing

It is well established that in some patients with an acoustic neuroma (vestibular schwannoma) useful hearing can be retained following removal of the tumor, but in others there is no hearing postoperatively even though the cochlear nerve is left apparently anatomically intact. In 1977, we began using click-evoked potentials during surgery to provide more immediate feedback about the status of the inner ear, cochlear nerve, and brain stem, to study whether this adjunct would help improve the ability to preserve hearing. The initial technique of monitoring has been reported in a previous publication. 

This paper describes the technique currently being used and summarizes our overall experience in 22 consecutive patients with unilateral acoustic neuromas in whom intraoperative monitoring was performed while attempting to preserve hearing. The results with patients having bilateral acoustic neuromas will be reviewed separately. In another publication, the mechanisms of hearing loss during surgery were studied in a series of detailed case reports.

Clinical Material and Methods

Patient Population

Table 1 records the series of all unilateral acoustic neuroma patients operated on by us via the suboccipital
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### Table 1

*Functional results following suboccipital microsurgical removal of acoustic neuroma*

<table>
<thead>
<tr>
<th>Tumor Size</th>
<th>Total Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small (&lt;2 cm)</td>
<td>Medium (2-3 cm)</td>
</tr>
<tr>
<td>no. of cases</td>
<td>18</td>
</tr>
<tr>
<td>good</td>
<td>18</td>
</tr>
<tr>
<td>fair</td>
<td>0</td>
</tr>
<tr>
<td>poor</td>
<td>0</td>
</tr>
<tr>
<td>died</td>
<td>0</td>
</tr>
</tbody>
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*Facial and auditory nerve functions are not included in the assessment of disability. Good: returned to full activity; fair: returned to most previous activity with some impairment; poor: significant residual disability.*

The 22 patients included 15 women and seven men, aged between 27 and 60 years. None had evidence of neurofibromatosis and all had preoperative speech discrimination scores of 35% or better. Tumor size was defined as the maximum diameter of the tumor outside the internal auditory meatus, as measured intraoperatively for the smaller tumors and from the computerized tomography scan for the larger tumors.

### Operative Techniques

In most of the patients in the early part of this series the tumor was removed through a suboccipital posterior fossa approach done in the semi-sitting position as described in previous publications. There was no major permanent morbidity related to this position but, because of the risk of hypotension in older patients, a modified supine position was developed (Fig. 1). Other advantages to this position soon became apparent: excellent visualization of the cerebellopontine angle (CPA), ease of tumor removal, and comfort of the operator. In addition, air embolism was no longer a concern. This position is now used for most CPA tumors.

The operating table is turned so the surgeon can sit behind the head with the feet under the table. The patient lies supine, with the shoulder that is ipsilateral to the tumor slightly elevated. If there is cervical spondylosis or limitation of neck motion, a more lateral position is used. The head is turned parallel to the floor, elevated, and held with a three-point skeletal-fixation headrest. During the operation, the line of sight to the brain stem may be altered by rotating the table from side to side.

All patients receive steroids preoperatively. Shortly after induction of anesthesia, an antibiotic and 10 to 20 mg of furosemide are given. During the initial exposure, 500 cc of 20% mannitol is administered. A paramedian incision is centered about 1 cm medial to the mastoid process. Pericranial tissue is taken from the occipital region to be used in closing the cerebellar convexity dura at the end of the operation. The craniectomy exposes the dura over the lateral two-thirds of the cerebellar hemisphere, and is carried over the edge of the transverse sinus above and the sigmoid sinus laterally. The dura is opened vertically, usually about 2 cm from the lateral edge of the exposure. The medial dura is kept intact. Superior, lateral, and inferior flaps of the dura are held back with sutures. The cerebellum is then gently elevated, arachnoid below the lower cranial nerves is opened, and cerebrospinal fluid (CSF) is allowed to drain. This will usually relieve any bulging of the cerebellum and allows exposure of the CPA with minimal retraction. The arachnoid should be opened enough to allow CSF to continue to drain during the operation. In some patients with large tumors, a portion of the lateral cerebellar hemisphere will be removed to facilitate the exposure.

