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Subcortical Somatosensory Evoked Potentials Fail to Detect Ischemia at C1–2

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A REPORT of a probable intraoperative ischemic event during stabilization of an odontoid fracture during which the cortical somatosensory evoked potentials (SEPs) showed a significant change in amplitude, whereas the subcortical potentials were unaffected.

Neurologic injury during surgery on the spine can be a devastating complication. Intraoperative neurophysiologic monitoring should provide warning of potential injury to the spinal cord. Measures can then be taken to reverse the cause of injury before the neurologic deficit becomes permanent. To avoid false-positives and false-negatives, neurophysiologic monitoring results should be interpreted in light of all the available information. This would include monitoring cortical and subcortical SEPs and interpreting the information gathered with a good understanding of the generators of each waveform recorded. We report a case during which subcortical potentials recorded over the mastoid process did not detect a transient disturbance to the cervicomedullary junction during bone drilling for C1–C2 screw fixation.

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Case Report

An 86-yr-old, 45-kg woman presented to an outside emergency room after a fall. Her injuries included a distal radius fracture, compression fractures of T6 and T7, and a type II odontoid fracture with displacement (fracture at the junction of the dens with the central body of the axis). A closed reduction and casting of the radius fracture was performed and the patient transferred to Hershey Medical Center for further treatment. No focal neurologic deficits were present. At admission, Gardner-Wells tongs were placed and cervical traction was applied. Cross-table cervical spine roentgenography showed inadequate reduction of the odontoid fracture. The patient was taken to the operating room for transarticular screw fixation of the odontoid fracture.

An awake fiberoptic intubation was performed without complications. After sedation with nitrous oxide (50%) and droperidol (5 mg), subdermal stainless steel needle electrodes (Nicolet Biomedical, Madison, WI) were placed at all electrode locations for monitoring bilateral posterior tibial (PTN) and ulnar nerve (UN) SEPs. A Viking II (Nicolet Biomedical) was used. Cortical (two channels, CZ-FPZ, and C3–C4) and subcortical (A1-FPZ) evoked potentials were monitored and were recorded using the same montage for both the PTN and the UN SEPs because the Viking II does not have the ability to automatically switch electrodes when changing from the PTN to the UN file. Recordings of peripheral nerve responses were attempted with electrodes placed at the popliteal fossa and Erb's point; however, this was unsuccessful. The low-pass filter was set at 30 Hz and the high-pass filter was set at 1,500 Hz. A stimulation rate of 4.7 Hz was used, and the stimulation intensity was set at 37 mA. Five hundred repetitions were used to obtain most of the waveforms. Right and left extremities were stimulated separately in an alternating fashion and the averages were updated continuously. A disposable ground-plate electrode (Nicolet Biomedical) was placed on the right shoulder.

General anesthesia was induced to obtain baseline SEPs before positioning. After induction of anesthesia, vecuronium was administered. Anesthesia was maintained with 65–70% nitrous oxide and a total of 250 μg fentanyl administered over the duration of the operation. Before positioning, PTN cortical SEPs were obtained bilaterally. A cortical and subcortical SEP could be elicited from stimulation of the left UN. Stimulation of the right UN generated a cortical evoked potential of much smaller amplitude and an unidentifiable subcortical evoked potential.

The patient was log-rolled into the prone position, her head was placed on a Mayfield head rest, and 33 kg of traction was applied to the Gardner-Wells tongs. The SEPs were checked immediately after positioning was completed. The PTN SEPs were absent after positioning; however, the UN SEPs were still present bilaterally. The UN SEP

waveforms were of better quality after positioning, possibly because of less electromyography artifact. Good alignment of the cervical spine was confirmed by fluoroscopy. Because the cervical instability was at the C1–C2 level and the UN SEPs were present, it was thought unlikely that the loss of the PTN SEPs represented an injury to the cervical spinal cord. All connections and wires appeared to be intact. The cause of the changes could not be identified. The surgeon proceeded with the planned procedure, and the UN SEPs were monitored exclusively.

