Reversible Evoked Potential Changes with Retraction of the Eighth Cranial Nerve

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Deafness is a recognized complication of operations in the cerebellopontine angle. It is the most common complication seen after microvascular decompression of the seventh cranial nerve for hemifacial spasm (1). The mechanism of injury has not been precisely defined but presumably relates to operative manipulation of the eighth cranial nerve and brainstem.

The functional integrity of the auditory pathway in the posterior fossa can be assessed by measuring brainstem auditory evoked potentials (BAEP), electrical responses to auditory stimulation that are made apparent in scalp recordings by averaging many epochs of electroencephalographic (EEG) activity time locked to the sensory stimulus (2-4). A normal BAEP is shown in Fig 1; the purported generators of designated peaks are listed in Table 1.

We have used BAEP to monitor auditory function during operations in the cerebellopontine angle (5). This report describes a case of reversible intraoperative BAEP obliteration clearly related to retraction of the eighth nerve and cerebellum. The evoked potential reappeared after repositioning of retractors and the patient's hearing was preserved.

Case Report

A 66-year-old, 85-kg woman with right hemifacial spasm was scheduled for retromastoid craniectomy and microvascular decompression of the seventh cranial nerve. Before surgery, BAEP were normal and audiograms were normal except for bilateral sensorineural reduction at high frequency. The patient was premedicated intramuscularly with meperidine, 50 mg, hydroxyzine, 50 mg, and atropine, 0.4 mg. Fentanyl, 100 μg, was given intravenously in the operating room before base line (preanesthesia) BAEP recording. We obtained BAEP using a MED 80 Biomedical Data System (Nicolet Biomedical, Inc, Madison, WI) especially configured for use in the operating room. Stimulation and recording parameters are shown in Table 2.

Anesthesia was induced with thiopental, fentanyl, and nitrous oxide in oxygen. Pancuronium provided muscle relaxation. Monitoring included electrocardiogram, esophageal stethoscope, intra-arterial blood pressure, nasopharyngeal stethoscope, and arterial blood gases. A normal BAEP is shown in Fig 1; the purported generators of designated peaks are listed in Table 1.

Fig 1. Normal BAEP. Cz-Ai: vertex to ipsilateral earlobe; Cz-Ac: vertex to contralateral earlobe. Peaks in upper wave form are labeled to show customary designations. Purported generators of these peaks are shown in Table 1.
TABLE 1
Purported Generators of BAEP Peaks*

I. Acoustic nerve
II. Cochlear nuclei (medulla)
III. Superior olive (pons)
IV. Lateral lemniscus (pons)
V. Inferior colliculus (midbrain)
VI. Medial geniculate (thalamus)
VII. Thalamocortical radiations

* Activity probably comes more from afferent volleys than from activity in nuclei. These generators have not been proven but seem clinically useful.

TABLE 2
Stimulus and Recording Parameters—BAEP

Stimulation
Site: bilateral ear insert transducers (Madsen Electronics, Inc, Buffalo, NY)
Clicks: alternating compression and rarefaction
Intensity: 60 dB SL
Duration: 200 µsec
Rate: 11.2 Hz
Contralateral masking: wideband pseudorandom noise, 35 dB HL

Recording
Site: channel 1, Cz-A1 (vertex to left earlobe); channel 2, Cz-A2 (vertex to right earlobe); ground: Fpz (midforehead)
Electrodes: 10-mm gold discs, applied with collodion and filled with conductive gel
Impedances: less than 3000 ohm
Filters: 30–3000 Hz
Sensitivity: ±25 µV full scale
Sweep time: 10.24 msec
Sampling rate for analog to digital conversion: 50,000 Hz
Repetitions per average: 2000

Fig 2. Intraoperative BAEP changes. Two columns show wave forms simultaneously recorded vertex to ipsilateral earlobe and vertex to contralateral earlobe.

Fig 3. Mean latencies and amplitudes for peaks I, III, IV–V, and V during four stages of operation: 1, preanesthesia; 2, anesthesia; 3, dura open; 4, dura closed.

Ryngeal temperature, precordial Doppler, and intermittent measurement of arterial blood gas tensions, hematocrit, and osmolarity. The patient was turned to the left lateral position and her head secured in a pin head holder. BAEP monitoring was continual except when momentarily suspended during use of the electrosurgical machine. Wave forms were plotted in the operating room and the poststimulus latency of peak V was measured digitally using a controllable cursor on the MED 80. The wave form was then stored on disc and a new average begun. To minimize the time between averages, additional measurements of the wave form were delayed until after surgery. Anesthesia and surgery were uneventful until retractors were placed on the cerebellum, and microvascular decompression of the seventh nerve was begun at 9:20 a.m. An increase in the poststimulus latency of peak V was noted 10 minutes later. No undue retractor pressure was apparent on visual inspection and the operation proceeded without alteration of retraction. Peak V latencies progressively increased until 10:16 a.m., when the BAEP past peak I was virtually obliterated (Fig 2). None of the other continuous monitors showed acute change. The surgeon then repositioned the retractor and the BAEP complex reappeared, although still distorted, at 10:21 a.m. Recovery of the wave form continued during the remaining 45 minutes.
of anesthesia but was incomplete. The patient awoke in the operating room neurologically intact, relieved of her hemifacial spasm and with clinically normal hearing.