Following placement of self-retaining cerebellar retractors, the operating microscope is positioned. All possible arterial vessels on the tumor capsule or adjacent cerebellum are preserved because they may supply the seventh and eighth nerves, the labyrinth, and in some cases the brain stem. The arachnoid over the posterior capsule is opened and reflected medially. An attempt is made to preserve arachnoid planes as a landmark for dissection. The lower cranial nerves (ninth, 10th, and 11th) are identified, and arachnoid and adjacent cerebellum are carefully dissected to aid the exposure of the inferior medial capsule. With larger tumors, the nerves are reflected off the tumor capsule.
and a small rubber dam is placed over them for protection during the rest of the operation.

In small tumors (less than 2 cm) the seventh and eighth nerve complex can usually be defined medially. Electric stimulation is used to confirm the position of the seventh nerve. The plane between the tumor and the seventh and eighth nerves is opened with a microdissector and microscissor. The tumor is carefully freed, keeping the strokes of the dissection parallel to the nerve. Vestibular nerve fibers entering the tumor capsule are divided. Once this dissection has been accomplished, the dura and bone over the internal auditory canal (IAC) are removed for a distance of no more than 10 mm. More lateral bone removal runs the risk of entering a semicircular canal. The dissection then proceeds, usually from medially to laterally, along the seventh and eighth nerves. Bleeding along the seventh and eighth nerves usually stops spontaneously.

In patients with medium and large tumors (2 cm or larger) the capsule is stimulated, especially superiorly, to determine if the facial nerve is in an unusual position. If the edge of the eighth nerve is not seen along the inferior capsule at the initial exposure, an intracapsular decompression is performed using the Cavitron ultrasonic surgical aspirator.* Dissection then begins inferiorly and medially, keeping intact as much of the arachnoid as possible. Working along the brain stem and inferior tumor capsule, one looks for the eighth nerve complex. As the dissection progresses, those portions of the brain stem and nerve that have been separated are covered with small strips of rubber dam for protection. The seventh nerve will usually be found superior and anterior to the eighth nerve. Typically, the tumor is most adherent to the seventh and eighth nerves just medial to the internal auditory meatus. The internal auditory canal is then exposed. After the attachment of the tumor to the superior and inferior margins of the meatus is divided, the tumor is carefully dissected from the seventh and eighth nerves, following the plane that had been defined intracranially. If the tumor is so large that the medial dissection is difficult, it may be better to define the seventh and eighth nerves in the canal and then follow them medially. Great care is taken in removing the lateral remnant of tumor which may extend beyond the edge of the area of bone removal.

**Intraoperative Monitoring**

Our technique for intraoperative monitoring is outlined in a previous report. Since then some modifications have been made. Figure 2 is a block diagram of the present system. After the patient has been anesthetized and positioned, the recording electrodes and earphones are placed and recordings are made continuously until the skin is closed. The electrocochleogram (ECoG) and the brain-stem auditory evoked potentials (BAEP's) are recorded on separate channels. For the ECoG, a needle-electrode is placed through the tympanic membrane to rest on the promontory of the medial wall of the middle ear. The only morbidity with this transtympanic electrode was a tear in the tympanic membrane in one patient, which later repaired itself spontaneously. A needle-electrode within the ipsilateral earlobe serves as the reference. The BAEP's are recorded between needle-electrodes at the vertex of the head and the ipsilateral earlobe. Forehead electrodes serve as ground. These potentials are first amplified by an isolation amplifier (fixed gain of 100, 3 dB down points: 0.0025 to 2.5 kHz) followed by a second amplifier with selectable gain and bandwidth. The overall bandwidth for the ECoG is usually 0.030 to 2.5 kHz and for the BAEP's 0.003 to 2.5 kHz.

Clicks are usually presented to the operated side at 80 dB nHL (0 dB nHL = mean threshold of a group of subjects with normal hearing). Broad-band masking noise at 45 dB nHL is presented continuously to the other ear to prevent any BAEP's due to acoustic crossover. Clicks are produced by sending 30 μsec electric pulses to the earphone. Because response amplitude may decline with higher click rates, while the noise level of an averager decreases with increasing number of trials, a click rate must be selected for each patient to maximize signal-to-noise ratio per unit of time. Typically, about 33 clicks/sec is used, with the click polarity that gives the larger response.

