

Intraoperative monitoring of spinal cord function

A review

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McEwen et al. (1975) reported an incidence of 0.7 percent acute neurological complications following operative treatment of scoliosis. At that time Harrington instrumentation was mainly used. However, despite major technical advances in spinal instrumentation and the introduction of evoked potential monitoring during the last decade, the incidence of neurological complications following spinal surgery appears to have increased to 1.6 percent (Dawson et al. 1991). Lowe (1987) stated that the Cotrel-Dubousset system has a three times higher rate of neurological complications than Harrington instrumentation. Sublaminar wiring techniques have been associated with an even higher incidence of neurological complications (Johnston et al. 1986, Forbes et al. 1991). Many surgeons now agree that the risk of sublaminar wiring is unacceptably high for the average uncomplicated case of idiopathic scoliosis (Drummond 1991).

Several techniques are now available for monitoring the function of the spinal cord during surgery. Among these are the intraoperative wake-up test, somatosensory evoked potential monitoring, and, more recently, motor evoked potential monitoring. The aim of monitoring is to detect impending damage to the cord in a reversible stage, to allow corrective measures in order to prevent permanent neurological damage.

Wake-up test

Intraoperative awakening to test voluntary motor function during scoliosis surgery was developed by Vauzelle et al. (1973). The wake-up test is highly specific for intraoperative motor function. However, it has several major limitations. Since the wake-up test requires the cooperation of the patient, it cannot be performed in mentally retarded or very young patients. The wake-up test provides only a snapshot of motor function, and spinal cord injury may occur before final distraction is

applied, resulting in irreversible spinal cord damage at the time of the wake-up test (Wilber et al. 1984). The test can be repeated only a limited number of times. There has been concern for the risks associated with sudden movements by the patient, which could cause accidental extubation or dislodgment of instruments, while sudden deep inspiration may cause venous air embolism. Despite these limitations it is currently the only intraoperative test of voluntary motor function. 74 percent of surgeons responding in a survey made by the Scoliosis Research Society reported that they always performed either a wake-up test in conjunction with evoked potential monitoring or a wake-up test when evoked potentials showed abnormal sensory conduction (Dawson et al. 1991).

Somatosensory evoked potentials

The monitoring of somatosensory evoked potentials (SSEP) during spinal surgery was introduced by Nash et al. (1977) and it has become widely accepted for monitoring spinal cord function during these procedures (Dawson et al. 1991); SSEP are the electrical response of the central nervous system to stimulation of a peripheral nerve. They depend on intact conducting pathways between the site of stimulation and the site of recording. The traditional view is that SSEP from lower limb nerves are transmitted exclusively via the dorsal columns. Recent studies suggest that lateral column tracts may transmit some of the impulse volleys involved in generating the SSEP signal (Greenberg et al. 1987, Halonen et al. 1989).

SSEP representing the ascending volley can be recorded along the pathway from the sensory or mixed nerve, spinal cord, subcortical structures and cerebral cortex (Figure 1). The time between application of the stimulus and appearance of an EP peak is called latency; it is measured in milliseconds. Latencies

