Somatosensory Evoked Potential Changes in Position-related Brain Stem Ischemia

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Neurologic deficits associated with the sitting position have occurred in patients undergoing posterior fossa craniotomy with the resultant recommendation that another position be used.¹ The park bench or three-quarters prone position has been advocated because of the low incidence of air emboli and the advantage of cerebellar displacement away from the brain stem by gravity.² An integral part of that position is flexion and rotation of the head to optimize exposure and minimize venous bleeding.² A patient is described in whom probable brain stem ischemia was demonstrated by somatosensory evoked potential monitoring despite technically correct park bench positioning and normal arterial blood pressure, intracranial pressure, and cerebral perfusion pressure.

REPORT OF A CASE

A 46-year-old woman presented with numbness and weakness of the left face and left arm. The patient had a mild left cranial nerve VII dysfunction, a mild right cranial nerve V weakness, and a bilateral hearing loss, with the left being greater than the right. A preoperative computerized tomography scan showed a large mass arising from the right cerebellopontine angle with displacement of the brain stem to the left and minimal enlargement of the lateral and third ventricles. A suboccipital approach to the tumor with the patient in the park bench position was planned. The patient was unmedicated, and arterial and central venous catheters were inserted under local anesthesia. Somatosensory evoked potentials (SEP) were monitored by gold-plated silver cup electrodes placed 2 cm posterior to positions C3 and C4. These electrodes were designated C3' and C4', respectively. An electrode was placed over the second cervical vertebral (SC2), and all electrodes were referenced to a disposable electrode at FP2 (International 10-20 system). The impedance of all electrodes was decreased to less than 2 kohms by dermal abrasion.

Prior to the induction of anesthesia, the location of each median nerve was determined using a transcutaneous stimulator. Sterile needle stimulating electrodes were placed near each median nerve and taped in place. Each median nerve was stimulated at a stimulus intensity slightly above motor threshold prior to anesthesia and at maximum stimulus intensity (19.9 mamps, 250 volts) after induction of anesthesia. One hundred twenty-eight stimuli were delivered and averaged using a Nicolet Med-80® signal averager (Nicolet Biomedical, Madison, Wisconsin). A time of 80 ms after the stimulus was evaluated. The sampling time was 0.312 ms/point. The somatosensory evoked potential for each upper extremity was determined prior to induction of anesthesia and before and after each change of position. Anesthesia was induced with fentanyl 25 µg/kg iv followed rapidly by pancuronium 0.1 mg/kg iv and after a brief period of hyperventilation, the trachea was intubated without difficulty, and nitrous oxide (50%) was administered. During the course of anesthesia, the end-tidal Pco2 was maintained between 25–30 mmHg as determined by a Capnograph® (Hewlett-Packard 72410A). Blood pressure, CVP, heart rate, and end-tidal CO2 were recorded continuously. The long axis of the body was always parallel to the floor, and all transducers were referenced to mid-thoracic level. After endotracheal intubation, the SEP waves had a slightly longer latency than prior to induction (Fig. 1A) but were otherwise unremarkable (Fig. 1B). A pin type head holder was placed, and the patient was placed in the park bench position with left side down and the head slightly flexed and rotated to the left. Minimal changes in blood pressure and heart rate occurred in response to surgical positioning. SEP determined for each extremity approximately 5 min after positioning demonstrated absence of cortical waves with preservation of the response at the second cervical vertebrae following stimulation of each median nerve (Fig. 1C). The head was returned to the neutral position and held manually in that position. Cortical SEP waves were present in response to each median nerve stimulation approximately 5 min after return of the head to the neutral position (Fig. 1D). The patient then was placed in the supine position, and a cerebrospinal fluid drain was placed in the left lateral ventricle. The blood pressure was 145/80 mmHg, with a mean pressure of 100 mmHg. The cerebrospinal fluid pressure was 10 mmHg and the cerebral perfusion pressure 90 mmHg. In the supine position, the patient's head was flexed manually to mimic the surgical position and held for approximately 5 min. Cortical SEP waves were absent in response to stimulation of either median nerve (Fig. 2B) despite hyperventilation (PaCO2, 27 mmHg) cerebral perfusion pressure (95 mmHg), intracranial pressure (10 mmHg), and PaO2 (150 mmHg). Return of the head to the neutral position resulted in a return of cortical SEP waves within 5 min (Fig. 2C). The surgical procedure was cancelled by the attending surgeon because of inability to place the patient in the desired surgical position. The patient awakened approximately 2 h later without change from preoperative neurologic status. No dramatic changes occurred in blood pressure or heart rate during either episode of head flexion.