With these recording methods, potentials from three major auditory structures that are at risk during surgery can be monitored: the cochlear microphonic from the hair cells of the inner ear (CM of the ECoG), the compound action potential of the auditory nerve (N-1 of the ECoG or Wave I of the BAEP's), and the potentials from the lower brain stem (Waves II to V of the

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* Cavitron ultrasonic surgical aspirator manufactured by Cooper Medical, Mountain View, California.
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BAEP's. In practice, Wave V is the largest of these waves and the one that is monitored.

Problems arise in distinguishing the different potentials in the ECoG. Two techniques can be used to distinguish N-1 from CM. Presenting masking noise and clicks to the same ear will attenuate N-1 more than CM. Reversing the click polarity will reverse the CM polarity, but generally not the N-1 polarity.

Another problem is distinguishing the CM from the stimulus artifact produced by the earphone as it generates the click. Manipulating the acoustic stimulus, as for CM and N-1, does not help, because they behave similarly. For this reason, in our first system (a dynamic earphone mounted next to the ipsilateral ear) we could not distinguish between these two (see Fig. 5). The approach we have taken has been twofold: 1) to develop an acoustic system that had a small stimulus artifact, and 2) to separate in time the production of the click (and stimulus artifact) and its arrival at the ear. Our most recent system consists of a shielded hearing aid receiver fitted into one end of a 6-in. (0.5 msec acoustic delay) piece of No. 10 polyvinyl tubing, the other end of which is positioned at the entrance to the ear canal in a custom-made ear mold. This mold also serves to limit background noise from reaching the ear. A No. 16 polyvinyl tube leads from the ear canal through the mold to a miniature microphone from which the output is monitored continuously on an oscilloscope to be certain the acoustic stimulus in the ear canal is stable.

Typically, the averager sampled 256 points, each with an 80-μsec sampling time. By starting the averager 8 msec before the presentation of the click, the background noise of the recordings can be assessed. An artifact reject system halts averaging whenever electrical noise causes the recording channels to exceed a pre-selected voltage level, as occurs, for example, during use of electrocautery.

By means of a computer system interfaced with the averager, the averaged waveforms can be taken repeatedly and automatically. Because the N-1 potential is often several times larger than Wave V, it can be detected after many fewer trials than can Wave V. We monitor both near their limits of detectability by first averaging a pre-set number of trials so that N-1 is just recognizable (even though Wave V may not be detectable). Next, the computer stores the averages in a buffer and clears the averager for another run. After a pre-set number of runs, a grand average is formed, stored on disc, and displayed so that Wave V can be assessed. We have recently added a feature that enables the computer, between averages, to plot on the terminal screen the amplitude and latency of the CM and N-1 for the previous 190 averages.

The waveforms in this series were stable and reproducible, could be recorded without encroaching on the operative field, and did not change with administration of anesthetic drugs. Temperature of the patient was monitored with an esophageal probe. Throughout the operation the view through the operating microscope was videotaped. On an analog recorder, the outputs of the probe tube microphone, the amplifiers and the pulse generator as well as the surgeon's voice were taped. A time-code generator continuously marked the time on the analog tape, the video tape, and the stored averages. With this system we could review off-line the surgical events and the evoked potentials that occurred at any time during the operation.

Results

Figure 3 includes the overall results of this series of 22 patients, listed in order of tumor size. The tumor was totally removed in all of these patients, and the cochlear nerve was preserved in all except one (Case 20) in whom it was so intimately involved with the tumor that it could not be saved. All patients had a good overall result and returned to normal activity.

As shown in Fig. 3, the chance of preservation of any hearing decreased with increasing tumor size. When hearing was assessed in terms of preservation of speech discrimination, the patients with the smallest tumors had the best results, with good preservation in three of the four patients with tumors of 1 cm or less. Two of eight patients with tumors of approximately 1.5 cm had good preservation, and two others had some hearing. Two of six patients with tumors 2 to 2.5 cm in size had good speech discrimination, while another had some sound perception. In one of the four patients with tumors of 3 cm or more there was modest preservation of speech discrimination.