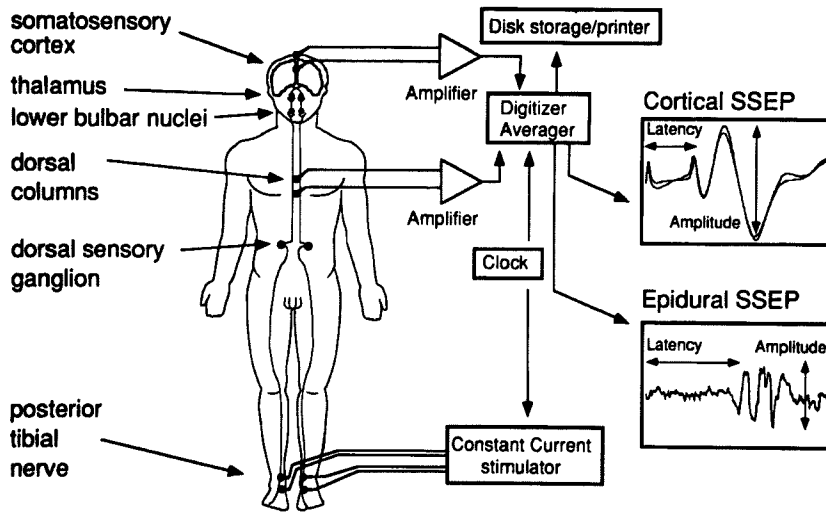


Figure 1. Equipment and electrode positions for intraoperative posterior tibial nerve evoked potential monitoring.

increase with height and age (Tsuji et al. 1981). After stimulation of the posterior tibial nerve at the ankle in normal subjects, the impulse reaches the sensorimotor cortex in about 40 msec. The cortical posterior tibial nerve SSEP shows its initial highest peak (P1) at 40 msec, which is followed by a negative peak (N1) with a latency of about 56 msec. Amplitudes can be measured from baseline to peak or from peak to peak. For spinal cord monitoring, peak-to-peak amplitudes are used, since they are less variable than baseline-to-peak measurements. The peak-to-peak amplitude of the first peaks of the P1-N1 primary cortical complex is highly variable among normal subjects, usually between 1 and 4 μ V. Epidural SSEP recorded at the upper thoracic level show a triphasic complex (Jones et al. 1983).

The principle of continuous spinal cord function monitoring with SSEP relies on the fact that changes in latency and amplitude indicative of spinal cord dysfunction occur early, while spinal cord injury is still in a reversible stage. There is ample data from experimental work in various injury or ischemia models that SSEP are sensitive indicators of impending spinal cord injury (Brodkey et al. 1972, Croft et al. 1972, D'Angelo et al. 1973, Kobrine et al. 1978, Griffiths et al. 1979, Dolan et al. 1980, Bennett 1983, Owen et al. 1989).

Intraoperative recording of SSEP

Stimulation sites and stimulation electrodes

To elicit SSEP for intraoperative monitoring periph-

eral nerves in the leg are stimulated. Usually the posterior tibial nerve at the ankle is used, less often the peroneal or sural nerve. It is also possible to stimulate the conus medullaris via an electrode inserted through an epidural catheter (Tamaki et al. 1981). Stimulation electrodes can be either subcutaneous needle electrodes or standard EEG disc surface electrodes, applied over the peripheral nerve. Subdermal needles have a constant electrical impedance. This allows a consistent stimulus delivery, which is important during long operations. Electrical stimuli used to elicit SSEP are usually square wave pulses of short duration (100–200 μ sec). The use of constant-current stimulators ensures constant stimulus delivery, even when electrode impedance changes over time.

Recording electrodes and recording sites

Standard silver-silver chloride surface electrodes filled with electrode jelly are used to record the signals from the different recording sites. Electrode impedance should be kept at a minimum (less than 2 k Ω) to prevent artifacts. Alternatively, stainless steel or platinum subdermal needles can be used for recording. Recording electrodes may be positioned along the ascending sensory pathway, in the popliteal fossa, the skin overlying the vertebral column, the neck and the scalp. The electrodes below the level of possible spinal cord injury serve as a check for appropriate stimulus entry into the sensory pathway. Cortical and subcortical SSEP are the most frequently used, because they are noninvasive and can be recorded preoperatively, intraoperatively and postoperatively. However, during the procedure, SSEP can also be recorded invasively from

electrodes placed in the epidural space (Forbes et al. 1991). These electrodes need to be positioned under direct vision by the surgeon, or inserted percutaneously before the operation via a Tuohy needle.

