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*Review articles*

# Intraoperative spinal cord monitoring of motor function with myogenic motor evoked potentials: a consideration in anesthesia

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## Introduction

Several surgical procedures are associated with the risk of spinal cord injury. Paraplegia and paraparesis remain devastating complications of thoracic and thoracoabdominal aortic surgery and spine surgery, although the incidence of such neurologic dysfunction varies widely between procedures. In particular, the reported incidence of paraplegia after thoracic and thoracoabdominal aortic surgery remains high, ranging from 2.4% to 40% [1–4]. Svensson et al. [1] reported that the overall incidence of neurologic deficits was 16% in 1509 patients who underwent thoracoabdominal aortic aneurysm surgery. Another report, of 605 patients by Crawford et al. [5], demonstrated a 4.4% incidence of paraplegia and a 5% incidence of paraparesis after thoracoabdominal aortic aneurysm surgery. The incidence of neurologic complications after the correction of scoliosis with spinal instrumentation has been shown to range between 0.4% and 1.6 % [6–8].

Intraoperative spinal cord damage can result from ischemia, disruption, compression, concussion, and distraction. Each of these insults can affect functional integrity, which may be neurophysiologically evident. These findings suggest the potential usefulness of the monitoring of evoked potentials. In fact, a large, multicenter retrospective study has demonstrated that teams experienced in spinal cord monitoring incur fewer neurologic deficits after scoliosis surgery compared with findings for teams with little monitoring experience [8]. Although the evidence that monitoring improves outcomes has not been established in prospective, randomized clinical trials, the intraoperative monitoring of the spinal cord with evoked potentials has gained widespread acceptance.

For monitoring the functional integrity of the spinal cord, somatosensory evoked potentials (SEPs) have been used widely, because the recording of SEPs is feasible under general anesthesia. However, because SEPs only reflect the functional integrity of sensory tracts, both false-positive and false-negative results have been reported for postoperative paraplegia [7–10]. Because motor evoked potentials (MEPs) are very sensitive to anesthetic-mediated suppression, the intraoperative recording of MEPs has been difficult under general anesthesia. However, recent progress in stimulation techniques has made the intraoperative recording of MEPs possible [11–13]. By monitoring MEPs intraoperatively, the functional integrity of motor tracts during surgical procedures can be monitored even under general anesthesia. This review will discuss recent advances in MEP monitoring techniques, the influence of anesthetic agents and physiologic parameters on myogenic MEPs, and the usefulness of intraoperative MEP monitoring during various surgical procedures.

## Limitations of somatosensory evoked potentials (SEPs)

Since the time of the first attempts to prevent intraoperative injury to the spinal cord during surgical correction of scoliosis using intraoperative neurophysiology in the late 1970s, SEPs have been widely used for spinal cord monitoring during operations that entail a risk of postoperative paraplegia. Intraoperative monitoring of

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SEPs was the only tool available at that time, and reliable methods to monitor motor pathways did not exist [14]. Therefore, SEPs were used for monitoring both sensory and motor pathways. However, these strategies can, of course, generate unreliable results in SEP monitoring.

During surgery for scoliosis, injury to the spinal cord is typically diffuse, affecting both ascending (sensory) and descending (motor) pathways. Therefore, monitoring spinal cord sensory pathways may reflect the functional integrity of both the sensory and motor pathways. In contrast, during the surgery for spinal cord tumors, surgeons can selectively damage either the motor or sensory pathways. Monitoring only one of these pathways was not sufficient.

During thoracoabdominal aortic aneurysm surgery, SEPs were also used to assess the adequacy of distal aortic pressure and to identify vessels critical to spinal cord blood supply. However, false-negative results (postoperative paraplegia despite unchanged intraoperative SEPs) have been reported [15–18]. SEPs can assess conduction in the ascending sensory pathways, which are located in the dorsal part of the spinal cord and supplied by the posterior spinal arteries. In contrast, the spinal motoneuronal system is located in the anterior horn gray matter and is supplied by the anterior spinal artery. Therefore, SEPs cannot reflect motor function and motor tract blood supply. To assess the functional integrity of motor tracts during this surgery, monitoring of MEPs was therefore required.