To suggest that patients with a relatively poor speech discrimination score did not have "useful" hearing is misleading since our patient with the largest tumor (Case 22) had a nonfunctioning opposite ear from an idiopathic sudden hearing loss 2 years earlier. Her postoperative hearing (speech discrimination score of 40%) has allowed her to conduct a conversation and talk on the telephone.

In 20 of the 22 patients, facial function was ultimately normal or nearly normal. Three of these patients had immediate weakness which recovered, and one had the onset of a delayed weakness (within 3 days) which subsequently recovered. One patient (Case 20) had only a fair recovery, and in another patient (Case 21) with a large tumor the facial nerve was lost during tumor removal. A hypoglossal anastomosis was done with a fair result.

At the bottom of Fig. 3 are tabulated the changes in the evoked potentials that were present by the end of the operation in each patient. Wave V was detectable and could be monitored in 17 patients. In only one of these patients did Wave V, N-1, and CM remain unchanged, and her speech discrimination score improved postoperatively. In all 16 others, Wave V decreased during the operation. In 13, including all eight patients with complete loss of hearing, Wave V was lost completely. In four with good hearing preservation (discrimination scores decreased by less than 30 percentage points), Wave V was found again in the postoperative
period. However, in two of the three patients with the largest reductions in speech discrimination (more than 50%), Wave V could still be detected at the end of the operation. In summary, unless Wave V is unchanged during operation, it is a poor predictor of the degree of eventual hearing preservation; its absence does not preclude preservation of good hearing.

The N-1 and CM results were much better correlated with hearing preservation than was Wave V. In all patients, N-1 could be monitored, including a few patients in whom Wave I was not detectable in the brainstem recordings. With our present system, CM could always be seen. Of the 11 patients who had some hearing postoperatively, all had a detectable N-1 by the time the skin was closed. In six of these patients CM was monitored, and in only one did a change in CM occur (Case 17, Fig. 4). In this one exception, both N-1 and CM disappeared about 35 minutes after tumor removal was completed; both had begun to recover by the end of the operation. Of the 11 patients with no postoperative hearing, all but one had lost N-1 by the end of the operation. In all seven of the patients in whom it was recorded, CM was either partially or completely lost. It would appear that loss of N-1 and impairment of CM without any signs of recovery correlate with total hearing loss. On the other hand, since the four patients with the greatest change in their speech discrimination scores had little or no change in their N-1 and CM recordings, preservation of N-1 and CM does not necessarily indicate good preservation of hearing.

A summary of the electrophysiological response changes as they occurred in the various stages of the operation is also presented in Fig. 3. Failure to obtain a satisfactory Wave V when the monitoring started was usually due to absence of the potential in the preoperative recording or occasionally to a technical problem, as in Case 6. In the first patients studied, CM was not recorded. No changes occurred prior to cerebellar retraction except those attributable to technical problems such as movement of the ear mold. With retraction of the cerebellum, changes in Wave V occurred in four of 16 patients in whom it could be measured, and all four had small tumors (less than 2 cm). In three it consisted of a small prolongation in latency or a decrease in...

Fig. 3. Summary of intraoperative monitoring in 22 patients with removal of unilateral acoustic neuromas. BAEP's = brain-stem auditory evoked potentials.
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FIG. 4. Plot of N-1 and CM amplitudes over the 2 1/2 hours following removal of the last piece of tumor tissue of Case 17. Upper arrow = tumor removal completed; lower arrow = termination of the operation. During the extrameatal dissection, the brain-stem auditory evoked potentials had become undetectable. Because a major change occurred in both N-1 and CM 35 minutes after the removal was completed, the cerebellopontine angle was reexplored and irrigated but no abnormality was found. It was postulated that this change might have been explained by vasospasm of the internal auditory artery. Amplitudes were measured from the pre-stimulus baseline to the peak of the CM and N-1 of the averages obtained successively on line. To the right are electrocochleogram waveforms obtained over adjacent intervals with and without masking noise at 80 dB nHL (0 dB nHL = mean threshold of normal hearing). The arrows point to the midpoint of the interval over which the two waveforms were obtained. From the top downward, the period over which each pair of waveforms was obtained were 0.5, 2.5, 2, 3, and 2 minutes, respectively. Because the auditory nerve potential (N-1) is more easily masked than are the inner ear potentials (CM or, possibly, the summating potential), the N-1 is taken as the negative deflection following the dotted line. Stimulus: 33/sec rarefaction clicks at 80 dB nHL from a hearing aid earphone within an ear mold placed in the ear canal. Positivity is plotted upward for all figures.