Signal processing

The amplitude of the EP signal is only a fraction of the background noise, consisting of electrical signals originating within the patient, such as EMG, ECG and EEG, and interference from electrical equipment present in the operating room. The signal from the recording electrodes is amplified and filtered to remove frequencies from the signal that do not contribute to the SSEP. Restricting the high-pass filter from 1 to 30 Hz reduces within-patient variability of intraoperative posterior tibial nerve SSEP, by removing low-frequency, high-amplitude components that do not contain information relevant to spinal cord conduction (Nuwer and Dawson 1984, Kalkman et al. 1991). Extracting the SSEP from the background noise is achieved by a signal-processing technique called signal-averaging. The principle of averaging is that the signal of interest is time-locked to the stimulus, while the background noise is random. Usually several hundred stimuli need to be averaged to extract the evoked potential waveform.

Influence of stimulus current and rate

Increasing the stimulus intensity increases SSEP amplitude. During anesthesia, SSEP amplitude increases linearly with increasing stimulus intensity until a plateau is reached at 20 mA (Nuwer and Dawson 1984). Cortical SSEP show a progressive decline in amplitude with increasing stimulus rates above 5 Hz (Nuwer and Dawson 1984). In most centers a stimulus rate between 3.1 and 5.1 Hz is employed during spinal cord SSEP monitoring. In patients with spinal cord injury of varying degrees, posterior tibial nerve SSEP could reliably be monitored only at stimulus rates of 1.1 or 2.1 Hz. Stimulation at a rate of 5.1 Hz had a markedly detrimental influence on amplitude in these patients (Schubert et al. 1987).

Artifacts

Various types of artifacts may be present in the SSEP waveform. Artifacts may originate within the patient, or arise from electrical sources outside the patient. These include stimulus artifacts from the current stimulator, line-frequency interference and artifacts caused by the use of diathermy. Diathermy can degrade an evoked potential within a few seconds. In general, artifacts should be eliminated at the source, rather than fil-

tered out. Measures to prevent SSEP artifacts in the operating room include the use of muscle relaxants, proper electrode preparation, grounding and positioning of other electrical equipment. Contamination of SSEP waveforms with diathermy artifact may be prevented by automatic interruption of the averaging process immediately before activation of the diathermy generator (Kalkman et al. 1991).

Influence of anesthetic agents and physiological factors on SSEP

In general, the administration of anesthetic drugs results in an increase in SSEP peak latencies and a decrease in peak-to-peak amplitudes. The changes associated with deepening anesthesia may be indistinguishable from spinal cord injury. It is therefore essential to be aware of the effects of various anesthetic drugs on SSEP in order to optimize signal strength. Changes in the level of anesthesia must be avoided to minimize variability.

Volatile anesthetic agents, e.g., halothane, enflurane and isoflurane, cause a marked, dose-related depression of amplitudes and latency prolongation of cortical SSEP. Waveforms recorded from the peripheral nerve are minimally affected, while epidural and subcortical peaks are relatively resistant to degradation by volatile anesthetics (McPherson et al. 1985, Salzman et al. 1986, Wolfe and Drummond 1988, Pathak et al. 1989). Nitrous oxide is a moderate depressant of SSEP amplitude, without effect on latency. However, it potentiates the depressant effects of volatile agents (Sebel et al. 1984, Sloan and Koht 1985). Provided that only low doses are used, SSEP monitoring is compatible with the use of halogenated anesthetics.

In contrast, *intravenous anesthetic drugs* have only minor effects on SSEP amplitude. SSEP can still be recorded when barbiturates are given in doses that produce a flat EEG (Drummond et al. 1987). An induction dose of the hypnotic propofol did not result in significant SSEP amplitude depression, and propofol/narcotic anesthesia produced higher amplitude SSEP than did nitrous oxide/narcotic anesthesia (Kalkman et al. 1991). *Etomidate* and *ketamine* increase SSEP amplitude (Kochs et al. 1986, Schubert et al. 1990). *Benzodiazepines* and *opioids* have minimal effects on evoked potentials (Schubert et al. 1987, Kalkman et al. 1988, Koht et al. 1988). *Muscle relaxants* have no direct effect on SSEP. However, by removing muscle artifact from the raw signal, they enhance the quality and reproducibility of intraoperative waveforms.

