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# Does Intraoperative Monitoring of Auditory Evoked Potentials Reduce Incidence of Hearing Loss as a Complication of Microvascular Decompression of Cranial Nerves?

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During a 14-month period, 129 individuals underwent 140 operations for microvascular decompression to relieve hemifacial spasm, disabling positional vertigo, tinnitus, or trigeminal neuralgia at our institution. Seven patients were operated upon twice on the same side and 4 were operated upon on both sides at different times. In each case, the brainstem auditory evoked potentials were monitored intraoperatively by the same neurophysiologist. In 75 of these operations, compound action potentials were also recorded from the exposed 8th nerve. Comparison of speech discrimination scores before the operation and at the time of discharge showed that at discharge, discrimination had decreased in 7 patients by 15% or more and increased in 4 patients by 15% or more, in 2 patients by as much as 52%. Essentially similar results were obtained when preoperative speech discrimination scores were compared with results obtained from the 87 patients who returned for a follow-up visit between 3 and 6 months after discharge. Only one patient lost hearing (during a second operation to relieve hemifacial spasm). Another patient (also operated upon to relieve hemifacial spasm) suffered noticeable hearing loss postoperatively, but had recovered nearly normal hearing by 4 months after the operation. Nine patients had an average elevation of the hearing threshold for pure tones in the speech frequency range (500 to 2000 Hz) of 11 dB or more at 4 to 5 days after the operation; 8 of these had fluid in their middle ears that most likely contributed to the hearing loss. Threshold elevations occurred at 4000 Hz and 8000 Hz in 19 and 29 ears, respectively. (*Neurosurgery* 24:257–263, 1989)

Key words: Hearing loss, Intraoperative monitoring, Evoked potentials, Microvascular decompression

## INTRODUCTION

Hearing loss is a serious complication of operations for microvascular decompression (MVD) of cranial nerves in the cerebellopontine angle (CPA) (3, 4). Although a unilateral loss of hearing may not lead to a major loss in quality of life, it causes a number of practical problems in everyday life, such as loss of directional hearing and increased difficulties in understanding speech in the context of background noise.

Injury to the auditory portion of the 8th nerve is almost always caused by stretching of the nerve from retraction of the cerebellum (4, 5, 13, 17, 18), by surgical manipulations, or by heat from electrocoagulation performed near the nerve (12, 13). The risk of hearing loss is greatest in operations to relieve disabling positional vertigo (DPV) and hemifacial spasm (HFS) (15), but decompression of the 5th cranial nerve for trigeminal neuralgia (TN) (4) and of the 9th cranial nerve (for glossopharyngeal neuralgia) also poses a significant risk of hearing loss (4, 5).

Although it may be reasonable to assume that better surgical technique and the introduction of intraoperative monitoring of auditory evoked potentials has reduced the incidence of hearing impairment as a complication of MVD operations (1–7, 12, 13, 16), few studies have been published that quantitatively assess the loss of hearing as a complication of such operations (3, 15).

The issue of how large a change in brain stem auditory evoked potentials (BAEPs) can be allowed before there is risk of causing measurable permanent hearing loss has been debated. Some investigators have suggested that the surgeon should only be made aware of changes in the evoked potentials that equal or exceed levels that may indicate permanent injury (3), while others have held the opinion that the surgeon should be informed about changes as soon as they become larger than the small variations that are normal for such potentials (12). The reason for communicating such information about small changes in the recorded potentials is that this information will make it possible for the surgeon to identify exactly which surgical manipulation affected the conduction in the auditory nerve, and thus will make it possible to intervene properly and with very little delay. Also, knowledge about which manipulations pose risk of injury to the 8th nerve is important in redesigning an operation that will decrease the risk of hearing loss in future operations. Although it cannot be proven that such small changes in evoked potentials are indicative of a noticeable risk of measurable loss, prolonged effects or increases in an effect with time may increase the risk of permanent neural deficit. For that matter, it is difficult to prove that any change in the evoked potentials, even total loss of the potentials, is indicative of permanent impairment of function. It is particularly difficult to assess the value of intraoperative monitoring of evoked potentials in operations in which complications occur with such low frequency as does hearing loss in MVD operations (15). For this reason, it is important that accurate assessments of hearing function be done before and after the operation to evaluate the results of intraoperative monitoring of auditory evoked potentials.

In the present study, we compare the results of detailed and accurate pre- and postoperative audiometric evaluations in patients who underwent MVD operations to relieve HFS, DPV, or TN.

