
Transcranial Motor-Evoked Potentials

2

Leslie C. Jameson

Key Learning Points

- The motor-evoked potential response (MEP) is an indirect complex polyphasic muscle response that requires a coordinated response of the motor neuron pathway and the muscle.
- Due to the motor pathway's blood supply, the MEP is more vulnerable to and a better indicator of adequacy of perfusion, particularly spinal cord perfusion.
- In addition to age, the ability to obtain MEP responses is impaired by pre-existing medical conditions (e.g., diabetes, hypertension, chronic spinal cord compression, spinal stenosis, nerve root injury, chronic hypoperfusion, brain injury, and genetic neuromuscular disease).
- MEPs are vulnerable to hypoperfusion and drug effects. Thus, the anesthesia caregiver is responsible for selecting an appropriate technique and maintaining adequate perfusion through maintenance of hemoglobin, blood pressure, and cardiac output.
- MEP change, loss or loss and recovery, has been shown to be a reliable predictor of immediate and long-term postoperative neurologic function.

Introduction

Motor-evoked potentials (MEPs) continue to be the most recent addition to routine intraoperative neurophysiologic monitoring (IOM). The importance of MEPs continues to expand primarily due to the ability to isolate perfusion-related neurologic function in the spinal cord. Initial reports of improved patient outcomes obtained with the use of somatosensory-evoked potential (SSEP) monitoring, primarily during scoliosis procedures in children and young adults, were quickly followed by case reports of isolated postoperative motor injury without SSEP or postoperative sensory changes. This reflected the reality of the anatomy and physiology of motor/sensory pathways in the brain and spinal cord [1]. MEP and SSEP pathways are located in different topographic and vascular regions of the cerebral cortex, brainstem, and spinal cord. MEP pathways are very complex and include the standard voluntary pyramidal and extrapyramidal networks. The more complex extrapyramidal network establishes additional motor connections including those to the cerebellum [2]. This complex and multiple synaptic architecture makes motor pathways more sensitive to ischemic insults than SSEP pathways [3].

Rare isolated motor injury without sensory changes after idiopathic scoliosis procedures was not the only driving force behind the widespread adoption of MEP monitoring. Increasing

L.C. Jameson, M.D. (✉)
Department of Anesthesiology, School of Medicine,
University of Colorado, 12401 E 17th Place,
Room 747, Aurora, CO 80045, USA
e-mail: leslie.jameson@ucdenver.edu

surgical volume and operative complexity in the central nervous system (CNS, spinal cord) and spine also fueled the need to independently assess motor function.

MEPs facilitate better intraoperative decision-making in all patient groups. As surgical techniques (instrumentation, diagnostic imaging, and intraoperative imaging) advanced and perioperative anesthetic management options improved, many patients who were at high anesthetic, surgical, and medical risk underwent new extensive surgical procedures. This increased risk of permanent and devastating neurologic complications. MEP monitoring became a favored method to help prevent complex surgical intervention from exceeding safe limits where the risk of the potential surgical adverse event exceeds possible functional gain [4]. New information suggests MEP monitoring, particularly in spine surgery, has a better correlation with good postoperative motor outcome than the use of SSEPs, and many experts advocate MEP monitoring for:

- Surgical correction of all axial skeletal deformities with instrumentation [5–8]
- Intramedullary spinal cord tumors [9–12]
- Intracranial tumors [13–15]
- CNS and spinal cord vascular lesions [16, 17]
- Seizure disorders [18]

MEP use continues to expand outside the area of neurosurgical and axial skeletal procedures to vascular procedures that put perfusion of the brain or spinal cord at risk like thoracoabdominal aneurysms, aortic arch procedures (both endovascular and open procedures) (see Chaps 39 and 40), and preemptive assessment of outcome in stroke [19–21].

Motor Pathway Blood Supply

To understand why MEPs provide essential information for surgical procedures where neural tissue perfusion is at risk, it is necessary to review the blood supply of the spinal cord and understand the relationship between ischemia, electrophysiology, and infarction. A detailed discussion is found in Chap. 40. The spinal cord is supplied by the anterior spinal artery (ASA) and

the posterior spinal arteries (PSAs). Spinal cord motor tracts are primarily supplied by the ASA, a vascular network that supplies the metabolically active anterior two-thirds to four-fifths of the spinal cord including the gray matter and anterior horn cells, all of which are more sensitive to ischemia [3, 22].

Both ASA and PSAs arise as branches of the vertebral arteries in the brainstem and then descend along the spinal cord providing perforators into the spinal cord. The ASA receives blood radicular arteries, which originate in the aorta [23]. Typically, there are three cervical and two thoracic arteries located at T2, 3 and T7-L4, with the Artery of Adamkiewicz (AA) providing about 75% of the blood supply to the anterior cord [3, 24]. The reduced number of radicular arteries, the increased distance traversed, and increased metabolic demand make areas of the spinal cord perfused by the ASA more susceptible to hypoperfusion. While axons are quite resistant to ischemia, the anterior cord contains many more cells and synapses, which explains the rapid changes seen in MEPs when inadequate perfusion occurs. Disruption of blood flow through these vessels due to mechanical or pressure changes rapidly leads to deterioration of MEPs and is used to prompt a change in management (e.g., improvement in systemic perfusion, cerebrospinal fluid drainage) [24–26].

The intracranial blood supply to motor areas is also vulnerable. Perforator arteries and lenticulostriate arteries supply the motor cortex and internal capsule; they arise from the middle cerebral artery. These vessels transverse a significant distance and are vulnerable to hypoperfusion with a decrease in cerebral perfusion pressure (CPP) from an increase in intracranial pressure (ICP) or cerebrospinal fluid pressure (CSFP) ($CPP = MAP - [ICP \text{ or } CSFP]$) or disruption of the source vessels (e.g., aneurysm or arterial-venous malformations (AVM)) or hypotension. The distance and caliber of these vessels creates a watershed area making motor function more vulnerable to hypoperfusion than the ascending sensory tracts [27, 28]. The normal spinal cord and brain will autoregulate blood flow to maintain normal perfusion. Autoregulation occurs with a CPP approximately between 50 and

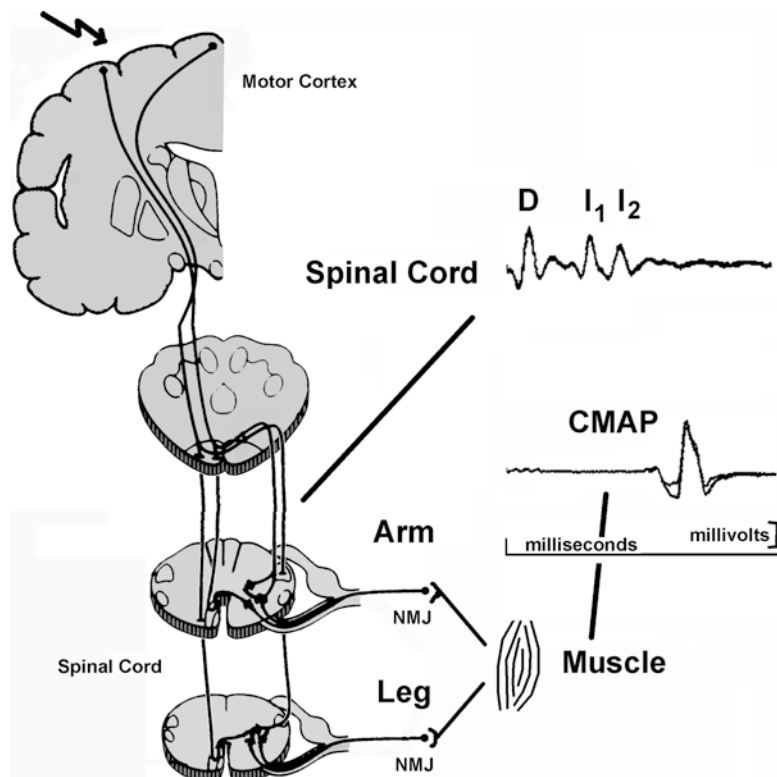
150 mmHg; specific individuals with long-term high (systemic hypertension) or low (infant) BP can be outside these limits. If the perfusion pressure falls below this range, autoregulation is lost and spinal cord blood flow is directly dependent on perfusion pressure. Hypoperfusion, as evident by a change in evoked potential activity, can also be caused by reductions in oxygen delivery (e.g., anemia, hypovolemia). MEP monitoring provides unique information about the functional status of the anterior spinal cord and internal capsule (see Chap. 21).

