

## Correlation Between Latency and Amplitude of Peak V in the Brainstem Auditory Evoked Potentials: Intraoperative Recordings in Microvascular Decompression Operations

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### Summary

Intraoperative prolongation of the latency and decrement of the amplitude of peak V of brainstem auditory evoked potentials (BAEP) were studied in 38 microvascular decompression operations in which prolongation of the latency of peak V exceeded 1.0 msec. Postoperative hearing tests of all patients were compared with their preoperative hearing tests.

Postoperative hearing loss was unrelated to the maximum prolongation of latency, but the amplitude decreased to lower values in patients with postoperative hearing loss compared to patients whose postoperative hearing was unchanged ( $P < 0.05$ ). Twelve (32%) of 38 patients whose latency of peak V was prolonged more than 1.0 msec and 11 (61%) of 18 patients whose amplitude of peak V decreased more than 40% during the operations had decreased hearing postoperatively. In all patients, a prolongation of the latency of peak V was always accompanied by a decrease in the amplitude of peak V. The decrement of the amplitude was greater in the patients with decreased postoperative hearing thresholds than in the patients with unchanged postoperative hearing thresholds.

The results of this study indicate that it would be valuable to monitor changes in the amplitude of peak V of BAEP in addition to monitoring the latency of peak V during operations where the VIIIth cranial nerve is manipulated.

**Keywords:** Amplitude; brainstem auditory evoked potentials; latency; microvascular decompression.

### Introduction

Intraoperative monitoring of brainstem auditory evoked potentials (BAEP) during operations in the cerebellopontine angle (CP angle) has been in routine use for a long time [2, 5, 6, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21]. Previously, the changes in the latencies of specific components of the recorded BAEP were used as indicators of intraoperative injuries to the auditory nerve, but more recently, changes in amplitude of the recorded evoked potentials have been recognized to be valuable signs of surgically induced injuries [12]. How-

ever, clinical criteria for changes of amplitude in predicting postoperative hearing have not been established. In the present study, changes in the latency and the amplitude of peak V of the BAEP were correlated with postoperative hearing loss to study the usefulness of intraoperative monitoring of the amplitude of peak V during operations in the CP angle in which the VIIIth cranial nerve may be manipulated to different degrees.

### Patients and Methods

The data for this study were obtained during routine intraoperative monitoring of BAEP in patients undergoing microvascular decompression (MVD) of cranial nerves. Thirty-eight patients in whom there was prolongation of the latency of peak V which exceeded 1.0 msec were selected from about 350 patients who had MVD operations performed during the 14-month period from March 21, 1995 to May 26, 1996 (Table 1).

Before the operation, all patients had audiometric tests for each ear including determination of pure tone thresholds and speech discrimination scores (SDS) using standard audiometric techniques. These tests were repeated postoperatively. A postoperative increase in hearing threshold of more than 10 dB in the speech frequency range of 500, 1000, 2000, and 4000 Hz, and/or a 15% decrease in the speech discrimination score were classified as a hearing loss

Table 1. *Patients Data*

Diagnosis	N	Postoperative hearing	
		Reduced	Unchanged
Trigeminal neuralgia	16	6	10
Hemifacial spasm	15	4	11
Disabling positional vertigo and tinnitus	6	2	4
Glossopharyngeal neuralgia	1	0	1
Total	38	12	26

in accordance with the criteria of the American Academy of Otolaryngology [1].

The patients were operated in the lateral decubitus position with a retromastoid craniectomy as described earlier [8, 9, 10]. After retraction of the cerebellum, the compressing vessel was dissected from the respective cranial nerve and small pieces of shredded Teflon were placed between the vessel and the nerve. During the operation, temperature of the patients was monitored continuously and maintained within  $37 \pm 0.5^\circ\text{C}$ .

