Changes in Motor Evoked Potentials during an Anterior Spinal Fusion for a Thoracolumbar Idiopathic Scoliosis.

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**Introduction:** One of the most feared complications associated with scoliosis surgery is paralysis, which has a reported incidence of 0.25% to 4%. (1) To allow for the earliest detection of cord injury, intraoperative assessment of spinal cord function is required. Commonly used assessment tools include the Stagnara “wake up” test and neurophysiologic modalities such as somatosensory evoked potential (SSEPs) and motor evoked potentials (MEPs).

**Case Description:** A 15 year old, 60 kg female with a history of a progressive left thoracolumbar idiopathic scoliosis presented for an anterior thoracolumbar spinal fusion (T 10-L3) with segmental anterior spinal instrumentation. Monitoring included standard ASA monitoring, with invasive arterial pressure and central venous pressure monitoring. A thoracic epidural catheter was placed at T7-T8 interspace for postoperative analgesia. Induction of general anesthesia was performed using propofol, fentanyl and rocuronium. Anesthesia was maintained with remifentanil infusion and isoflurane at 0.4% with nitrous oxide 50% in oxygen. Neurophysiologic monitoring included both SSEPs and MEPs.

Surgery proceeded uneventfully, with laminectomies performed at all the disc levels between T10 and L3. Just prior to the placement of the stainless steel rotation rods, changes in the form of a decrease in the amplitude of the MEPs in the lower extremities bilaterally was noticed. SSEPs, however, did not manifest any changes compared to the baseline records. At that time, the mean arterial blood pressure (MAP) was in the 70’s (with no major changes compared to prior readings during the surgery). Oxygen saturation was 98% and esophageal temperature was 37°C. Volume expansion was started using intravenous crystalloids. The end tidal isoflurane was found to be 0.6% so it was decreased back to 0.4%. The MAP was increased to the 90’s. In spite of these measures, the MEPs showed no improvement. An arterial blood gas (ABG) checked few minutes before the incident showed a Hemoglobin (HB) of 8.9 gm/dl and a hematocrit of 28%. A Stagnara “wake up” test showed absence of right lower extremity movement. The patient was reanesthetized and one unit of packed red blood cells (420 cc) was transfused. Toward the end of the transfusion, the MEPs showed a marked improvement in amplitude. Repeat recheck ABG showed a HB of 9.8 gm/dl and a hematocrit of 30%. At the end of the surgery, the patient was coaxed awake.

A subsequent neurological examination showed no deficit. She was transferred to the Intensive Care Unit (ICU) in stable condition. In the ICU, epidural patient controlled analgesia (PCA) was started using a solution of bupivacaine 0.1% and fentanyl 2 mcg/cc, with no bolus given.

Two hours after the start of epidural PCA, the patient was unable to move her lower extremities. The mean blood pressure was in the 60’s at that time, oxygen saturation was 98%. A sensory level at T11 was demonstrated. Epidural PCA was discontinued and an IV PCA of morphine was started. A bolus of IV fluids was given. Methylprednisolone bolus followed by an infusion was started and dopamine infusion was titrated to maintain the mean blood pressure in the 80’s. An MRI was performed and did not show any acute changes. The patient did not show improvement, in spite of the above measures. Another ABG showed a HB of 9.3 gm/dl and a hematocrit of 29.3%. A second unit of PRBC (370cc) was given. The patient gradually started to improve. Near the end of the blood transfusion, lower extremities motor power returned to normal. Repeat recheck ABG showed a HB of 12.2 gm/dl and a hematocrit of 37.7%.

**Discussion:** SSEPs monitor the dorsal portion of the spinal cord, while MEPs monitor the anterior two-thirds of the spinal cord. It is possible to have ischemia of the anterior segment, which is responsible for motor function, without disturbing the posterior blood supply that lies dorsally and feeds the sensory tract.
All volatile agents produce a dose-dependent increase in cortical SSEP latency and reduction in the amplitude. (2) MEPs are abolished at therapeutic concentrations of the halogenated inhalational agent. Non anesthetic factors that affect intraoperative monitoring includes spinal cord blood flow, minute ventilation, HTC level, and body temperature. In a primate model, hematocrit levels of below 15% have shown to decrease SSEP amplitude and increase latency. Hematocrit data for MEPs are not available. (3) We responded to the decrease in the amplitude of MEPs by increasing the mean arterial pressure and reducing the anesthetic agents. Since the wake up test showed the presence of motor deficit, we transfused PRBCs. The patient responded to this intervention and the MEPs returned to baseline. During the postoperative period, the loss of motor power might have been multifactorial. However, the patient again responded to the transfusion of PRBCs. As a conclusion, we think the HTC threshold at which blood transfusion should be initiated in these cases may be higher than expected in presumably otherwise healthy teenagers.

Refs:
1. Mooney III JF et al., J Pediatric Orthopedic 2002
2. Clapcich AJ et al., Anesth Analg 2004