## Motor evoked potentials (MEPs)

### *Spinal MEPs vs myogenic MEPs*

MEP is a strong candidate for the intraoperative monitoring of the spinal cord, because it provides a method for monitoring the functional integrity of descending motor pathways. MEPs can be elicited by transcranial stimulation, by direct stimulation of the motor cortex, or by the stimulation of the descending motor tracts at the level of the spinal cord. Stimulation can be either magnetic or electric. Magnetic stimulation is undoubtedly better than electrical stimulation in conscious patients because it is not painful. However, magnetic transcranial stimulation requires continuous access to the head, and small displacements of the magnet result in considerable amplitude variability. Furthermore, MEPs eleicited by magnetic stimulation are considered to be more sensitive to anesthetic depression than those elicited by electrical stimulation [19,20]. Therefore, magnetic stimulation is currently not used for the intraoperative monitoring of MEPs under general anesthesia. Instead, electrical stimulation, especially transcranial electrical stimulation, is mainly used for spinal cord monitoring.

Motor evoked responses can be recorded from the spinal cord (spinal MEPs), or from muscles (myogenic MEPs) (Fig. 1). Merton and Morton [21] first described myogenic MEPs after transcranial stimulation. In awake subjects, spinal MEPs contain a corticospinal "D wave" and then a series of "I waves" generated indi-

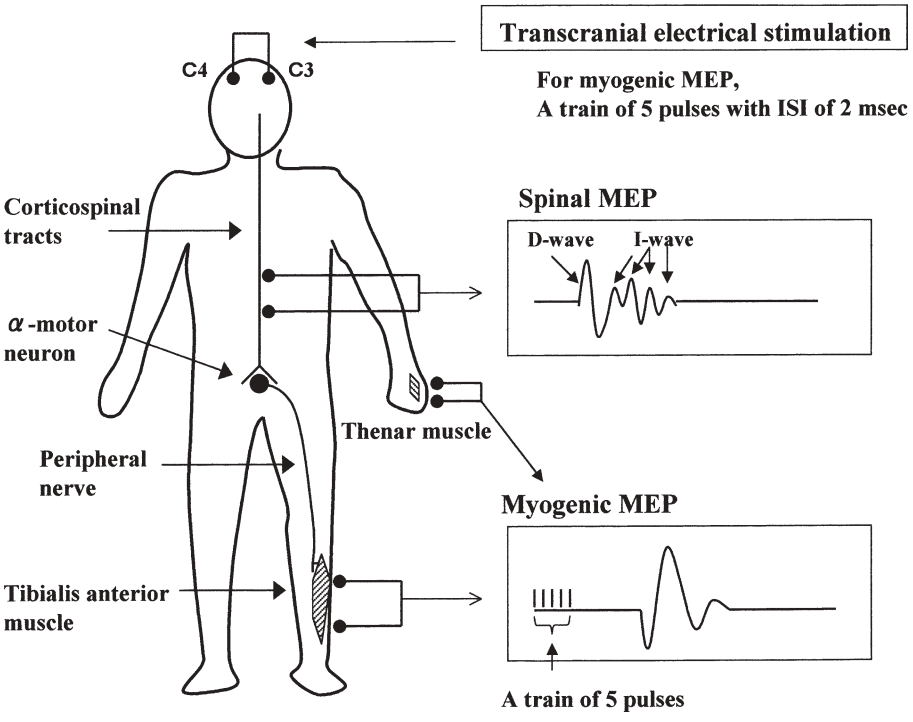


**Fig 1.** Schema of motor evoked potentials (*MEPs*). *ISI*, interstimulus interval

rectly by cortical synapses. These descending corticospinal volleys summate to depolarize spinal motor neurons, producing muscle responses. However, during general anesthesia, anesthetic agents suppress cortical and anterior horn synapses, and this suppression eliminates the I waves of spinal MEPs and myogenic MEPs. Only the D wave is resistant to anesthesia and could be used to monitor corticospinal tract integrity during general anesthesia [22].

