Neuromonitoring for Scoliosis Surgery

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KEYWORDS

• Anesthesia • Pediatrics • Scoliosis • Evoked potentials • Electromyography • TIVA • Propofol

KEY POINTS

- Somatosensory evoked potentials (SSEPs) are more sensitive to inhalation agents, with decreases in amplitude and increases in latency, compared with intravenous agents, such as propofol, ketamine, dexmedetomidine, and opioids.
- Ketamine and etomidate may be used to augment SSEPs.
- Motor evoked potentials (MEPs) are the modality of choice for monitoring motor tract function, are easily abolished by inhalational agents, and negate the use of full neuromuscular blockade.
- Patients with immature neural pathways or preexisting neuromuscular disease may have abnormal baseline SSEP recordings.
- Maintenance of adequate physiologic parameters for normal neuronal functioning is critical to intraoperative neuromonitoring (IONM) during scoliosis repair.

INTRODUCTION

The management of the pediatric patient presenting for scoliosis repair places many demands on pediatric anesthesiologists. These procedures are fraught with complications and require strict attention to acid-base status, hemodynamic fluctuations, coagulation, and temperature maintenance with constant neurologic monitoring to assess for neurologic injury to the spinal cord and nerve roots. Neurologic injury resulting in postoperative paralysis or sensory loss is an uncommon yet devastating and unpredictable complication of spine surgery.[1] The goal of IONM is to assess the integrity of neural pathways that may become compromised during a procedure from direct injury to the spinal cord or nerves during instrumentation, from excessive traction placed on the spinal cord, or from inadequate perfusion of the spinal cord. IONM facilitates the identification of neural irritation or injury along a time frame that allows

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for corrective anesthetic and surgical interventions. IONM also aids in defining the nature of the injury so that the surgical procedure may be completed while minimizing the risk of further neurologic injury.[2] In order to fully understand how anesthetic choices and management influence IONM during scoliosis surgery and how this may affect neurologic outcomes, it is necessary to understand how the various types of neurophysiologic monitors (SSEPs, MEPs, and electromyography [EMG]) provide an assessment of neuronal functioning, how individual anesthetic agents can affect each type of neuromonitoring technique, and how physiologic parameters can alter normal neuronal function. In doing so, it becomes evident that the anesthetic principles and considerations are similar for providing anesthetic care to adult and pediatric patients for scoliosis repair and that the immature neural development of young pediatric patients or those with preexisting neurologic deficits may render neurophysiologic monitoring more unreliable and sensitive to anesthetic techniques.[3]

BACKGROUND

Scoliosis is a multidimensional deformity of the thoracolumbar spine resulting from a lateral and rotational deformity of the spine that occurs at an incidence of 1% to 2%.[4] The degree of scoliosis is quantified by the Cobb angle, which is measured by the intersection of perpendicular lines extending from lines along the vertebral body at the superior and inferior margins of the spine deformity.

Scoliosis can be categorized as idiopathic, congenital, or neuromuscular. Idiopathic scoliosis can be further subdivided based on age of onset (**Table 1**). Despite extensive research, the cause and pathogenesis remain unknown, although leading hypotheses center on a multifactorial origin.[5,6] Adolescent idiopathic scoliosis is the most common variant seen, with an incidence of 1% to 3% in children aged 10 to 16 years. The vast majority of these patients can be managed with conservative therapy. Congenital scoliosis, a defect noted at birth that occurs from vertebral or costal maldevelopment, occurs in approximately 1 of every 1000 live births.[7] Animal studies have postulated that congenital scoliosis may be linked to maternal toxin exposure during fetal development. The rate of disease progression is rapid in the first 5 years of life and again during puberty, coinciding with stages of rapid spine growth. Neuromuscular scoliosis is commonly associated with patient conditions listed in **Table 1**. Because severity of symptoms is associated with progression of this disease, surgical correction is usually undertaken when the Cobb angle is greater than 50° in those considered skeletally mature and greater than 40° in those with skeletal immaturity to arrest progression.

