
Intraoperative Loss of Auditory Function Relieved By Microvascular Decompression of the Cochlear Nerve

John B. Wahlig, Anthony M. Kaufmann, Jeffrey Balzer,
Thomas J. Lovely and Peter J. Jannetta

ABSTRACT: Background: Brainstem auditory evoked potentials (BAEP) are useful indicators of auditory function during posterior fossa surgery. Several potential mechanisms of injury may affect the cochlear nerve, and complete loss of BAEP is often associated with postoperative hearing loss. We report two cases of intraoperative auditory loss related to vascular compression upon the cochlear nerve. **Methods:** Intra-operative BAEP were monitored in a consecutive series of over 300 microvascular decompressions (MVD) performed in a recent twelve-month period. In two patients undergoing treatment for trigeminal neuralgia, BAEP waveforms suddenly disappeared completely during closure of the dura. **Results:** The cerebello-pontine angle was immediately re-explored and there was no evidence of hemorrhage or cerebellar swelling. The cochlear nerve and brainstem were inspected, and prominent vascular compression was identified in both patients. A cochlear nerve MVD resulted in immediate restoration of BAEP, and both patients recovered without hearing loss. **Conclusion:** These cases illustrate that vascular compression upon the cochlear nerve may disrupt function, and is reversible with MVD. Awareness of this event and recognition of BAEP changes alert the neurosurgeon to a potential reversible cause of hearing loss during posterior fossa surgery.

RÉSUMÉ: Perte de la fonction auditive peropératoire soulagée par décompression microvasculaire du nerf cochléaire. Introduction: Les potentiels évoqués auditifs du tronc cérébral (PÉATC) sont des indicateurs de la fonction auditive qui sont utiles pendant la chirurgie de la fosse postérieure. Une lésion du nerf cochléaire peut survenir par différents mécanismes et une perte complète des PÉATC est souvent associée à une perte auditive postopératoire. Nous rapportons deux cas de perte auditive peropératoire reliée à une compression vasculaire du nerf cochléaire. **Méthodes:** Les PÉATC ont été surveillés peropératoire dans une série consécutive de plus de 300 décompressions microvasculaires (DMV) effectuées sur une période de 12 mois récemment. Chez deux patients qui subissaient un traitement pour névralgie du trijumeau, les ondes ont disparu complètement pendant la fermeture de la dure-mère. **Résultats:** L'angle ponto-cérébelleux a été réexploré immédiatement. Il n'y avait par d'évidence d'hémorragie ou d'oedème cérébelleux. Le nerf cochléaire et le tronc cérébral ont été inspectés et une compression vasculaire évidente a été identifiée chez les deux patients. Une DMV du nerf cochléaire a provoqué une restauration immédiate des PÉATC et les deux patients ont récupéré sans perte auditive. **Conclusion:** Ces cas illustrent qu'une compression vasculaire du nerf cochléaire peut affecter sa fonction et qu'elle est réversible par DMV. La connaissance de cette complication et l'observation des changements des PÉATC alertent le neurochirurgien à une cause réversible potentielle de perte auditive pendant la chirurgie de la fosse postérieure.

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The relative efficacy and safety of microvascular decompression (MVD) for a variety of cranial nerve compression syndromes has been well established.¹⁻⁵ While postoperative neurological deficits have been uncommon, hearing loss has been a serious albeit infrequent complication of MVD for trigeminal neuralgia. Various series have quoted incidences of hearing loss between 0-18%.^{1,6-9} Several intraoperative events may result in cochlear nerve dysfunction, including surgical manipulation, retraction, and thermal injury from irrigation fluids.¹⁰⁻¹³ Intraoperative monitoring of brainstem auditory evoked potentials (BAEP) has been widely employed to detect alteration of auditory function which may respond to adjustment of

surgical technique, and has successfully reduced the occurrence of postoperative hearing loss.¹⁴⁻¹⁶

MVD procedures for the treatment of cranial nerve compression syndromes have been carried out at the University of

From the Department of Neurological Surgery, University of Pittsburgh Medical Center, Presbyterian University Hospital, Pittsburgh, Pennsylvania, (J.B.W., J.B., T.J.L., P.J.J.); Division of Neurosurgery, Department of Clinical Neurosciences, University of Calgary, Calgary, Alberta, (A.M.K.)