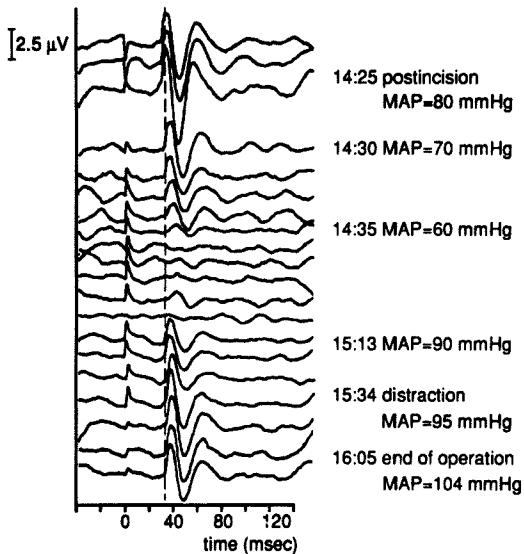


Figure 2. Loss of posterior tibial nerve SSEP during institution of controlled hypotension in a 14-year-old girl suspended in the Cotrel traction frame. The SSEP changes occurred before any spinal instrumentation had been performed. After reduction of traction and increase of mean arterial blood pressure, the SSEP waveform returned.

Physiological factors

Maintenance of patient temperature, adequate blood pressure, acid-base, and electrolyte status are important for minimizing SSEP variability.

Temperature. Hypothermia reduces conduction velocity in peripheral nerves and increases synaptic delay. Linear correlations were found between patient temperature and evoked potential latencies (van Rheineck Leyssius et al. 1986, Markand et al. 1990). Amplitudes of cortical SSEP did not change with moderate hypothermia during cardiopulmonary bypass (van Rheineck Leyssius et al. 1986).

Arterial blood pressure. Cerebral blood flow and spinal cord blood flow are maintained within narrow limits for changes in mean arterial pressure between 50 and 150 mmHg by autoregulatory mechanisms. A direct relationship between SSEP latency or amplitude and blood pressure has not been established in the uncompromised spinal cord, and there is no evidence that controlled hypotension to a mean arterial pressure of 60 mmHg in itself changes SSEP. However, when blood flow to the spinal cord flow is already compromised as a result of compression or distraction of the spine, the effect of hypotension becomes additive, which may result in SSEP abnormalities due to spinal cord ischemia (Brodkey et al. 1972, Griffiths et al. 1979, Grundy et al. 1982). Figure 2 shows a loss of

cortical posterior tibial nerve SSEP during institution of controlled hypotension in a 14-year-old girl undergoing Cotrel-Dubousset instrumentation for scoliosis.

Acid-bases status. Changes in end-tidal $p\text{CO}_2$ between 20 and 50 mmHg do not result in clinically significant alterations of cortical SSEP (Schubert and Drummond 1986, Kalkman et al. 1991).

Clinical experience with SSEP monitoring during spinal surgery

Spinal cord injury during surgery may be the result of mechanical injury due to distraction or compression during instrumentation, or of disruption of vessels critical to spinal cord blood supply, either by spasm or ligation. Many reports on SSEP monitoring from single centers, presenting from 55 to over 1200 patients each, have been published (Nash et al. 1977, Engler et al. 1978, LaMont et al. 1983, Brown et al. 1984, Tamaki et al. 1984, Whittle et al. 1984, Dinner et al. 1986, Veilleux et al. 1987, York et al. 1987, Bieber et al. 1988, Roy et al. 1988, Abel et al. 1990, Halonen et al. 1990, Forbes et al. 1991, Hicks et al. 1991, Kalkman et al. 1991).