Table 1
Scoliosis classification and associated conditions

Scoliosis Classification	Associated Conditions
Idiopathic	Infantile (0–3 y) Juvenile (4–10 y) Adolescent (>10 y)
Congenital	Bony deformity Neural tube defects
Neuromuscular	Cerebral palsy Poliomyelitis Muscular dystrophy Spinal muscular atrophy Neurofibromatosis

ANATOMY OF THE SPINAL CORD AND PATTERNS OF INJURY

The blood supply of the spinal cord is organized segmentally both along the longitudinal axis of the spinal cord as well as cross-sectionally. Longitudinally, paired posterior spinal arteries supply the posterior third of the spinal cord whereas a single anterior spinal artery supplies the anterior two-thirds of the spinal cord.[8] Longitudinally, the paired posterior arteries and the collateral circulation that exists from the subclavian and intercostal arteries provide some redundancy in blood flow for the posterior third of the cord, making the dorsal columns less likely to suffer ischemic insult. Segmentally, sulcal arteries branch from the anterior spinal artery and penetrate into the spinal cord to supply the gray matter of the anterior horn.[9] The blood flow through the anterior spinal artery is not continuous because the collateral circulation from iliac and intercostal arteries is widely variable. Watershed areas along the thoracic spine can be attributed to this lack of collateral circulation. Because of this flow variability along the anterior spinal artery, segmental medullary and radicular arteries arising from the aorta facilitate perfusion for the lower thoracic and lumbar spinal cord. The most significant of these medullary arteries is the artery of Adamkiewicz, which usually anastomoses with the anterior spinal artery between T8 and L3, and is the primary source of blood supply for the lower two-thirds of the spinal cord. As a result of this vascular anatomy, the thoracic spinal cord receives less overall blood supply than the cervical and lumbosacral regions, placing the thoracolumbar area at increased risk for hypoperfusion when manipulation of the spinal column or aorta occurs.[10]

Cerebral and spinal cord blood flow follow the same principles of autoregulation and response to hypoxia, hypercarbia, and temperature. Spinal cord perfusion is dependent on the arterial blood pressure minus the central venous pressure or the cerebrospinal fluid pressure, whichever of the latter two is higher.

Neurologic injury during spine surgery can occur from a multitude of causes and is the most concerning complication associated with repair. Injury can involve nerve roots as well as the spinal cord with a permanent deficit, such as quadriplegia, as one catastrophic outcome. A previous combined analysis by the Scoliosis Research Society and the EuroSpine in 1991 reported on 51,000 surgical cases and noted an overall injury occurrence of 0.55%.[11] Distraction of the spine accounts for the highest risk of spinal cord injury. Direct trauma from surgical manipulation, damage to vasculature with surgical exploration, and positional issues can also lead to spinal cord ischemia. Patient conditions associated with a higher incidence of neurologic injury include combined anterior and posterior repair, neuromuscular scoliosis, and significant kyphosis.[12–14] Other studies point to neurologic injury occurring at an incidence of 0.5% to 1% of all cases. A 2011 analysis of data submitted to the Scoliosis. Research Society puts the incidence of new neurologic deficit (NND) associated with spine surgery at 1%, with revision cases having 40% higher incidence of neurologic injury when compared to primary cases. Pediatric cases versus adult cases reported an approximately 60% higher incidence of NND (1.32% vs 0.83%). Cases with implants doubled the chance of developing a neurologic deficit in the perioperative period. The cohort with the highest rate of NND's at 2.5' were pediatric patients undergoing revision with implants.[15]

Positioning injuries for scoliosis repair can range from isolated neuropathies along the extremities to quadriplegia, with one study reporting a prevalence of ulnar neuropathy at 6.2% with occurrence at a higher frequency related to prone positioning and in those whose arms were abducted greater than 90°.[16,17] The presentation of spinal cord injury can be varied given the separate blood supply (discussed previously).

Selective insult to the posterior blood supply can result in sensory deficits with intact motor function. Impaired anterior cord perfusion can result in flaccid paralysis with impairment in temperature and pain (spinothalamic tracts) but intact proprioception and sensation (dorsal columns) and is known as anterior spinal artery syndrome.[18]

Intraoperative neurophysiologic monitoring allows assessment of the integrity of the spine through the surgical period with real-time feedback to allow for interventions if needed, all with the goal of minimizing neurologic injury. All potentials are graded on their respective amplitudes, latencies, and shape. With respect to injury, expected neurophysiologic findings center on a decrease in amplitude potentials and increase in latency caused by decreased impulse transmission from damaged axons. Isolated latency changes are rare and are usually associated with hypothermia and/or hypercarbia. Significant findings requiring intervention include unilateral or bilateral amplitude changes of greater than 50%.