Technical Aspects of MEP Monitoring

MEPs are elicited by transcranial stimulation of the motor cortex using an electrical or a magnetic technique. The stimulation creates motor neuron depolarization and a descending response that traverses the corticospinal tracts and eventually generates a measurable response either in the form of muscle activity (compound muscle action poten-

tial, CMAP) or a wave propagation along the corticospinal tract (Direct wave or D wave) (Fig. 2.1). In humans, the exact structural connections that are activated by evoked potential stimulation have not been clearly defined. Structures involved in voluntary motor activity in animal models have been described. Recordings from deep brain (DB) electrodes used for stimulation and recording in patients being treated for epilepsy or movement disorders have led to better definition of motor transmission and its interaction with sensory function [29]. Use of magnetic stimulation, the only technique available for eliciting MEPs from awake humans, has allowed simulation to occur with simultaneous recordings of electroencephalography (EEG) and electromyography (EMG) as paired responses. These data suggest that the variability in MEP recordings is due to normal variations in inhibition and facilitation in the corticospinal and cortical pathways [30]. Much of the latency seen in MEPs is due to the slower conducting areas of the spinal pathway, which may explain the MEP sensitivity to hypoperfusion and anesthetic drugs (see Chap. 19) [31, 32]. Continued

Fig. 2.1 Depiction of the neurologic response pathway with motor-evoked potentials. Stimulation of the motor cortex (arrow) results in a response that is propagated through the brain and spinal cord to cause a muscle contraction. The response typically is recorded near the muscle as a compound muscle action potential (CMAP) or EMG. The response can also be recorded over the spinal column as a D wave followed by a series of I waves (high frequency repetitive discharges from the corticospinal fibers) (from Jameson and Sloan [33]; with permission)



investigation using DB electrodes for therapy in movement and seizure disorders will lead to a clearer picture of the motor pathways activated by diagnostic transcranial MEPs during surgery and may lead to a better understanding of the difficulties in eliciting responses.

All IOM MEP responses require continuity of the pathway since disruption of any component will change the measured response. Responses are affected by health of the neuron (e.g., peripheral neuropathy associated with diabetes), strength of the stimulus or number of neurons contributing to the response, propagation distance (height), sex, and temperature. Standard intraoperative transcranial electrical MEP monitoring in anesthetized patients uses a high-voltage electrical stimulus (measured in volts) to stimulate pyramidal cells of the motor cortex. This produces a wave of depolarization that is estimated to activate only 4–5% of the corticospinal tract. The motor pathway descends through the motor cortex, crosses the midline in the brainstem, and descends in the ipsilateral anterior funiculi of the spinal cord (Fig. 2.1) [2, 33].

Attempts to stimulate spinal cord motor tracts and then record neurogenic motor-evoked potentials (NMEPs) from peripheral nerves were done with stimulating electrodes placed into the epidural space (see Chap. 6) [8, 34]. An alternate, but less successful method was to use needle electrodes placed near the lamina of the appropriate spinal segment. Beginning in the 1990s, this technique for obtaining responses was instituted to eliminate the difficulties associated with the effects of anesthesia on the cerebral motor cortex when trying to elicit MEPs. NMEPs have largely been abandoned as a motor response since current evidence indicates that NMEPs are not mediated by the same motor pathways as MEP but instead by antidromic conduction in sensory pathways. Thus, NMEPs are not a motor response at all [32, 35]. Direct cortical or spinal cord stimulation using a strip electrode placed directly on the spinal cord or cerebral cortex to stimulate motor pathways continues to be used to map or identify neural tissue with motor functionality. A detailed treatment of spinal cord motor mapping techniques with grid electrodes is found in Chaps 9 and 36.

Table 2.1 Effect of varying the interstimulus interval (ISI) and the stimulus pulse duration on the threshold stimulus. Threshold stimulus, which can be in volts or mAmps (mA), is the energy required to produce a response in 50% of the patients

ISI (ms)	Pulse duration (ms)		
	0.1 ms	0.2 ms	0.5 ms
	Mean motor threshold (mA)		
2	158±67	105±33	76±26
3	140±55	97±33	64±20
4	126±56	91±35	61±19
5	179±74	120±45	83±31

Stimulus was applied at C3/C4. All combinations of ISI and pulse duration are significantly different from each other at the P value of <0.001. The lowest mean motor threshold occurred at an ISI of 4 ms and pulse duration of 0.5 ms (adapted from Szelényi et al. [36])

MEP stimulation utilizes a train of usually 3–7 electrical pulses of 100–500 V intensity (maximum 1000 V) applied through corkscrew electrodes most commonly placed a few centimeters anterior to the somatosensory recording electrodes at C3'–C4' (International 10–20 system). Standard stimulus pulse durations are 0.2 ms with an interpulse or interstimulus interval (ISI) (period between stimuli) between 2 and 4 ms (Table 2.1). Corkscrew scalp electrodes increase the electrode surface area and reduce the risk of burns from the high-energy stimulus. Manipulation in the number of stimuli, ISI, pulse duration, pulse strength or intensity, and stimulating electrode locations allows for adequate cortical neuron depolarization. Parameter changes overcome some of the impediments to propagation such as the anesthetic effect on the anterior horn cell synapse, preexisting neuropathy and myelopathy, distance of the motor cortex from the stimuli, loss of motor neurons, comorbid conditions, and age. The time required to obtain a MEP is generally less than 10 s. Multiple organizations have published best practice algorithms that in their hands produce the best signals [36]. ISI manipulation is frequently cited as a critical stimulus parameter to adjust to optimize MEP acquisition (Table 2.1) [7, 36, 37].

Once stimulation has occurred, a reliable and easily detected response is required for monitoring purposes. The response typically used is the CMAP recorded from muscle groups in the

extremities, although percutaneous epidural D and I waves [38], can be used to confirm a response (Fig. 2.1). D waves, direct activation of the corticospinal neurons [38], have a variable success rate and following them as a sole source of monitoring is currently uncommon except in specific surgical procedures such as intramedullary spinal cord tumors [39, 40].

Standard muscle responses differentiate laterality and therefore localize neural tissue at risk. These CMAP or EMG responses are recorded using needle or skin electrodes that are placed in hand muscles of the thenar eminence (abductor or flexor pollicis brevis), in muscles of the lower extremities (gastrocnemius, tibialis anterior, and abductor hallucis brevis), and trunk muscles (intercostals, rectus abdominis). The “best” (largest and most reproducible) specific muscle response below the site of the surgical procedure is selected to be followed [36, 40–46]. In our organization, acceptable CMAP responses are polyphasic with a consistent latency and an amplitude greater than 150–200 μV . We will continue to follow lesser responses but inform the surgeon that the information is not reliable. Direct motor mapping in the spinal cord or cerebral cortex requires needle placement in the muscle groups that are innervated by the areas being stimulated (e.g., homunculus hand representation—abductor or flexor pollicis brevis). This includes those muscles innervated by the cranial nerves (e.g., cranial nerve VII: orbicularis oculi or oris).