Both cortical and epidural SSEP appear to be sensitive to changes in spinal cord function during spinal surgery. In some early series, no specific measures were taken after recognition of the SSEP changes, and these changes reliably predicted the occurrence of new neurological deficits (Tamaki et al. 1981, Grundy et al. 1982, Dinner et al. 1986). In most series, immediate measures were taken to restore spinal cord conduction after SSEP were lost, such as reducing distraction, removal of instrumentation and increasing blood pressure to restore blood supply to the spinal cord. This often resulted in the prompt return of SSEP and prevented postoperative neurological complications.

Criteria for intervention

When significant impairment of spinal cord function occurs intraoperatively, there is usually a rapid increase in latency paralleled by a decrease in amplitude, and eventually a total loss of SSEP. No prospective studies have been performed to assess the validity of criteria used for intervention. On empirical grounds it has been advocated that an increase in latency of more than 10 percent of the first cortical peak P1 or a decrease in cortical PIN1 peak-to-peak amplitude of more than 50 percent should be an indication for intervention (Brown et al. 1984, Nuwer 1986). These criteria are adhered to in most centers (Dawson et al. 1991). Unfortunately, several authors reported sponta-

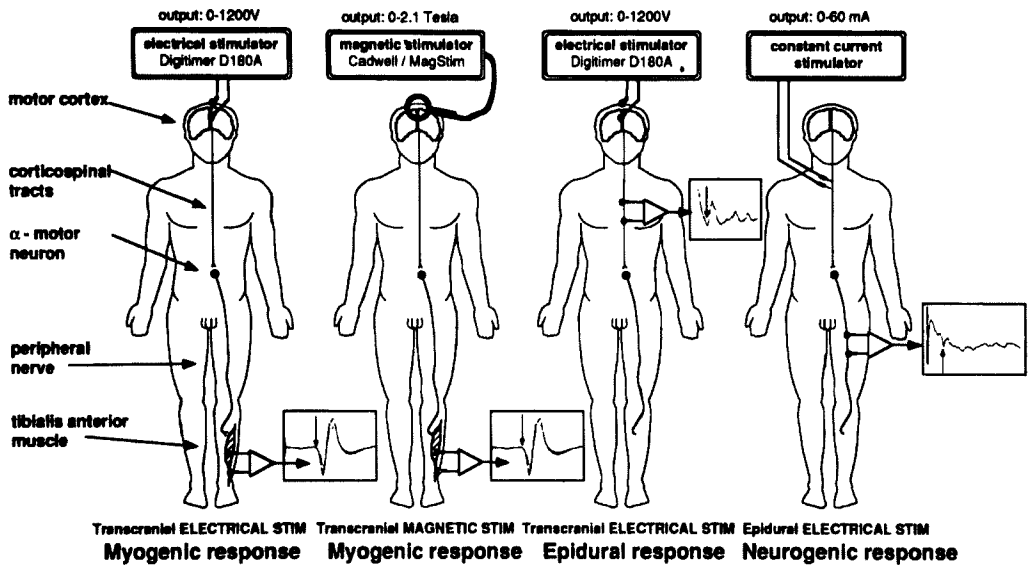


Figure 3. Equipment and electrode positions for various methods of intraoperative motor evoked potential monitoring.

neous amplitude variability of cortical posterior tibial nerve SSEP to be as high as 40–50 percent (York et al. 1987, Lubicky et al. 1989). In the individual patient, variability can be reduced by maximizing signal strength by selecting proper anesthetic regimens and high-pass filtering, to remove frequencies below 30 Hz (Kalkman et al. 1991).

