Original Article

BAER suppression during posterior fossa dural opening

Christopher B. Shields, Lisa B. E. Shields, Yi Dan Jiang, Tom Yao, Yi Ping Zhang, David A. Sun

Norton Neuroscience Institute, Norton Healthcare, 210 East Gray Street, Suite 1102, Louisville, KY 40202, ‘Impulse Monitoring Inc., 10420 Little Patuxent Parkway, Suite 250, Columbia, MD 21044, USA

E-mail: *Christopher B. Shields - cbshields1@gmail.com; Lisa B.E. Shields - LBES@earthlink.net; Yi Dan Jiang - djiang@impulsemonitoring.com; Tom Yao - Tom.Yao@nortonhealthcare.org; Yi Ping Zhang - yipingzhang50@gmail.com; David A. Sun - David.Sun@nortonhealthcare.org

*Corresponding author

Received: 30 March 14   Accepted: 25 January 15   Published: 09 April 15

Access this article online
Website: www.surgicalneurologyint.com
DOI: 10.4103/2152-7806.154775

Abstract

Background: Intraoperative monitoring with brainstem auditory evoked responses (BAER) provides an early warning signal of potential neurological injury and may avert tissue damage to the auditory pathway or brainstem. Unexplained loss of the BAER signal in the operating room may present a dilemma to the neurosurgeon.

Methods: This paper documents two patients who displayed a unique mechanism of suppression of the BAER apparent within minutes following dural opening for resection of a posterior fossa meningioma.

Results: In two patients with anterior cerebellopontine angle and clival meningiomas, there was a significant deterioration of the BAER soon after durotomy but prior to cerebellar retraction and tumor removal. Intracranial structures in the posterior fossa lying between the tumor and dural opening were shifted posteriorly after durotomy.

Conclusion: We hypothesized that the cochlear nerve and vessels entering the acoustic meatus were compressed or stretched when subjected to tissue shift. This movement caused cochlear nerve dysfunction that resulted in BAER suppression. BAER was partially restored after the tumor was decompressed, dura repaired, and bone replaced. BAER was not suppressed following durotomy for removal of a meningioma lying posterior to the cochlear complex. Insight into the mechanisms of durotomy-induced BAER inhibition would allay the neurosurgeon’s anxiety during the operation.

Key Words: Brainstem auditory evoked responses, durotomy, posterior fossa brain tumor, internal auditory meatus

INTRODUCTION

Intraoperative monitoring provides early warning signals of potential danger that enable the surgeon to avoid neural damage. Brainstem auditory evoked responses (BAER) depicts the transmission of electrical activity of the auditory pathway from the cochlea to the inferior colliculus. BAER has been used to monitor neurological function in several neurosurgical operations such as microvascular decompression for trigeminal/glossopharyngeal neuralgia, hemifacial spasm, and resection of benign cerebellopontine angle or clival tumors. BAER signals may be suppressed intraoperatively by several physiological...
causes of signal changes including: (i) Decreased core temperature, (ii) masking the auditory stimulus by the sound of bone drilling, (iii) noisy electrical background, (iv) dislodgement of the auditory stimulators, and (v) the collection of fluid between the stimulator and inner ear. However, BAER have been reported and widely believed to be unchanged in the presence of drugs used for induction and maintenance of general anesthesia. Following the exclusion of biological and technical factors, any alterations of BAER signals are related to irritation and/or damage of the anatomical pathway related to hearing.

Accurate interpretation of electrophysiological changes intraoperatively is crucial, particularly with respect to differentiating false negative and false positive responses. Following the onset of abnormal BAER, the initial challenge is to identify changes arising from either technical or biological causes. Abnormal BAER responses are not invariably permanent, however, may be temporary due to cochlear nerve stretching during cerebellar retraction, cochlear ischemia caused by systemic hypotension, cold irrigation, or arterial kink. The need to immediately recognize and correct the causes of early BAER changes is critical to avoid permanent neurological deficits. The identification of such findings is based on ruling out and/or correcting systemic physiological changes, which requires a close collaboration between neuromonitoring technicians, the surgeon, and the anesthesiologist.