CMAPs can be difficult to obtain from patients at both extremes of age, elderly and young children. In addition to age, adults often have preexisting conditions such as diabetes, hypertension, chronic spinal cord compression, nerve root injury, chronic hypoperfusion, and axonal conduction changes that reduce CMAP responses [47]. Children, particularly those under 6 years, have an immature CNS that makes obtaining a motor response challenging [48]. CMAP responses can be difficult to obtain in procedures that are performed on patients with substantial neurologic deficits from preexisting brain injury (e.g., cerebral palsy) and genetic diseases that impair muscle function (e.g., Duchene muscular dystrophy, Charcot-Marie-Tooth). Recent com-

prehensive review articles address these issues and offer solutions to help the IOM team obtain signals [49]. Often the most critical decision in obtaining MEP responses, particularly in those with known neurologic, metabolic or muscular diseases, is the selection of the anesthetic management (see Chap. 19).

When spinal cord D and I wave responses are used, they do not differentiate laterality and D waves do not involve a synapse. The D wave correlates with the number of functioning fibers of the corticospinal tract responding to the stimulus. Thus, D wave amplitude changes have significance. D waves are more commonly used during intramedullary spinal cord surgery where recording electrodes are placed by the surgeon in the field [33, 50, 51]. Another alternate method of producing a motor response is the Hoffmann reflex (H-reflex). It is the electrical equivalent of the spinal cord reflex elicited by a tendon percussion knee jerk and monitors the sensory and motor efferent axons as well as the spinal gray matter and components of the reflex arc [50]. Discussion of this response is outside the scope of this chapter (see Chap. 8). CMAPs are by far the most common measure of the MEP response. The literature evaluating D, I, and H waves is very limited.

CMAPs can demonstrate considerable variability even in normal awake subjects [32, 52]. The variability is magnified during general anesthesia [31, 53]. Most organizations establish standardized criteria for a minimum baseline amplitude (difference between positive and negative peaks), complexity (number of positive and negative wavelets) but not latency (time from stimulus to response). This is necessary to prevent false-positive monitoring alert when the signal changes. Without these waveform components, a reliable signal was never present. It assures the surgeon that the MEP responses will be a reliable measure of function throughout the procedure. MEP responses are presented in Fig. 2.2. What constitutes a CMAP change that must be acted upon has not been universally defined. Permanent loss, a straightforward event, is strongly correlated with permanent neurologic injury, whereas patients who experience temporary loss or alerts (a predetermined decrease in

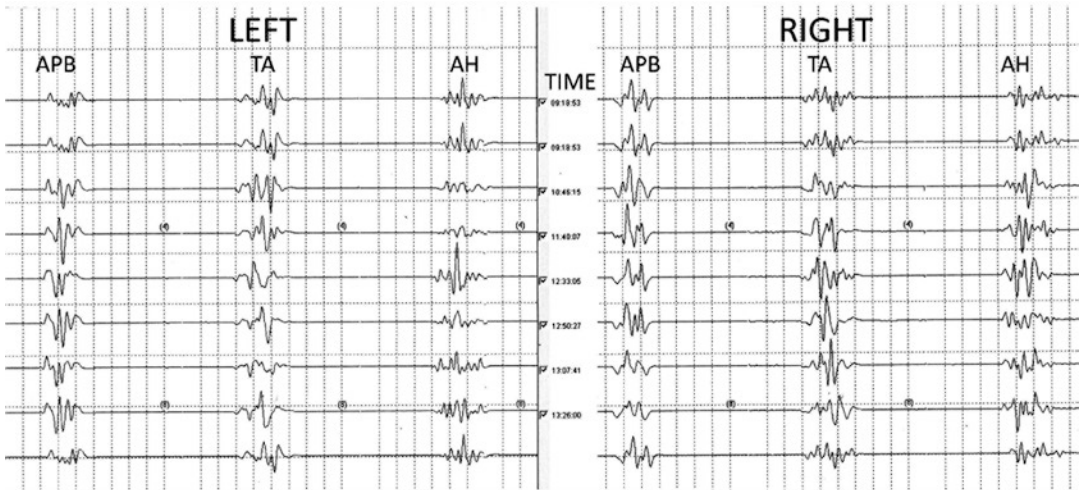
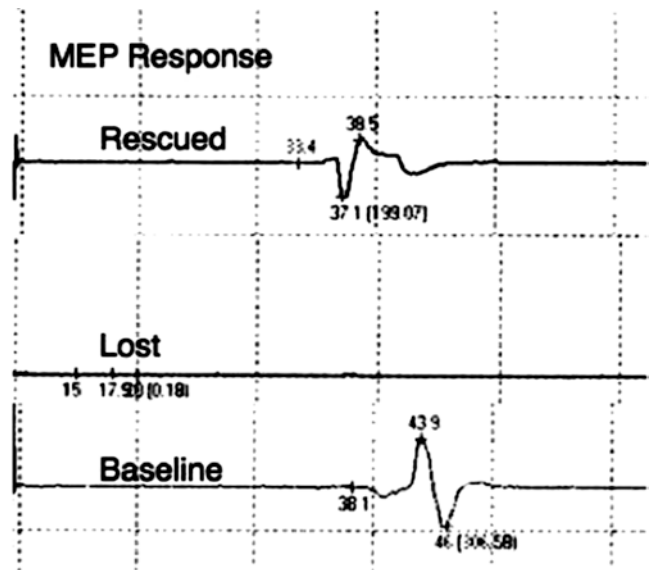


Fig. 2.2 Standard normal MEP responses. The CMAP response, a large polyphasic wave, is obtained from the upper extremity traditionally using the abductor pollicis brevis (APB) and from the lower extremity using tibialis anterior (TA) and abductor hallucis (AH) brevis. Two

lower extremity muscle groups are used due to the increased difficulty obtaining a consistent response particularly in adults. Other upper and lower extremity muscles can be used depending on the needs of the specific patient. Obtained from the author’s archive

Fig. 2.3 Normal MEP baseline responses and acute injury. Placing the patient prone resulted in the loss of responses. There was a recovery of response to baseline configuration after adjusting the head position, increasing blood pressure and eliminating residual desflurane. Obtained from the author’s archive



amplitude and/or latency) frequently have normal function at the end of the procedure and ultimately regain full motor function. Some IOM groups use the presence or absence of a CMAP response as their sole criteria for notifying the surgeon about a problem. This criterion allows the use of muscle relaxants as a component of the anesthetic, which is a common surgical request to eliminate patient movement at an inopportune

time. Other suggested criteria include increases in stimulus strength greater than 50–100 V, changes in stimuli number or pattern required to elicit an MEP, or a significant decrease in CMAP amplitudes (usually >80%) from the initial responses (without muscle relaxant). All are considered significant changes by some individuals (Fig. 2.3). Signal recovery after these changes is reassuring and usually predicts normal postop-

erative motor function. Loss of CMAP responses requires notification of the surgeon and anesthesiologist to correct, when possible, the physiologic issues contributing to the MEP change (see Chap. 20) [1, 54–59].

Application of MEP Monitoring

Monitoring during structural spine and spinal cord surgery is customarily multimodal and includes SSEPs, MEPs, and electromyography (EMG, free running, and stimulated). MEP monitoring is considered essential whenever spinal cord function is at risk. Thus, MEP monitoring is usually performed during structural spine surgery from C1 to cauda equina whenever the risk of cord injury due to stretch, compression, or vascular damage [56, 60] could occur. “At risk” situations also include any surgery where a compromise of spinal cord perfusion or direct injury to motor tracks or nerve roots could occur. Consensus opinion is that the evidence supports MEP monitoring in the following specific spine procedures:

- Spinal deformities with scoliosis greater than 45° rotation
- Congenital spine anomalies
- Resections of intramedullary and extramedullary tumors
- Extensive anterior and/or posterior decompressions in spinal stenosis with myelopathy
- Functional disturbance of the cauda equina and/or individual nerve roots

However, the evidence does not meet the level 1 standard (large randomized, placebo-controlled, double-blind studies). The evidence is based on a large case series and meta-analysis (level 2, 3 evidence) where MEP changes predicted immediate postsurgical neurologic findings [1, 40, 54–56, 58, 59, 61–63]. A recent evidence-based analysis by the American Academy of Neurology and American Clinical Neurophysiology Society is strongly supportive of IOM in spine surgery [46, 56].