The quest for appropriate spinal cord monitoring techniques dates back to the early 1960s when Harrington[19] introduced instrumentation to allow for correction of spinal column deformities. A retrospective analysis performed by the Scoliosis Research Society in 1974 found that from 1965 to 1971, neurologic complications occurred at a rate of 0.72%, with partial or irreversible injury occurring in 0.65% in this patient cohort.[13] This discussion is an overview of the commonly used intraoperative monitoring techniques used today; readers are referred to several excellent reviews and studies cited in the References for more detailed information.[1–3,21,24,26,32]

WAKE-UP TEST

The wake-up test has historically been considered the first method to assess the functional integrity of the motor tracts during spine surgery and remains the standard for assessing global motor function. Developed by Stagnara and Vauzelle in 1973, anesthesia was reversed after implant placement and the patient was allowed to emerge intraoperatively from anesthesia with assessment of motor function in the lower extremities.[20,21] A major limitation of this form of testing is that although it can localize injury along the motor pathway, it can only do so for a single point in time.[22] An anesthetic technique tailored for rapid emergence should be a part of the anesthetic plan in those undergoing spinal fusion. A preoperative anesthetic consultation is imperative to decrease anxiety, to inform the patient and family about the details of the wake-up process, and to answer questions related to emergence, pain, and recall. Performance of the wake-up test first entails removing the anesthetic from the patient. The operating room environment should be made conducive to wake-up with minimized noise and activity. With indications of emergence, the anesthesiologist remains at the head of the bed asking the patient to follow a set of commands, such as “move your hands” or “move your feet,” with operating room personnel assessing the upper and lower extremities. Once the patient has followed the indicated commands, the anesthetic is then reintroduced.

The wake-up test is still performed today, although it occurs more commonly in the face of changing neurophysiologic findings. There seems little debate that SSEP and MEP changes likely correlate with compromised spinal cord function with much higher sensitivity and specificity than the wake-up test.[23] Risks associated with the wake-up test include the potential for recall, increased surgical time, and potential for accidental tracheal extubation. Risks can be further compounded by delay in wake-up, resulting in a potential increase in time from diagnosis to treatment in those with actual injury. Practically, the wake-up test may offer therapeutic benefit in patients with potential spinal cord compromise because the hemodynamic changes associated with

an intraoperative wake-up (increase in blood pressure) would have a positive affect on spinal cord perfusion.

SOMATOSENSORY EVOKED POTENTIALS

SSEP monitoring became widely adopted in the 1980s and is currently the mainstay for intraoperative monitoring during scoliosis repair (**Fig. 1**). Tamaki and Yamane[24] and Nash and colleagues[25] first reported its use in the late 1970s. SSEPs monitor the integrity of the dorsal column-medial lemniscus pathway, which mediates proprioception, vibration, and tactile discrimination. The dorsal column medial lemniscus pathway comprises afferent axons from the periphery, which ascend via the dorsal columns and synapse at the lower medulla, where they cross the midline and form the medial lemniscus. Second- and third-order neurons project from the thalamus to the primary sensory cortex. Pain and temperature are not mediated by this process and are instead mediated through the spinothalamic system. SSEPs involve