We report two patients harboring posterior fossa meningiomas lying anterior to the cochlear nerve in whom the BAER was suppressed within a short time after durotomy and cerebrospinal fluid (CSF) drainage. We hypothesize that the durotomy induced structural shifts that resulted in cochlear ischemia caused by direct kinking or stretching of the cochlear nerve or its arterial supply. An additional patient is also reported in whom the tumor was located posterior to the cochlear nerve. This patient did not display BAER depression following durotomy. Thus, BAER inhibition was dependent on tumor location. Prompt exclusion of potential systemic and anesthetic factors that may cause BAER suppression and a clear recognition of the novel cause described in this report will minimize the neurosurgeon’s concern that a serious neurological event has occurred and allow the operation to proceed with assurance.

**CASE REPORTS**

**Case #1**

A 49-year-old female (height: 5'11''); weight: 230 pounds; body mass index [BMI]: 32.1) presented with right facial numbness and slight difficulty with mastication and swallowing. Examination demonstrated decreased sensation to touch in her right V1 and V2 distributions as well as decreased hearing from the right ear. The magnetic resonance imaging (MRI) scan revealed a 1.4 × 2.7 × 1.7 cm right cerebellopontine angle (CPA) tumor with clival extension and anterior indentation of the pons that lay anterior to the internal auditory meatus [Figure 1a].

Anesthesia was induced utilizing sufentanil and rocuronium, maintained with sevoflurane, and supplemented with fentanyl and propofol as needed. The patient was placed in a park bench position, her head was fixed in a Mayfield clamp, and her face was directed at an angle of 45° to the floor. BAER and electromyography (EMG) were used to monitor facial, cochlear, glossopharyngeal, vagus, accessory, and hypoglossal nerve functions continuously using a Caldwell Cascade device (Caldwell Laboratories, Kennewick, WA). Monitoring was performed by a certified neurophysiologist from the neuromonitoring company Impulse Monitoring, Inc. (Columbia, MD). An individual with a doctoral degree at Impulse Monitoring, Inc. supervised the technologist. The protocols of this company included quality control and routine techniques used to determine noise from signal.

EMG recordings were unaffected throughout the operation. Auditory stimuli (Caldwell Laboratories, Kennewick, WA), delivered through foam earphones (Elymotic Research Inc, Elk Grove Village, IL) were inserted into the ear canals, sealed with bone wax to hold the earphones in position, and covered with waxes to hold the earphones in position, and covered with

**Figure 1:** (a) Case #1: MRI showing a 1.4 x 2.7 x 1.7 cm meningioma in the right CPA extending anteriorly into Meckel’s cave. The arrow depicts the internal auditory meatus, and the asterisk denotes the tumor site. (b) Case #2: MRI demonstrating a 2.8 x 1.3 x 2.8 cm meningioma arising from the right side of the clivus. In each case the internal acoustic meatus was located posterior to the tumor, with the cochlear nerve and vessels located adjacent to the tumor. The arrow depicts the internal auditory meatus, and the asterisk denotes the tumor site. (c) Case #3: MRI showing a 2.9 x 3.2 x 3.6 cm meningioma lying adjacent to the right sigmoid sinus, posterior to the internal auditory meatus. The arrow depicts the internal auditory meatus, and the asterisk denotes the tumor site.
Tegaderm film (3M Healthcare, St. Paul, MN). BAER recordings used conventional averaging techniques that were induced by rectangular rarefaction sound stimulation of 11.1 Hz at 90 dB. Masking noise was applied on the contralateral side to that being tested to eliminate a “crossover” response. The active electrode was placed on the ipsilateral mastoid, reference electrode on the vertex, with the forehead serving as ground. The bandpass was set within 50–2000 Hz. Impedance of the recording electrodes was maintained at <5 kΩ. A total of 2000 signals administered at 11.2 Hz were averaged per recording to reduce interference from electric noise in the operation room. BAERs were alternatively obtained from each side with each response recorded for 3 min. The intact side served as control. Continuous monitoring was performed throughout the operation with no interval between samples. Noise was differentiated from physiologic responses by repetitive recordings obtained during continuous monitoring.