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Reprint requests to: Anthony M. Kaufmann, Department of Clinical Neurosciences, University of Calgary, 12th Floor, Foothills Hospital, 1403 - 29th Street N.W., Calgary, Alberta, Canada T2N 2T9

Pittsburgh since 1972, and intraoperative monitoring of BAEP has been routinely employed since 1984. During a recent 12-month period, three neurosurgeons (PJJ, TJJ, and AMK) performed over 300 of these procedures, 212 for trigeminal neuralgia. In two cases, BAEP were suddenly lost during final dura closure. Successful intraoperative measures were employed to restore normal responses and maintain postoperative hearing. The mechanism of acute cochlear nerve microvascular compression is described, and the importance of attention to intraoperative monitoring changes is highlighted.

PATIENT 1

A 66-year-old gentleman with a three-year history of typical trigeminal neuralgia ($V_{2,3}$), refractory to medical therapy, underwent a retro-mastoid craniectomy and MVD. A branch of the petrosal vein that coursed between the rostral fascicles of the trigeminal nerve was coagulated and divided. Also, an anterior inferior cerebellar artery (AICA) branch compressing the lateral aspect of the nerve was mobilized, and shredded Teflon® felt implants were placed between the artery and nerve. During the MVD, BAEP were stable and latency increases were

less than 1 millisecond. However, during closure of the dura, there was spontaneous loss of BAEP (Figure 1).

The dura was reopened and no hemorrhage or swelling was encountered. The cerebellum was gently elevated and the flocculus sharply dissected away from the vestibular-cochlear nerve. The AICA was found to be compressing the cochlear nerve and its root entry zone, before coursing up between the seventh and eighth cranial nerves towards the trigeminal nerve where it was previously decompressed (Figure 2). A MVD of the cochlear nerve was performed. The BAEP then rapidly returned towards baseline (Figure 1). The dura was then closed and the procedure completed. Postoperatively the patient awoke neurologically intact and demonstrated no evidence of hearing loss, as compared to pre-operative assessment (Table).

PATIENT 2

A 55-year-old man presented with a ten-year history of typical trigeminal neuralgia ($V_{2,3}$), refractory to medical treatment. Preoperative audiometry and BAEP disclosed a mild hearing deficit (Table). At surgery, a large arterial loop was incidentally noted caudal to the eighth nerve, although this was not approached or manipulated. The trigeminal nerve was compressed by a loop of the superior cerebellar artery, and a MVD was performed. BAEP were variable with latency delays of up to

