

## Case report

# Neurophysiological monitoring under anesthesia to position a child with extreme lumbar spine flexion for MRI and CT scan

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## Summary

A novel application of neurophysiological monitoring enabled us safely to anesthetize and position a child with severe lumbosacral spine flexion for diagnostic MRI and CT scan. We conducted a propofol-based anesthetic to optimize somatosensory (SSEP) and transcranial electric motor (tceMEP) evoked potential amplitudes, thereby facilitating dynamic neurological monitoring while fully extending the patient supine. In cases outside the operating room involving extraordinary changes in patient position, anesthesia providers may consider utilizing neurophysiological monitoring.

**Keywords:** monitoring; neurophysiological; evoked potentials; somatosensory; transcranial electric motor

## Introduction

Neurophysiological monitoring of somatosensory (SSEP) and, more recently, transcranial electric motor (tceMEP) evoked potentials are commonly used to assess the functional integrity of the spinal cord in anesthetized patients undergoing spine surgery (1–6). We report a novel application of multimodality SSEPs and tceMEPs that enabled us safely to anesthetize and

position a child with severe lumbosacral spine flexion for diagnostic MRI and CT scan.

## Case report

A 13 year-old Vietnamese boy with a 10 month history of progressive substernal chest pain, back pain, and severe lumbosacral flexion presented for diagnostic MRI and CT. His medical history included mild gastritis, asthma, and a 10 kg weight loss. He reported no symptoms of bowel or bladder dysfunction. He presented in full flexion with his head resting between his legs as shown in Figure 1. Although his family reported that he was able to

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**Figure 1**  
Patient in severely flexed position.

extend and lie supine for several minutes each day, he rested and performed most activities in this extremely flexed posture. His weight was 52 kg, T 35.8°C, HR 60 b·min<sup>-1</sup>, BP 96/54, and RR 20 b·min<sup>-1</sup>. On physical examination, his extremities had normal sensation, strength, and reflexes, bilaterally. His spine showed no lateral curvature, no sacral dimpling, and no evidence of spinal dysraphism. Sternal palpation elicited localized areas of pain. Laboratory values, including an ESR of 12, were normal as were his chest and abdominal radiographs, ECG, and echocardiogram. Esophageal and gastric endoscopy, conducted in an unknown position at an outside institution, revealed mild gastritis. Although the largely negative clinical findings raised our suspicion of malingering, conversion, or psychosis, our remote concern of intra/paraspinal pathology warranted MRI of the lower thoracic, lumbar, and sacral spine. Further CT imaging was obtained for complete skeletal evaluation. We utilized neurophysiological monitoring at the outset of the combined radiological procedures because we wanted some indication that the extended position would not result in spinal cord compromise and possible injury over time.