Temporal resolution of the recorded responses was 2400 samples per second using the Cadwell machine (sampling rate; http://www.cadwell.com/products/ionm/cascade/cascade.htm). BAER latency changes and interpeak latencies of wave I to III, III to V, and I to V were monitored.

Baseline bilateral BAER was recorded following induction of anesthesia [Figure 2a]. Latencies on the right (tumor side) were slightly delayed, likely related to mass effect of the tumor (measurements on the tumor side: I = 1.81 ms, III = 4.31 ms, V = 6.11 ms, I-III = 2.5 ms, III-V = 1.8 ms, I-V = 4.3 ms versus corresponding measurements on the nontumor side: I = 1.5 ms, III = 4 ms, V = 5.87 ms, I-III = 2.5 ms, III-V = 1.87 ms, I-V = 4.37 ms [Figure 2a]. All changes in BAER latency and amplitude were compared with the baseline BAER after induction of anesthesia.

A craniotomy was created with bone removal flush with the transverse and sigmoid sinuses and inferiorly to the foramen magnum. When mastoid air cells in bone were exposed, bone wax was applied to avoid seepage of blood into the middle ear. This avoided BAER suppression due to conduction deficits by preventing blood from entering the middle ear. There were no air cells exposed after dura was opened, so there was no chance of CSF

Figure 2: Consecutive recordings showing BAER suppression for 14 minutes after dural opening and before placement of cerebellar retraction (traces 2-4). Waves I and the IV-V complex were lost during dural opening in Cases #1 and #2. Partial return of BAER occurred during wound closure. (a) Case #1: Diminished amplitudes of peaks I, II, and III with delayed latency of the wave IV-V complex became apparent after dural opening (traces 2-4). The wave IV-V complex improved during dural closure and bone flap replacement (traces 6-10). There was no return of wave I. (b) Case #2: Soon after dural opening a loss of wave III became apparent, and the latency of peaks I and V were prolonged (traces 2-4). Wave I was restored after tumor removal, dural closure, and mesh replacement (traces 6-8). There was no return of wave V. Left BAER remained intact throughout the operations in Cases #1 and #2. (c) Case #3: There was no increase in the latency and no amplitude suppression of wave I during or following the durotomy (traces 2-4)
entering the middle ear. The dura was tense, indicating a high intracranial pressure (ICP). The dura was opened in a circular manner up to the sinus margins, resulting in cerebellar herniation through the dural opening with resultant anatomical shift of posterior fossa structures. Following gentle cerebellar retraction, the subarachnoid cisterns were decompressed by CSF removal that caused relaxation of the cerebellum. Within several minutes after dural opening and cisternal drainage, BAER signals demonstrated deterioration ipsilateral to the tumor as manifested by a prolonged latency and flattened waves. BAER demonstrated that the latency of peak I increased by >10% with an amplitude reduction of >75%. When the dura was completely opened, only peak V persisted throughout the operation (V = 6.65 ms). This pattern remained throughout tumor resection. The tumor was resected from the surface of the skull base, and the surgeon believed it was completely resected. After the dura was closed and the bone flap reconstructed, improvement of wave I occurred although it did not return to baseline. After the dura was closed, recovery of electrophysiological function was prominently noted by improvement of wave I that further improved after the bone flap was reapplied. The latency of wave V demonstrated minimal improvement (I = 1.81 ms, III = 4.93 ms, V = 6.81 ms) [Figure 2a].

The surgical pathology demonstrated a World Health Organization (WHO) grade I meningioma. A postoperative MRI scan revealed a small amount of residual tumor in the anterior aspect of Meckel’s cave that was not recognized intraoperatively. At the patient’s 3-week postoperative visit, she complained of headaches, nausea, and vomiting. There was also increased numbness and tingling of the right side of her face. When hearing was tested several weeks postoperatively, it was unchanged compared with preoperatively. A computed tomography (CT) scan revealed ventriculomegaly and transependymal absorption of CSF and, therefore, the patient underwent placement of a ventriculoperitoneal shunt.