Later, the findings in both of these cases suggest that the loss of neural function may be due to cochlear ischemia.

Dissection along the region of the cochlear nerve between the brain stem and the internal meatus resulted in frequent decreases in the evoked potentials. Decreases in Wave V were particularly common. Ten (77%) of 13 patients had significant decreases in Wave V as compared to seven (37%) for N-1. In four of the 10 patients Wave V decreased without any disturbance in N-1. Usually the reason for the decrease in Wave V was not apparent. An exception was seen in Case 6: an abrupt change occurred as the capsule was being elevated from a position ventral to the brain stem (Fig. 5). In two patients there was abrupt loss of N-1 and CM, followed by recovery over several minutes. In one patient (Case 21) there was abrupt loss of N-1 with recovery which was associated with a slight change in CM; in three patients in whom only N-1 was recorded, there was abrupt loss without recovery in two and recovery in one (Fig. 6). On two occasions dissection of the cochlear nerve from the inferior capsule caused a significant change in N-1, which recovered after the dissection was stopped.

The timing of the exposure of the tumor and neurovascular bundle within the IAC depends upon the size of the tumor and how easily a dissection plane can be identified between the tumor capsule and the surrounding neural structures. No new changes in Wave V were
FIG. 6. Successive electrocochleogram waveforms obtained from Case 21 during extrameatal dissection of the inferolateral pole of her 3.5-cm tumor. A similar change in N-1 with recovery had occurred 2 hours earlier when working in this same region. When this change in N-1 occurred, dissection in the area was stopped and recovery of the potential was seen. The times at the right indicate the intervals over which each waveform was obtained. No brain-stem auditory evoked potentials were detectable preoperatively or intraoperatively. Stimulus: 22/sec rarefaction clicks at 80 dB nHL (0 dB nHL = mean threshold of normal hearing) from a dynamic earphone mounted next to the ear. The large stimulus artifact that occurred within the first 0.3 msec has been suppressed. It cannot be determined whether the oscillatory activity during the first 1 msec is stimulus artifact or CM. Because it changed little with reversal of the click polarity, the large negative potential at about 2 msec was identified as N-1.

initiated during drilling of bone over the IAC. In two patients a gradual loss of Wave V, which had started prior to drilling, continued. In half of the patients there were no changes in N-1. Most of the changes in N-1 involved only minor alterations in wave shape and were probably insignificant. Both patients with a major amplitude loss (more than 30%) were found to have intimate involvement of the tumor with the auditory nerve and ultimately their hearing was lost. In three patients (Cases 13, 17, and 18) the amplitude of N-1 increased slightly when the canal was opened (Fig. 7), but only one retained hearing with significant speech discrimination.

As the final dissection of the capsule from the seventh and eighth nerves was being done in the IAC, there were still 17 patients in whom N-1 could be recorded. Changes occurred in 12 of them but in two these were slight. There were no new independent changes in Wave V. Three patterns of significant change in N-1 occurred during this part of the operation. In one pattern there was a rapid loss of N-1 (over less than 10 minutes) as the tumor was being separated from the nerves, followed by equally rapid recovery. Wave V was lost during this period without recovery later during the operation, but would reappear in the postoperative period. Two of these patients (Cases 3 and 14) retained their hearing and, in the third (Case 8), N-1 abruptly disappeared during closure. In the second pattern, the N-1 change occurred as the tumor was being dissected from the most lateral recess of the IAC. In all three cases where this occurred the hearing outcome was poor. In two (Cases 9 and 18), the potentials were lost with no hearing postoperatively. In the third patient (Case 10), N-1 amplitude fluctuated, even sometimes growing larger, but speech discrimination decreased from 78% to 12% postoperatively. The third pattern, one of gradual hearing loss, was seen in three patients; it progressed to complete loss in one patient (Case 15) but stabilized with a partial loss in the other two, both of whom had good hearing (Cases 2 and 5). In one patient (Case 20), the auditory nerve had to be sacrificed in order to completely remove the tumor.