Obtaining MEPs remains challenging in some patient populations. They often require an altera-

tion in anesthetic management to obtain adequate waveforms—a point that necessitates negotiations between the anesthesiologist, surgeon, and IOM team. Many of the older prospective series used SSEPs and EMG but only rarely MEPs due to this issue. In one study of 1055 adult patients undergoing cervical spine surgery between 2000 and 2005, MEP studies were attempted and obtained in only 26 of 1055 patients due to the perceived difficulties [61]. These were the highest risk patients for spinal cord injury. With the current relatively routine availability of total intravenous anesthesia (TIVA) based on propofol, MEPs can be relatively easy to obtain (see Chap. 19). When used during spine procedures, MEPs had 100% sensitivity, 96% specificity, and a positive predictive value of 96% for postoperative motor changes [61]. Adults with cervical myelopathy had about a 12% incidence of only MEP alerts (no EMG or SSEP changes). These alerts were usually followed by resolution after alterations in anesthetic and surgical management occurred. Nonetheless, these authors believed that the MEP monitoring provided 100% sensitivity and 90% specificity [64].

MEP changes are relatively infrequent [7] in pediatric procedures. One group reported that in 172 pediatric spinal deformity corrective procedures, there were 15 intraoperative MEP alerts, all of which resolved with changes in management. None of the patients (MEP-alert and MEP-unchanged patients) had new neurologic deficits. This group concluded that MEP monitoring alone was adequate for spinal deformity surgery with a sensitivity of 1.0 and a specificity of 0.97. Patients with persistent MEP changes had immediate postoperative motor deficits. SSEP changes, when present, lagged significantly behind the MEP changes and often did not predict outcome [65]. For adults with spinal cord myelopathy, one of the diagnostic criteria includes changes in MEPs prior to surgical intervention; consequently, baseline studies, post anesthesia, and repositioning are strongly recommended [66, 67].

Consensus opinion supports and studies suggest that the use of intraoperative spinal cord motor mapping improves long-term motor function in intramedullary spinal cord tumor resection

(see Chap. 36) [68, 69]. The MEP is the only reliable monitor of motor pathways and is an early predictor of impending damage to the cord due to the precarious blood supply. For an anterior approach to an intramedullary spinal cord tumor resection, focal injury to the anterior spinal vasculature or motor tracts is generally detected only minutes after a hypoperfusion event; this is considerably faster than with SSEP monitoring alone [11, 68, 69].

During intracranial procedures, direct cortical stimulation is likewise used to map motor function and to delineate the demarcation between tumor and functional tissue. This can be performed using electrode strips, direct hand-held device, or a Penfield motor stimulation technique. Although the Penfield technique is often preferred in awake patients, the pulse train technique used for MEP is associated with less stimulation-related seizures and is more effective in producing CMAP responses during general anesthesia. The motor stimulation may replace or augment awake craniotomy procedures in the supratentorial area when eloquent areas (e.g., speech) or motor pathways (e.g., internal capsule, motor cortex, and premotor cortex) are at risk [70–72]. In large clinical studies, sensitivity and specificity are reported to be between 90% and 100%, respectively; however, in some reports, Broca's area had a reported specificity of only 64% and Wernicke's area of just 18% [73].

Excess stimulation strength can cause direct activation of structures at a distance from the stimulus location. Focal stimulation often involves manually stimulating portions of the cortex or inserting a strip electrode under the dura. The usual pattern of transcranial stimulation is performed at about 1/10th the MEP stimulus strength. A pattern of gradually increasing stimulus is applied. Recent large case reports have documented that MEP monitoring assists in delineating the edge between tumor cells and functioning neural tissue. In a study of 404 patients, all with a low-grade glioma, MEP mapping substantially reduced the number and severity of permanent motor deficits while increasing the number of total resections. One hundred of these 100 patients had temporary

motor deficits and only 4 patients had deficits (1%) remaining 3 months after surgery. Total or subtotal tumor resection was done in only 11% of patients prior to motor mapping but 69.8% after motor mapping was initiated [74]. A number of other groups have published similar reports and noted that the long-term outcome is significantly improved by more extensive tumor resection in both children and adults for all supratentorial tumors [72, 75, 76]. Neurologic injury to the posterior fossa can have devastating consequences. Motor mapping is an effective way to identify both tumor margins and safe resection zones, areas between cranial nerve nuclei, or entry zones into the floor of the fourth ventricle. Stimulation can be either transcranial or, more frequently, direct brainstem stimulation [77].

Intracranial aneurysms and arteriovenous malformations can result in areas of hypoperfusion during the endovascular embolization, resection, or temporary and permanent clipping. MEPs identify hypoperfusion in motor areas and adjacent areas perfused by vessels involved in the vascular lesion. Identification of MEP change followed by therapeutic intervention appears to substantially reduce permanent injury. Two large studies with 108 and 129 patients undergoing supratentorial aneurysm clippings found that in cases where MEPs were unchanged, none of the patients had deficits. One study confirmed adequate flow with MEPs and with microvascular Doppler ultrasonography. Both studies reported between 13 and 33% of patients had reversible MEP changes; these patients had no neurologic changes immediately after the procedure or had only transient neurologic changes from which they fully recovered. Patients with permanent MEP change (about 20%) had permanent neurologic deficits, some quite severe [16, 78, 79]. Small case series generally support these findings. The neurosurgical community has reported improved outcomes during aneurysm occlusion on basilar, vertebral, and middle cerebral artery aneurysms when using MEP monitoring. Publications report MEP changes occur rapidly and better reflect long-term outcomes when the involved vessels provide perfusion to motor pathways.

Contribution of Anesthesiology to Effective MEP Monitoring

Without the cooperation and support of the anesthesia care provider, producing MEP responses and detecting changes is not possible. Most treatment options, when MEP change occurs, are in the hands of the anesthesia caregiver. MEP change is not only initiated by surgical activity but by physiologic management and anesthetic drug choices. Any event that will impact neural function can impact MEP waveforms (see Chap. 19). This reality stresses the importance of the team effort, cooperation between the surgeon, anesthesiologist, and IOM technologist.

Hypotension is of particular interest since deliberate hypotension to reduce blood loss was once considered a management technique, particularly in the idiopathic scoliosis procedures in children and during aneurysm clipping. There is a growing appreciation that the presumed lower limit of autoregulation is not always adequate for tissues undergoing surgical stress [80]. Mean BP that is adequate for a young adult patient may not be adequate for an older adult with many coexisting diseases. Hence, increasing or maintaining systemic perfusion pressure effectively treats many impending hypoperfusion injuries (Fig. 2.4).

The acceptable lower limit for hemoglobin has come under question. Current recommendations by the blood banking community are to allow hemoglobin to be as low as 7 g/dL during acute blood loss, particularly in healthy patients [81]. However, anemia can be compensated only within the limits of the patient's physiologic ability to increase cardiac output to maintain local tissue perfusion. Neurologic tissue has a high metabolic demand and may have compromised perfusion due to pre-existing systemic disease (hypertension, vascular disease, poor cardiac output, surgical stress, and inflammation) as well as regional compression (spinal cord stenosis, surgical activity, position, acute injury). Thus the acceptable lower limit of blood pressure and hemoglobin is unlikely to be the same for all situations and is poorly predictable. MEP monitoring allows a functional assessment

of the combination of blood pressure and oxygen-carrying capacity. Consequently it assesses the adequacy of perfusion in specific patients under specific surgical conditions. When IOM signals deteriorate, increasing the systemic blood pressure to the patient's preoperative value or higher is the most common and most effective response the anesthesia care team can provide. Transfusion is also an effective therapeutic intervention when appropriate. Maintenance of "normal" physiologic conditions within the brain and spinal cord can be difficult but results in the ideal monitoring conditions and the best neurologic outcomes.