The techniques of intraoperative monitoring of BAEP have been described previously in detail [12]. The sound stimuli used were clicks delivered through earphones at an intensity of 90 dB peak equivalent sound pressure level (PeSPL) at rate of 19.3 pps (pulses per second). Either condensation or rarefaction clicks were used, and the click polarity that gave the largest amplitude of the BAEP was chosen. BAEP were recorded from needle electrodes placed at the vertex and at the neck at the level of the C3 vertebrae as a noncephalic reference. This way of recording BAEP provides a record with a distinct peak V, which is used as an indication of injury to cochlear nerve since it is the most prominent and most easily identified intraoperatively [12]. The recorded potentials were amplified (50,000 times) with filters set at 10 to 3000 Hz, and the averaged BAEP were digitally filtered using a W50 zero-phase digital filter [12]. The baseline BAEP was obtained from preoperative recordings, done after the patients were anesthetized but before the operations began. From these several records, we selected the one with the highest amplitudes. During the operation, the BAEP were recorded continuously and the latencies and amplitudes of peak V were compared with those of the baseline BAEP. When the changes in peak V of the BAEP were larger than those that usually occur spontaneously (about 0.25 msec), the surgeon was informed of the changes.

The analysis of the recorded BAEP for the present study was done off line using the same digital filter that was used intraoperatively and using computer programs to automatically identify the different peaks and to print their latencies. The amplitude of peak V was measured between peak V and the following large vertex negative peak. The ratio of that amplitude value to that of the baseline recording was expressed as a percentage.

## Results

Twelve (32%) of 38 patients had postoperative hearing loss (Table 1), according to the criteria given above, but only two of those patients had pronounced hearing loss. In twenty-six patients hearing was unchanged postoperatively.

The amplitude of peak V decreased below 40% of its preoperative value in 18 patients, and 11 (61%) of those patients showed postoperative hearing loss. A chi-square test of these results (Table 2) showed that the change was significant (Fisher's exact test:  $P < 0.05$ ).

The maximum prolongation of latency in each patient during the operation did not correlate with incidence of postoperative hearing loss (Student's t-test:  $P = 0.18$ , Fig. 1A), but in patients with postoperative hearing loss the amplitude of peak V decreased to smaller values ( $P < 0.05$ , Fig. 1B).

Table 2. Correlation Between Changes of Latency or Amplitude and Postoperative Hearing

	Total	Postoperative hearing	
		Reduced	Unchanged
Max. of latency $>1.0$ msec	38	12	26
Min. of amplitude $<40\%$	18	11	7

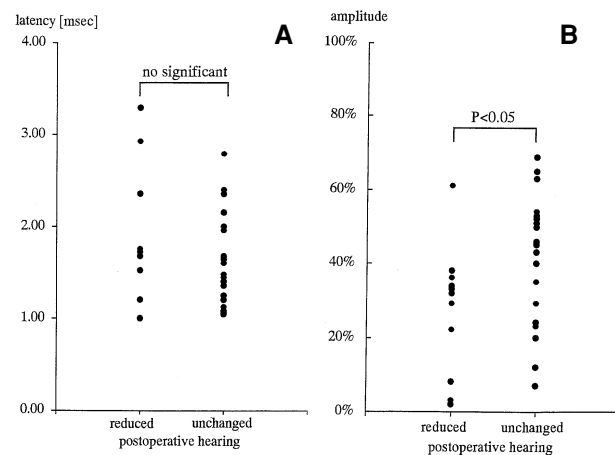


Fig. 1. (A) The maximum prolongation of the latency of peak V that occurred during each operation, in patients who had postoperative hearing loss (left hand column), and in patients whose hearing was unchanged after the operation (right hand column). (B) The lowest values of the amplitude of peak V that was recorded during each operation (in percentage of the preoperative values) in patients who had postoperative hearing loss (left hand column), and in patients whose hearing was unchanged after the operation (right hand column)

The linear regression of the relationship between the prolongation of latency in peak V and its amplitude (in percentage of preoperative values) was steeper (Student's t-test:  $P < 0.05$ ) in the patients with reduced postoperative hearing (Fig. 2A) than in the patients with no change in postoperative hearing (Fig. 2B).

Representative cases are described below.