During closure of the wound, two patients exhibited abrupt loss of N-1. In one (Case 8), CM appeared to be unchanged but N-1 never recovered and there was no hearing postoperatively. The other patient (Case 17) had loss of both N-1 and CM, which led to reexploration of the operative field, but no abnormality was seen. Slowly, CM recovered as did N-1 (see Fig. 4), and excellent hearing was preserved.

Discussion

Several reports record preservation of hearing following removal of an acoustic neuroma using microsurgical techniques. The middle fossa approach has been described in detail, but, because that route has the disadvantages of being associated with a higher incidence of facial weakness and having limited access to the posterior fossa, we prefer to use the suboccipital approach. The semi-sitting po-
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The vestibular nerve and may leave the cochlear nerve intact, particularly when the lesion is small. This finding was confirmed in our patients with preoperative speech discrimination scores greater than 35%, where it was possible to remove the tumor completely in all patients and retain the cochlear nerve anatomically in all but one case. In view of the reports that speech discrimination scores can improve postoperatively, as we found in two patients (Cases 5 and 7), attempts to preserve hearing even for patients with poor speech discrimination are reasonable. We made such attempts in three patients not included in this series, who had nearly normal threshold for tones with poor discrimination scores preoperatively, but we were unable to improve discrimination or preserve any hearing.

The degree of preoperative hearing loss does not have a strict relationship to the size of the tumor. However, it is clear that the chance to save hearing is better with the smaller tumors. In reports of patients in whom it has been possible to preserve some hearing, the tumor has usually been 2 cm or less in diameter. In our experience, if the tumor is 1 cm or less there is about a 75% chance and if it is 1.5 to 2.5 cm there is a 25% to 33% chance of having good preservation of hearing postoperatively (speech discrimination score over 35%). Rarely has preservation of hearing been reported following removal of tumors larger than 3 cm in diameter. (Our Case 22 was previously reported in the discussion of a paper by Fischer, et al.)

Because of its larger signal to noise ratio, ECoG has the great advantage over the BAEP's of providing a more rapid feedback about the status of the compound action potential of the auditory nerve (N-1 or Wave 1). Typically a clear N-1 record could be obtained in less than 5 seconds. Such rapid feedback might have altered the outcome in the cases reported by Raudzens and Shetter in which the BAEP's were described as changing too fast (within 3 minutes or less) to allow feedback.

Monitoring potentials from the inner ear (CM), auditory nerve (N-1), and brainstem (Wave V) permits localization of dysfunction, if we assume a simple sequential model of the relationship between these three potentials: change in one potential would result in changes in all later potentials without affecting earlier potentials. Of course, there can be concurrent insults at more than one level. Our results are generally consistent with this formulation. Changes in CM were always associated with changes in N-1 and Wave V, and major changes in N-1 were always associated with Wave V alterations. On the other hand, Wave V could change in isolation, and N-1 could change without CM changes. The only exceptions occurred during drilling of the bone of the IAC. Slight changes in the configuration of N-1 occurred without corresponding changes in Wave V. Because removal of this bone changes the electrical characteristics of the tissues nearby the generation site of N-1, these changes in the configuration of N-1 might be due to alterations in the electrical properties of the media through which the potentials are conducted, and not due to any change in the functioning of the auditory nerve. Raudzens and Shetter noted routinely during drilling a temporary increase in the latencies and a decrease in the amplitude of the BAEP's that recovered. We have made similar observations which later were clearly due to the masking effect from the noise associated with drilling. If a complete loss of function should occur during drilling it probably means the labyrinth has been injured, although we have not seen this. Domb and Chole reviewed the anatomy in this area and the possibility of injury to the posterior semicircular canal during the bone removal.