During drilling for placement of screws for fixation of the C1–C2 joint, the amplitude of the left ulnar cortical SEP (C3–C4) decreased from 0.50 (baseline value) to 0.17 μV, a change of 66%, with no significant change in latency. The subcortical SEPs were unchanged in amplitude or latency. The amplitude returned to baseline within 5 min, with temporary cessation of drilling. Amplitude decreases of more than 50% were seen in the cortical evoked responses three additional times during drilling. The amplitudes recovered each time after cessation of the drilling.(fig. 1) No changes were noted in the latency of the potentials. At no time were changes in amplitude or latency seen in the waveforms recorded at A1-FPZ. No changes in anesthetic technique or depth occurred during the drilling. In addition, no significant hemodynamic or temperature changes occurred. The systolic blood pressure remained between 120–135 mmHg, the pulse was 62–68 beats/min, the oxygen saturation was ≥ 98%, and the total estimated blood loss for the case was 60 ml.

Both the cortical and the subcortical UN SEPs were present at the conclusion of the case. The PTN SEPs were still absent. Neurologic status was unable to be evaluated immediately postoperatively secondary to sedation from intraoperative anesthetics. However, within 5 h the patient was awake and moving all extremities on command with no weakness noted. She was easily weaned from the ventilator and extubated on postoperative day 1. She began ambulating on postoperative day 6 and was weaned from a cervical collar at 6 weeks.

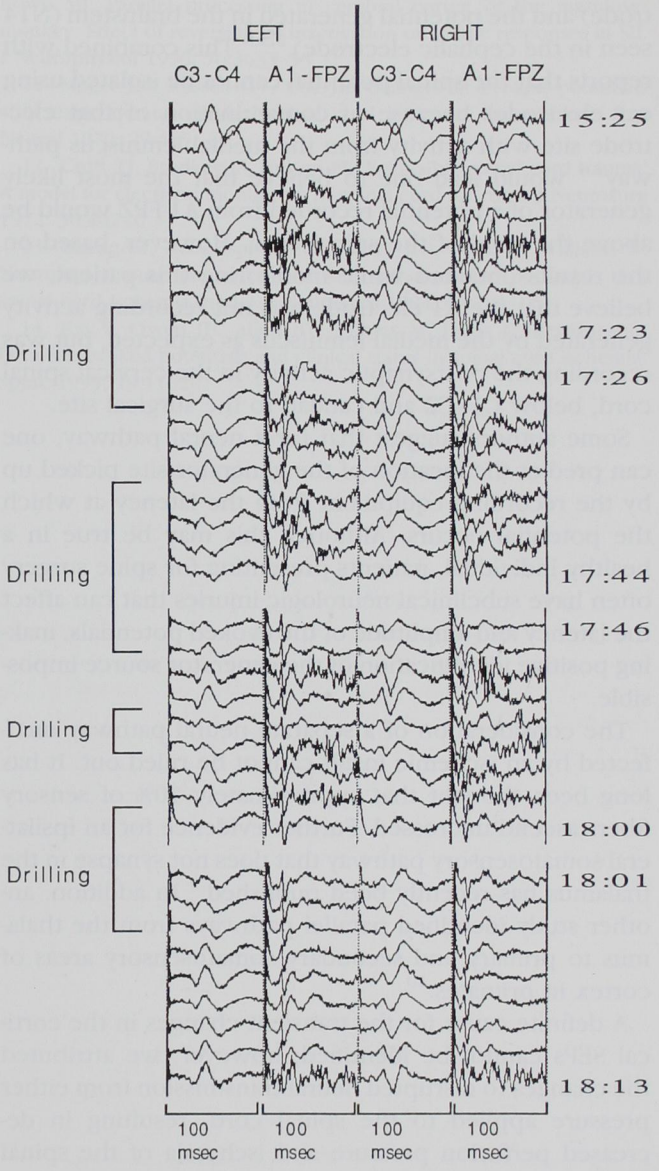

Fig. 1. Waterfall layout of the ulnar nerve cortical and subcortical somatosensory evoked potentials (SEPs) during placement of the transarticular screw at C1–C2. The periods of decreased amplitude of the cortical potentials associated with drilling are bracketed. Note that there is no significant change in the subcortical SEPs and that there is a gradual recovery of the cortical SEPs after each drop in amplitude.

Discussion

We describe a patient in which multiple, reversible decreases in amplitude of greater than 50% were detected in the cortical SEP (C3–C4), with no change noted in the subcortical SEP (A1-FPZ). This discrepancy between the cortical and subcortical potentials is possible if ischemia develops cephalad to the generator of the subcortical response. Alternatively, the subcortical response may travel in a completely separate neural pathway that is not affected by the insult. This is an important concern because the subcortical potential is more resistant to the depressive effects of anesthetic agents[1,2] and, therefore, is considered a more reliable intraoperative monitor.

