



Failure of Sequential Compression Device Detected by Neuromonitoring during Minimally Invasive Posterior Scoliosis Surgery

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J Neuroanaesthesiol Crit Care 2023;10:125–127.

Abstract

Intraoperative neuromonitoring is recommended as standard practice for corrective scoliosis surgery. Common methods include somatosensory-evoked potentials (SSEPs) and transcranial motor-evoked potentials (TcMEPs), which have been shown to have a high diagnostic accuracy in detecting new neurological deficits postoperatively. Sequential compression devices (SCDs) are a common method for thromboprophylaxis in spine surgery and are not known to have many device-related complications. To date, there have been no reports of lower extremity ischemia secondary to SCD deflation failure detected by multimodality neuromonitoring during minimally invasive posterior spine surgery. We, therefore, present a case report of an 18-year-old male with adolescent idiopathic scoliosis who underwent minimally invasive posterior spinal fusion with instrumentation. Intraoperative decrease in SSEPs and TcMEPs were noted in the left leg shortly after incision before any instrumentation or reduction occurred. Further examination revealed that the left leg was hypoperfused compared with the right leg and that the left SCD was not properly deflating. Bilateral SCDs were removed, and perfusion and neuromonitoring returned to baseline immediately. Bilateral SCDs and the machine were replaced, and neuromonitoring remained within normal limits for the rest of the surgery. The patient had no postoperative neurologic or vascular deficits. Early detection of lower extremity ischemia by neuromonitoring resulted in the prompt identification and addressing of SCD malfunction, sparing devastating neurological and vascular injury to the patient's leg. This case reinforces the importance of neuromonitoring within spine surgery.

Keywords

- ▶ neuromonitoring
- ▶ scoliosis
- ▶ sequential compression device

Introduction

Intraoperative monitoring of spinal cord activity has been recommended as a standard practice for corrective scoliosis surgery to prevent the rare but devastating possibility of

postoperative paralysis or paresis due to damage to the spinal cord.¹ Intraoperative neuromonitoring to assess neurological functioning can be obtained with the use of somatosensory-evoked potentials (SSEPs), motor-evoked potentials (MEPs), or

article published online
May 14, 2023

DOI <https://doi.org/10.1055/s-0043-1764297>.
ISSN 2348-0548.

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electromyography.¹ Within deformity surgery, transcranial MEPs (TcMEPs) and SSEPs are highly sensitive and specific tests (91% vs. 96%, respectively, for TcMEPs and 84 and 98%, respectively, for SSEPs), and the combination of these tests results in greater diagnostic accuracy in detecting new neurological deficit postoperatively.^{2–4}

Sequential compression devices (SCDs) are a common method of thromboprophylaxis in spine surgery.⁵ SCDs have a low failure rate (3–8%) and minimal device-related complications.⁶ We present for the first time a case of unilateral lower extremity ischemia secondary to SCD deflation failure detected by SSEP and TcMEP neuromonitoring.

Case Report

An 18-year-old male with progressive idiopathic scoliosis presented for T3 to T11 scoliosis correction using minimally invasive posterior spinal fusion with instrumentation. He demonstrated a 52-degree right thoracic curvature and a 14-degree left thoracic curvature. He had no other significant history. He did not have any neurological or vascular deficit before surgery.

SCDs were positioned on the patient's bilateral calves for deep vein thrombosis prophylaxis. The patient underwent intravenous anesthetic induction with propofol, vecuronium, and fentanyl. A 7.5 internal diameter cuffed endotracheal tube was placed under laryngoscopy guidance. Anesthetic depth was monitored via clinical signs (i.e., lack of movement, no response to painful stimuli) and change in vital signs (i.e., heart rate, blood pressure). The patient was maintained on at least 0.5 minimum alveolar concentration of sevoflurane and a combination of ketamine, sufentanil, and fentanyl to allow neuromonitoring yet provide adequate anesthesia and analgesia. No complications with anesthesia were noted.

For SSEPs, stimulation was from the ulnar nerve for the upper limbs and the posterior tibial nerve at the medial malleolus for the lower limbs. Recording electrodes were placed per the standard 10–20 system at FpZ, C3', Cz', C4', and mastoid with the ground placed at the shoulder. A monophasic square pulse with an amplitude of 25 mA was used for the upper limbs and 45 mA for the lower limbs. The pulse width for both the upper and lower limbs was 300 ms, and a repetition rate of 4.13 Hz. All four limbs were interleaved. TcMEPs were recorded from the abductor pollicis brevis tied to the abductor digiti minimi for the upper limbs and the anterior tibialis and adductor hallucis for the lower limbs. Stimulation was delivered via two needled electrodes placed just in front of the C3 and C4 of the 10–20 system. The anode was just in front of C4 for left muscle responses and just in front of C3 for right muscle responses. Stimulation parameters included a monophasic square pulse of 250 V amplitude for the right and left responses, a pulse width of 500 ms, an interstimulus interval of 75 ms, and a train of 7 pulses. SSEPs and MEPs demonstrated good morphology and reproducibility of the

potentials and had baseline latencies near normal limits (►Fig. 1).