## METHODS

### *Patients*

The results of the present study were obtained from a total of 140 MVD operations in 129 patients who were operated upon consecutively during the 14-month period from March 1, 1986 to May 19, 1987, and in whom intraoperative monitoring of evoked potentials was performed by the same neurophysiologist (A.R.M.). Table 1 shows the number of patients and operations that were included in the present study.

In none of the cases was intraoperative monitoring cancelled for technical reasons. The patients who were studied had essentially normal pure tone thresholds before the operation, except for such types of hearing losses that are attributable to age or to exposure to noise. The patient's preoperative speech discrimination scores were between 84 and 100%.

*Pre- and postoperative evaluations*

All audiometric tests were performed in a sound-insulated room by a certified audiologist using standard modern equipment. The speech discrimination scores were obtained using recorded test lists (NV-6; Northwestern University). In addition, the tympanic membranes were examined using an otomicroscope, and cerumen was removed if present preoperatively.

*Operative procedure and intraoperative monitoring*

All patients were operated upon using the microvascular decompression (MVD) technique described by Jannetta (8–11). With the patient placed in the lateral decubitus position and the head fixed in a three-pin headholder, a retromastoid craniectomy was performed. The cerebellum was retracted and the cranial nerve to be decompressed was exposed. After identification of the compressing vessel or vessels, these were dissected from the nerve and a soft implant (shredded Teflon) was placed between the vessel and the nerve. In operations to relieve DPV, the offending vessel was often found only after the paraflocculus had been dissected from the 8th nerve. Usually a balanced anesthetic regimen of a narcotic and nitrous oxide together with a muscle relaxant was used in operations on patients with TN or DPV. Since facial electromyographic potentials were monitored intraoperatively in patients undergoing operations for HFS, it was not possible to use long-lasting muscle relaxants, so in these patients, inhalation anesthesia was used, usually with isoflurane and nitrous oxide, supplemented by small amounts of narcotics (12, 14).

Intraoperative monitoring of evoked potentials was performed using techniques that have been described previously in detail (12, 13), so only a brief description will be given here. Click sounds were generated by applying 100-$\mu$s square waves to miniature stereo earphones at a rate of 10 or 19 pulses per second. The recorded potentials were amplified using standard AC amplifiers with filter settings of 3 to 3,000 Hz (6 dB points) and roll-off of 6 dB per octave. The recorded potentials were averaged and digitally filtered by an LSI 11/73 microprocessor to be viewed on an oscilloscope together with a baseline recording that was obtained after the patient was anesthetized but before the beginning of the operation. In most cases, an interpretable record could be obtained on the basis of 30 seconds of recording.

In 62 of the operations for decompression of the 9th and 7th nerves, and in 13 of the cases in which the 5th cranial nerve was decompressed, a recording electrode was placed directly on the 8th nerve or close to it when it became exposed, for recording of the compound action potentials (CAPs). The reference electrode for these recordings was placed on the shoulder (12, 13).

Before the 8th nerve was exposed, BAEPs were continuously compared with baseline BAEPs, to facilitate detection of any effect the surgical manipulation might have on neural conduction in the auditory nerve. Usually, the latencies of either peak III or peak V of the BAEPs were compared. When CAPs were recorded directly from the 8th nerve, they were used for monitoring conduction in the 8th nerve, and changes in both latency and wave form of the CAPs were used to estimate changes in conduction in the auditory nerve. In all instances, the surgeon was informed of changes in the far-field potentials (BAEPs) or near-field potentials (CAPs) that were larger than those that may occur spontaneously (about 0.25 ms). In most cases, intervention was done as soon as a shift in latency was recognized, and only in exceptional cases was intervention delayed until the delay reached 0.75 ms.

TABLE 1
*Patient Data*

| Patients and Procedures | n |
|---|---|
| Hemifacial spasm | |
| Total number of patients | 44 |
| Bilateral MVD[a] | 1 |
| Second operations | 2 |
| Total MVD procedures | 47[b] |
| Patients who had had MVD previously | 4 |
| Disabling positional vertigo and tinnitus | |
| Total number of patients | 51 |
| Patients operated on both sides | 3 |
| Patients who underwent second operations on the same side | 5 |
| Total MVD procedures | 59 |
| Patients who had had MVD previously | 7 |
| Patients who had had vestibular nerve section | 2 |
| Trigeminal neuralgia | |
| Total number of patients | 34 |
| Total MVD procedures | 34[c] |
| Patients with previous MVD | 2 |
| Total all procedures | 140 |

[a] MVD, microvascular decompression.