The BAEP wave forms recorded during surgery were subsequently measured using the cursor program on the MED 80. Latencies and amplitudes for all identifiable peaks (Fig 3) were recorded and grouped according to stages of the clinical procedure (before anesthesia, after anesthesia before opening of the dura, from opening to closing of dura, from closure of dura to conclusion of anesthesia). Tables and graphs describing these BAEP measurements individually and during the four designated time periods were generated using the PROPHET computer system (7).

The relative stability of peak I throughout the operation contrasted with the increases in latency and decreases in amplitude of all subsequent peaks. Wave forms were less variable during anesthesia before opening of the dura than in the awake state. Latency shifts during operative manipulation were greater in wave forms referenced to the ipsilaterial ear for peaks I and II but greater in wave forms referenced to the contralateral ear for peak V, suggesting that retractor pressure may have been affecting the brainstem as well as the eighth nerve. An audiogram done 4 days after surgery showed hearing to be the same as before surgery in the left ear and the same in the right ear except for a slight decrease in hearing level at 2000 Hz. The BAEP done 4 days after surgery showed normal latency for peak V but newly increased latencies for peaks II and III. Eight months later, the patient was clinically well. She had no recurrence of hemifacial spasm and no clinically evident hearing loss. Audiogram at this time showed no change from the audiogram done 4 days after surgery, but the BAEP showed some lessening of the latency prolongation for peaks II and III.

Discussion

Hearing loss ipsilateral to the site of operation occurred in 18 (7.9%) of the 229 patients treated at our institution between 1971 and 1979. Eight patients were completely deaf, eight patients had profound hearing loss, and two patients had mild deficits (1). The incidence of hearing loss was higher early in the series and has diminished over the years with increased experience, improvements in operating techniques, and the use of better implant materials.

This case is of interest because virtually complete obliteration of the BAEP, apparently due to pressure from a retractor in the posterior fossa, was shown to be reversible. We cannot be sure that this patient would have lost her hearing had the initial retractor pressure been maintained throughout the operation, but it seems likely that damage related to retraction of neural structures may account for some of the deafness that occurs after operations in the cerebellopontine angle. The minimal decrease in hearing acuity at 2000 Hz and the latency shifts of BAEP peaks II and III seen 4 days and again 8 months after surgery in this patient could well have been due to retractor pressure during microvascular decompression of the seventh cranial nerve. It is not inconceivable that more prolonged retraction (and more prolonged BAEP obliteration) might have been associated with greater loss of hearing.

The BAEP can be altered by several factors other than compromised neurologic function (8). Temperature, stimulus parameters, and filter settings on recording devices should be kept constant if BAEP changes are to reflect the effects of surgical manipulation. In contrast to sensory evoked potentials of intermediate or long latency (9–13), the short latency BAEP are affected relatively little by anesthetic agents and by physiologic changes of the kind usually seen during anesthesia and surgery. Anesthetics can nevertheless alter BAEP (14) and anesthetic depth should be kept relatively constant during critical monitoring periods.

We still do not know how much BAEP alteration can be tolerated over what period of time without risking permanent loss of function. Nevertheless, BAEP monitoring may help to reduce the incidence of deafness after operations in the cerebellopontine angle by warning of compromised function in a timely fashion so that appropriate interventions can be made to preserve hearing.

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Coronary-Artery Spasm after Myocardial Revascularization

Coronary-artery spasm was investigated in six patients who had had unexpected hemodynamic collapse within 2 hours after cardiopulmonary bypass for myocardial revascularization. All six had profound hypotension and recurrent ST-segment elevation in electrocardiographic leads II, III, and aVF. All had either normal or noncritical luminal irregularities of dominant right coronary arteries and more than 75% occlusions in the left coronary circulation. Right-coronary-artery spasm, which was reversed after intracoronary nitroglycerin, was demonstrated angiographically in one patient; a patent right coronary artery was found at autopsy in another patient. Three patients died despite large intravenous doses of nitroglycerin. Two patients who had been unresponsive to intravenous nitroglycerin recovered after direct infusion of nitroglycerin into the right coronary artery. Coronary-artery spasm immediately after myocardial revascularization may cause circulatory collapse and death; although the spasm may be refractory to usual therapy, it may respond to intracoronary nitroglycerin. (Buxton AE, Goldberg S, Harken A, Hirshfeld J Jr, Kastor JA: Coronary-artery spasm immediately after myocardial revascularization: recognition and management. N Engl J Med 1981;304:1249-53)

Influence of Hemodilution on Potency of Neuromuscular Blockers

Cumulative dose-response curves of succinylcholine, pancuronium, and tubocurare were constructed in 45 patients during general anesthesia (methohexitone, 70 to 100 mg; droperidol, 2.5 to 10 mg; fentanyl in increments of 0.05 to 0.1 mg as required; nitrous oxide, 75% in oxygen; normoventilation) maximal electrical stimulation (100 to 120 V, 0.2 msec, 0.2 Hz) of the ulnar nerve. In 28 adult patients up to 1000 ml of venous blood was withdrawn before operation and replaced immediately with dextran 40 (Rheomacrodex 10%). The control group consisted of 17 patients not undergoing hemodilution. In patients with hemodilution, the potencies of succinylcholine, pancuronium, and tubocurare were increased, as shown by a parallel shift to the left of cumulative dose-response curves, following normovolemic hemodilution before operation. This is attributed to an increase in cardiac output, a decrease in protein binding and, in the case of succinylcholine, to a decrease in cholinesterase activity. (Schuh FT: Influence of hemodilution on the potency of neuromuscular blocking drugs. Br J Anaesth 1981;53:263-4)