False-positive and false-negative SSEP results

The incidence of false-positive results appears to be related to the experience of the monitoring team or an inability to maintain constant levels of anesthesia. Although the impact of false-positive results is not as serious as false-negative ones, they may cause anxiety to the surgical team and, possibly, incomplete surgical correction when instrumentation is removed. Strictly speaking, the occurrence of SSEP abnormalities during a spinal surgical case, with recovery of the signal and intact postoperative motor function following specific therapeutic measures, cannot be considered a false-positive result, since there is no objective way to ascertain whether the spinal cord actually was ischemic.

A false-negative SSEP result is the occurrence of a major neurological deficit, despite unchanged SSEP throughout the operation. Several case reports have been published describing false-negative results with SSEP monitoring (Ginsburg et al. 1985, Lesser et al. 1986, Ben-David et al. 1987). However, some of these reports have been criticized for their lack of reliable baseline responses or failure to monitor the pathway at

risk (Salzman et al. 1986, Young 1986, Friedman and Grundy 1987). Moreover, a neurological injury that evolves slowly in the postoperative period cannot be predicted by any form of intraoperative monitoring, since the monitor will only detect changes in spinal cord function while it is being applied. Nonetheless, the 1991 Survey of the Scoliosis Research Society revealed that 5 of 30 major neurologic deficits occurring after spinal surgery were not detected by SSEP monitoring (Dawson et al. 1991).

Motor evoked potentials

Since SSEP from lower extremity nerves are conducted primarily in the dorsal columns and probably also in the dorsolateral tracts, selective damage to the descending cortico-spinal motor tracts and anterior horn cells may go undetected when only sensory conduction in the spinal cord is monitored. Recently, several systems have become available for monitoring the motor pathways during surgery (Figure 3).

Transcranial stimulation of the motor cortex can be performed using specifically designed stimulators. Since the resistance of the skull is very high, conventional constant current stimulators are unable to deliver enough current to the motor cortex through the intact scalp. Transcranial electrical stimulation was introduced by Merton and Morton (1980). Brief high-voltage pulses (up to 1200 V) are delivered to the scalp via normal EEG electrodes. Transcranial mag-

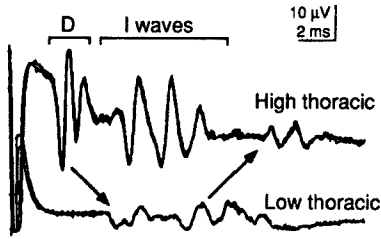


Figure 4. Motor evoked potentials to transcranial electrical stimulation recorded from the epidural space in a 14-year-old girl undergoing Cotrel-Dubousset instrumentation for scoliosis. The high thoracic electrode was positioned at the T1-2 level, the low thoracic electrode at T12-L1. Reproduced with permission from Hicks R G et al. Br J Anaesth 1992; 69: 130-136.

netic stimulation allows contactless stimulation of the motor cortex, by inducing a current in brain tissue underlying the magnetic coil. The motor response to both types of transcranial stimulation can be easily recorded as a compound muscle action potential (CMAP) from limb muscles using standard EMG techniques (Edmonds et al. 1989, Zentner 1989). This *myogenic* motor response has an amplitude of several millivolts in awake human subjects. The signal can also be recorded from the spinal cord before it traverses the lower motor neuron, using an electrode positioned in the epidural space (Boyd et al. 1986, Hicks et al. 1991). The epidural waveform consists of an initial direct or D-wave, followed by several indirect I-waves (Figure 4). The D-wave is the result of direct activation of pyramidal cells in the motor cortex, while the I-waves are the result of transsynaptic activation of the corticospinal tract by cortical interneurons (Burke et al. 1990).

Another approach to monitoring the motor pathways during surgery is to stimulate the spinal cord, using needles positioned in the base of the spinous process and record a neurogenic motor evoked potential (NMEP) from the sciatic nerve, either at the ischial tuberosity or in the popliteal fossa (Owen et al. 1991). The amplitude of the NMEP peak is about 1 µV, therefore it is necessary to average responses of 100 or more spinal cord stimuli to isolate the signal from the background noise. It is important to realize that the NMEP signal is not entirely specific for the motor tracts, since it contains components produced by antidromic conduction in sensory tracts (Su et al. 1992).