[b] One patient who was deaf on the affected side before surgery and one patient who was not tested before leaving the hospital are excluded.

[c] Two patients who had no postoperative tests are excluded, as is one patient who was deaf before the operation.

## RESULTS

*Audiometric evaluations*

Only one of the patients in the present study lost hearing as a result of the operation, which was a second operation to relieve HFS (the first operation was performed elsewhere). In addition, one patient (who was also operated upon to relieve HFS) acquired a moderate hearing loss that consisted of a reduction in speech discrimination from 96% preoperatively to 56% 1 week postoperatively. While the first patient's hearing loss was permanent, the second patient's hearing had nearly returned to its preoperative level (84% speech discrimination) by his follow-up visit at 4 months. None of the other patients in this study suffered any noticeable sensorineural hearing loss as a result of the operations they underwent.

One patient's speech discrimination score decreased by 20%, and another patient's by 12%, when tested at the time of discharge from the hospital (Fig. 1). Both patients had middle ear effusion after the operation. The speech discrimination scores obtained at follow-up from the 23 patients who returned for audiometric evaluation were not noticeably different from those obtained at the time of discharge from the

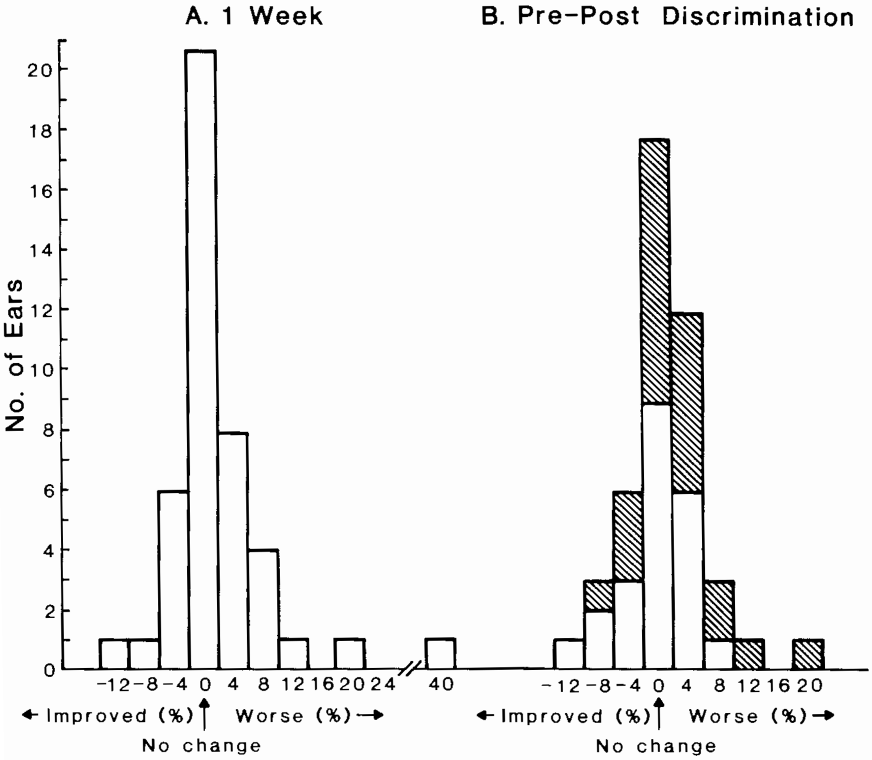


FIG. 1. *A*, differences in the speech discrimination scores obtained preoperatively and at the time of discharge in patients who were operated upon to relieve hemifacial spasm. *B*, same as *A*, but obtained at the follow-up visit at 4 months for the 23 patients who returned for this visit (*open areas*).