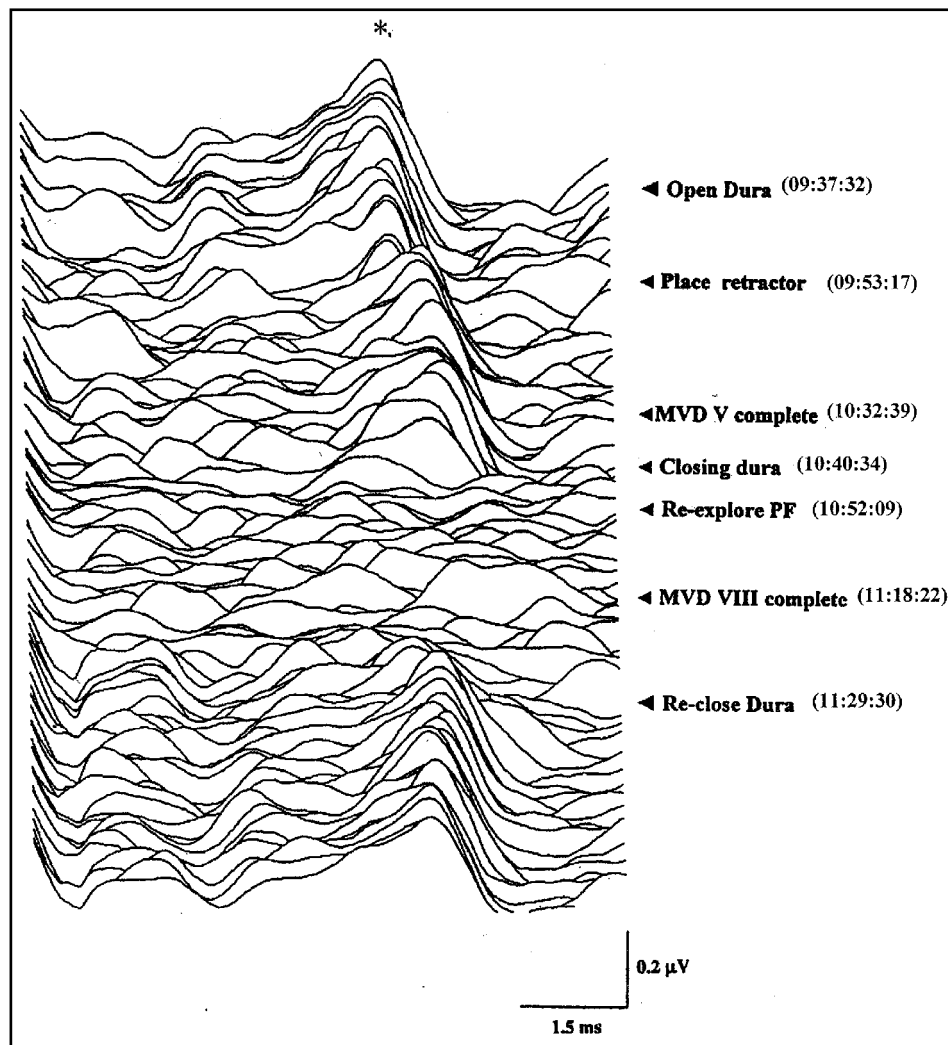


Figure 1: Ipsilateral brainstem auditory evoked potentials (BAEP) recorded in Patient 1. Alterations of the IV/V waveform complex (*) seen in relation to operative events. MVD – microvascular decompression; PF – posterior fossa; V – trigeminal nerve; VIII – cochlear nerve.

Table: Pre- and Post-operative audiology; PTA = pure tone average (dB); SDS = speech discrimination score (%@ dB) Tests performed within one week perioperatively.

Patient	Test	Side	Pre-Op	Post-Op
1	PTA	R	8	7
	SDS	R	96@ 58	96@ 47
	PTA	L	12	7
	SDS	L	96@ 52	96@ 47
2	PTA	R	17	20
	SDS	R	80@ 65	86@ 60
	PTA	L	10	17
	SDS	L	84@ 55	86@ 57

1.25 milliseconds that corrected with adjustment of cerebellar retraction. No significant amplitude reduction occurred. While the dura was being closed, the BAEP suddenly decreased and disappeared.

The dura was reopened and no swelling or hemorrhage was encountered. Vascular compression of the cochlear nerve was suspected as the cause of cochlear nerve dysfunction. The cerebellum was mobilized and retracted from the lower cranial nerves with additional arachnoid dissection. The rostral knuckle of the vertebral artery was found to be severely compressing the cochlear nerve at the brainstem. The BAEP improved somewhat with initial retraction of the cerebellum and then dramatically improved with MVD of the cochlear nerve, returning to preoperative baseline by the completion of closure. In the recovery room hearing was grossly intact, and postoperative audiometry performed one week later demonstrated no significant change in the patient's pre-established baseline mild hearing deficit (Table).