**Case #2**

A 33-year-old male (height: 5’9”; weight: 219 pounds; BMI: 32.3) presented with unstable gait and the sensation of heaviness and numbness of his feet. He also complained of paresthesias of the right occipital scalp. Examination revealed ataxia with a tendency to lean to the right and a positive Romberg sign. Hearing was intact bilaterally. MRI revealed a 2.8 × 1.3 × 2.8 cm anteriorly situated CPA and clival meningioma with displacement of the anterior pons [Figure 1b].

Anesthesia, surgical preparation, and BAER and EMG monitoring were the same as described in the previous patient. The baseline BAER from each side were nearly identical. Measurements on the right (tumor side) were: I = 1.65 ms, III = 4 ms, V = 6.18 ms, I-III = 2.35 ms, III-V = 2.18 ms, I-V = 4.53 ms versus I = 1.5 ms, III = 4 ms, V = 5.71 ms, I-III = 2.5 ms, III-V = 1.71 ms, I-V = 4.21 ms on the left (nontumor side) [Figure 2b]. A depression of BAER recordings became apparent during the 14 min following dural opening manifested by latency of peak I being delayed by >10%, total loss of waves II, III and IV, however, wave V was preserved (Figure 2b: traces 2-4). Explanation of BAER suppression at this point of the operation was not apparent and, therefore, an extensive evaluation of physiologic causes was performed. Twenty minutes after dural opening, with no other explanation for the BAER suppression identified, cerebellar retractors were inserted and the operation progressed. Resection of the fibrous meningioma was technically difficult with frequent intraoperative warnings of BAER suppression. The permanent loss of wave V was likely secondary to damage to the brainstem during surgical resection. The risk of permanent brainstem damage prompted the surgeon to abort the operation before a complete tumor resection was attained. After the dura was repaired, BAER latency responses partially recovered, especially wave peak I. Peak I had returned to normal in latency and amplitude soon after the bone flap substitute was placed (Figure 2b: Traces 5-8). Removal of the cerebellar retractors may have led to some improvement of the BAER. However, since BAER suppression occurred prior to retractor insertion, some return of function may be related to decreased angulation or improved blood supply to the cochlear nerve. BAER on the left served as controls and did not change throughout the entire operation.

The tumor was a WHO grade I meningioma. A postoperative MRI scan demonstrated resection of >50% of the tumor that decreased the mass effect on the pons. The patient developed a right 6th nerve palsy postoperatively that was treated with ocular therapy. Facial nerve function remained intact, and hearing was unchanged compared with preoperatively. However, there was minimally decreased hearing from the right ear.

**Case #3**

A 60-year-old female (height: 5’3”; weight: 120 pounds; BMI: 23.23) with right CPA meningioma was compared with the two previous patients. This patient described a several month history of episodic posterior occipital headaches as well as neck pain. The MRI scan revealed a 2.9 × 3.2 × 3.6 cm tumor lying adjacent to the right sigmoid sinus, posterior to the internal auditory meatus (IAM) [Figure 1c]. Hearing was intact bilaterally.

Anesthesia, surgical approach, durotomy, and exposure of the tumor were identical to Cases #1 and #2. The baseline BAER from each side was recorded with measurements on the right (tumor side) were: I = 1.51 ms, III = 4 ms, V = 5.9 ms, I-III = 2.49 ms, III-V = 1.90 ms, I-V = 4.39 ms versus I = 1.48 ms, III = 3.75 ms, V = 5.71 ms, I-III = 2.27 ms,
III-V = 1.86 ms, I-V = 4.23 ms on the left (nontumor side) [Figure 2c]. Although the amplitude of the BAER waves were slightly lower on the side of the retrometal meningioma (even at baseline), these changes were likely caused by mild extrinsic pressure on the cochlear nerve on the side of the tumor. However, following durotomy, BAER responses were not further suppressed, nor were there any changes in the BAER latencies or amplitudes soon after dural opening.

The tumor arose from the sigmoid sinus. The surgeon considered the surgery to be a gross total removal.