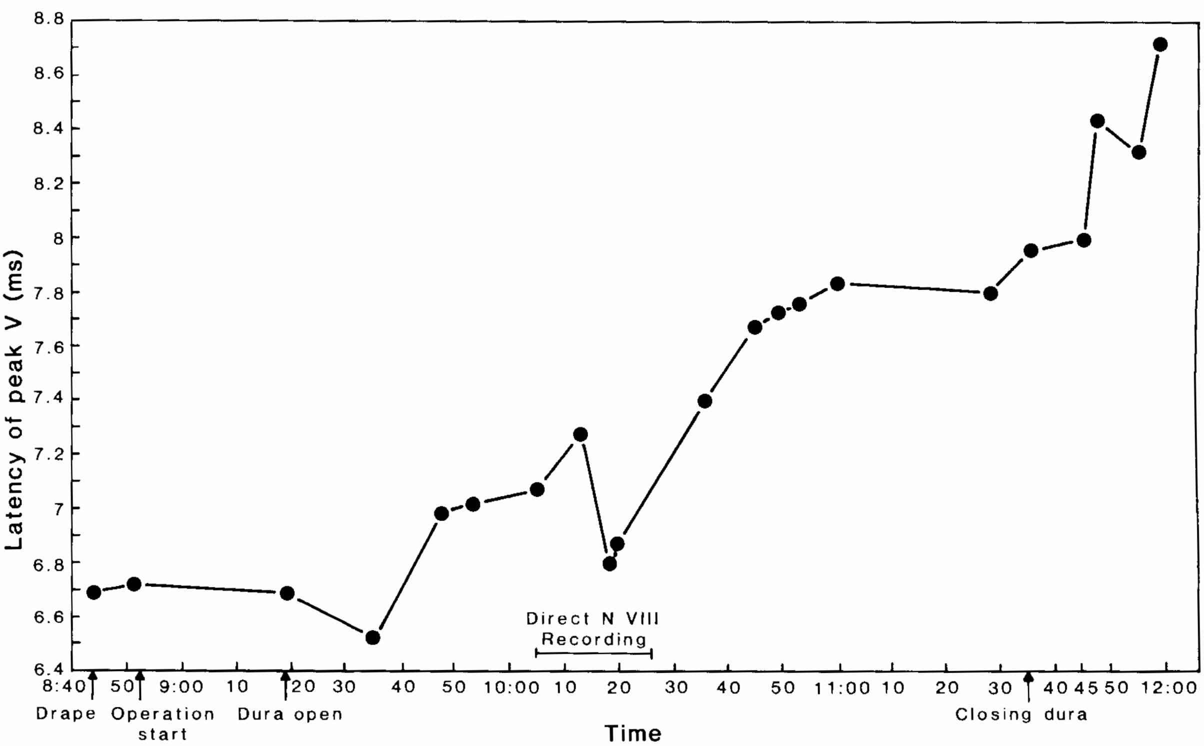


FIG. 2. Changes in the latency of peak V of the BAEP in a patient who lost hearing as a complication of the operation, as recorded during the operation.

hospital. The patient who had a 20% decrease in speech discrimination at the time of discharge did not return for postoperative evaluation.

In 17 ears, the pure tone thresholds in the speech frequency range (500, 1000, and 2000 Hz) obtained at a postoperative visit 3 to 6 months after discharge were elevated 10 dB or more at one or more of these frequencies; two patients had average threshold elevations of 17 and 22 dB, respectively, at 500, 1000, and 2000 Hz when tested 5 days after the operation, but they both had middle ear effusion at that time.

The pure tone threshold increased more than 11 dB at 4000 Hz in 10 ears and 8000 Hz in 16 ears at the time the patients left the hospital, at which time they had clear signs of middle ear effusion. Only 5 of the 16 patients tested 3 to 6 months after the operation had that degree of hearing loss at 8000 Hz, indicating that the middle ear effusion contributed to the hearing losses measured at the time of discharge from the hospital.

In 5 of the 51 patients (59 operations) who were operated upon to relieve DPV, speech discrimination scores had decreased 15% or more at the time of discharge, but only 3 ears showed a threshold elevation of 10 dB or more in the speech frequency range (500, 1000, and 2000 Hz). Of the 9 patients who did not return at 3 months for the postoperative visit, only 1 patient had a hearing loss of more than 10 dB at discharge. At 4000 and 8000 Hz, 4 and 5 ears, respectively,