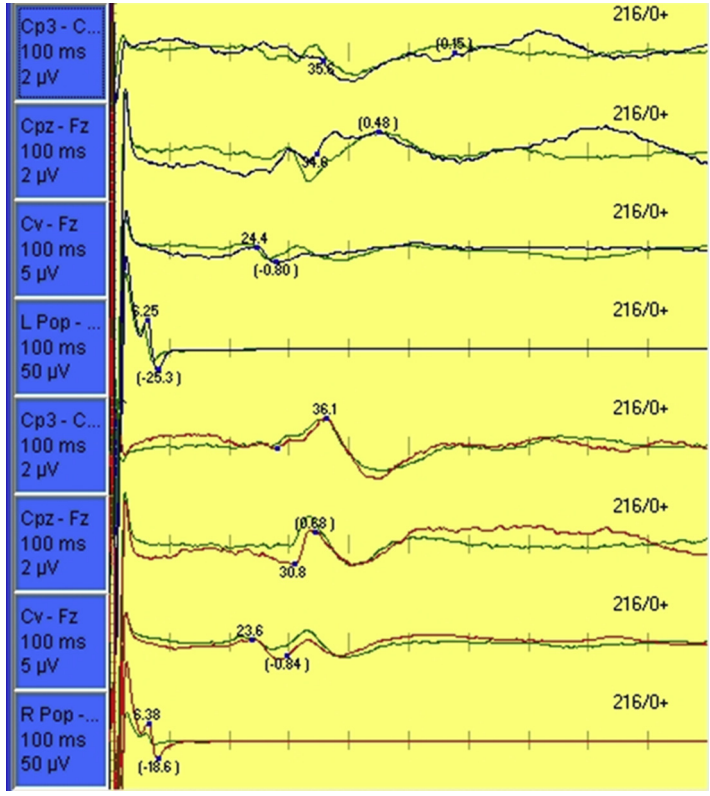

Fig. 1. SSEP recording. This recording was taken from the left side (blue tracings) and right side (red tracings) with control (green tracings). The total time of this trace is exhibited in the second line of each waveform and in this example is 100 ms. The abbreviations Cp3, Cpz, Cv, and L Pop refer to active electrode positions whereas C and Fz are the reference electrodes. 216/0 Refers to the average of 216 waveforms over the rejected waveforms during the time period measured (100 ms). The various voltages listed reference the sensitivity of the waveform. These numbers listed above are the latency associated with the examination whereas the numbers listed in parenthesis are the amplitudes.

stimulation of the peripheral nerve at fixed intervals distal to the surgical site, leading to signal propagation from the periphery to the primary sensory cortex. These cortical and subcortical signals are then recorded via scalp electrodes. The amplitude and latency of the responses are measured and then averaged with a comparison to baseline recordings to assess the potential for neurologic injury.[20] Changes are considered significant if the amplitude is decreased by more than 50% and/or the latency is increased by 10%.[26]

In addition, testing at the level of the brachial plexus can give insight into potential limb ischemia or nerve compression due to patient positioning, stretch injury to nerves, or during surgical manipulation. Any reduction of 50% in amplitude and/or a 10% increase in latency should cause personnel to investigate for potential neurologic defect.[20,26] Anesthetic agents have been noted to affect SSEPs (**Table 2**).[27,28]

Regarding safety and efficacy, a large multicenter study by Nuwer evaluated the efficacy of SSEP monitoring in diagnosing neurologic injury and found a statistically significant reduction in the total number of neurologic deficits (0.55% v 0.72%). He further pointed out that definite neurologic injury in the face of stable SSEPs occurred at a rate of just 0.063%. Although SSEP specificity in the detection of neurologic defects approaches 99%, a major limitation of SSEP monitoring is that this modality can only monitor the ascending dorsal columns. Specific patient conditions, such as neuromuscular scoliosis, cerebral palsy, and Down syndrome, have all been monitored reliably.

No information should be inferred on the integrity of the motor tracts or nerve roots from SSEP monitoring. Multiple reports of motor paresis after procedures with unchanged intraoperative SSEPs contributed to the search for other modalities to allow for improved intraoperative monitoring of the motor tracts of the spine.[23,29–32]

MOTOR EVOKED POTENTIALS

Mertin and Morton[33] revolutionized spinal cord monitoring in 1980 by demonstrating that single-pulse voltage applied transcranially could result in contralateral motor activity, marking the first time integrity of the corticospinal tract could be assessed. Translating these findings to the operating room was difficult given the

Table 2
Anesthetic agents and somatosensory evoked potential effects

Anesthetic Agents	Amplitude	Latency
Volatile agents • Isoflurane • Desflurane • Sevoflurane	↓↓	↑↑
Barbiturates	↓	↑
N_2O	↓	↔
Midazolam	↓	↔
Propofol	↔	↔
Dexmedetomidine	↔	↔
Opioids	↔	↔
Etomidate	↑	↔
Ketamine	↑↑	↔

Symbols: ↓, decrease; ↑, increase; ↔, no change.

exquisite sensitivity of this single-pulse technique to volatile anesthetics.[34] This difficulty remained until introduction of multiphase techniques in the mid-1990s. Incorporation of propofol into anesthesia practice was also occurring over this period.[35,36]