The impact of dexmedetomidine on MEP monitoring deserves special comment. Propofol infusion syndrome [82] is diagnosed primarily in pediatric patients and can prove fatal (see Chap. 19). Thus, substituting dexmedetomidine for propofol as the "recommended" TIVA hypnotic when IOM is required has been advocated. Early literature reports suggested that its use caused no negative physiologic effect or impairment in MEP monitoring [83–85]. Two recent carefully performed studies found a clinically and statistically significant attenuation in the amplitudes of MEPs when the targeted plasma concentrations of dexmedetomidine exceeded 0.6–0.8 ng/mL [83, 86, 87]. Another study in which dexmedetomidine was administered in combination with propofol was discontinued by the safety monitoring board. Reduction or loss of MEPs occurred in healthy pediatric spines when both drugs were used in any combination [83]. Dexmedetomidine also has a long context-sensitive half-life consequently wakeup times can be prolonged.

Risk of MEP Monitoring

MEP monitoring is not without risk. The US Food and Drug Administration has specified relative MEP contraindications. The most common concern was direct cortical thermal injury (kindling), but over the last 18 years only two cases of cortical thermal injury have been reported. In a 2002 survey of the literature,

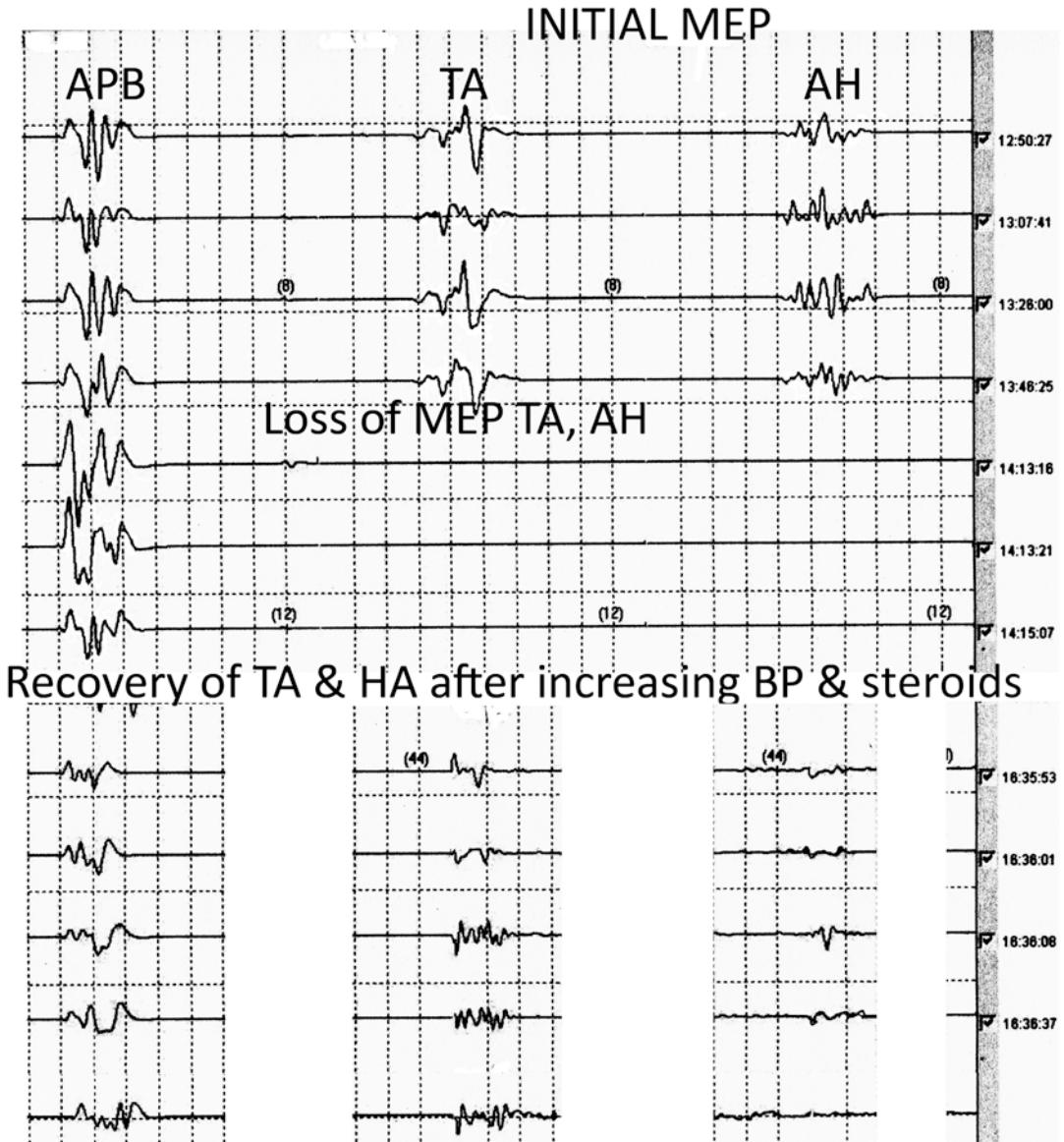


Fig. 2.4 Recovery of MEP responses after intraoperative loss. During a posterior cervical fusion of C5 to T4 the patient abruptly lost MEP responses in both lower extremities. After BP elevation and steroid administration, MEP responses returned only on the left. Patient

had weakness on the left, which resolved over 2 weeks. On the right, the patient had a dense hemiparesis that had not changed 3 months after surgery. (Tibialis anterior–TA, Adductor hallucis–AH) Obtained from the author’s archive

published complications included tongue laceration ($n=29$), cardiac arrhythmia ($n=5$), scalp burn at the site of stimulating electrodes ($n=2$), jaw fracture ($n=1$), and awareness ($n=1$) [88]. Placing a bite block between both molars can ameliorate tongue laceration. Notably no new-

onset seizures, epidural hematomas, or infections from epidural electrodes or movement injuries (e.g., surgical, joint dislocation), neuropsychiatric disease, headaches, and endocrine abnormalities have been reported. Relative MEP contraindications include epilepsy, a cortex

lesion, skull defects, high intracranial pressure, an intracranial apparatus (electrodes, vascular clips, and shunts), cardiac pacemakers, or other implanted pumps. The most common patient identified side effect is sore muscles [89, 90]. Needle placement will lead to bleeding and bruising at the insertion site. Infection is always possible. The prevalence of major and minor problems is astonishingly low.

Conclusion

Clearly the goal of intraoperative monitoring is to provide the greatest degree of assistance to the operative team for optimal intraoperative decision-making. The current literature suggests that MEP monitoring provides excellent specificity and sensitivity whenever motor tracts are involved. As such, the real question for consideration is which of the techniques available should be used to complement MEP monitoring in individual patients.

