Loss of Transcranial Electric Motor Evoked Potentials during Pediatric Spine Surgery with Dexmedetomidine

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Case report: Intraoperative neurophysiologic monitoring using transcranial electric motor evoked potentials (TcMEP) has been increasingly utilized to reduce the risk of spinal cord injury during corrective spine surgery. Since inhalational anesthetic agents considerably depress TcMEP amplitude in a dose dependent manner (1,2), total intravenous anesthesia (TIVA) techniques with propofol as a central component have been advocated to optimize TcMEP monitoring during spine surgery (3). Due to its ability to decrease propofol requirements during TIVA, we often use dexmedetomidine, an alpha-2 agonist, as an adjunct to TIVA for procedures requiring TcMEP monitoring. We report a pediatric patient who experienced deterioration of TcMEP signal during intraoperative use of dexmedetomidine. 13-yr-old, 90 kg obese girl with kyphoscoliosis presented for T2 to L3 posterior spinal fusion. After inhalation induction, intubation was facilitated with administration of fentanyl 100 $\mu$g and propofol 100 mg. TIVA was initiated immediately after intubation with propofol 200 $\mu$g.kg$^{-1}$.min$^{-1}$, remifentanil 0.5 $\mu$g.kg$^{-1}$.min$^{-1}$ and dexmedetomidine 0.5 $\mu$g.kg$^{-1}$.hr$^{-1}$. No dexmedetomidine loading dose was given. One hour after starting TIVA (propofol, dexmedetomidine and remifentanil) the patient was placed in the prone position and baseline TcMEP was obtained (Figure 1). During the course of exposure over the next 1.5 hours, TcMEP amplitude from upper and lower extremities gradually decreased in size and eventually disappeared after two hours. By comparison, ulnar and posterior tibial nerve cortical somatosensory evoked potential amplitudes and latencies remained within baseline range, suggesting that the TcMEP changes did not reflect global iatrogenic injury to the spinal cord. The patient’s vital signs at the time of TcMEP disappearance were similar to baseline vital signs. The infusion rates of propofol, remifentanil, dexmedetomidine were 100 and 0.5 $\mu$g.kg$^{-1}$.min$^{-1}$, and 0.5 $\mu$g.kg$^{-1}$.hr$^{-1}$ respectively. The dexmedetomidine infusion was discontinued and TcMEP reappeared within approximately 30 minutes, gradually increased in size.

Discussion: TIVA has been increasingly used during spine procedures to provide optimal intraoperative neurophysiologic monitoring with minimal interference. We use dexmedetomidine as an adjunct to TIVA because of its sedative, analgesic and neuroprotective properties. Addition of dexmedetomidine to our TIVA regimen enabled us to reduce infusion rates of propofol. When the amplitude of TcMEP was totally lost, we went through the following differential diagnosis: equipment failure, direct injury of the spinal cord, decreased spinal cord perfusion and excessive doses of TIVA agents (propofol, remifentanil, dexmedetomidine). Neuromonitoring equipment were checked by neurophysiologist were functional. Surgical manipulation of the spine was excluded as a cause, as there was generalized loss of TcMEP, including in the upper extremities (Figure 1). Spinal cord hypoperfusion as a result of decreased cardiac output appeared unlikely as mean arterial pressures before and after loss of TcMEP were stable and similar. Excessive doses of TIVA agents were therefore to felt to be the reason for the loss of TcMEP amplitude. As remifentanil has minimal effects on TcMEP even at high infusion rates and the propofol infusion rate at the time of signal deterioration was relatively low (100 $\mu$g.kg$^{-1}$.min$^{-1}$), we elected to discontinue the infusion of dexmedetomidine. The return of TcMEP signal two hours after dexmedetomidine
was discontinued supports our impression that excessive dexmedetomidine was responsible for the deterioration in TcMEP signal.