DISCUSSION

BAEP have been consistently used at the University of Pittsburgh Medical Center for all microvascular decompression cases since 1984. The well described technique involves placement of scalp needle electrodes and a bilateral earphone to

deliver alternating 95 dB auditory clicks at 17.5 Hz. The observational interval is 10 msec, with 1024 target trials, and a sample rate of 10,000 Hz.¹² Prior to the initiation of routine intraoperative monitoring, the risk of hearing loss during MVD procedures was 2.6%, and thereafter decreased to 0.6%.¹ Other centers have reported similar reliance upon this technology.^{10,11,14,16,17} Despite these observations, it has been difficult to prove the actual efficacy of BAEP in preventing hearing loss during microvascular decompression procedures.^{10,14,18} Nevertheless, we have made efforts to respond to changes of BAEP with adjustment of retraction, further arachnoid dissection, or mobilization of vessels causing apparent cochlear nerve compression. These interventions are made for any increased latency approaching 1 millisecond, as prolonged delays between 1.5 and 2.5 milliseconds carry a significant risk of hearing loss.^{14,19}

The complete intraoperative loss of BAEP is strongly, although not absolutely, predictive of hearing loss if not immediately reversed.^{10,14} Figure 3 presents selected BAEP tracings at key times during the surgical procedure in Patient 1, and clearly demonstrates loss of all waves. While wave V is typically followed during intraoperative monitoring, all preceding waves were also lost in the two presented patients, while contralateral responses remained unchanged. This supports the concept that the ipsilateral signal loss was related to cochlear nerve compression, since waves I and II are generated by the distal and proximal portions of the cochlear nerve.¹² Unfortunately, computerized records of BAEP in Patient 2 were lost. However, the intraoperative events and BAEP changes in both cases were alike. The immediate recovery of BAEP following cochlear nerve MVD is highly suggestive that such vascular compression may produce physiologic loss of auditory function.

In the patients presented, the intraoperative loss of auditory function apparently due to vascular compression of the cochlear nerve. In Patient 1, the direct manipulation of the AICA loop

