A Rare Presentation of Motor-Evoked Potential Stimulation-Induced Intraoperative Seizure in a Pediatric Patient

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J Neuroanaesthesiol Crit Care 2023;10:219–221.

Intraoperative neurophysiological monitoring (IONM) is routinely used to prevent neurological morbidity during spine and spinal cord surgeries. Among all IONM techniques, transcranial motor-evoked potential (MEP) stimulation is commonly utilized. Lip and tongue bite injuries, cardiac arrhythmias, seizures and bleeding, hematoma, and minor scalp burns at the stimulation site are widely reported complications associated with MEP stimulation.[1] The incidence of intraoperative seizures following transcranial MEP stimulation is rare, ranging from 0.03 to 0.8% for spine surgery.[2,3]

An 8-year-old male child (weight: 25 kg; height: 135 cm; body mass index: 21.6) presented with painless swelling in the mid-lower back since birth. There was no history of discharge from the swelling. The child was able to walk and run without support, and no other deformities were noted. On examination, a 4×3 cm swelling was covered by full-thickness skin in the midline at the lumbar region 7 cm above the natal cleft. Motor, sensory, and cranial nerve examinations were within normal limits. The bladder and bowel control were intact. Other systemic examination was normal. Magnetic resonance imaging -lumbosacral spine revealed low-lying conus ending at L5 to S1 with a 2.7 cm focal syrinx at L3 to 4 levels. The thickened fatty filum terminale was terminating at S3 to 4. The fibrous tract extended from the subcutaneous plane to the low-lying cord at L5 to S1. The child was scheduled for L3 to S2 laminectomy and excision of the fibroneural stalk, followed by sectioning of the fatty filum terminale in a prone position with IONM. The following IONM modalities were requested: MEP, bulbocavernosus reflex (BCR), and root stimulation.

After placing standard monitors, inhalational induction was performed using sevoflurane. A 20-gauge peripheral line was established, anesthesia was deepened with propofol and fentanyl, paralyzed using atracurium, and intubated with a 5.5 mm endotracheal tube. A soft bite block was placed to prevent MEP stimulation-induced tongue bite and lip injuries. Anesthesia was maintained with total intravenous anesthesia using propofol (150–250 µg/kg/min) and fentanyl infusion (1–2 µg/kg/h) and was titrated to maintain a bispectral index (BIS Covidien, Minneapolis, Minnesota, United States) between 40 and 50.

MEP responses were recorded (CADWELL Cascade IOMAX, Kennewick, WA 99336, USA.) using transcranial electrical stimulation of the motor cortex using four corkscrews placed at C1/C2 and 1.5 cm anterolateral to C1 and C2 (labeled as C1'/C2'), respectively. Five sets of lower limb muscles (quadriceps, tibialis anterior, abductor hallucis, extensor digitorum brevis, soleus, and external anal sphincter) were monitored bilaterally. First, a train of five pulse stimulations with an intensity of 100V, pulse width of 500 µs, and interstimulus interval of 3ms was used. Because there were no recordings from either stimulation montage, the intensity was increased to 120V. At 120V, a low-amplitude response was observed in the right lower limb muscles. Raising the stimulation intensity to 130V brought mild improvement. Therefore, the pulse count was increased to six using the C1'/C2' montage, which induced a bilateral response from the muscles, and was established as a baseline. BCR responses were obtained from the anal sphincters at 25mA current. A total of 13 transcranial electrical stimulations were performed over 40 minutes from the time of positioning to 20 minutes after the start of surgery to record

DOI https://doi.org/10.1055/s-0043-1776786.
ISSN 2348-0548.

Thieme Medical and Scientific Publishers Pvt. Ltd., A-12, 2nd Floor, Sector 2, Noida-201301 UP, India

the MEP. Seizure episodes occurred after the 12th MEP stimulation.

Twenty minutes after the start of surgery, the BIS suddenly increased to 73 from a baseline of 51 and remained high despite increasing the depth of anesthesia with a propofol bolus and increasing the propofol infusion from 200 to 250 µg/kg/min. During this period, high-amplitude, high-frequency electroencephalographic (EEG) waveforms were noted on the BIS monitor. As there was electromyogram (EMG) contamination, we could not appreciate the spike activity. As intense pain stimulation can cause β arousal on EEG, a bolus of fentanyl (1 µg/kg) was administered. Other causes of intraoperative seizures, such as hypoxia, hypercarbia, hypocapnia, and hypoglycemia, were ruled out. The surgeons and neurophysiology team were informed, cautery use was stopped, and the IONM stimulator and the monitor were turned off. The blowing of air by the forced-air warming device (3M Bair Hugger, St. Paul, Minnesota, United States) can also create an artifact (as it was placed over the upper body close to the head and neck); hence, it was also turned off. Despite these measures, the BIS continued to be high. No hemodynamic changes or abnormal movements were observed or felt in the trunks or limbs during this period. Examination of the face under the drapes revealed twitches over the forehead and both cheeks. The possibility of MEP stimulation-induced delayed focal seizures was considered, and 1 mg midazolam was administered. The BIS decreased to 35 to 40 for 5 minutes and then increased (70–73) with the reappearance of facial twitches. After administration of another 1.5 mg of midazolam, the BIS decreased to and remained between 35 and 45 till the end of surgery, with no further facial twitches. As the episode lasted for a prolonged duration (>15 minute), we decided to avoid further MEP stimulation and continue with root stimulation. The child was extubated at the end of surgery, and no neurological deficits were noted. The patient was observed for delayed seizures in the recovery room for 2 hours. The patient's postoperative course was uneventful, and he was discharged on the 5th postoperative day.