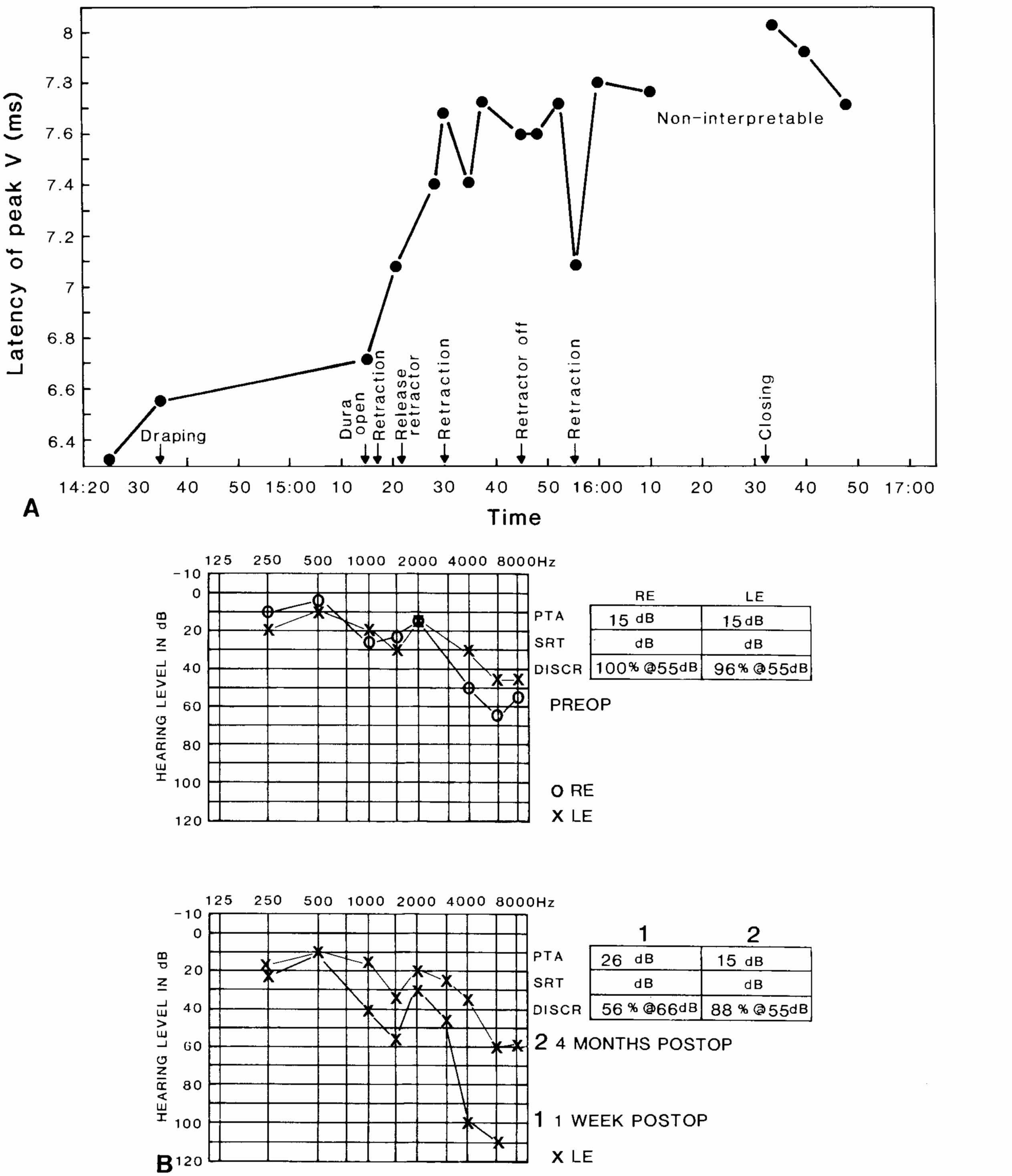


FIG. 3. *A*, changes in the latency of peak V of the BAEP recorded during an operation for left hemifacial spasm in a patient who acquired a moderate hearing loss as a result of the operation. *B*, audiograms obtained before the operation, on the sixth postoperative day, and several months postoperatively.

had threshold elevations of 10 dB or more. Two patients experienced considerable improvement in hearing (more than 15 dB) postoperatively, which was most likely the result of relieving the pressure on the auditory nerve that had been caused by the blood vessel compressing the vestibular portion of the 8th nerve.

None of the 34 patients operated upon for TN had a decrease in speech discrimination of more than 15% at the time they were discharged from the hospital, and only 3 patients had a hearing loss of more than 10 dB (1 patient had a loss of 45 dB) in the speech frequency range. These test results were obtained at the time the patients were discharged from the hospital, and there were clear signs of middle ear effusion in these patients.