Six weeks later the patient underwent surgery in the left lateral position with the head secured with a pin-type head holder in the neutral position. Cardiovascular and evoked potential monitoring was conducted as described previously. The SEPs generated by stimulating each median nerve was essentially unchanged by induction of anesthesia and surgical positioning. Specifically, both cortical and spinal cord responses (SC2) were present bilaterally. A right partial temporal lobectomy was performed to minimize the need for brain retraction for surgical exposure. Approximately 2 h after surgical incision, the cortical response to left median nerve stimulation changed and became unrecordable over a period of approximately 1 h without change in wave recorded at SC2. Cortical and spinal cord (SC2) response in response
to right median nerve stimulation were unchanged. Immediately after recognition of SEP change, all brain retractors were removed. The attending surgeon noted swelling of the tumor mass with further rotation of the brain stem to the left despite surgical removal of tumor bulk. The cortical SEP to left median stimulation (right brain stem) was absent at the end of the procedure, and the patient awakened with a profound left hemiparesis. The hemiparesis did not clear postoperatively.

**DISCUSSION**

This case demonstrates both the potential for position-related brain stem ischemia in the park bench position and the inadequacy of intracranial pressure monitoring and physical signs in assessing brain stem function in anesthetized patients with a mass directly compressing the brain stem. Although brain stem dysfunction related solely to positioning has been demonstrated by a change in respiratory pattern, the almost universal use of controlled ventilation during posterior fossa surgery removes this sensitive physical sign. Furthermore, reliance on hemodynamic observations suggesting a Cushing reflex (hypertension and bradycardia) also can be misleading. In our patient, despite a clear indication of brain stem ischemia during the initial surgical attempt shown by failure of the SEP above the upper spinal cord, a Cushing’s response (hypertension and bradycardia) was not elicited.

The decision to abort the initial attempt of surgery based on SEP changes was made by the attending neurosurgeon. A particular anatomic approach to this difficult tumor had been planned based on a distinct surgical position and with retraction of brain in a certain direction to enhance exposure. Rather than proceed in a surgical position and anatomic orientation not thoroughly considered, the procedure was postponed.

The electrode placement and monitoring parameters used for SEP were selected for “near field waves,” i.e., those waves generated near the electrode. The persistence of evoked activity originating in upper spinal cord and recorded over the second cervical vertebra (SC2) and the loss of “near field” waves previously recorded by scalp electrodes localized the deficit above the cervical spinal cord. The anatomic substrates of the waveform of the SEP has been well investigated. The SEP is carried in the posterior columns of the spinal cord, descissates in the medulla, and then is carried via the medial lemniscus to the thalamus and then to the somatosensory cortex. The pattern of early potentials recorded at the second cervical vertebrae without cortical waves has been dem-
onstrated in patients without brain stem function. There is ample evidence that changes in the SEP reflect changes in O₂ delivery to neural tissue, and failure of return to SEP to normal is associated with permanent neurologic changes. In our patient, a postoperative neurologic deficit incorrectly could have been attributed to surgical manipulation based on the location of the tumor and expected surgical difficulty had the initial surgical attempt not been aborted.

Several points should be emphasized regarding the SEP monitoring during this case. Institution of the monitoring prior to induction of anesthesia allowed documentation that brain stem function was not compromised by either of anesthesia induction or intubation of the trachea. The use of a monitoring electrode over the second cervical vertebrae demonstrated that the stimulating and averaging equipment were operating correctly and failure of the SEP occurred above the level of the cervicomедullary junction. Care was taken to not introduce or change anesthetic agents that alter the SEP during the critical time of positioning. This allowed rapid determination that alterations in SEP were related solely to position changes. The correlation of cortical SEP changes and neurologic outcome following the definitive operation supports our belief that the patient would have suffered irreversible neurologic damage due to positioning during the initial surgical attempt.

This case supports the importance of surgical positioning including head flexion and rotation in neurologic deficits in patients undergoing surgery for posterior fossa masses. Also, we have previously demonstrated that neurologic dysfunction unrelated to intracranial pathology can be demonstrated and prevented during neurosurgical procedures. Many contributing factors to intraoperative neurologic deficits have been suggested, such as venous obstruction, decreased cerebral perfusion pressure, and brain stem retraction. A decrease in cerebral perfusion pressure was ruled out in this patient during the initial surgical attempt. Although venous engorgement cannot be ruled out completely, restriction of venous outflow would be expected to have raised the intracranial pres-

sure. Direct pressure on the brain stem caused by head flexion appears the most likely cause of brain stem dysfunction.

In summary, dysfunction of the brain stem may occur during positioning for posterior fossa exploration in the presence of normal blood pressure and intracranial pressure. Patients with mass lesions of the posterior fossa distorting the brain stem may benefit from SEP monitoring during both positioning and surgery.

REFERENCES