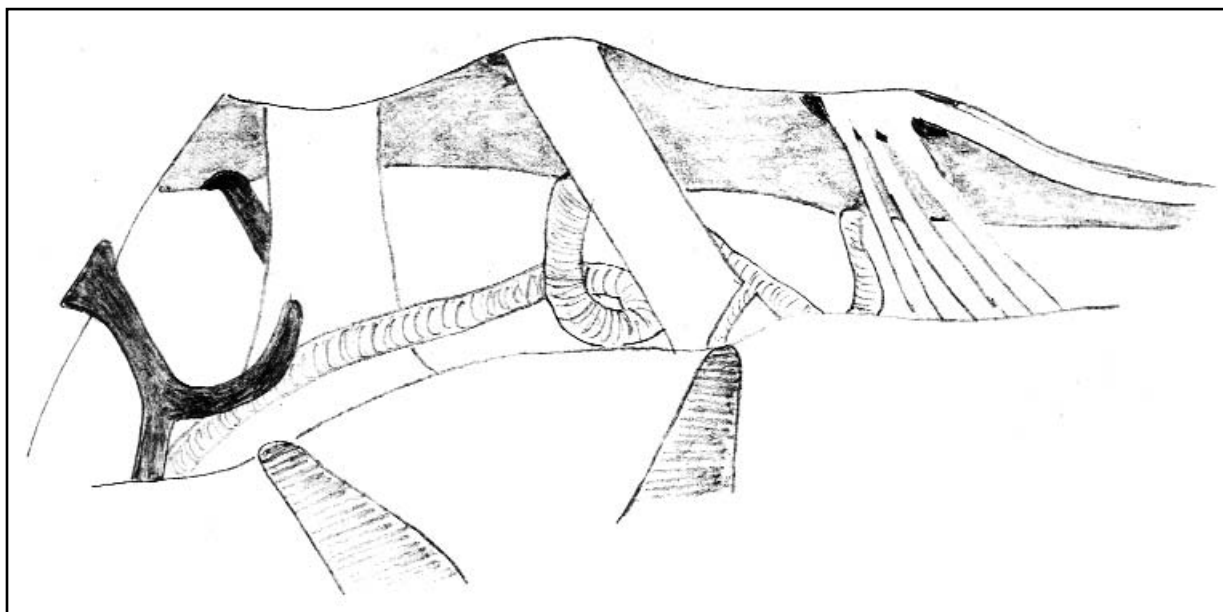


Figure 2: Diagram of operative findings in Patient 1. Microvascular decompression of right trigeminal nerve (V) resulted in shift of anterior inferior cerebellar artery (AICA), accentuating vascular compression upon cochlear nerve (VIII). Retractors (R); petrosal vein (pv); lower cranial nerves (IX-XI); brainstem (BS); cerebellum (Ce).

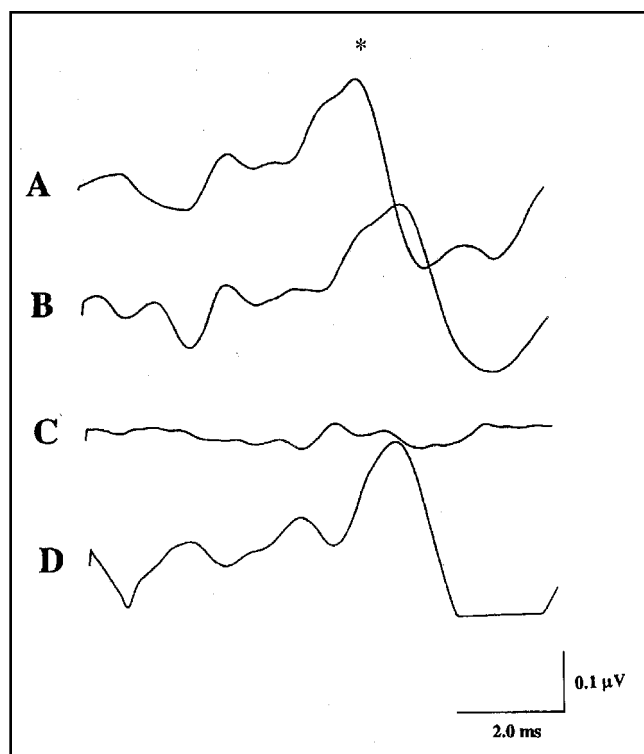


Figure 3: Brainstem evoked potentials recorded in response to click stimulation of the operative side ear during (A) exposure of the CP angle, (B) decompression of the trigeminal nerve, (C) upon closure of the dura after trigeminal nerve decompression, and (D) after re-exploration of the CP angle and subsequent decompression of the cochlear nerve. Note that not only does the IV/V complex of the BAER disappear, but so do the earlier generated waves of the response.

during trigeminal nerve MVD may have produced compression upon the cochlear nerve. In Patient 2, shift of the ectatic vertebral artery following arachnoid dissection and cerebellar retraction may have aggravated the degree of compression upon the cochlear nerve. It is not clear whether the loss of BAEP during dural closure was due to sudden compression of the cochlear nerve precipitated by re-accumulation of cerebrospinal fluid or related to vascular compression caused during arachnoid or vascular manipulations during the trigeminal nerve MVD. Nevertheless, we found that in this setting of the dramatic BAEP loss, predictive of post-operative deafness, surgical intervention to alleviate vascular compression upon the cochlear nerve may be effective to normalize neurophysiologic cochlear nerve function.

SUMMARY

Intraoperative monitoring of BAEP effectively identifies physiological dysfunction of the cochlear nerve, and thereby alerts the neurosurgeon to take corrective measures. The two cases presented illustrate intraoperative loss of auditory function

apparently due to exacerbation of vascular compression of the cochlear nerve. Appropriately directed cochlear nerve MVD effectively alleviated this potential cause of hearing loss.

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