### *Intraoperative changes in evoked potentials*

The changes in the latency of peak V in the BAEPs recorded intraoperatively in the patient who lost hearing during an operation for HFS (Fig. 2) were noticeable during retraction of the cerebellum, dissection of the facial nerve, and manipulation during decompression of the nerve. These changes were, however, no larger than have been seen in many other patients who did not acquire any measurable hearing loss as a result of the operation. In addition, the CAPs recorded from the 8th nerve in this patient during the intradural part of the operation showed only a small increase in latency (about 1.1 ms). The latencies of peak V of the BAEPs started to increase while the dura was being closed (Fig. 2), and progressed during closure of the wound, so that just before the skin was closed, the BAEP wave form was highly distorted and identification of the individual waves was difficult. The injury to the 8th nerve that caused permanent hearing loss in this patient thus seems to have occurred after completion of the intradural part of the operation, and could be the result of a shift in the cerebellum or of compression in the cerebellopontine angle that either compromised the blood supply to the cochlea or stretched or compressed the 8th nerve. The relatively slow change in BAEPs makes the latter explanation more plausible, since an interruption in the blood supply to the cochlea would most likely have caused rapid obliteration of BAEPs.

At the time that this patient was operated upon, it was not clear to us that injuries to hearing can occur after completion of the intradural part of the operation. In such cases, recording of BAEPs was continued to the end of the operation solely for research purposes; the surgeon was not notified of these changes, and consequently, no attempts were made to intervene.

In another patient who experienced hearing loss, the BAEPs recorded intraoperatively (during an operation to relieve HFS) showed large increases in the latency of peak V during retraction of the cerebellum, before the 8th nerve was exposed. These changes were at least partially reversed when the retraction was released (Fig. 3*A*). However, as soon as even slight retraction was applied again, the latency of peak V of the BAEPs promptly increased. The CAPs that were recorded directly from the 8th nerve were small, and technical difficulties prevented optimal placement of the recording electrode. The decision was made, therefore, to rely on the recording of BAEPs for monitoring during the rest of the operation. Manipulations in connection with MVD of the facial nerve resulted in large increases in the latency of peak V, and although the surgeon modified his procedure as soon as these changes appeared, the increase in the latency of peak V of the BAEPs accumulated during the intradural part of the operation, so that at the end of this period, the latency was about 1.5 ms longer than it had been when the BAEPs were recorded before the operation. The change in pure tone thresholds is shown in the audiogram in Figure 3*B*.

Changes in the BAEPs and the CAPs recorded from the 8th nerve in a patient who acquired transient hearing loss with operation to relieve DPV are shown in Figure 4, *A* and *B*. It was retraction of the cerebellum that caused the change in the latency of the BAEPs (Fig. 4*A*), and both the latency and the wave form of the CAPs recorded from the 8th nerve changed as well (Fig. 4*B*). Release of the retractor resulted in the potentials approaching the baseline with regard to both latency and wave form, although they did not reach baseline, and at the end of the MVD there was a shift in the latency of the CAPs of about 1 ms. During closure of the incision, when recording of BAEPs was resumed, the latency of peak V was about 1.5 ms greater than preoperatively. The pre- and postoperative audiograms of this patient (Fig. 4*C*) revealed a slight hearing loss at high frequencies at the time of discharge from the hospital, but at the follow-up visit at 3 months, hearing had actually improved relative to the preoperative test results.

## DISCUSSION

The results of the present study show that loss of hearing is a rare complication of MVD operations, even when the target of the operation is the 8th nerve itself. Not surprisingly, second operations pose a greater risk to hearing than initial operations, and the risk of hearing loss is greater during operations to decompress the 7th or 8th cranial nerves than during operations for decompression of the 5th nerve.

It would be of value to know the relationship between the change in the evoked potentials and the risk of permanent hearing loss; however, the present study cannot answer that question. The only patient in the present study who lost hearing had an increasing latency of peak V of the BAEPs at the end of the operation. The patient who had significant hearing loss at the time of discharge from the hospital displayed such large variations in BAEPs that at times the recording could not be interpreted with any degree of certainty. Nevertheless, this patient's hearing had returned to nearly preoperative values at the time of his follow-up visit at 4 months.