The pathology was a WHO grade I meningioma. A postoperative MRI scan demonstrated a near complete resection, with only a minimal degree of enhancement observed along the sigmoid sinus. There was also evidence of a heterogeneous attenuation of the right cerebellum indicating a cerebellar infarct and hydrocephalus. The patient was managed with a ventricular drain and rehabilitation. There was no hearing loss postoperatively.

**DISCUSSION**

BAER is used to monitor auditory pathways intraoperatively from the cochlear nerve to the inferior colliculus. baseline BAER in our patients was intact preoperatively due to the gradual adaptation of cochlear function during the tumor expansion. In Cases 1 and 2 the meningiomas were located anterior to the cochlear nerve complex so that dural opening caused displacement of posterior fossa structures including the cochlear nerve that was pushed against the posterior margin of the IAM. An alternative but less likely mechanism may be suppression of BAER caused by vascular compression (internal auditory artery or loop of the anterior inferior cerebellar arteries) inducing partial ischemia of the auditory pathway. These hypotheses are supported by partial BAER recovery after durotomy and bone flap replacement. Furthermore, the BAER was unaffected following durotomy in Case 3 when the meningioma was situated posterior to the internal auditory meatus. Cases 1 and 2 were obese (BMI > 32). ICP is elevated in obese patients, particularly when they are placed in a semiprone position due to gravity. Excessive ICP elevation in the obese patient and lateral head fixation may enhance structural shifts that cause BAER deterioration following durotomy.

BAER is used to monitor auditory nerve and brainstem function clinically and experimentally. BAER monitoring helps the surgeon avoid potential irreversible tissue damage. Alterations in BAER response have been associated with ICP elevation and dural opening during surgery for Chiari I malformations with improvement occurring after dural repair. This report is the first to describe BAER suppression induced by durotomy causing structural shifts that are not caused by systemic effects or direct surgical manipulation of the acoustic structures. Waves I and II are generated from the proximal and distal cochlear nerve. In Cases 1 and 2, there was considerable suppression of wave I soon after durotomy, suggesting that the site of pathology was at the cochlear nerve. Insofar as there was no direct brainstem pressure or distortion induced by dural opening, it is unreasonable to imply that later waves (II-V) might be the primary site of damage. However, incomplete recovery of the later waves in Case 2 was presumably caused by concomitant intraoperative brainstem damage.

We have described two patients with meningiomas located anterior to the IAM who demonstrated BAER suppression soon after durotomy and prior to direct surgical manipulation of the cochlear nerve complex. Instead of being alarmed or aborting surgery prior to tumor resection, neurosurgeons must be aware of this mechanism of “unexplained” BAER suppression and should continue the operation without delay.

**ACKNOWLEDGMENTS**

The authors acknowledge Norton Healthcare, Louisville, KY for their continued support.
REFERENCES

1. Abramson M, Stein BM, Pedley TA, Emerson RG, Wazen JJ. Intraoperative BAER monitoring and hearing preservation in the treatment of acoustic neuromas. Laryngoscope 1985;95:1318-22.

2. Banoub M, Tetzlaff JE, Schubert A. Pharmacologic and physiologic influences affecting sensory evoked potentials: Implications for perioperative monitoring. Anesthesiology 2003;99:716-37.

3. Biacabe B, Chevallier JM, Avan P, Bonfilis P. Functional anatomy of auditory brainstem nuclei: Application to the anatomical basis of brainstem auditory evoked potentials. Auris Nasus Larynx 2001;28:85-94.

4. Chen L. Detection of ischemia in endovascular therapy of cerebral aneurysms: A perspective in the era of neurophysiological monitoring. Asian J Neurosurg 2010;5:60-7.

5. Dannenbaum M, Lega BC, Suki D, Harper RL, Yoshor D. Microvascular decompression for hemifacial spasm: Long-term results from 114 operations performed without neurophysiological monitoring. J Neurosurg 2008;109:410-5.

6. Hammerschlag PE, Berg HM, Prichep LS, John ER, Cohen NL, Ransohoff J. Real-time monitoring of brainstem auditory evoked response (BAER) during cerebellopontine angle (CPA) surgery. Otalaryngol Head Neck Surg 1986;95:538-42.