References¹

1. *Raynor BL, Bright JD, Lenke LG, Rahman RK, Bridwell KH, Riew KD, et al. Significant change or loss of intraoperative monitoring data: a 25-year experience in 12,375 spinal surgeries. *Spine*. 2013;38:E101–8.
2. Waxman S. Control of movement. In: Waxman SG, editor. *Clinical neuroanatomy* 27/E. 27th ed. New York: McGraw Hill Professional; 2013. p. 183–94.
3. Hickey R, Sloan TB, Rogers JN. Functional organization and physiology of the spinal cord. In: Porter SS, editor. *Anesthesia for surgery of the spine*. New York: McGraw-Hill; 1995. p. 15–39.
4. Fehlings MG, Houldon D, Vajkoczy P. Introduction. Intraoperative neuromonitoring: an essential component of the neurosurgical and spinal armamentarium. *Neurosurg Focus*. 2009;27(4):E1.
5. Pelosi L, Lamb J, Grevitt M, Mehdi SM, Webb JK, Blumhardt LD. Combined monitoring of motor and somatosensory evoked potentials in orthopaedic spinal surgery. *Clin Neurophysiol*. 2002;113(7):1082–91. Epub 2002/06/29.
6. MacDonald D, Zayed Z, Khoudair I, Stigsby B. Monitoring scoliosis surgery with combined multiple pulse transcranial electric motor and cortical somatosensory-evoked potentials from the lower and upper extremities. *Spine*. 2003;28(2):194–203.
7. Hsu B, Cree AK, Lagopoulos J, Cummine JL. Transcranial motor-evoked potentials combined with response recording through compound muscle action potential as the sole modality of spinal cord monitoring in spinal deformity surgery. *Spine (Phila Pa 1976)*. 2008;33(10):1100–6. Epub 2008/05/02.
8. *Minahan RE, Sepkuty JP, Lesser RP, Sponseller PD, Kostuik JP. Anterior spinal cord injury with preserved neurogenic ‘motor’ evoked potentials. *Clin Neurophysiol*. 2001;112(8):1442–50. Epub 2001/07/19.
9. Deletis V, Sala F. Intraoperative neurophysiological monitoring of the spinal cord during spinal cord and spine surgery: a review focus on the corticospinal tracts. *Clin Neurophysiol*. 2008;119(2):248–64. Epub 2007/12/07.
10. Yanni DS, Ulkatan S, Deletis V, Barrenechea IJ, Sen C, Perin NI. Utility of neurophysiological monitoring using dorsal column mapping in intramedullary spinal cord surgery. *J Neurosurg Spine*. 2010;12(6):623–8.
11. Morota N, Deletis V, Constantini S, Kofler M, Cohen H, Epstein FJ. The role of motor evoked potentials during surgery for intramedullary spinal cord tumors. *Neurosurgery*. 2010;41(6):1327–36.
12. *Sala F, Bricolo A, Faccioli F, Lanteri P, Gerosa M, Sala F, et al. Surgery for intramedullary spinal cord tumors: the role of intraoperative (neurophysiological) monitoring. *Eur Spine J*. 2007;16 Suppl 2:S130–9.
13. Mikuni N, Okada T, Enatsu R, Miki Y, Hanakawa T, Urayama S, et al. Clinical impact of integrated functional neuronavigation and subcortical electrical stimulation to preserve motor function during resection of brain tumors. *J Neurosurg*. 2007;106(4):593–8.
14. Neuloh G, Pechstein U, Schramm J, Neuloh G, Pechstein U, Schramm J. Motor tract monitoring during insular glioma surgery. *J Neurosurg*. 2007;106(4):582–92.
15. *Neuloh G, Bogucki J, Schramm J. Intraoperative preservation of corticospinal function in the brainstem. *J Neurol Neurosurg Psychiatry*. 2009;80(4):417–22.
16. Szelényi A, Langer D, Kothbauer K, De Camargo AB, Flamm ES, Deletis V. Monitoring of muscle motor evoked potentials during cerebral aneurysm surgery: intraoperative changes and postoperative outcome. *J Neurosurg*. 2006;105(5):675–81. Epub 2006/11/24.
17. Neuloh G, Schramm J. Monitoring of motor evoked potentials compared with somatosensory evoked potentials and microvascular Doppler ultrasonography in cerebral aneurysm surgery. *J Neurosurg*. 2004;100(3):389–99.
18. Neuloh G, Bien CG, Clusmann H, von Lehe M, Schramm J. Continuous motor monitoring enhances functional preservation and seizure-free outcome in surgery for intractable focal epilepsy. *Acta Neurochir (Wien)*. 2010;152(8):1307–14.
19. Corti M, Patten C, Triggs W. Repetitive transcranial magnetic stimulation of motor cortex after stroke: a

¹ Asterisks indicate key references.

- focused review. *Am J Phys Med Rehabil.* 2012;91(3):254–70.
20. Nascimbeni A, Gaffuri A, Imazio P, Nascimbeni A, Gaffuri A, Imazio P. Motor evoked potentials: prognostic value in motor recovery after stroke. *Funct Neurol.* 2006;21(4):199–203.
 21. Woldag H, Gerhold LL, de Groot M, Wohlfart K, Wagner A, Hummelsheim H. Early prediction of functional outcome after stroke. *Brain Inj.* 2006;20(10):1047–52.
 22. Waxman S. Spinal cord. In: Waxman SG, editor. *Clinical neuroanatomy* 27/E. 27th ed. New York: McGraw Hill Professional; 2013. p. 43–147.
 23. Crawford ES, Svensson LG, Hess KR, Shenaq SS, Coselli JS, Safi HJ, et al. A prospective randomized study of cerebrospinal fluid drainage to prevent paraplegia after high-risk surgery on the thoracoabdominal aorta. *J Vasc Surg.* 1991;13:36–45.
 24. Schurink GWH, Nijenhuis RJ, Backes WH, Mess W, de Haan MW, Mochtar B, et al. Assessment of spinal cord circulation and function in endovascular treatment of thoracic aortic aneurysms. *Ann Thorac Surg.* 2007;83(2):S877–81. discussion S90–2.
 25. Okita Y. Fighting spinal cord complication during surgery for thoracoabdominal aortic disease. *Gen Thorac Cardiovasc Surg.* 2011;59(2):79–90.
 26. Wan IY, Angelini GD, Bryan AJ, Ryder I, Underwood MJ. Prevention of spinal cord ischaemia during descending thoracic and thoracoabdominal aortic surgery. *Eur J Cardiothorac Surg.* 2001;19(2):203–13.
 27. Sakuma J, Suzuki K, Sasaki T, Matsumoto M, Oinuma M, Kawakami M, et al. Monitoring and preventing blood flow insufficiency due to clip rotation after the treatment of internal carotid artery aneurysms. *J Neurosurg.* 2004;100(5):960–2.
 28. Horiuchi K, Suzuki K, Sasaki T, Matsumoto M, Sakuma J, Konno Y, et al. Intraoperative monitoring of blood flow insufficiency during surgery of middle cerebral artery aneurysms. *J Neurosurg.* 2005;103(2):275–83.
 29. Ghitani N, Bayguinov PO, Vokoun CR, McMahon S, Jackson MB, Basso MA. Excitatory synaptic feedback from the motor layer to the sensory layers of the superior colliculus. *J Neurosci.* 2014;34(20):6822–33.
 30. Ferreri F, Pasqualetti P, Maatta S, Ponzo D, Ferrarelli F, Tononi G, et al. Human brain connectivity during single and paired pulse transcranial magnetic stimulation. *Neuroimage.* 2011;54(1):90–102.
 31. Firmin L, Muller S, Rosler KM. A method to measure the distribution of latencies of motor evoked potentials in man. *Clin Neurophysiol.* 2011;122(1):176–82.
 32. Tsutsui S, Yamada H, Hashizume H, Minamide A, Nakagawa Y, Iwasaki H, et al. Quantification of the proportion of motor neurons recruited by transcranial electrical stimulation during intraoperative motor evoked potential monitoring. *J Clin Monit Comput.* 2013;27(6):633–7.
 33. Jameson LC, Sloan TB. Monitoring of the brain and spinal cord. *Anesthesiol Clin.* 2006;24(4):777–91.
 34. Toleikis JR, Skelly JP, Carlvn AO, Burkus JK. Spinally elicited peripheral nerve responses are sensory rather than motor. *Clin Neurophysiol.* 2000;111(4):736–42.
 35. Amassian VE, Stewart M, Quirk GJ, Rosenthal JL. Physiological basis of motor effects of a transient stimulus to cerebral cortex. *Neurosurgery.* 1987;20(1):74–93.
 36. *Szelényi A, Kothbauer KF, Deletis V. Transcranial electric stimulation for intraoperative motor evoked potential monitoring: stimulation parameters and electrode montages. *Clin Neurophysiol.* 2007;118(7):1586–95.
 37. *Deletis V. Basic methodological principles of multimodal intraoperative monitoring during spine surgeries. *Eur Spine J.* 2007;16 Suppl 2:S147–52.
 38. Houlden DA, Schwartz ML, Tator CH, Ashby P, MacKay WA. Spinal cord-evoked potentials and muscle responses evoked by transcranial magnetic stimulation in 10 awake human subjects. *J Neurosci.* 1999;19(5):1855–62.
 39. Costa P, Peretta P, Faccani G. Relevance of intraoperative D wave in spine and spinal cord surgeries. *Eur Spine J.* 2013;22(4):840–8.
 40. Gavaret M, Jouve JL, Pereon Y, Accadbled F, Andre-Obadia N, Azabou E, et al. Intraoperative neurophysiologic monitoring in spine surgery. *Developments and state of the art in France in 2011.* *Orthop Traumatol Surg Res.* 2013;99(6 Suppl):S319–27.
 41. Fernandez-Conejero I, Deletis V. Transcranial electrical stimulation and monitoring. *J Neurosurg.* 2014;120(1):291–2.
 42. Joksimovic B, Damjanovic A, Damjanovic A, Rasulic L. Transcranial electric stimulation for intraoperative motor evoked potential monitoring: dependence of required stimulation current on interstimulus interval value. *J Neurol Surg A Cent Eur Neurosurg.* 2015;76(3):190–8.
 43. Ukegawa D, Kawabata S, Sakaki K, Ishii S, Tomizawa S, Inose H, et al. Efficacy of biphasic transcranial electric stimulation in intraoperative motor evoked potential monitoring for cervical compression myelopathy. *Spine (Phila Pa 1976).* 2014;39(3):E159–65.
 44. Yellin JL, Wiggins CR, Franco AJ, Sankar WN. Safe transcranial electric stimulation motor evoked potential monitoring during posterior spinal fusion in two patients with cochlear implants. *J Clin Monit Comput.* 2016;30(4):503–6 [Epub ahead of print].
 45. Kobayashi S, Matsuyama Y, Shinomiya K, Kawabata S, Ando M, Kanchiku T, et al. A new alarm point of transcranial electrical stimulation motor evoked potentials for intraoperative spinal cord monitoring: a prospective multicenter study from the Spinal Cord Monitoring Working Group of the Japanese Society for Spine Surgery and Related Research. *J Neurosurg Spine.* 2014;20(1):102–7.
 46. Ney JP, van der Goes DN, Nuwer M, Emerson R, Minahan R, Legatt A, et al. Evidence-based guideline update: intraoperative spinal monitoring with somato-