The subcortical potential recorded in a given patient may be generated by postsynaptic activity in the cervical spinal cord, activity in the caudal medial lemniscus, or activity just below the thalamus.[3,4,5,6,7] Both the medial lemniscus and the subthalamic areas are above the level of potential spinal cord injury for the operation discussed. Because our recording electrode was on the mastoid process and referenced to a cephalic electrode (FPZ), we initially postulated that the most likely generator of the potential we were recording was from activity in the medial lemniscal pathway. This is based on studies that show SEPs recorded from the neck and referenced to FPZ reflect an interaction between the spinal-generated potential (N13 seen in the cervical elec-

trode) and the potential generated in the brainstem (N14 seen in the cephalic electrode).[3,5,8] This combined with reports that the spinal potential cannot be isolated using ear electrodes because of contamination of that electrode site with activity from the medial lemniscus pathway[3,4] would lead one to believe that the most likely generator of a potential recorded from A1-FPZ would be above the level of the spinal cord. However, based on the results obtained while monitoring this patient, we believe that the A1 electrode was not recording activity generated by the medial lemniscus as expected, but was recording the postsynaptic activity in the cervical spinal cord, below C1–C2 and caudad to the surgical site.

Some authors suggest that for a neural pathway, one can predict the location of the generator site picked up by the recording equipment from the latency at which the potential occurs. Although this may be true in a healthy individual, patients presenting for spine surgery often have subclinical neurologic injuries that can affect the latency and amplitude of the evoked potentials, making positive identification of the generator source impossible.

The consideration of a separate neural pathway unaffected by an ischemic insult cannot be ruled out. It has long been thought that approximately 10% of sensory fibers ascend uncrossed. Further evidence for an ipsilateral somatosensory pathway that does not synapse in the thalamus has recently been published.[9] In addition, another study identified parallel pathways from the thalamus to primary and secondary somatosensory areas of cortex in primates.[10]

A definite cause for the transient changes in the cortical SEPs cannot be identified; however, we attributed the changes to disrupted neural transmission from either pressure applied to the spinal cord, resulting in decreased perfusion pressure and ischemia of the spinal cord, or local heating of the tissue during drilling. Although there were no postoperative deficits corresponding to the changes noted intraoperatively, animal studies validate the sensitivity of SEPs to acute spinal cord injury.[11,12,13] The latencies of the responses remained unchanged throughout the operation; however, the initial response to ischemia may result in a decrease in amplitude without a shift in latency.[14]

The changes seen in the cortical SEPs during this procedure are unlikely to be anesthetic related because no changes in the anesthetic technique or boluses of drugs were administered during the period of time in which the changes occurred. Cortical ischemia is also an unlikely cause of the varying amplitude because there were no significant changes in blood pressure, pulse, oxygenation, or erythrocyte volume.

We also believe it unlikely that the changes in the cortical SEPs were related to artifact. If the loss of amplitude of the cortical potentials was related to artifact generated by the use of the drill, one would expect the cortical and subcortical potentials to be affected equally. This was not the case. In addition, the amplitude of the SEPs changed gradually, both when it decreased and when it returned to baseline. If the changes in amplitude were purely artifact, then one would expect amplitudes to return to baseline immediately when the drilling stopped. This further suggests that the changes of the cortical SEPs were related to ischemia and not to artifact from the use of the drill.

In conclusion, reversible decreases in amplitude of the cortical SEPs during drilling of the cervical spine did not occur in the subcortical SEPs recorded from electrodes at A1-FPZ. Although the changes seen in this patient were not sufficiently serious to affect neurologic outcome, they do suggest that these subcortical responses are an insufficient monitor for ischemia during posterior spine surgery performed in the region of C1–C2. The generator of the potential recorded from A1-FPZ may be caudal to the area of the spinal cord at risk or may arise from a separate, parallel neural pathway that is unaffected by the insult. Interpretation of intraoperative neurophysiologic monitoring must include consideration of the generators of the evoked potentials and their anatomic location and anesthetic, physiologic, and surgical manipulations and changes.

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