### Case 1

Fig. 3A shows typical intraoperative recordings of BAEP in a patient with left hemifacial spasms, in whom a prolongation of the latency of peak V and a decrease of the amplitude occurred after retraction of the cerebellum. The latency and amplitude recovered gradually towards the end of the operation. The decrease in the amplitude occurred concomitantly with the prolongation of the latency (Fig. 3B). The patient's postoperative hearing was unchanged when tested one

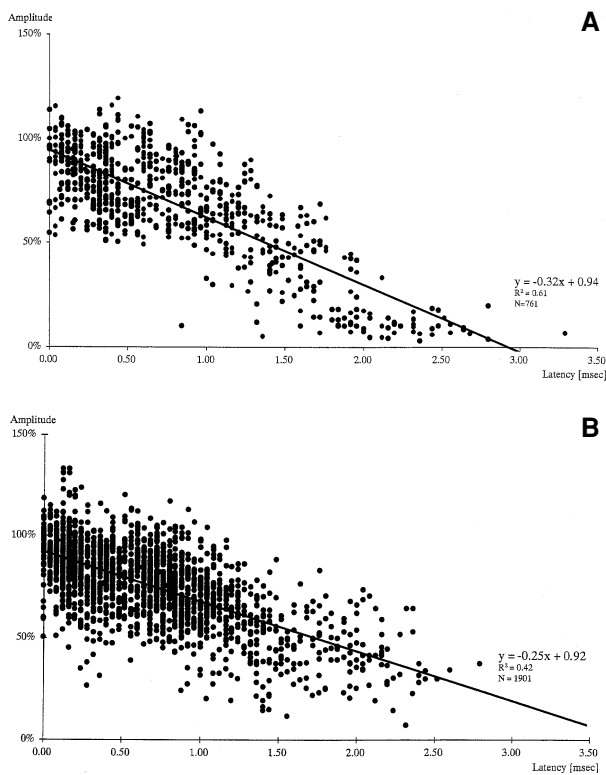


Fig. 2. Amplitude of peak V (in percentage of preoperative values) as a function of the prolongation of the latency of peak V. (A) Data from patients whose hearing had deteriorated during the operation. (B) Data from patients who had unchanged hearing after the operation

week after the operation even though the prolongation of latency of peak V reached 2.16 msec and the minimum amplitude was 35% of the baseline amplitude at one point during the operation.

Case 2

This patient, who was operated on for left tinnitus, is the only patients in whom the amplitude of the BAEP never decreased below 40%, nevertheless she had a deterioration of hearing postoperatively. The changes of the latency and amplitude of peak V of BAEP occurred during manipulation of the VIIIth nerve (Fig. 4A). The course of decrease in the amplitude followed the course of prolongation of the latency (Fig. 4B). The maximum prolongation of latency was 1.52 msec and the minimum amplitude was 61% of baseline. The patient's postoperative hearing threshold was increased by 5–15 dB in frequency range of speech, but her speech discrimination was only slightly impaired, dropping from 100 to 92% (Fig. 4C).

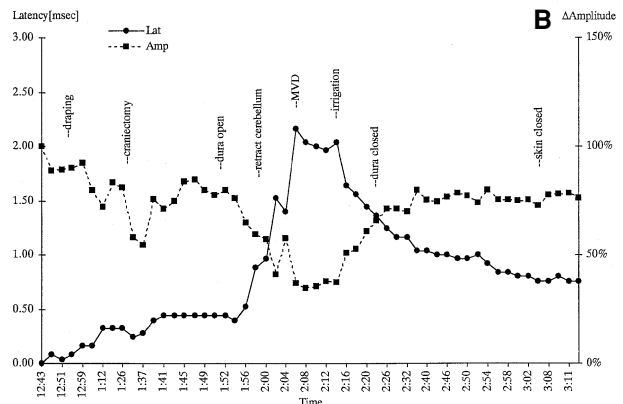
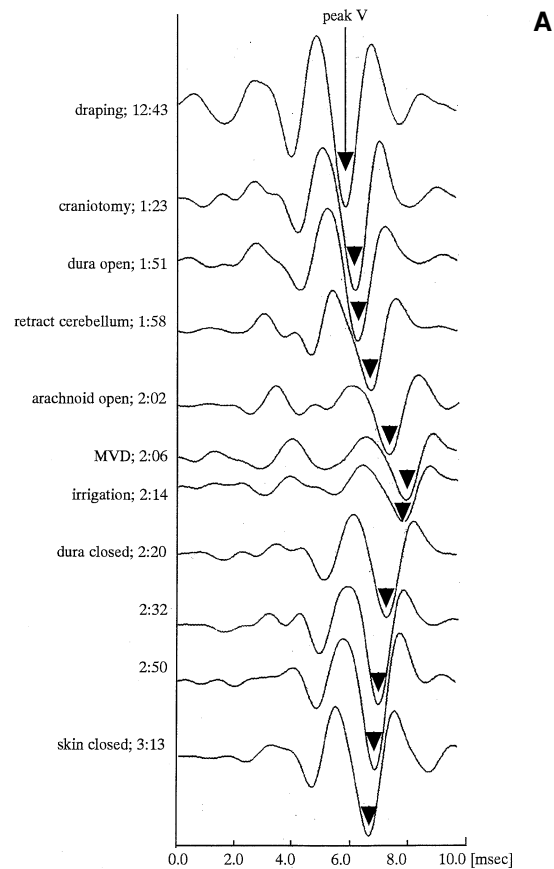


