[NM-244] Anesthesia For Awake Craniotomy And Deep Brain Stimulation In Traumatic Brain Injury-Induced Hemi-dystonia

Jani J, Reddy S
Children's National Medical Center , Washington , District o, USA

Deep brain stimulation (DBS) is a neurosurgical technique aimed at improving functional neurologic disorders. These disorders, such as Parkinson’s disease, dystonia, and essential tremors, are not associated with obvious structural changes in brain anatomy. To facilitate the required neurophysiologic microelectrode recording (MER), DBS is commonly performed via an “awake” craniotomy as preserved brain function is essential for accurate lead placement.

Our patient was a 24 year old male with a history of traumatic brain injury (TBI) as an infant with resultant post-traumatic stroke, hydrocephalus, and hemi-dystonia. To facilitate initial head frame placement and CT imaging, a low-dose (0.5 mcg/kg/hr) dexmedetomidine infusion was initiated with additional boluses of midazolam and fentanyl as needed to ensure patient comfort while maintaining spontaneous respirations. The surgeon also infiltrated pin sites for the head frame with local anesthetic. Spontaneous respiratory effort was critical because, once the head frame was placed, access to the patient’s airway in order to assist or control ventilation would have been difficult. During this initial management, the patient neither become apneic nor experienced any pain.

After CT scan completion, the patient was transported back to the operating room where additional access lines were placed under the same low-dose dexmedetomidine infusion and the addition of a 100 mcg/kg/min propofol infusion. The patient was positioned, and after infiltration of the surgical site with local anesthetic, incision was made without any patient reaction to painful stimuli and with preservation of spontaneous respiration. Supplemental oxygen and end tidal carbon dioxide monitoring were available at all times via nasal cannula. During MER, the propofol infusion was discontinued, and the patient readily followed commands under the dexmedetomidine infusion. The only anesthetic complication encountered during the procedure was a decrease in heart rate greater than 20% of baseline values; the dexmedetomidine infusion was decreased to 0.3 mcg/kg/hr and a dobutamine infusion at 1-2 mcg/kg/min was implemented to prevent further bradycardia. Despite this, the patient maintained hemodynamic stability and continued to follow commands. Once MER was complete, the propofol infusion was restarted for surgical site closure.

There is currently a paucity of literature describing DBS for TBI-induced dystonias. Here we present one such indication for DBS and awake craniotomy. As the indications for DBS are expanding, the creation of a standard protocol guiding the complex anesthetic management for these cases will likely be of benefit. As safely used in our case, dexmedetomidine will potentially be a key anesthetic agent for these types of procedures as it causes minimal to no respiratory depression, maintains hemodynamic stability, and possesses analgesic properties with opioid-sparing effects.

References:

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