This correspondence highlights the variable presentation of intraoperative seizures following MEP stimulation. Intraoperative seizures involving only the face without truncal/limb movements in nonparalyzed children without a hemodynamic response have not been reported. The monitor graph trend (►**Fig. 1A** and **B**) revealed a normal heart rate and blood pressure response with isolated elevation of EEG and EMG values, followed by a moderate increase in end-tidal carbon dioxide (3–4 minutes after the EEG and EMG elevation), confirming the possibility of seizure-induced increased metabolic activity. Normalization of the BIS value to the baseline value after the administration of midazolam confirmed our diagnosis.

If a seizure occurs, whether to continue or discontinue MEP stimulation is based on the risk of a seizure versus the benefits of MEP monitoring in warning about possible central motor pathway injury.[4] In our case, we decided to abandon MEP stimulation and continue with the root stimulation because of prolonged seizure episodes. Similar to the present case, there have been reports of delayed seizures following MEP stimulation.

Certain patient-related and anesthesia-related risk factors have been implicated in intraoperative seizures during MEP stimulation.[4,5] Patients with epilepsy, cortical lesions, raised intracranial pressure, and those on proconvulsive medications are patient-related risk factors that can induce intraoperative seizures after MEP stimulation. Anesthetic-related risk factors, such as the administration of sevoflurane, ketamine, and etomidate, have been implicated in intraoperative seizures.[4–6] Propofol has convulsive and anticonvulsant properties.[6]

In this case, the BIS was within normal limits (40–45) after sevoflurane induction and the first 20 minutes of propofol infusion. Therefore, we ruled out the possibility of propofol or sevoflurane-induced seizure. We did not administer other

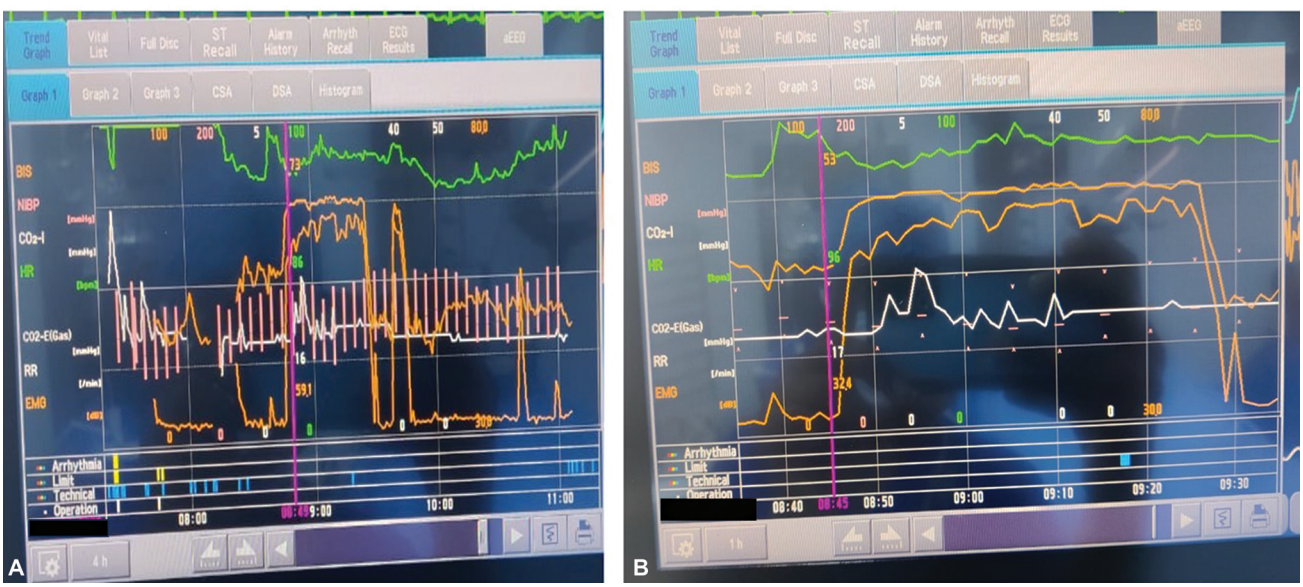

Fig. 1 (**A**) The vital trends *(blood pressure, heart rate, respiratory rate, end-tidal carbon dioxide)* and the processed electroencephalogram (EEG) and electromyogram (EMG) over a period of 4 hours; (**B**) The zoomed view of vitals and EEG and EMG during the seizure episode (20 minutes).

anesthetic agents that could trigger seizures and ruled out all physiological and metabolic causes of seizures. Hence, MEP stimulation was considered to be the cause of seizures in this case. A recent systematic review presented some recommendations for certain stimulation parameters, such as the number of pulses (1–9), number of trains (1, 2, or recurrent train stimulation), pulse duration (0.05–0.5 milliseconds), and interstimulus interval (1–5 milliseconds) based on the previous studies.[7] The intensity used in this case was from 100V to 130V. All the stimulation parameters were within the safe recommended thresholds.

All IONM monitors have an inbuilt facility to monitor raw EEG; monitoring raw EEG can help the neuroanesthesiologist diagnose and treat the intraoperative seizure (seizure spikes) as early as possible.

Conclusion

This case report highlights the variable presentation of seizures following MEP stimulation in a child undergoing excision of the fibroneural stalk and sectioning of the fatty filum terminale. Whenever IONM is used, we recommend using raw EEG for prompt diagnosis and treatment of intraoperative seizure. Vigilant monitoring, effective team communication, and prompt treatment of seizures helped to prevent morbidity.

Funding
None.

Conflict of Interest
None declared.

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