Fig. 3. (A) Vertex positive peaks of brainstem auditory evoked potentials (BAEP) during the operation are shown as a downward deflection. BAEP changes related to retraction of cerebellum during the operation for hemifacial spasm. Prolonged and decreased peak V returned toward normal after closing dura (arrows; peak V). (B) Differences from baseline in the latency and amplitude of peak V during the operation of the case shown in Fig. 3A, who experienced no hearing loss postoperatively

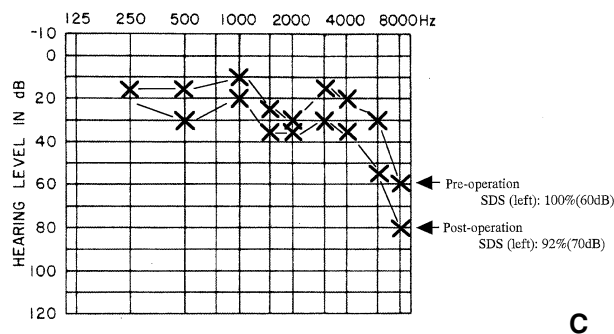
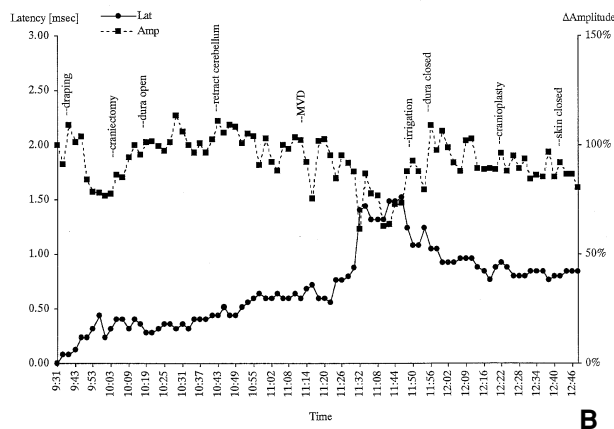
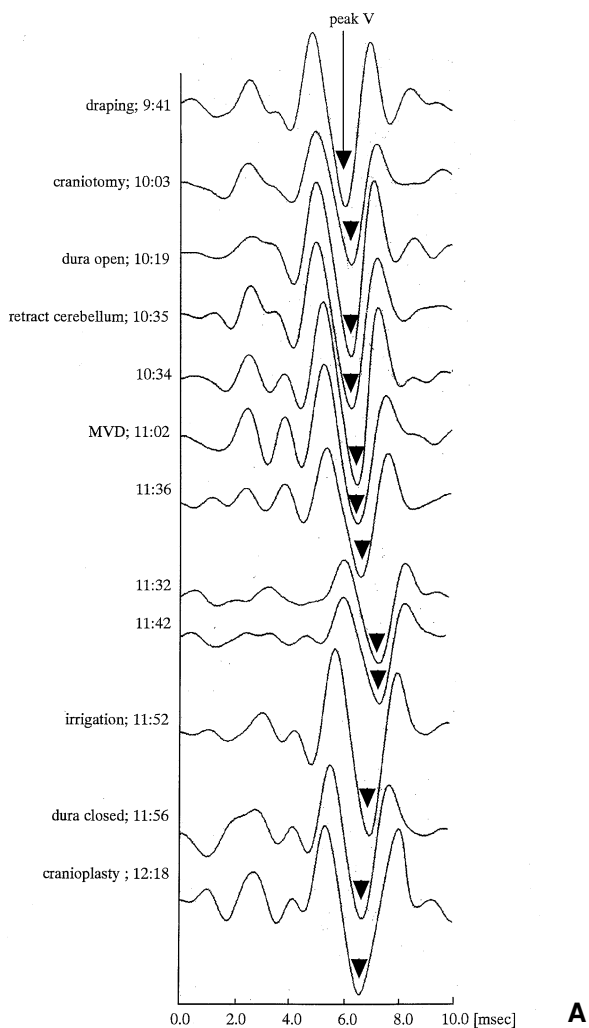