Motor Evoked Potential Spinal Evoked Potential T5 → Soleus Muscle T5 → L2

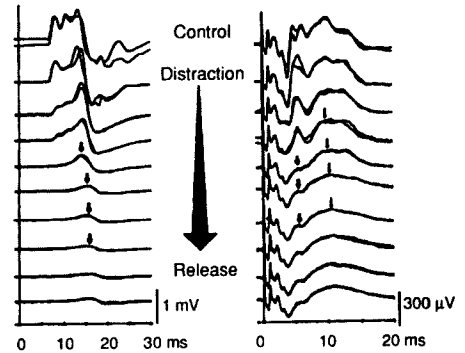


Figure 5. The effect of spinal distraction on myogenic and spinal cord responses to thoracic spinal cord stimulation in cats. Note the rapid disappearance of the muscle MEP and minimal change in spinal MEP. Reproduced with permission from Machida M et al. Spine 1989; 14 (7): 687-91.

Sensitivity of motor evoked potentials to spinal cord injury

Several experimental studies, using a variety of spinal cord injury models, have shown that MEP are extremely sensitive and specific for motor tract injury (Levy et al. 1986, Konrad et al. 1987, Fehlings et al. 1989). Unilateral transection of the cord with sparing of the dorsal columns completely abolished the compound muscle action potential (CMAP) to spinal cord stimulation on the ipsilateral side only. There is some evidence that MEP recorded directly from the spinal cord are less sensitive to spinal cord injury than MEP recorded from the peripheral nerve or muscle. Machida et al. (1988) showed that cross-clamping of the thoracic aorta in dogs above the level of the Adamkiewicz artery resulted in the rapid disappearance of the myogenic response to thoracic cord stimulation, but left the spinal cord response intact. The same authors studied the effect of spinal distraction in cats and observed that the myogenic response is more sensitive to over-distraction with or without derotation than the response recorded from the spinal cord (Machida et al. 1989; Figure 5).

Effects of anesthetics on MEP

Introduction of motor evoked response monitoring techniques in spinal surgery is hampered by the fact that MEP are extremely sensitive to depression by anesthetic drugs. The myogenic transcranial motor response is completely abolished even with very low concentrations of volatile anesthetic agents, such as isoflurane (Calancie et al. 1991, Kalkman et al. 1991; Figure 6).

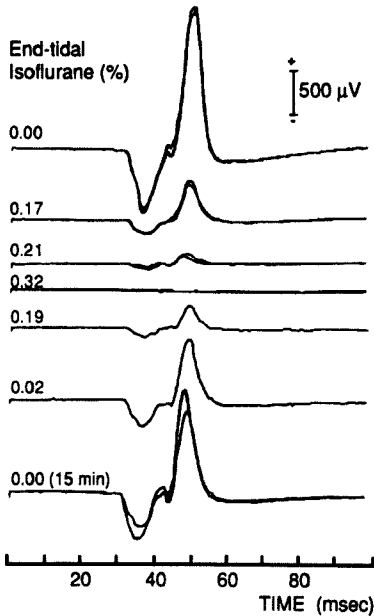


Figure 6. Effect of isoflurane on myogenic MEP to transcranial electrical stimulation in a patient anesthetized with fentanyl/nitrous oxide. Reproduced with permission from Kalkman C J et al. *Anesth Analg* 1991; 73: 410-415.

Nitrous oxide is also a powerful depressant of myogenic MEP (Zentner 1989). Nevertheless, most authors have reported modest success with MEP monitoring during a nitrous oxide/narcotic anesthetic technique. Intravenous anesthetic drugs, like the benzodiazepines, barbiturates and propofol, all produce marked depression of the myogenic MEP, while etomidate and ketamine have little or no effect (Ghaly et al. 1990a, Ghaly et al. 1990b, Kalkman et al. 1992). The neurogenic MEP to spinal cord stimulation is also depressed by propofol, suggesting that anesthetic drugs depress MEP not only at the motor cortex but also at the level of the spinal cord (Peterson and Mongan 1991).