7. Hett DA, Smith DC, Pilkington SN, Abbott TR. Effect of temperature and cardiopulmonary bypass on the auditory evoked response. Br J Anaesth 1995;75:293-6.

8. Hojlund J, Sandmand M, Sonne M, Mantoni T, Jorgensen HL, Belhage B, et al. Effect of head rotation on cerebral blood velocity in the prone position. Anesthesiol Res Pract 2012;2012:647258.

9. Lapinsky SE, Posadas-Calleja JG, McCullagh I. Clinical review: Ventilatory strategies for obstetric, brain-injured and obese patients. Crit Care 2009;13:206.

10. Legat AD. Mechanisms of intraoperative brainstem auditory evoked potential changes. J Clin Neurophysiol 2002;19:396-408.

11. Lumenta CB, Reschovsky K, Bock WJ. Effects of cerebellar retraction on brainstem auditory evoked potentials in an experimental animal model of cerebellopontine angle tumor. Surg Neurol 1989;31:246.

12. Mom T, Gabrillargues J, Gilain L, Chazal J, Kemeny JL, Vanneauville G. Anatomy of the vestibulo-acoustico-facial neurovascular pedicle. Importance of therapeutic management of vestibular schwannomas. Neurochirurgie 2002;48:387-97.

13. Mustain WD, al-Mefty O, Anand VK. Inconsistencies in the correlation between loss of brain stem auditory evoked response waves and postoperative deafness. J Clin Monit 1992;8:231-5.

14. Notley SV, Bell SL, Smith DC. Auditory evoked potentials for monitoring during anaesthesia: A study of data quality. Med Eng Phys 2010;32:168-73.

15. Polo G, Fischer C, Sindou MP, Marneffe V. Brainstem auditory evoked potential monitoring during micrvascular decompression for hemifacial spasm: Intraoperative brainstem auditory evoked potential changes and warning values to prevent hearing loss-prospective study in a consecutive series of 84 patients. Neurosurgery 2004;54:97-104.

16. Rizvi SS, Goyal RN, Calder HB. Hearing preservation in microvascular decompression for trigeminal neuralgia. Laryngoscope 1999;109:591-4.

17. Scheller BC, Daunderer M, Piga G. General anesthesia increases temporal precision and decreases power of the brainstem auditory-evoked response-related segments of the electroencephalogram. Anesthesiology 2009;111:340-55.

18. Seifert E, Lamprecht-Dinnen A, Asfour B, Rotering H, Bone HG, Scheld HH. The influence of body temperature on transient evoked otoacoustic emissions. Br J Audiol 1998;32:387-98.

19. Sekiya T, Moller AR. Effects of cerebellar retraction on the cochlear nerve: An experimental study on rhesus monkeys. Acta Neurochir (Wien) 1988;90:45-52.

20. Simon MV. Neurophysiologic intraoperative monitoring of the vestibulocochlear nerve. J Clin Neurophysiol 2011;28:566-81.

21. Simon TA, Kahler D, Simon WE, Fox C, Li J, Palta J, et al. An MLC calibration method using a detector array. Med Phys 2009;36:4495-503.

22. Sindou M, Gimbert E. Decompression for Chiari type I malformation (with or without syringomyelia) by extreme lateral foramen magnum opening and expansile duraplasty with arachnoid preservation: Comparison with other technical modalities (Literature review). Adv Tech Stand Neurosurg 2009;34:85-110.

23. Tiainen M, Kovala TT, Takkunen OS, Roine RO. Somatosensory and brainstem auditory evoked potentials in cardiac arrest patients treated with hypothermia. Crit Care Med 2005;33:1736-40.

24. Wind JJ, Leonetti JP, Raffin MJ, Pisanský MT, Herr B, Triemstra JD, et al. Hearing preservation in the resection of vestibular schwannomas: Patterns of hearing preservation and patient-assessed hearing function. J Neurosurg 2011;114:1232-40.

25. Zamel K, Galloway G, Kosnik EJ, Raslan M, Adeli A. Intraoperative neurophysiologic monitoring in 80 patients with Chiari I malformation: Role of duraplasty. J Clin Neurophysiol 2009;26:70-5.