Fig. 4. (A) Prolonged and decreased peak V due to MVD for tinnitus returned toward normal after closing dura. (arrows; peak V) (B) Differences from baseline in the latency and amplitude of peak V during the operation of the case shown in Fig. 4A, who experienced slight postoperative hearing loss. (C) Audiogram obtained before the operation and one week postoperatively in Case 2

Case 3 and 4

A patient who was operated on for DPV and who experienced pronounced postoperative hearing loss, had a large increase in the latency and decrease in the amplitude of peak V during decompression of the VIIIth nerve (Fig. 5A). The changes in the BAEP reversed only partially after that manipulation of the VIIIth nerve was stopped, and large changes both in the latency and the amplitude remained until the end of the operation (Fig. 5B). The maximum prolongation of the latency was 2.92 msec and the amplitude decreased to 3% of the baseline value. This patient suffered a pronounced hearing loss postoperatively with

no speech discrimination (a total loss of useful hearing) (Fig. 5C).

The BAEP of another patient who suffered severe hearing loss after an operation for hemifacial spasm had a prolongation of the latency of peak V of 3.29 msec and a decrease of the amplitude to 2% of the baseline value during the operation. This patient had a decrease in speech discrimination from 84% to 44% after the operation.

The findings in these two patients are examples which show that longer prolongations of the latency of BAEP and greater reductions in amplitude are associated with greater postoperative hearing loss.