The patient was placed in prone position, and all pressure points were padded. Using lateral fluoroscopy, a midline incision was made extending from T2 to T12. Shortly after the incision, SSEPs were noted to be decreased in the left leg, followed by a reduction in TcMEPs in the left leg. Examination revealed that the left leg appeared ischemic compared with the right with pulse oximetry of 85% and that the left SCD was not appropriately deflating. The wound was covered in a sterile fashion, and the drapes were removed. Bilateral SCDs and SCD machine were removed, and neuromonitoring electrodes were replaced. SSEPs and TcMEPs returned to baseline within 30 minutes after identifying the malfunctioning SCD. The patient was repped and draped, new SCDs and device were placed, and the procedure was completed without complication. Immediate postoperative examination and follow-up at 2 weeks, 5 weeks, and 6 months were without any neurological or vascular defects in the bilateral lower extremities.

Discussion

Intraoperative neuromonitoring has previously identified cases of lower extremity ischemia during spine surgery.^{7,8} In both the cases, the cause of ischemia was identified as malpositioning of the patient while in prone position which caused temporary occlusion of the femoral artery.^{7,8} Neuromonitoring was able to quickly identify the lack of perfusion to the extremity and prompt repositioning of the patient was undertaken, which avoided any long-term consequences.^{7,8}

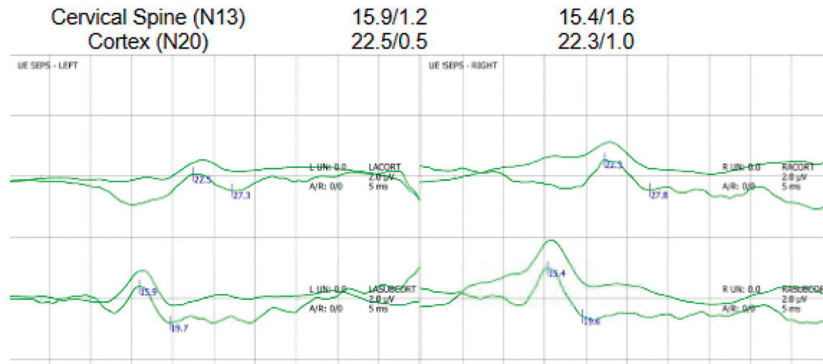
In our case, lower limb ischemia was also detected by SSEPs and TcMEPs but was found to be secondary to prolonged compression by the SCDs. Commercially available SCDs are typically designed to deliver 45 mm Hg pressure for 12 seconds with 48 seconds of deflation time (ArjoHuntleigh, Sweden). Intermittent use of SCDs has been shown to have no consequences for pressures up to 70 mm Hg for 130 seconds.⁹ It is not known, however, what damage may occur for sustained pressure for a longer period of time, for which its use was not intended for. Given that minimally invasive spine corrective surgery operative time has been reported to range between 4.2 and 8.78 hours, the prolonged time the device was left inflated may have had an accumulation of pressure to result in the point of limb ischemia.¹⁰ This may have resulted in more severe neurological and vascular insults to his leg if it had not been detected via neuromonitoring. The authors hope this case serves as a reminder of the potential benefits of neuromonitoring, especially in the recent discussion of implementing neuromonitoring as the standard of care for spinal deformity surgery. Finally, providers should be aware of recently developed checklists to optimize response to intraoperative neuromonitoring events, which can be expertly reviewed elsewhere.^{11,12}

Median Nerve Evoked Potential

Results: Beginning at 09:43, on stimulation of each median nerve the N13 and N20 were clearly defined and reproducible.

Latency (msec)/Amplitude (microV)

Left Median Nerve Right Median Nerve

**Posterior Tibial Nerve Evoked Potential**

Results: Beginning at 09:42, on stimulation of each posterior tibial nerve the N10, N30 and P37 were clearly defined and reproducible.

Latency (msec)/Amplitude (microV)

Left PTN Right PTN

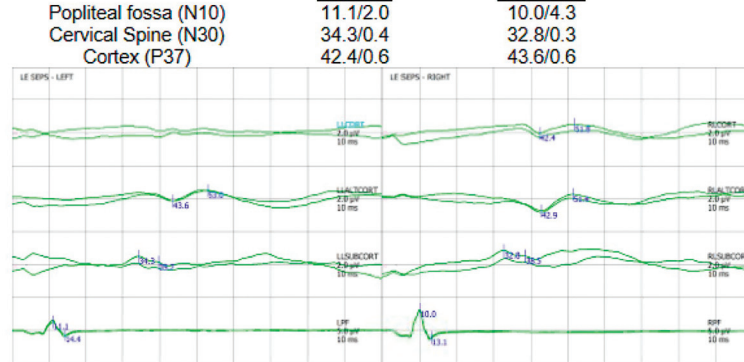


Fig. 1 Baseline somatosensory-evoked potentials (SSEPs). Example of SSEP neuromonitoring data demonstrating stimulation of the median nerve (top) and posterior tibial nerve (bottom). Waveforms demonstrated good morphology and reproducibility of the potentials (graph), and stimulation of the electrodes were near normal parameters (latency [ms]/amplitude [micro-V]).

Conflict of Interest

None declared.

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