Currently, transcranial stimulation can occur via magnetic pulse or via electrical energy (**Fig. 2**). The multipulse technique relies on a train of 5 to 7 short successive electrical pulses applied over the scalp, causing pyramidal cell activation and summation at the anterior horn, resulting in alpha motor neuron firing of skeletal muscle.[36] Transcranial MEPs (TcMEPs) can be recorded at multiple levels. Direct (D) waves are recorded through epidural electrodes.[37] This recording of D waves is not routinely used because electrodes need to be placed into the epidural space. TcMEPs are recorded more commonly as compound muscle action potentials (cMAPs) via surface electrodes or subdermal needles placed in peripheral muscles.[38] Monitoring commonly occurs in adductor pollicus, adductor hallucis, and tibialis anterior. The control cMAP is taken in the upper extremity for comparison to the lower extremity cMAP. Determining which TcMEP changes are significant remains difficult because of the large variability seen in the response to stimulation under anesthesia.[39] The

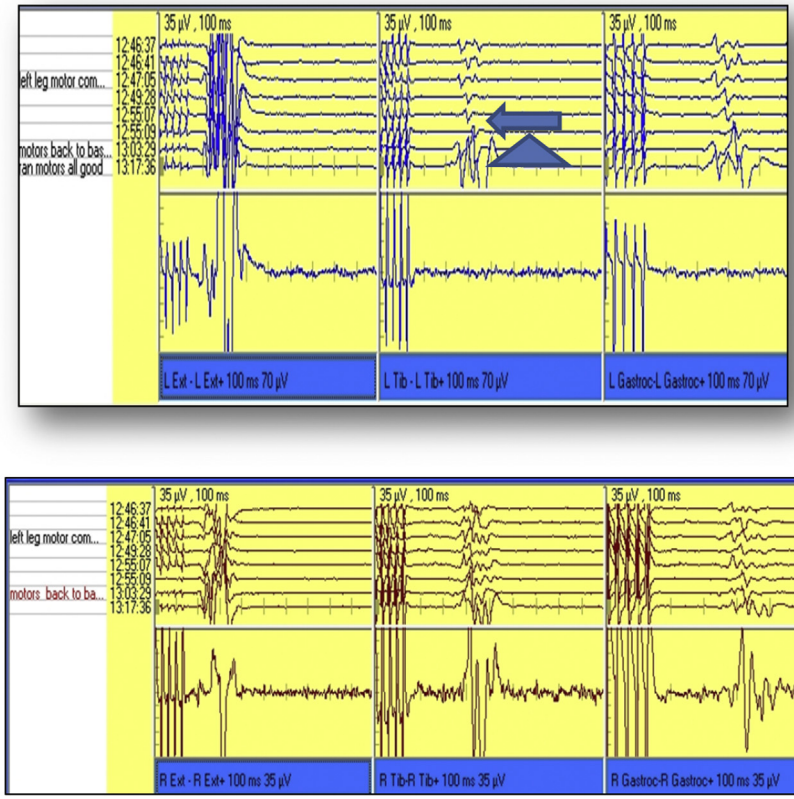

Fig. 2. TcMEP—exhibits a TcMEP tracing and associated change on the left tibial electrode (L tib) only signified by the arrow. Sensitivity of the waveform noted to be 35 µV over an examination time of 100 ms. The terminology, *R Ext – R ext*, refers to the active electrode versus the reference. TcMEP to baseline with intervention as noted by the triangle along the right side of the body. *Upper panel* is L side tracings, *Lower panel* is R side tracings.

most common methods for evaluation are using criteria similar to SSEP monitoring, where a threshold decrease signals potential injury, or evaluating TcMEP as all-or-none method.

Monitoring TcMEPs has several advantages. TcMEPs are exquisitely sensitive to spinal cord impairment and are able to detect spinal cord impairment an average of 5 minutes before SSEPs in a study by Schwartz.[23] TcMEPs are also sensitive to blood pressure changes given the blood supply to the anterior spinal cord. A major limitation with this monitoring modality is cMAPs' exquisite sensitivity to volatile anesthetics.[40] Other limitations include avoidance or limitation in the use of nondepolarizing muscle relaxants as well as the need for intermittent testing because patient movement makes operating conditions less than ideal.[41,42]

ELECTROMYOGRAPHY

Nerve root injuries are some of the most common neurologic deficits seen after scoliosis surgery, accounting for 65% of all NNDs.[11] SSEPs do not have the specificity or sensitivity to identify individual nerve root injury because they assess multiple nerve roots simultaneously. EMG assesses for potential nerve injury by electrical stimulation along the pedicle track or screw with placement of recording needle electrodes in specific muscles innervated by nerve roots (**Table 3**). A normal EMG has low-amplitude, high-frequency activity. EMG can be classified as free-running or triggered EMG.

