A Delayed Surgeon Is a Dismayed Surgeon

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Nitrous oxide, dexmedetomidine, somatosensory evoked potentials

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Abstract

This case discusses a pharmacodynamic interaction between dexmedetomidine and nitrous oxide, resulting in decreased amplitude signals of somatosensory evoked potential measurements.

Case

A resident on the second day of her neuroanesthesia rotation was posted on a case involving general anesthesia with somatosensory evoked potential (SSEP) monitoring for a C4-C6 fusion on an elderly female. The patient had a history of hypertension and coronary artery disease (CAD).

Induction of anesthesia with propofol, fentanyl, and succinylcholine was uneventful. Anesthesia was maintained with a dexmedetomidine infusion and 0.5 minimum alveolar concentration (MAC) sevoflurane. The pins were placed and the patient was positioned prone. Good-quality baseline SSEP signals were obtained. Two hours into the case, the neuromonitoring technician reported "we've lost the SSEPs," which was confirmed by the covering neurologist. Physiologic and hemodynamic causes of SSEP loss were ruled out and retractors were removed without improvement in the SSEP signals. The surgeon feared spinal cord compromise and decided to perform a "wake up test" with the patient still prone in pins to rule out a true neurologic deficit.

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The anesthesia resident turned off the sevoflurane and decreased the dexmedetomidine infusion rate to allow the patient to be awake for a neurologic exam. The patient still had not emerged enough to follow commands 30 minutes later despite an undetectable level of end-tidal sevoflurane. The anesthesia attending was paged and quickly noticed the nitrous oxide flowmeter showed a 2:1 nitrous oxide-to-oxygen ratio. The embarrassed anesthesia resident admitted that he had turned on the nitrous oxide because he was afraid 0.5 MAC of an inhalational agent wasn't enough to prevent awareness and had forgotten it was on. He had also used nitrous oxide because he was concerned that using other inhalational anesthetics would decrease the blood pressure significantly below the baseline mean arterial blood pressure and he was worried about the patient's history of CAD and the risk for visual loss due to the prone position. The nitrous oxide was discontinued, the patient woke up and moved all extremities on command, and then was reanesthetized with sevoflurane. The surgical procedure resumed with an irate surgeon complaining loudly and at length. That evening the attending called the crestfallen resident to review the effect of inhalational anesthetics on evoked potentials (EPs) and reassure him that all anesthesiologists have been in the situation of wondering why the patient won't wake up and then realizing that the nitrous oxide is still on.

Discussion

SSEP monitoring is a noninvasive technique used to assess the integrity of the somatosensory system intraoperatively without having to perform a "wake up test." Continuous SSEP monitoring can warn a surgeon about impending neurologic injury and allow intervention before a permanent injury occurs. Common procedures that utilize SSEP monitoring includes spinal surgery, scoliosis repair, spinal fractures, intracranial procedures (aneurysms), and carotid endarterectomy. SSEPs monitor pathways supplied by the posterior spinal artery, thus it is still possible to miss motor tract injuries if SSEP alone are monitored.[1] For this reason, SSEPs are usually combined with motor evoked potentials (MEPs) to monitory anterior spinal artery pathways. Needles are placed in peripheral nerves and repeated electrical stimulations of 1 to 2 HZ are given. These electrical stimulations travel from the periphery to the central nervous system in a stepwise fashion via dorsal root ganglia, first order neurons (ipsilateral posterior column), second-order neurons (cross to contralateral side), medial lemniscus, thalamus, third-order neurons and sensory cortex. The electrical impulses can be monitored at several points along this circuit, with the potential sites generally classified as cortical or subcortical potentials.

The latency and amplitude of neuromonitoring signals are the most commonly utilized data. Signal latency is defined as the time from peripheral electrical stimulation to peak voltage at the measurement site. Amplitude is the voltage measurement from the peak apex to the next trough. Changes in baseline amplitude and latency intraoperatively may be indicative of compromised sensory pathways. By definition,

a decrease in amplitude of >50% or an increase in latency of >15% is considered a significant change. SSEP amplitude and latency changes lasting for >15 minutes may lead to permanent neurologic changes. Typically, interventions in response to decreased amplitude or increased latency are aimed at improving spinal cord perfusion. Surgical retractors are often removed to improve blood flow. Physiologically, blood pressure and oxygen delivery are optimized. Often SSEP signal changes are a result of anesthetic drugs, therefore, anesthesia-related signal changes must be considered in the differential diagnosis.

Unfortunately for anesthesia providers, many drugs that are commonly used intraoperatively can negatively impact SSEP monitoring (see Table 24.1). Inhalational anesthetic agents all have a dose-dependent decrease in amplitude and increase in latency.[2] However, up to 0.5 MAC isoflurane is compatible with satisfactory SSEP signals.[2] Nitrous oxide decreases SSEP cortical amplitude with little or no effect on latency. At equipotent concentrations, nitrous oxide results in more profound changes in cortical SSEPs than any other inhalational anesthetic agent.[1] In the scenario above, it was the resident's use of nitrous oxide that lead to the SSEP signal changes and subsequent "wake up test."

Intravenous anesthetic agents can impact SSEP signals as well. Propofol minimally decreases SSEP signals and is often the anesthetic drug of choice in these types of cases. Etomidate has been shown to increase amplitude and latency, therefore, infusions of etomidate may help augment SSEP signals.[3] Similarly, ketamine has been demonstrated to significantly increase signal amplitude (no change in latency) and can be utilized to improve SSEP recordings.[4] Intravenous opioids such as fentanyl, remifentanil, and sufentanil cause minimal changes in latency and amplitude, making them attractive choices as part of a balanced anesthetic.[1] Because many anesthetic drugs can alter SSEP signals, maintenance of anesthesia should be kept as consistent as possible intraoperatively. Large changes in anesthetic management or boluses of drugs that effect SSEP should be avoided to prevent creating a delayed and dismayed surgeon.

Table 24.1 Effect of Anesthetic Drugs on Somatosensory Evoked Potential Parameters

Drug	Latency	Amplitude
Isoflurane	Increased	Decreased
Sevoflurane	Increased	Decreased
Desflurane	Increased	Decreased
Nitrous oxide	No change	Decreased (significant)
Etomidate	Increased	Increased
Ketamine	No change	Increased
Midazolam	No change	Decreased
Fentanyl/remifentanil	Increased (minimal)	Decreased (minimal)
Propofol	None	Decreased (minimal)

Take-Home Points

- SSEP monitoring is a minimally invasive technique for assessing the integrity of sensory patients in anesthetized patients.
- SSEP signals are affected by many drugs used perioperatively and care must be taken to avoid significant changes in anesthetic management intraoperatively.
- Administration of nitrous oxide will significantly decrease the amplitude of SSEP signals.
- Changes in SSEP amplitude and/or latency can be cumulative with coadministration of certain additional anesthetic agents.
- It is very easy to turn on the nitrous oxide and forget that it is on!

Summary

Interaction: pharmacodynamic
Substrates: dexmedetomidine and nitrous oxide
Site of action: sensory pathways involving dorsal root ganglia, first-order neurons (ipsilateral posterior column), second-order neurons (cross to contralateral side), medial lemniscus, thalamus, third-order neurons, sensory cortex
Clinical effect: additive decrease in amplitude of somatosensory evoked potentials

References

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