Postoperative paraplegia with preserved intraoperative somatosensory evoked potentials

Case report

HOWARD H. GINSBURG, M.D., ANDREW G. SHETTER, M.D., AND PETER A. RAUDZENS, M.D.
Barrow Neurological Institute, Phoenix, Arizona

Intraoperative somatosensory evoked potentials (SSEP's) are being used with increasing frequency to monitor neurological function during spinal surgery. The authors report a case of postoperative paraplegia that occurred despite preserved intraoperative SSEP's in an achondroplastic dwarf who underwent correction of a congenital kyphoscoliosis. Surgeons and anesthesiologists involved with SSEP monitoring should be aware that false-negative results may occur with this technique.

KEY WORDS: somatosensory evoked potentials · intraoperative monitoring · scoliosis · paraplegia

Intraoperative somatosensory evoked potentials (SSEP's) have been used increasingly in recent years to monitor neurological function during scoliosis surgery and other high-risk spinal operations. Numerous cases have been reported in which early recognition of SSEP latency and amplitude changes appeared to have prevented permanent neurological deficits by alerting the surgeon to the need for appropriate corrective action. We report a case of postoperative paraplegia that occurred despite preserved intraoperative SSEP's.

Case Report

This 12-year-old female achondroplastic dwarf with a progressive kyphosis at the thoracolumbar junction underwent a two-stage surgical procedure with intraoperative SSEP monitoring. Her deformity was first noted at birth. In July, 1974, at 3 years old, she had a 65° thoracolumbar kyphosis at the T11–L3 vertebral level, with severe hypoplasia of the anterior bodies of L-1 and L-2. This had progressed to 103° by September, 1982. A severe gibbus was present, with a hemivertebra at L-1 (Fig. 1). The patient was normally active with occasional numbness and weakness of the lower extremities. There was no bowel or bladder dysfunction. Neurological examination was normal.

Operations. In February, 1983, a first-stage anterior release procedure with correction through a left thoracoabdominal approach was carried out with SSEP monitoring. Segmental vessels were ligated from T-11 to L-3. A Pinto distractor was inserted between T-10 and L-3. The left ninth rib and the left fibula were excised and used to strut-graft the kyphotic deformity, which was corrected to 58°. Postoperatively, the patient was placed in a halo wheelchair and a removable thoracolumbar sacral orthosis was used for support. The neurological examination remained normal.

Three weeks later, a fusion procedure was performed with SSEP monitoring. Harrington compression instrumentation was inserted from T-10 to L-4 on the left and distraction instrumentation was placed from T-9 to L-4 on the right (Fig. 2). Clearly identifiable SSEP's persisted during and after instrumentation.

The level of anesthesia, body temperature, and blood pressure remained stable. The patient awakened in the operating room in the prone position. She was paraplegic with no appreciation of pinprick below T-12. Other sensory modalities were not tested. The patient was immediately reanesthetized, the rods were removed, and partial laminectomies at T9–10 and L3–4 were carried out. A dural laceration at L-4 was repaired. There had been no cerebrospinal fluid leak. No other
Intraoperative somatosensory evoked potentials

**FIG. 1.** Left: Lateral thoracolumbar film taken at 3 years old showing a kyphosis of 65°. Center and Right: Thoracolumbar films, lateral (center) and anteroposterior (right) views, taken at 12 years old showing progression of the kyphosis to 103°.

**FIG. 2.** Lateral films showing posterior instrumentation with anterior rib and fibula strut grafts. Intraoperative film (left), and films at 3 months (center) and 6 months (right) postoperatively.

intraspinal pathology was noted. Immediately postoperatively, the patient remained paraplegic with absence of all sensory modalities below T-12, including proprioception and perception of pinprick, vibration, and deep pressure. The anal sphincter was flaccid without volitional contraction. Six months later, sensation had returned to the entire right lower extremity and to portions of the left hip. Hip flexor strength was graded at 2/5 on the right and 1/5 on the left. No other lower-extremity motor activity was present, including the bladder.

**Somatosensory Evoked Potential Monitoring.** The SSEP’s were recorded preoperatively and during the three separate surgical procedures. Intraoperative monitoring was limited to cortical SSEP’s generated by 10.0-mAmp stimulation of the individual peroneal nerves and was recorded from the contralateral somatosensory cortex. The preoperative SSEP’s also included the lumbar response recorded from L-1 to L-3 and the response from T-6 to T-10. Two thousand individual responses were averaged on an NIC CA 100* and two successive waveforms were superimposed to demonstrate reproducibility. The stimulus was a square-wave electrical pulse of 300-μsec duration delivered at a rate

* Nicolet monitoring system, Model NIC CA 1000, manufactured by Nicolet Biomedical Instruments, Madison, Wisconsin.
H. H. Ginsburg, A. G. Shetter and P. A. Raudzens

FIG. 3. Recordings at the first-stage anterior procedure. Preoperative somatosensory evoked potentials (SSEP's) were recorded from lumbar and contralateral cortical recording sites. This was a normal study. Intraoperative SSEP's recorded from the contralateral sensory cortex continued unchanged. The patient was neurologically normal postoperatively.

of 4.1/sec. The bandpass filters were set at 30 and 1500 Hz, and a 60-Hz notch was incorporated. The recording electrodes were silver discs with interelectrode impedances of less than 5000 ohms. Both latency and amplitude measurements were made of the primary cortical response.