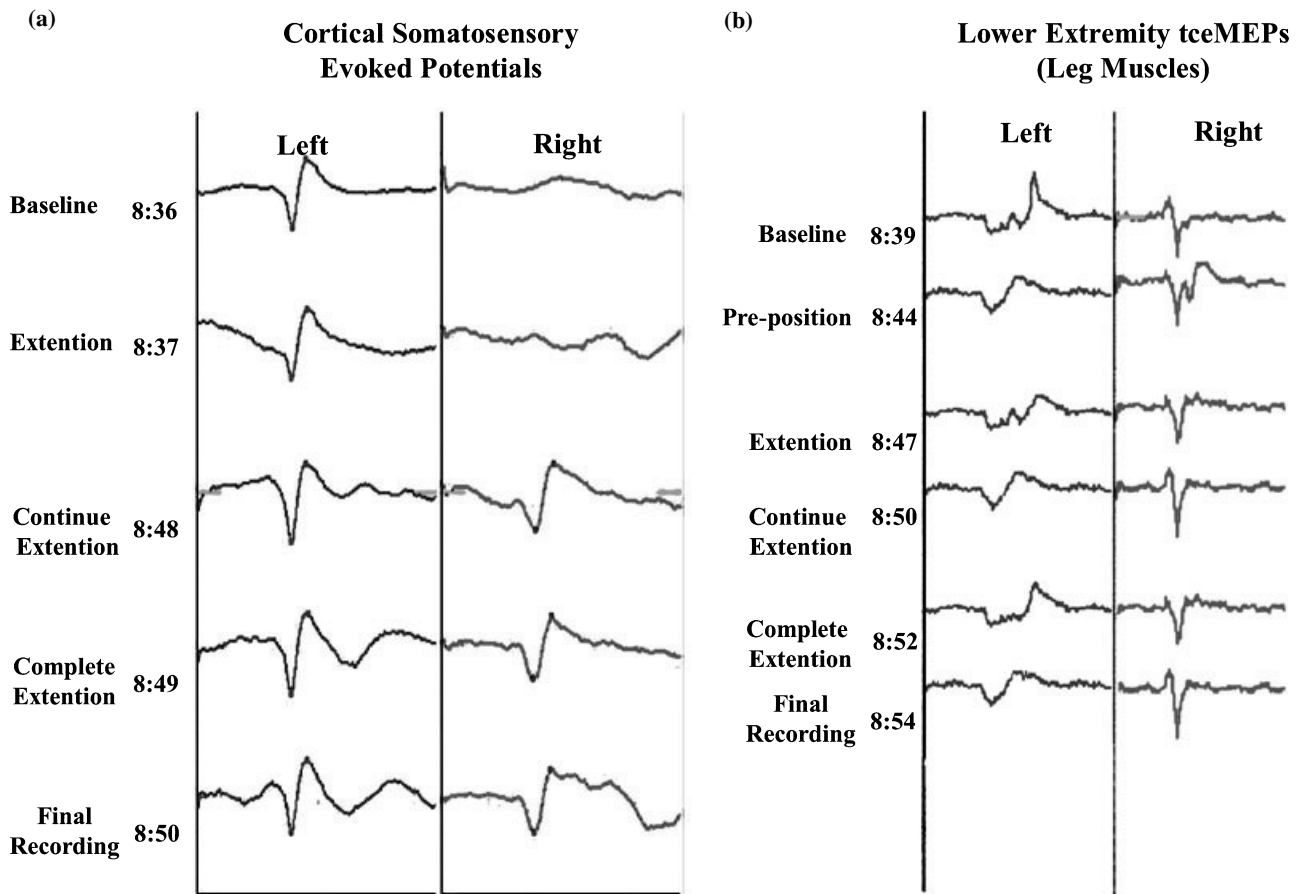
With the patient in flexed position, general anesthesia was induced and maintained with intravenous propofol (150 + 50 mg and 150 µg·kg<sup>-1</sup>·min<sup>-1</sup>). Spinal cord monitoring was performed continuously from anesthesia induction through extension and supine positioning by recording lower

extremity efferent tceMEPs and afferent SSEPs. Cortical and subcortical SSEPs were elicited to a 35 mA, 300 µs square wave electrical pulse presented to interleaving left and right posterior tibial nerves at a rate of 4.7 s<sup>-1</sup>. A commercially available neurophysiology instrument (Nicolet Endeavor; Nicolet Biomedical, Madison, WI, USA) was used for all SSEP stimulation and recording. Transcranial electric motor evoked potentials were recorded over both tibialis anterior and gastrocnemius muscles following a brief duration, high voltage (400–1000 V) anodal electrical stimulus train (pulse width = 50 µs, *n* = 3–7, interpulse interval 1–5 ms). The multipulse stimulus was delivered between two corkscrew-type electrode (A-Gram Co.; Glenn Rock, NJ, USA) inserted over motor cortex regions at C1 and C2 (international 10–20 system), respectively. Stimuli were delivered through a transcortical stimulator (D185; Digitimer, Ltd, Welwyn Garden City, Hertfordshire, UK) with responses recorded on the same system used for monitoring SSEPs.

Figure 2 presents stack plots of the cortical posterior tibial nerve SSEPs (Figure 2a) and tceMEPs (Figure 2b) from baseline flexion to full extension under anesthesia. As seen, the left baseline posterior tibial nerve SSEP was characterized by clear waveform morphology and normal amplitude and latency consistent with propofol anesthesia, whereas that for the right side was absent. Conversely, tceMEPs recorded from lower extremity leg muscles were present and symmetrical, bilaterally. The slightly different morphological character of the left *versus* right tceMEP responses is attributed to needle electrode placement differences with the left closer to the tibialis anterior muscle and the right closer to the gastrocnemius muscle.

Under continuous and vigilant guidance with neurophysiological monitoring, the patient was then extended at the waist. During extension the previously normal posterior tibial nerve SSEPs remained unchanged on the left; however, those on the right which had been absent at baseline, reemerged with amplitude and latency progressing to normal values and becoming essentially symmetrical with the left side.