- sensory and transcranial electrical motor evoked potentials: report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology and the American Clinical Neurophysiology Society. *Neurology*. 2012;79(3):292–4.
47. Deiner SG, Kwatra SG, Lin H-M, Weisz DJ. Patient characteristics and anesthetic technique are additive but not synergistic predictors of successful motor evoked potential monitoring. *Anesth Analg*. 2010;111(2):421–5.
 48. Lieberman JA, Lyon R, Feiner J, Diab M, Gregory GA. The effect of age on motor evoked potentials in children under propofol/isoflurane anesthesia. *Anesth Analg*. 2006;103(2):316–21.
 49. *Sala F, Manganotti P, Grossauer S, Tramontano V, Mazza C, Gerosa M. Intraoperative neurophysiology of the motor system in children: a tailored approach. *Childs Nerv Syst*. 2010;26(4):473–90.
 50. Leppanen RE. Intraoperative monitoring of segmental spinal nerve root function with free-run and electrically-triggered electromyography and spinal cord function with reflexes and F-responses. A position statement by the American Society of Neurophysiological Monitoring. *J Clin Monit Comput*. 2005;19(6):437–61.
 51. *Jameson LC, Sloan TB. Neurophysiologic monitoring in neurosurgery. *Anesthesiol Clin*. 2012;30(2):311–31.
 52. Wassermann EM. Variation in the response to transcranial magnetic brain stimulation in the general population. *Clin Neurophysiol*. 2002;113(7):1165–71.
 53. Sloan TB. Anesthesia and the brain, does it matter? *Anesthesiol Clin North America*. 2002;20:1–27.
 54. Davis SF, Corenman D, Strauch E, Connor D. Intraoperative monitoring may prevent neurologic injury in non-myelopathic patients undergoing ACDF. *Neurodiagn J*. 2013;53:114–20.
 55. Avila EK, Elder JB, Singh P, Chen X, Bilsky MH. Intraoperative neurophysiologic monitoring and neurologic outcomes in patients with epidural spine tumors. *Clin Neurol Neurosurg*. 2013;115(10):2147–52.
 56. *Nuwer MR, Emerson RG, Galloway G, Legatt AD, Lopez J, Minahan R, et al. Evidence-based guideline update: intraoperative spinal monitoring with somatosensory and transcranial electrical motor evoked potentials: report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology and the American Clinical Neurophysiology Society. *Neurology*. 2012;78(8):585–9.
 57. Gavaret M, Trebuchon A, Aubert S, Jacopin S, Blondel B, Glard Y, et al. Intraoperative monitoring in pediatric orthopedic spinal surgery: three hundred consecutive monitoring cases of which 10% of patients were younger than 4 years of age. *Spine*. 2011;36(22):1855–63.
 58. Eager M, Shimer A, Jahangiri FR, Shen F, Arlet V. Intraoperative neurophysiological monitoring (IONM): lessons learned from 32 case events in 2069 spine cases. *Am J Electroneurodiagnostic Technol*. 2011;51(4):247–63.
 59. Malhotra NR, Shaffrey CI. Intraoperative electrophysiological monitoring in spine surgery. *Spine*. 2010;35(25):2167–79.
 60. Sutter M, Deletis V, Dvorak J, Eggspuehler A, Grob D, Macdonald D, et al. Current opinions and recommendations on multimodal intraoperative monitoring during spine surgeries. *Eur Spine J*. 2007;16 Suppl 2:S232–7.
 61. Kelleher MO, Tan G, Sarjeant R, Fehlings MG. Predictive value of intraoperative neurophysiological monitoring during cervical spine surgery: a prospective analysis of 1055 consecutive patients. *J Neurosurg Spine*. 2008;8(3):215–21.
 62. Sutter MA, Eggspuehler A, Grob D, Porchet F, Jeszenszky D, Dvorak J. Multimodal intraoperative monitoring (MIOM) during 409 lumbosacral surgical procedures in 409 patients. *Eur Spine J*. 2007;16 Suppl 2:S221–8.
 63. Eggspuehler A, Sutter MA, Grob D, Jeszenszky D, Porchet F, Dvorak J. Multimodal intraoperative monitoring (MIOM) during cervical spine surgical procedures in 246 patients. *Eur Spine J*. 2007;16 Suppl 2:S209–15.
 64. Kim DH, Zaremski J, Kwon B, Jenis L, Woodard E, Bode R, et al. Risk factors for false positive transcranial motor evoked potential monitoring alerts during surgical treatment of cervical myelopathy. *Spine (Phila Pa 1976)*. 2007;32(26):3041–6.
 65. Haghighi SS, Mundis G, Zhang R, Ramirez B. Correlation between transcranial motor and somatosensory-evoked potential findings in cervical myelopathy or radiculopathy during cervical spine surgery. *Neurol Res*. 2011;33(9):893–8.
 66. Wilson JR, Fehlings MG, Kalsi-Ryan S, Shamji MF, Tetreault LA, Rhee JM, Chapman JR. Diagnosis, heritability, and outcome assessment in cervical myelopathy: a consensus statement. *Spine (Phila Pa 1976)*. 2013;38(22S):S76–7.
 67. Wilson JR, Barry S, Fischer DJ, Skelly AC, Arnold PM, Riew KD, et al. Frequency, timing, and predictors of neurological dysfunction in the nonmyelopathic patient with cervical spinal cord compression, canal stenosis, and/or ossification of the posterior longitudinal ligament. *Spine (Phila Pa 1976)*. 2013;38(22 Suppl 1):S37–54.
 68. Quinones-Hinojosa A, Gulati M, Lyon R, Gupta N, Yingling C, Quinones-Hinojosa A, et al. Spinal cord mapping as an adjunct for resection of intramedullary tumors: surgical technique with case illustrations. *Neurosurgery*. 2002;51(5):1199–206. discussion 206–7.
 69. Cheng JS, Ivan ME, Stapleton CJ, Quinones-Hinojosa A, Gupta N, Auguste KI. Intraoperative changes in transcranial motor evoked potentials and somatosensory evoked potentials predicting outcome in children with intramedullary spinal cord tumors. *J Neurosurg Pediatr*. 2014;13(6):591–9.