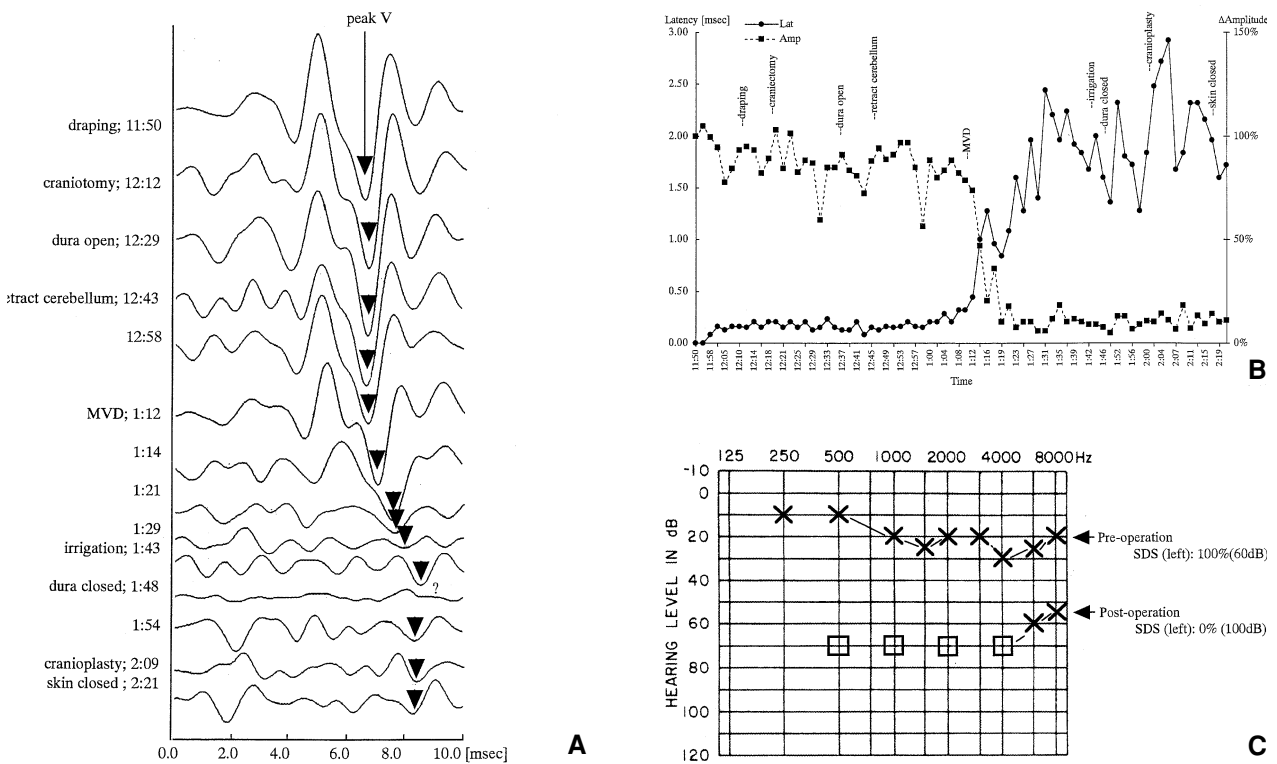


Fig. 5. (A) Peak V of BAEP was distorted strongly while manipulating the VIIIth nerve and showed poor recovery during the operation for DPV. (arrows; peak V). (B) Differences from baseline in the latency and the amplitude of peak V during the operation of the case shown in Fig. 5A, who showed pronounced hearing loss postoperatively. (C) Audiogram obtained before the operation and one week postoperatively in Case 3

**Discussion**

The value of changes in the latency of peak V of the BAEP in intraoperative monitoring for hearing preservation during operations where the auditory nerve is being manipulated is based on the assumption that a prolongation of the conduction time in the auditory nerve is a valid indicator of injury to the auditory nerve. However, the mechanism for decrease in conduction velocity as a result of surgical manipulation is unknown.

The amplitude of the compound action potentials (CAP) that can be recorded from a nerve in response to a transient activation is proportional to the number of nerve fibers that conduct nerve impulses [4]. Therefore, a decrease in the amplitude of the CAP as a result of manipulation may be assumed to be the result of a conduction block in a certain number of nerve fibers. The amplitude of the various components of the BAEP (including peak V) may also be presumed to be related to the number of nerve fibers in the auditory nerve that conduct nerve impulses, but this relationship is prob-

ably more complex than that of the CAP recorded from the auditory nerve [12]. The decrease in the amplitude of peak V probably does not correlate to the number of conducting nerve fibers of the auditory nerve to the same extent that the amplitude of the CAP of the nerve is.

Physiologic factors such as body temperature and the anesthetics that are used may affect the latency of the different components of the BAEP, but these factors are not likely to have a consistent effect on the amplitude [7, 20]. When the amplitude decreases without prolongation of the latency, change in perfusion (e.g. from hypotension) should be considered as the most likely cause [6]. This means that the amplitude of the various components of the BAEP might be more significant and therefore more valuable than the latencies as an indicator of injury to the auditory nerve. However, the amplitude shows a large degree of spontaneous variability which may be related to recording conditions, electrode impedance and a number of unknown factors [12]. Furthermore, the averaged amplitude will decrease if the latencies of the BAEP change

during the time that the recorded potentials are being acquired, because the peaks shift in time during the time that the responses are averaged.

The present study demonstrates that the amplitude of peak V decreases in a similar way as the prolongation of the latency. The fact that the degree of decrement in the amplitude of peak V tends to be steeper in the patients with postoperative hearing loss than in those whose hearing is unchanged indicates that a significant progressive decrease of the amplitude accompanied by only a slight prolongation of the latency of peak V could be a warning of a risk of postoperative hearing loss.