With neurophysiological evidence of improved neurological function on the right side while in the supine position, we felt that the patient would not be at risk of neurological damage with prolonged



**Figure 2**

Posterior tibial nerve somatosensory (a) and transcranial electric motor (b) evoked potentials preceding, during and following lumbo-sacral extension for diagnostic MRI.

extension. Neurophysiological monitoring was discontinued and the patient was given vecuronium and fentanyl and then intubated for the studies. MRI of the spine showed no spinal cord or paraspinal abnormalities. There were four lumbar vertebrae, which is considered a normal variant. After the MRI study, the patient was transported to CT imaging and then to the PACU where he was extubated and recovered. He rested in the supine position. One hour later he was transported to the ward where he returned to his flexed position.

## Discussion

The routine use of neurophysiological monitoring has helped reduce the incidence of neurological insults during corrective spine surgery (1–6). In the case presented, multimodality SSEPs and tceMEPs

were used to ensure patient safety during prolonged lumbosacral extension and supine positioning for his diagnostic MRI and CT scan.

Intraoperative sensory and/or motor evoked potential changes can result from surgical insult, spinal cord ischemia, and nonspecific physiological and pharmacological factors (6). Since most common anesthetic drugs and sedatives effect cortically based efferent and afferent responses, the anesthetic technique is of paramount importance. All volatile anesthetics and nitrous oxide produce dose-dependent changes in SSEPs and tceMEPs (6–10). In patients with preexisting central nervous system disorders who may have reduced SSEP or tceMEP amplitudes at baseline, these anesthetics may hinder the ability to detect clinically significant changes (1). Propofol, however, has minimal effects on neurological monitoring and was central to our anesthesia plan (10,11).

During patient positioning under propofol-based anesthesia, the right posterior tibial nerve SSEP was unexpectedly absent at immediate postinduction baseline, thereby suggesting at least some form of spinal nerve root or cord transmission compromise in the extreme, flexed position. With our knowledge of the patient's ability to transiently extend at home, we proceeded with monitored extension and demonstrated the emergence of the right posterior tibial nerve SSEP, thereby obtaining supportive evidence of flexion-induced compromise. Although the actual mechanism for the absent unilateral SSEP is unknown, it is plausible that severe flexion had caused excessive stretch at either the L5-S1 spinal nerve roots or the right sciatic nerve and that this was reversible with extension. The absence of concomitant spinal motor tract abnormality as evidenced by the normal and unaltered tceMEP data, adds further credence to a peripheral nerve *versus* spinal cord origin for the absent right SSEPs. Lastly, and in keeping with these physiological data, the MRI confirmed normal spinal cord and pelvic anatomy.

We were extremely cautious in our approach to this patient with an extraordinary presentation. Although we are unaware of any cases of spinal/paraspinal tumors, abscesses, vascular malformations or cysts, manifest by back pain and severe lumbosacral flexion deformity (and without significant neurological or primary radiographic findings), we are aware of a pediatric spinal tumor presenting with leg discomfort and a mild gait abnormality (12). Further, we know that tumors of the spinal cord can mimic psychiatric disorders and that physicians can misdiagnose organic spinal disease as conversion disorder (13,14). The risk of misdiagnosis may be increased when there are cultural or language differences (14), such as in this case presentation of a Vietnamese boy treated in our quaternary North American hospital.

By careful exclusion then, the extreme lumbosacral flexion was attributed to a conversion disorder. The patient received a series of three intercostal blocks with triamcinolone and bupivacaine to treat his concurrent musculoskeletal chest pain and costochondritis. His condition improved and he was able to walk upright. Unfortunately, the child reverted to his flexed posture after 3 months and he required readmission for continued weight loss and depression.

In summary, neurophysiological monitoring may be used to ensure patient safety during positioning when there is the potential for neurological insult. Neurophysiological monitoring was indispensable in this child about to undergo a diagnostic MRI and CT when the etiology of his flexion remained uncertain. Within the first several minutes of the case, we became confident that the patient would not be at risk for potential spinal cord injury during the 4 h anesthetic. In addition, we were able to document reversal of compromised physiological transmission. This anesthetic required considerable advance planning and it highlights the anesthesiologist as a consultant who can help coordinate the efforts of a team of professionals (neurophysiologists, orthopedic surgeons, and radiologists) to ensure optimal care.

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