Muscle relaxation. Ideally, myogenic MEP should be recorded in the absence of neuromuscular blockade. However, during surgery in the vicinity of the exposed spinal cord or nerve roots, sudden patient movement may be hazardous. Fortunately, it is possible to titrate the administration of muscle relaxants to such a level that contraction of muscles is greatly diminished while the recording of CMAPs from the leg muscles is still possible (Edmonds et al. 1989, Calancie et al. 1991, Kalkman et al. 1992).

In summary, it appears that anesthetic techniques based on nitrous oxide/narcotic and those employing etomidate or ketamine allow recording of reproducible

MEP, while the use of potent volatile anesthetics is contraindicated. There is some evidence that myogenic MEP to transcranial magnetic stimulation are more susceptible to the effect of anesthetic drugs than transcranial electrical MEP (Kalkman et al. 1992). This might be explained by the fact that magnetic stimulation activates motor pathways transsynaptically, while electrical transcranial stimuli can produce direct activation of corticospinal axons.

Techniques for improving MEP amplitude

The amplitude of myogenic MEP in awake subjects is increased several-fold by voluntary muscle contraction in the muscle under study (Ackermann et al. 1991). Unfortunately, this method of producing facilitation is not possible in the anesthetized patient. However, there are several approaches to increasing the MEP amplitude by increasing the level of excitatory input to the motor neuronal system. The application of paired stimuli with interstimulus intervals of 1-3 msec results in temporal summation of excitatory postsynaptic potentials at the α -motor neuron and produces a larger muscle response (Inghilleri et al. 1990). Another approach is to produce afferent conditioning of the MEP by applying sensory stimulation (500 Hz pulse train) to the segmental dermatome, corresponding to the muscle studied prior to delivering the stimulus (Kasai et al. 1992).

Clinical experience

Clinical experience with MEP is still limited to a few centers. Edmonds et al. (1989) recorded reproducible MEP to transcranial magnetic stimulation in 9 of 11 patients during scoliosis surgery. Zentner (1989) recorded transcranial electrical MEP during operations on the spinal cord and found good agreement between MEP changes (50 percent amplitude decrease) and postoperative neurological status. Jellinek et al. (1991) recently reported their experiences with MEP monitoring during propofol anesthesia. Although the responses were severely depressed by this anesthetic regimen, they were able to record reproducible responses, even in those patients with preexisting neurological deficits. Owen et al. (1991) have used NMEP monitoring in 300 cases. They reported that the NMEP were more reliable (less within-patient variability) than SSEP. Since there were no postoperative neurological deficits in the spinal surgery patients, the authors were unable to comment on the sensitivity and specificity of NMEP during spinal surgery. However, 5 of the 16 neurosurgical cases awoke with motor paraplegia, which was predicted by the loss of NMEP, but preservation of SSEP.

Conclusions

During the last decade, SSEP monitoring has become established as a reliable indicator of spinal cord dysfunction during spinal instrumentation. However, SSEP may produce false-negative results in patients with local compression or selective ischemia of the motor pathways. Monitoring conduction in descending motor pathways may provide useful additional information and prevent the occurrence of false-negative results of spinal cord monitoring. Before MEP monitoring on a routine basis can be advocated, several questions need to be addressed. What are the optimal stimulation and recording sites? How can we optimize intraoperative MEP amplitude and minimize within-patient variability? Which anesthetic techniques are compatible with the recording of myogenic or neurogenic MEP? Nonetheless, it is highly likely that the addition of one of the currently available MEP modalities will complement SSEP monitoring, and thus improve the safety of spinal surgery by allowing early and reliable detection of impending spinal cord damage.

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