Despite the many studies of the relationship between the change in the evoked potentials and the risk of permanent hearing loss, it is still not known what degree of peak V delay or decrease in amplitude is clinically important. Fluctuations in latency of peak V up to 1.0 msec are frequently seen in operations in the CP-angle, and these changes are probably not associated with direct surgical threat to auditory or brainstem function [3, 16, 17]. Among the patients that we studied in whom the latency of peak V was prolonged more than 1.0 msec, 12 of 38 patients (32%) showed postoperative hearing loss. On the other hand, a loss of greater than 50% of peak V amplitude has been described earlier as potentially important [16]. In this study, a loss of greater than 40% of peak V amplitude occurred in 18 patients of which 11 (61%) had postoperative hearing loss. Therefore a decrease in amplitude of peak V of more than 40% seems to be a better criterion of injury for the purpose of predicting the risk of postoperative hearing loss than use of prolongation in latency alone.

It is important to note that this study is limited to patients in whom the latency of peak V was prolonged 1.0 msec or more intraoperatively. The results of hearing loss can therefore not be taken as representative for MVD operations in our institution (which has been assessed earlier [14]). The results do, however, indicate that a latency change of more than 1.0 msec indeed poses a substantial risk of postoperative hearing loss (12 of 38 patients).

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## Comments

The leadership of the Pittsburg team in MicroVascular Decompression (MVD) for neurovascular conflicts is well-known. In this article the authors add new insights to our knowledge, on the neurophysiologic control by BAEP monitoring of this surgery which may be dangerous for hearing.

Increase in *Latency* of Peak V (of more than 1–0 msec.) is classically considered as the main warning-sign of hearing threatening during MVD surgery. This work shows that a decrease in *Amplitude* of peak V (of more than 40%) is also an indicator of injury of the cochlear nerve. The authors draw attention to the fact that appearance of a significant reduction in amplitude of peak V is a better criterion of risk of postoperative hearing loss than prolongation of latency alone.

It is now well established that the infratentorial-supracerebellar route for approaching the trigeminal nerve, and the infero-lateral cerebellar route for approaching the VII-VIII nerve complex, make stretching of the cochlear nerve minimal and therefore keyhole-MVD little risky for hearing. Nevertheless if surgery has to manipulate the VIIth nerve or to work in its vicinity or close to its vessels, intraoperative BEAP monitoring is very useful, if not mandatory. A good knowledge of the “warning-signs” by both the neurophysiologist and the neurosurgeon is of prime importance.

*M. Sindou*

These authors have studied a single component of the BAEP (wave V) during 38 microvascular decompressions and now describe details of their findings only in those patients in which a minimum latency delay of 1 msec was observed. There main finding is that in this subgroup (with a latency delay of more than 1 msec) only 32% of the patients had hearing decrease but 61% of 18 patients with amplitude decrease had hearing decrease. Contrary to the usual practice of mainly observing latency delays as warning criterion during AEP monitoring they now suggest that observing the amplitude might also be useful. This is of relevance for those neurosurgeons who try to monitor acoustic nerve function during neurovascular decompression and acoustic neurinomas.

These patients with a minimum change of 1 msec were picked from a large series of 350 microvascular decompression operations monitored. The significance of hearing loss was defined according to the criteria of the American Academy of Oto-Laryngology. The degree of amplitude loss considered significant was arbitrarily set at 40%. Applying these limits of abnormality the amplitude loss was significantly more significant than latency delay. The study also contains other interesting findings, described previously: You may not have hearing loss despite severe latency prolongation. You may have hearing decrease although the amplitude never decreased below 40%. The discussion on the possible reasons for amplitude loss is brief but interesting enough.

The paper is richly illustrated and if the editor wishes, a few recommendations could be given as to reduce the number of Tables.: The content of Table 2 is contained in the text. This is a well written and concise paper. It illuminates a detail which may be important for the clinical monitoring in posterior fossa surgery. The expertise of the authors is well known

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