Our goal in monitoring patients undergoing neurosurgical operations is to avoid any measurable hearing loss as a complication of the operation, not just to avoid deficits noticeable to the patient as a decreased ability to understand spoken English; these latter deficits were defined by the American Academy of Ophthalmology and Otolaryngology (AAOO) in 1959 as an average 25 dB hearing loss at 500, 1000, and 2000 Hz (1). More recently, the American Academy of Otolaryngology (AAO) determined that a change of up to $\pm 10$ dB at the speech frequencies (500, 1000, 2000, and 3000 Hz) and/or a 15% decrease in discrimination score for phonetically balanced words is not considered a change in hearing (2).

Nevertheless, even speech discrimination scores may not give an accurate picture of the patient's hearing ability immediately after an operation, as many patients still are troubled by severe headache at that time, and thus may not be able to concentrate as well during the hearing test as they had before the operation. For these reasons, a threshold elevation measured only 4 to 5 days postoperatively may not be indicative of hearing loss caused by surgical trauma to the auditory nerve.

In a previous study (15), in which intraoperative monitoring of auditory evoked potentials was only performed during 39 of 143 operations, one of the 39 monitored patients suffered a profound hearing loss as a complication of the operation, while 3 (2.8%) of the 104 patients in whom monitoring was

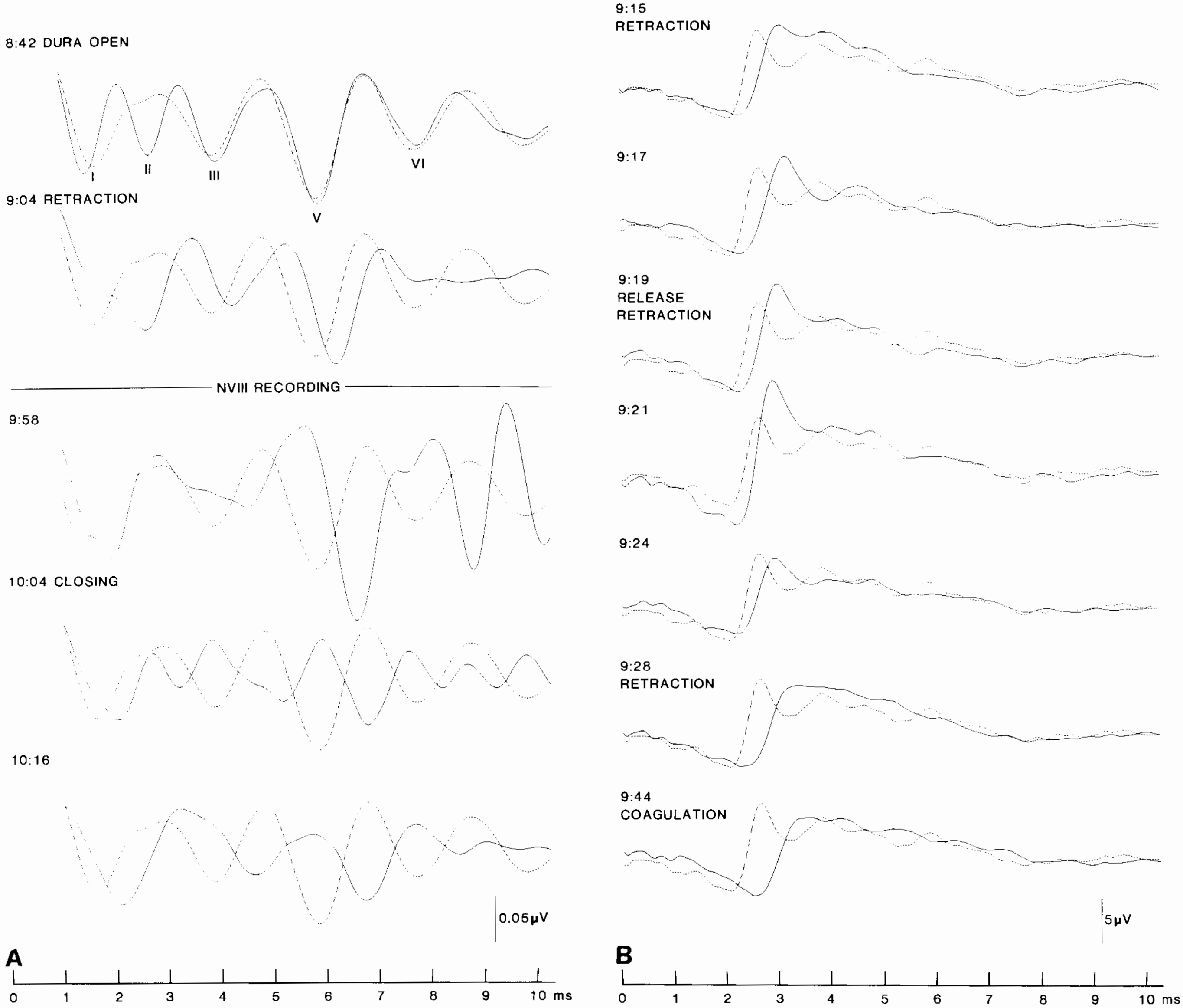


FIG. 4. *A*, sample BAEPs recorded during an operation for right-sided disabling positional vertigo in a patient who acquired a slight elevation of her hearing threshold as a result of the operation. *B*, compound action potentials recorded from the exposed 8th nerve in the same patient. The broken lines show baseline recordings.