During extrameatal dissection, changes are more common in the Wave V than in N-1, probably because the dissection involves the part of the auditory nerve distal to the portion that generates N-1. Any local disturbance in the auditory nerve would not change N-1, but would disrupt conduction through this part of the nerve and be reflected in latency and amplitude change in the later brain-stem peaks (Wave V). When the dissection was intracanalicular, any new change in Wave V tended to occur abruptly with a loss of N-1, since it is likely that the nerve was being injured close to the site of generation of the N-1 potential. These observations support the concept that N-1 is generated primarily by the afferent auditory fibers where they initially become myelinated in the vicinity of the habenulae perforata.

We can only speculate about why we frequently found slowly progressive amplitude and latency changes in Wave V that had no implications for the ultimate hearing outcome. One possibility is a reversible alteration in the conduction properties of the auditory nerve fibers due to partial ischemia of the nerve from compression of its microvasculature or mechanical distortion of the axons. A second consideration is temperature effects. Lowering the local temperature in normal animals reversibly prolongs latency. However, this effect is too small to account for our observations, unless we speculate about other factors such as increased sensitivity to temperature changes due to alterations induced by the tumor or lower temperatures at the operative site than at the temperature probe (esophagus) due to exposure to room air. Anesthesia is probably not the cause, because a patient with no changes in the evoked potentials (Case 7) received the same anesthetic agents at the same dosages as many others who did show changes in Wave V.

An alteration in the CM suggests that the inner ear has been disturbed. This must occur indirectly, since the surgery itself does not involve the cochlea. Among the several possible explanations for this effect (involve-
ment of the middle ear, the efferent nerve fibers to the cochlea, the facial nerve fibers to the stapedius muscle, the cochlear aqueduct, or the endolymphatic duct), compromised blood flow through the internal auditory artery (IAA) seems to be the most likely, given the fact that the principal blood supply to the cochlea, the IAA, accompanies the seventh and eighth nerves within the IAC. Decreased blood flow through the IAA could cause loss of CM due to ischemia of the cochlea. The decreased functioning of the hair cells of the inner ear would in turn result in less activity of the auditory nerve and a smaller N-1. This artery also supplies the auditory nerve throughout most of its extramedullary course, so that changes in N-1 that occur with changes in CM could also be due to a direct effect upon the nerve’s blood supply. There is support for the concept of ischemia from experimental work. In guinea pigs it has been shown that occlusion of the IAA results in loss of CM and N-1 potentials. If this idea is correct, then the loss of CM and N-1 during closure in Case 17 (Fig. 3) would be consistent with delayed ischemia, possibly due to vasospasm of the IAA. The changes in N-1 that occurred without any detectable change in CM are unlikely to result from compromised blood flow through the IAA but rather from some other mechanism, such as mechanical disruption of the nerve’s function or localized ischemia at the nerve level alone.

One of the crucial stages in the operation that this study has identified is removal of the tumor from the lateral aspect of the IAC, since on several occasions the auditory nerve potential was lost during this phase. Cases of acoustic neuromas have been described in which the tumor was invading the cochlea through the cribriform area. It is therefore possible that dissecting the lateral remnant of tumor avulsed cochlea nerve fibers. Perhaps in the future, high-resolution imaging techniques will allow precise assessment of the relationship between the most lateral extent of the tumor, the cochlea, and the auditory nerve.

It is difficult to compare our study to other reports because most monitor only BAEP’s and not ECoG. Hardy, et al., described the use of BAEP’s in two patients with acoustic neuromas. In one patient with a 2-cm lesion, a “small amount of tumor” was left adherent to the cochlear nerve when it was noted that Wave I decreased in amplitude and the Wave V latency increased. In the second patient, an intracanalicular tumor was removed through a subtemporal approach with no change in BAEP’s, but there was some loss of hearing. Most of the other series include all posterior fossa procedures with only a few acoustic neuroma operations.