Preoperative SSEP's showed well resolved waveforms. Latency values after left and right peroneal nerve stimulation from lumbar, thoracic, and contralateral cortical recording sites were: 8.4 and 8.4 msec (lumbar), 11.4 and 12.6 msec (thoracic), and 24.6 and 25.7 msec (cortical). The normal unanesthetized adult values at the Barrow Neurological Institute are: 12.4 ± 2.15 msec (lumbar), 15.04 ± 2.62 msec (thoracic), and 28.00 ± 4.48 msec (cortical) (within 2.5 standard deviations for 62 observations). The short latency times in our patient were due to the reduced length of the limbs, since central conduction times were normal.

During the first-stage anterior procedure, cortical somatosensory responses persisted intraoperatively (Fig. 3). The latency values varied from 21.4 to 26.0 msec on the left and from 21.7 to 25.7 msec on the right. The anesthetic was limited to halothane in concentrations below 1% with small supplemental doses of fentanyl. The patient's temperature remained above 36.5°C and the systolic blood pressure was above 90 torr. There were no postoperative neurological deficits.

Three weeks later, SSEP's were recorded during the second-stage posterior procedure with instrumentation and fusion, using similar stimulus and recording parameters (Fig. 4). Initial latency and amplitude values were 29.4 msec and 0.12 μV on the left and 28.6 msec and 0.09 μV on the right. These figures were somewhat prolonged over those seen postoperatively and during the first-stage procedure, but we do not consider this abnormal, since latency delays of 4 to 5 msec are routinely seen in the anesthetized patient, depending upon the depth of anesthesia. At the end of the operation, 5½ hours later, well resolved cortical responses persisted. The latencies and amplitudes were 29.2 msec and 0.19 μV on the right and 30.4 msec and 0.21 μV on the left.

On emergence from anesthesia, the patient was found to be paraplegic. Immediate rod removal and exploration of the operative site was performed. For a 15-minute period during reexploration, systolic blood pressure was 70 torr. No other blood pressure fluctuations were noted. The temperature remained above 36°C. During the 3 hours required for the third operation, there was gradual deterioration in SSEP latency and amplitude. By the end of the procedure, no reproducible cortical response could be identified. Two days later, postoperative SSEP's failed to demonstrate any cortical response. On SSEP recording 6 months postoperatively (Fig. 5), no cortical response could be identified following left peroneal nerve stimulation. A low-amplitude
Intraoperative somatosensory evoked potentials

FIG. 4. Intraoperative somatosensory evoked potentials generated by peroneal nerve stimulation were recorded from contralateral cortical sites (8:15 a.m.). Well resolved cortical responses were identified at the end of the procedure despite postoperative paraplegia (1:45 p.m.). The cortical responses deteriorated during the next 3 hours (2:30 p.m.), and by the conclusion of the third operation no clear-cut responses could be identified (4:40 p.m.).

A response of 37.9 msec was reproducible following right peroneal nerve stimulation. This result is consistent with the sensory deficits observed.

Discussion

It is well established that SSEP's are primarily or exclusively an indication of dorsal column integrity and do not provide information about the more clinically relevant motor pathways. However, it has been hoped that any intraoperative factors affecting descending motor pathways would produce concurrent changes in dorsal column activity which could be detected by SSEP monitoring.

Our report documents a case in which a patient with well preserved reproducible intraoperative SSEP's awoke from surgery with paraplegia. The SSEP's gradually deteriorated and were eventually lost during emergency reexploration, but the onset of clinical paraplegia preceded loss of the electrical signal by a minimum of 3 hours. The cause of paraplegia in this situation was presumably vascular infarction of the conus medullaris or cauda equina. MacEwen, et al., surveyed patients operated on by members of the Scoliosis Research Society, and reported that 30% of those with a major neurological complication had a severe associated kyphosis.

False-positive SSEP's (that is, a change in the electrical signal with no change in neurological function) are not infrequent during intraoperative monitoring, and are usually due to technical problems or environmental factors unrelated to surgical manipulation. These include equipment failure, electrode movement, alterations in blood pressure, depth of anesthesia, and temperature changes. False-negative SSEP's (that is, preservation of the electrical signal despite significant neurological deficit) are much less common, but their consequences are more serious. The explanation for the false-negative recordings seen in our case is not clearly apparent, but several possibilities exist. It is conceivable that a progressive spinal cord infarction developed which initially involved anterior portions of the cord and only later affected the dorsal columns. When the patient awakened from Harrington rod placement, she was paraplegic with preserved SSEP's, and it was impossible to perform a detailed sensory examination due to the urgent need for reexploration; however, at that time some clinical dorsal column function may have remained. Following her recovery from rod removal, it was apparent that all sensory modalities in the legs were absent, but by this time the SSEP's had been lost as well.

Another explanation for false-negative SSEP's may be the nonphysiological nature of the repetitive high-intensity electrical pulses used to stimulate the peripheral nerves. Under certain circumstances, somatosensory cortical activity might be evoked by strong
Six months postoperatively, somatosensory evoked potentials showed prolonged latency following right peroneal nerve stimulation. There was no detectable response to left peroneal nerve stimulation. Electrical stimulation, but not by other forms of stimulation such as touch, proprioception, or vibration. Some viable sensory neurons must have remained immediately postoperatively in our patient despite her anesthesia below the T-12 level, since she eventually experienced partial return of sensation in both legs.

Regardless of the etiological factors involved, it is important for surgeons, anesthesiologists, and neurologists performing SSEP monitoring to realize that severe motor deficits can occur after surgery despite intraoperative preservation of a clear-cut electrical signal. The frequency with which this phenomenon occurs is not known, and will only become apparent as further experience is gained with electrophysiological monitoring of the spinal cord during surgery.

References