not done suffered profound hearing loss. In comparison, only 1 patient (0.7%) lost hearing, and none suffered profound hearing loss, as a result of the 140 operations reported in this paper (13 of which were second operations), in which intraoperative monitoring of auditory evoked potentials was done in all cases. The patient in this study who suffered a complete loss of hearing had the largest changes in BAEPs after closing of the dura, during which no interaction was believed to have resulted in this change. Thus, none of the 140 operations resulted in any profound hearing loss as a result of the intradural manipulations. When this is compared with the results of the earlier study (15), it seems reasonable to assume that intraoperative monitoring of auditory evoked potentials has contributed to this improvement in preservation of hearing during MVD operations. Perhaps the greatest benefit of intraoperative monitoring of auditory evoked potentials, however, has been that it makes it possible to identify with greater precision which step in the operation has caused the injury, so that the surgeon may modify the operative procedure accordingly. The experience that severe changes in BAEPs can occur after closure of the dura has taught us to continue to monitor BAEPs after the dura is closed.

In conclusion, the risk of hearing loss in MVD operations is very small, with or without intraoperative monitoring of auditory evoked potentials. Nevertheless, we believe that the information provided the surgeon by intraoperative monitoring of auditory evoked potentials contributes to keeping the risk of permanent hearing loss low, and that the true incidence of such losses can only be determined if accurate audiometric examinations are performed before and after the operation.

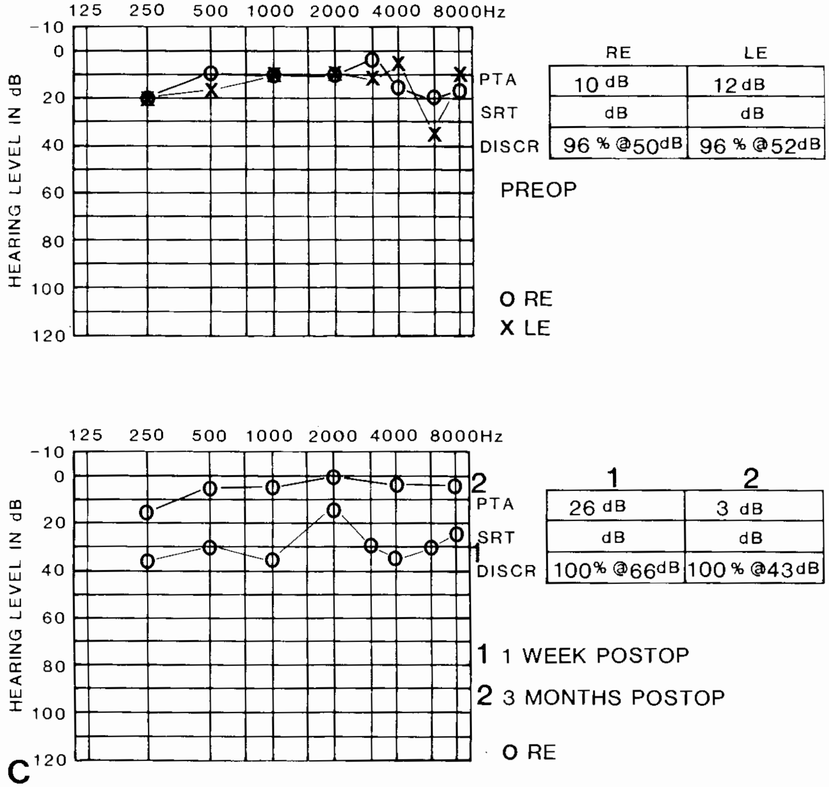


FIG. 4. *C*, audiogram showing change in hearing threshold of the same patient.

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