70. Balogun JA, Khan OH, Taylor M, Dirks P, Der T, Carter Snead III O, et al. Pediatric awake craniotomy and intra-operative stimulation mapping. *J Clin Neurosci*. 2014;21(11):1891–4.
71. Ringel F, Sala F. Intraoperative mapping and monitoring in supratentorial tumor surgery. *J Neurosurg Sci*. 2015;59(2):129–39.
72. Bello L, Riva M, Fava E, Ferpozzi V, Castellano A, Raneri F, et al. Tailoring neurophysiological strategies with clinical context enhances resection and safety and expands indications in gliomas involving motor pathways. *Neuro Oncol*. 2014;16(8):1110–28.
73. Trinh VT, Fahim DK, Maldaun MV, Shah K, McCutcheon IE, Rao G, et al. Impact of preoperative functional magnetic resonance imaging during awake craniotomy procedures for intraoperative guidance and complication avoidance. *Stereotact Funct Neurosurg*. 2014;92(5):315–22.
74. Bertani G, Fava E, Casaceli G, Carrabba G, Casarotti A, Papagno C, et al. Intraoperative mapping and monitoring of brain functions for the resection of low-grade gliomas: technical considerations. *Neurosurg Focus*. 2009;27(4):E4.
75. Sanai N. Emerging operative strategies in neurosurgical oncology. *Curr Opin Neurol*. 2012;25(6):756–66.
76. Sanai N, Berger MS. Glioma extent of resection and its impact on patient outcome. *Neurosurgery*. 2008;62:753–6.
77. Morota N, Ihara S, Deletis V. Intraoperative neurophysiology for surgery in and around the brainstem: role of brainstem mapping and corticobulbar tract motor-evoked potential monitoring. *Childs Nerv Syst*. 2010;26(4):513–21.
78. *Szelényi A, Kothbauer K, de Camargo AB, Langer D, Flamm ES, Deletis V. Motor evoked potential monitoring during cerebral aneurysm surgery: technical aspects and comparison of transcranial and direct cortical stimulation. *Neurosurgery*. 2005;57(4 Suppl):331–8.
79. Neuloh G, Schramm J. Motor evoked potential monitoring for the surgery of brain tumours and vascular malformations. [Review] [126 refs]. *Adv Tech Stand Neurosurg*. 2004;29:171–228.
80. Edmonds Jr HL. Multi-modality neurophysiologic monitoring for cardiac surgery. *Heart Surg Forum*. 2002;5(3):225–8.
81. Goodnough LT, Levy JH, Murphy MF. Concepts of blood transfusion in adults. *Lancet*. 2013;381(9880):1845–54.
82. Marik PE. Propofol: therapeutic indications and side-effects. *Curr Pharm Des*. 2004;10(29):3639–49.
83. Mahmoud M, Sadhasivam S, Salisbury S, Nick TG, Schnell B, Sestokas AK, et al. Susceptibility of transcranial electric motor-evoked potentials to varying targeted blood levels of dexmedetomidine during spine surgery. *Anesthesiology*. 2010;112(6):1364–73.
84. Ansel DJ, Aherne A, Soto RG, Carrion W, Hoegerl C, Nori P, et al. Successful intraoperative spinal cord monitoring during scoliosis surgery using a total intravenous anesthetic regimen including dexmedetomidine. *J Clin Neurophysiol*. 2008;25(1):56–61.
85. Koruk S, Mizrak A, Kaya Ugur B, Ilhan O, Baspinar O, Oner U. Propofol/dexmedetomidine and propofol/ketamine combinations for anesthesia in pediatric patients undergoing transcatheter atrial septal defect closure: a prospective randomized study. *Clin Ther*. 2010;32(4):701–9.
86. Mahmoud M, Sadhasivam S, Sestokas AK, Samuels P, McAuliffe J. Loss of transcranial electric motor evoked potentials during pediatric spine surgery with dexmedetomidine. *Anesthesiology*. 2007;106(2):393–6.
87. Bala E, Sessler DI, Nair DR, McLain R, Dalton JE, Farag E. Motor and somatosensory evoked potentials are well maintained in patients given dexmedetomidine during spine surgery. *Anesthesiology*. 2008;109(3):417–25.
88. Legatt AD. Current practice of motor evoked potential monitoring: results of a survey. *J Clin Neurophysiol*. 2002;19(5):454–60.
89. *Macdonald DB, Skinner S, Shils J, Yingling C. Intraoperative motor evoked potential monitoring: a position statement by the American Society of Neurophysiological Monitoring. *Clin Neurophysiol*. 2013;124(12):2291–316.
90. Macdonald DB. Intraoperative motor evoked potential monitoring: overview and update. *J Clin Monit Comput*. 2006;20(5):347–77.

Questions

- Which of the following does NOT decrease the likelihood that MEP can be acquired in the OR
 - Very young age
 - Diabetes
 - Long-standing hypertension
 - Myelopathy
 - All of the above
- During surgery, MEP change in the tibialis anterior that is NOT resolved by the conclusion of surgery correlates with
 - Loss of proprioception in the feet
 - Loss of vibration sense in the hands
 - Loss of motor function in the leg
 - Loss of speech discrimination
 - Loss of visual acuity
- Which of the following has been associated with EMG monitoring?
 - Epidural D waves
 - H reflex
 - Stimulation of cranial nerve VII during an acoustic neuroma

- d. Stimulation of the posterior tibial nerve
 - e. Neurogenic motor-evoked potentials
4. Which of the following are associated with deterioration of MEP muscle responses during surgery?
- a. Inhalational anesthesia
 - b. Hypotension
 - c. Anemia
 - d. Administration of muscle relaxant
 - e. All of the above
5. Compared to SSEP
- a. MEP has the same vascular supply in the spinal cord
 - b. MEP has more synapses in the spinal cord than SSEP
 - c. MEP is supplied by the posterior spinal artery while the SSEP is supplied by the anterior spinal artery
- d. The MEP is less sensitive to ischemia in the spinal cord
 - e. All of the above
6. When MEP responses are lost during surgery, the most frequent rescue technique is
- a. Change to volatile anesthesia
 - b. Decrease blood pressure
 - c. There is no effective therapy
 - d. Increase BP to preoperative values or higher
 - e. None of the above

Answers

- 1. c
- 2. c
- 3. c
- 4. e
- 5. b
- 6. d