Free-running and spontaneous EMGs are passive continuous EMGs and primarily used to map and assess nerve root function. Trauma to nerve roots causes depolarization with a subsequent muscle action potential in the muscles monitored. This sustained "burst" on the EMG is an asynchronous wave and can imply use of irrigation, contact of the nerve root, abrupt traction, and/or stretch injury. Long and sustained bursts imply nerve root irritation and potential risk for injury with the need for prompt action by the operative team (increasing blood pressure, release of distraction, and removal of hardware).[43]

Triggered EMG or stimulus-evoked EMG is primarily used to assess pedicle screw placement and cortical integrity of the vertebra. This is based on the principle that the conduction of an electrical stimulus between bone and soft tissue is relatively high. With cortical perforation, the resistance to the electrical stimulus drops significantly, resulting in cMAPs seen at very low voltage. Triggered cMAPS with unusually low voltage on EMG imply incorrect pedicle screw placement and the need for re-evaluation by a surgeon.[44,45] Pedicle screw malposition occurs in approximately 5% to 15% of cases.[46] In a large retrospective analysis of 1078 patients, Raynor and colleagues[47] found threshold levels less than 2.8 mA were 100% specific for cortical breech with sensitivity of 8.4%. Specificity decreased to 99% in those where the threshold was less than 4 mA but the sensitivity increased 4-fold. Current

Table 3
Electromyography nerve roots and corresponding muscles monitored

Nerve Root	Muscles Monitored
C8-T1	Adductor pollicis
T2-T6	Intercostals
T6-T12	Rectus abdominus
L3-L4	Vastus lateralis
L4-L5	Anterior tibialis
S1-S2	Gastrocnemius

recommendations include the use of EMG in conjunction with radiography and palpation for optimized pedicle screw placement.

Regarding anesthetic agents, EMG is resistant to their effects and, as such, there are few limitations to maintain adequate monitoring conditions besides limiting or avoiding neuromuscular blocking agents.

ANESTHETIC EFFECTS ON NEUROPHYSIOLOGIC MONITORING

Neurophysiology and its use in scoliosis repair provide multiple challenges for anesthesiologists attempting to ensure patient comfort and safety while providing an anesthetic that minimally affects monitoring techniques. The impact of anesthetics on neurophysiologic recordings cannot be overstated. All anesthetics depress synaptic activity and axonal conduction in a dose-dependent manner with prominent alterations seen in cortically generated responses.[48] The difference in the severity of the decreased amplitude and increased latency seen from anesthetics relate to an individual agent's lipid solubility, which has traditionally been considered a gauge of an anesthetic's potency. Generally speaking, increasing lipid solubility resulted in increased cortical depression.[28]

The neurophysiologic effects of the commonly used volatile anesthetics are summarized as follows. Isoflurane, sevoflurane, and desflurane all produce an initial excitation with increased alpha wave activity. With increased exposure, slowing occurs with eventual burst suppression noted. All halogenated inhalational agents produce dose-dependent decrease in amplitude and increase in latency for SSEPs with cortical responses affected to a larger degree than subcortical and peripheral nerve responses.[41] Although isoflurane is most potent given its lipophilicity, studies with sevoflurane and desflurane suggest similar effects on EEG and potential recordings. Doses up to 0.5 minimum alveolar concentration (MAC) can be used if subcortical responses are adequate, whereas the use of cortical SSEP recordings restricts use of these anesthetics.[41] With increasing concentrations of halogenated agents, a prominent effect on the anterior horn is noted with cMAP responses being eliminated.[49] Concentrations as low as 0.2 MAC largely abolish TcMEPs, relegating these agents suboptimal for use in cases where IONM is used.[40,49,50]

Nitrous oxide (N_2O) causes profound reduction in amplitude with increased latency in all neurophysiologic monitoring with suppression of cortical responses that mimic halogenated agents. Given its synergistic effects on SSEPs when combined with volatile anesthetics, use of this insoluble agent should be limited, although techniques with N_2O and opioids have been described.[35,51]

Intravenous opioids produce minimal depression of cortical SSEPs and TcMEP recordings. Studies have shown mild amplitude decreases and latency increases with opioids thought secondary to the action at the μ receptor via G protein–mediated activity, resulting in depressed electrical excitability.[41,52] Considering their minimal neurophysiologic effects and superior analgesic properties, an opioid-based anesthetic for scoliosis cases requiring monitoring seems beneficial.