Grundy, et al., monitored BAEP’s in 54 operations on 50 patients having surgery in the CPA. Most of the operations were for microvascular decompression of cranial nerves. Of the seven acoustic neuroma patients, no BAEP’s could be recorded in three, and the eighth nerve was divided in the other four. In 22 patients, changes in BAEP’s occurred with retraction of the cerebellum, and usually resulted with repositioning or removal of the retractors. They also reported that the status of the BAEP’s at the conclusion of the operation correctly predicted the patient’s ability to hear postoperatively. In our series, two patients (Cases 6 and 10) had good BAEP’s at the end of the operation and yet had major loss of speech discrimination. On the other hand, several of our patients had no waves beyond N-1 at the end of the operation and yet had good hearing (we had one exception, Case 16, and Allen, et al., also reported a similar patient). The presence of good hearing despite the intraoperative loss of all the BAEP’s after Wave I is not entirely surprising in view of the fact that many patients with acoustic neuromas (such as Cases 1, 18, 21, and 22) and multiple sclerosis may have good hearing despite absence of recognizable waves beyond Wave I.

Raudzens and Shetter reported intraoperative monitoring of BAEP’s in 46 patients (27 for neoplastic or vascular lesions, 14 for microvascular decompression, and five for vestibular nerve section). There were no changes in 34 patients. Seven patients developed prolonged latencies and reduced amplitude of Wave V with surgical manipulation or retraction in the vicinity of the eighth nerve and, in these patients, hearing was not significantly affected. Four had abrupt loss of all wave forms beyond Wave I, and had a profound hearing loss postoperatively. This differs from our experience with four patients (Cases 2, 3, 14, and 17), who had good hearing despite no intraoperative recovery of waves beyond N-1. The explanation for this discrepancy may be related to the location of the insult, since in our cases the changes usually occurred while the tumor was being dissected off the nerve in or near the IAC and were associated with transient changes in N-1. On the other hand, Raudzens and Shetter were for the most part operating for other types of pathology that apparently did not involve N-1, and were probably at or near the brain stem. They speculated that the mechanism responsible for changes in the BAEP’s involved ischemia of the cochlear nerve or cochlear nucleus.

Our study does not answer the question as to how useful intraoperative monitoring of ECoG and BAEP’s is in improving hearing preservation during removal of an acoustic neuroma; however, it does suggest that other adjuncts may be needed to aid in preserving hearing. Certainly for the cases where no changes occurred, monitoring was unnecessary. In some patients in whom an abrupt major change occurs without recovery, it will not be possible to save hearing, but correlation of the changes with the events during operation may help to identify the cause of deterioration. For example, the problem of abrupt hearing loss with the removal of tumor at the lateral end of the IAC was documented, as were changes with blood vessel manipulation. In other patients, the change in the monitored potentials served as a warning, and the method of dissection was revised; in some of these patients, recovery of the evoked responses occurred.
Preservation of hearing in acoustic tumors

Because of the rapid feedback already available for N-1, it is unlikely that improvements in monitoring the ECoG will help. There is room for improvement in detection of the BAEP's. Direct recording from the eighth nerve has been reported. This technique may be useful in small tumors. However, it is not clear that preservation of hearing would be improved by more rapid feedback about Wave V, since it seems that the state of Wave V is not closely related to hearing preservation. A more profitable direction for future work may be in trying to understand the various mechanisms underlying intraoperative hearing losses, so that specific ways of dealing with each can be developed. Monitoring of evoked potentials can then be used not only for alerting the surgeon that a critical situation is developing, but can also suggest why it is happening in order that specific remedies may be instituted. For example, changes in CM suggest ischemia involving the IAA. Perhaps an investigation of vasospasm due to manipulations in the region of the eighth nerve would be helpful in developing a specific treatment for avoiding irreversible losses in hearing during surgery for acoustic neuroma.

Addendum
Since this detailed analysis was completed, 12 more patients have been monitored. In the total group of 34 patients, correlation of tumor size with postoperative hearing was as follows: discrimination scores of more than 35% were found in five of seven with 1-cm tumors, six of 14 with 1.5-cm tumors, two of nine with 2- to 2.5-cm tumors, and one of four with tumors of 3 cm or more. All patients had a good overall result and returned to normal activity.

References

Manuscript received March 1, 1984.
This work was supported in part by U.S. Public Health Service Grant 5P01 NS13126.
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