Intraoperative Loss of Somatosensory-evoked Potentials Predicts Loss of Spinal Cord Function

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Somatosensory-evoked potentials (SSEP) can be used to monitor spinal cord function during operations on the spinal column1-3 and within the spinal canal.4-7 Studies in animals have shown that spinal cord injuries of sufficient severity to irreversibly obliterate these potentials consistently produce major neurologic deficits.8,9 This report describes the correlation, in a patient, between intraoperative loss of SSEP and permanent injury to the spinal cord.

REPORT OF A CASE

A 52-year-old man, previously healthy except for mild obstructive airway disease, had a 14-month history of intermittent weakness, numbness, and pain in the right leg. He also suffered progressive constipation and urinary incontinence. Examination revealed partial loss of motor function in hip flexors and extensors, with strength graded as 2/5 on the right and 3/5 on the left. Distal muscle groups were intact. Vague dullness to pin-prick extended up both legs to the level of the inguinal ligaments. Deep tendon reflexes were hyperactive in the right lower extremity, with clonus, and the right toe upgoing. Myelography showed a lesion, thought to be a vascular malformation, on the dorsal aspect of the cord. It extended from the level of the eighth thoracic vertebra to the top of the first lumbar vertebra. On arteriography, numerous small feeding vessels were seen at multiple levels, but attempts to demonstrate the entire lesion were unsuccessful.

SSEP elicited by separate stimulation of the left posterior tibial nerve, right posterior tibial nerve, and right median nerve were recorded in the operating room before induction of anesthesia, using the method described in the Appendix. Only the responses to median nerve stimulation were completely normal, but reproducible waveforms were obtained with stimulation at each of the three sites.

Anesthesia was induced with 3 mg/kg thiopental, 5 μg/kg fentanyl, and nitrous oxide 65%. Pancuronium provided skeletal muscle relaxation, and cardiovascular responses to stimulation were controlled with small doses of thiamethaphan. The patient was turned to the left lateral decubitus position, and SSEP were recorded continually throughout the operation. Electrocardiogram, intra-arterial blood pressure, and nasopharyngeal temperature were monitored continuously. Central venous pressure, arterial blood-gas tensions, hematocrit, and serum osmolality, sodium and potassium were measured at various intervals.

FIG. 1. SSEP elicited by stimulation of the right posterior tibial nerve. The first waveform, abnormal but reproducible, was recorded before opening of the dura. The second, recorded three hours later, showed increasing loss of definition. The third waveform, recorded after five hours of intradural operation, was not distinguishable from an average recorded without somatosensory stimulation. Peaks are labeled N for negative and P for positive, with numbers representing nominal poststimulus latencies. Note that actual latencies, particularly those of later peaks, are delayed far beyond their nominal values. These latency shifts are due in part to neurologic abnormality, in part to the effects of anesthesia. The artifact seen in the last two tracings probably stems from spontaneous EEG rhythms. This kind of artifact is now memorized by using pseudoranandom interstimulus intervals, so that rhythmic EEG patterns are eliminated by the averaging process.

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As the multiple small vessels feeding the AVM were sacrificed, SSEP elicited by stimulation of either posterior tibial nerve were progressively distorted. There was no dominant feeding vessel suitable for test occlusion. After 14 hours of operation, SSEP produced by stimulation of the lower extremities were completely lost (fig. 1). In contrast, responses to stimulation of the median nerve remained stable throughout. The patient awoke in the recovery room with a dense posterior column deficit. He had only minimal motor function in the lower extremities. SSEP recorded on the second day after surgery showed no response to stimulation of either posterior tibial nerve. Hopes for partial recovery became untenable ten days postoperatively when the patient developed herpes infection at the operative site and the spinal cord lesion became complete.

DISCUSSION

This case report adds to the collective body of knowledge about correlations between intraoperative alterations in evoked potentials and neurologic outcomes. SSEP were irreversibly obliterated, and the patient was left paraplegic.

We still have no quantitative description of the absolute tolerance limits for intraoperative SSEP changes short of irreversible obliteration. Marked distortion and even complete loss of sensory-evoked potentials may be consistent with preservation of neurologic function, provided the changes are reversible.\textsuperscript{1,11,12} We have seen recovery of brainstem auditory-evoked potentials after complete obliteration lasting 177 minutes.\textsuperscript{13} The maximum duration of evoked potential obliteration compatible with recovery is unknown. Absolute tolerance limits for changes in other physiologic variables monitored during anesthesia and operation, such as arterial blood pressure, heart rate, and electrocardiogram, are similarly lacking. For each of these variables, however, complete and irreversible loss of the signal is invariably associated with an untoward outcome.

When changes in either neurologic or cardiovascular parameters warn of deteriorating function, interventions can often be undertaken to optimize physiologic stability and minimize the risks of permanent injury.\textsuperscript{1-3,11,12} Monitoring, however, cannot absolutely assure freedom from injury. Our patient’s AVM presented no major feeding vessel suitable for test occlusion, and the multiple small vessels that had been sacrificed when SSEP were lost could not be reopened. The seemingly unavoidable unfortunate outcome in this case provides evidence that irreversible intraoperative obliteration of SSEP may in patients, as in experimental animals,\textsuperscript{8,9} predict postoperative loss of neurologic function.

APPENDIX

SSEP were recorded using a Nicolet MED-80 Biomedical Data System (Nicolet Biomedical, Inc., Madison, Wisconsin). Constant current electrical shocks, 250 $\mu$A in duration, were delivered to each posterior tibial nerve at the ankle and to the right median nerve at the wrist. For stimulation, pairs of subdermal platinum electrodes (Grass Medical Instruments, Quincy, Massachusetts) were positioned along the course of each nerve, 3 cm apart with the cathode proximal. Stimulus intensity was adjusted to provide a painless twitch before anesthesia and increased to 19.9 mA during anesthesia. The stimulus interval was 1.2 s. Gold cup electrodes, 10 mm in diameter, were placed according to the International Ten Twenty System, applied to the scalp with collodion, and filled with conductive gel. Electrode impedances were less than 3,000 ohm. Recording was in three bipolar derivations: 2 cm behind Cl to F2, 2 cm behind C2 to F2, and 2 cm behind C3 to FZ. The analog electroencephalographic (EEG) signal, amplified to $\pm 50$ $\mu$V full scale, was filtered with a bandpass of 1–1,500 Hz, then sampled at 3,906 Hz for analog-to-digital conversion. To extract the sensory-evoked potential from background EEG activity, 128 epochs, 262 ms in duration and precisely time-locked to the sensory stimulus, were averaged. The averaged waveforms were displayed on an oscilloscope, measured using a controllable cursor, plotted, then stored on disc.

REFERENCES