Ketamine, via its N-methyl-D-aspartate receptor inhibition, and etomidate, via its γ-aminobutyric acid A ($GABA_A$) receptor inhibition, differ from halogenated agents in that they cause increases in cortical amplitudes of SSEP and MEP, making them agents of choice when monitoring responses to stimulation are difficult.[53,54] Ketamine provides superb analgesia and hypnosis, but its use must be weighed against potential dissociative effects and its effects on patients with intracranial pathology. Etomidate can be used as a constant infusion to enhance SSEP cortical recordings, but lack of analgesia, potential for enhanced seizure activity, and adrenal suppression are factors to consider with its use.[55]

Benzodiazepines and midazolam, in particular, can be used for cortical SSEP monitoring because only mild effects are seen when used at induction doses.[56] These agents also seem to have minimal effect on subcortical and peripheral responses. Use of benzodiazepines in TcMEP monitoring results in significant depression of motor potentials, necessitating caution if midazolam is used for induction.[56,57]

Propofol does not result in amplitude enhancement. This agent produces amplitude depression on induction with isoelectric EEGs seen in those given large doses. This is transient given propofol's rapid metabolism. This rapid metabolism makes propofol an excellent agent for total intravenous anesthesia (TIVA) and for rapid titration of anesthetic depth, thereby minimally effecting evoked potentials.[50]

Dexmedetomidine is a specific α_2-receptor agonist that provides anxiolysis and analgesia without depression of respiration. At clinically relevant doses, there is little effect on neurophysiologic monitoring.[58–60] This ability to minimally affect IONM in combination with its MAC-sparing ability for volatile agents makes dexmedetomidine an appealing adjunct when cases require IONM.[61,62] There are 2 reported cases of significant impairment on neurophysiologic monitoring because of dexmedetomidine's effect on TcMEP, but further analysis of the study revealed dosing well above the clinical recommendations.[63–65]

NEUROPHYSIOLOGIC CONSIDERATIONS FOR SPECIAL POPULATIONS

In neuromuscular scoliosis and associated conditions, such as cerebral palsy and Down syndrome, IONM is still possible although there is debate about its utility and reliability in patients with these conditions. SSEP monitoring has been successfully accomplished in this patient population with rates approaching 85% to 95%, but failure rates with TcMEP monitoring in those with cerebral palsy ranged from 40% to 60% based on the severity of their cerebral palsy.[66–69] Congenital scoliosis occurs at a time when a still developing nervous system may be encountered, potentially rendering IONM less reliable. Recent prospective data, however, document reliable and successful IONM in infants and young children using a TIVA technique.[70,71] When encountering scoliosis patients who fall outside the common diagnosis of idiopathic scoliosis, addition of ketamine and/or etomidate to the anesthetic plan should be considered to assist neurophysiologic monitoring.

SUMMARY

The intraoperative management of patients presenting for scoliosis repair presents many challenges for anesthesiologists. Along with normal intraoperative and perioperative concerns for a procedure that involves hemodynamic fluctuations, potentially large intraoperative blood losses, and long operating times in the prone position, there is the added challenge of providing an anesthetic regimen that permits neurophysiologic monitoring to assess for intraoperative neurologic compromise. Commonly used anesthetic techniques for scoliosis repair include combinations of opioid with propofol infusions to allow for SSEP and TcMEP monitoring. If volatile agents are administered, they should be used in low concentrations with communication to the neurophysiologist. Patients in whom there may be difficulty obtaining reliable intraoperative signals because of preexisting neurologic deficits or because of immature neurologic development may require the use of etomidate or ketamine infusions to improve SSEP amplitudes. Anesthesiologists taking care of these patients must have a comprehensive understanding of the effects of anesthetic agents on monitoring techniques, including SSEPs, TcMEPs, EMG, and the intraoperative wake-up test. Appropriate anesthetic regimens should allow for rapid emergence in case of the need to wake a patient

intraoperatively to assess neurologic function and should therefore use anesthetic agents that are known to have minimal effects on the monitoring technique used.

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