For the recording of spinal MEPs (D wave), anesthetic depression is less than that occurring with myogenic MEPs. However, epidural electrodes have to be inserted invasively for recording spinal MEPs. Furthermore, because spinal MEPs assess only conduction in the corticospinal tracts, spinal MEPs are resistant to ischemia. Spinal MEPs may disapper slowly after the interruption of spinal cord blood flow. In a rabbit model of spinal cord ischemia, de Haan et al. [23] demonstrated that myogenic MEPs disappeared within 2 min after aortic occlusion, whereas spinal MEPs required 11 min to decrease 50% in amplitude. These findings suggest that the observed time between the onset and detection of spinal cord ischemia with spinal MEPs may be too long to allow prompt intervention.

In contrast, myogenic MEPs require no invasive electrode placement and have been shown to be highly sensitive in predicting paraplegia. Although myogenic MEPs are more sensitive to anesthetic depression than spinal MEPs, and the use of neuromuscular blockade is limited, the intraoperative recording of myogenic MEPs has become clinically feasible after the introduction of the multipulse stimulation technique. Currently, myogenic MEP after transcranial electrical stimulation with a train of pulses is a popular tool for the intraoperative monitoring of spinal cord motor function.

*Myogenic MEPs after multipulse stimulation*

Myogenic MEPs elicited by single pulse stimulation have been shown to be very sensitive to suppression by most the anesthetic agents [24]. To overcome anesthetic-induced depression of myogenic MEPs, multiple-stimulus setups, with paired pulses or a train of pulses for stimulation of the motor cortex have recently come into use (Fig. 2). When descending impulses are inhibited, the temporal accumulation of several excitatory postsynaptic potentials (EPSPs) is required to bring motor neurons from the resting state to the firing threshold [25,26]. Kalkman et al. [11] examined the effect of paired transcranial electrical stimulation on myogenic MEPs in anesthetized patients and demonstrated that maximum amplitude augmentation was observed with interstimulus intervals between 2 and 5 ms. When the interstimulus interval was increased to 7 ms, no further augmentation occurred. Pechstein et al. [27] applied transcranial high-frequency electrical stimulation in patients anesthetized with propofol and alfentanil, and indicated that a minimum stimulation frequency of 200 Hz (interstimulus interval, 5 ms) was required to elicit myogenic MEPs. Multipulse transcranial stimulators providing a train of up to ten stimuli are now available commercially. A train of three to six pulses, with an interstimulus interval of 2 ms (500 Hz), is the recommended setup for transcranial electrical stimulation under general anesthesia.

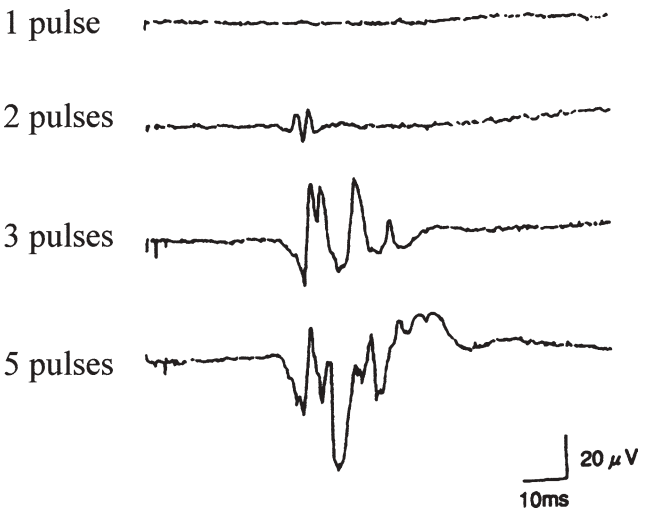


**Fig 2.** Myogenic motor evoked potentials (MEPs) in response to transcranial electrical stimulation with a single pulse, paired pulses, or a train of three or five pulses under propofol and fentanyl anesthesia. The interstimulus interval was set at 2 ms (500 Hz)

**Table 1.** Effects of anesthetics on myogenic motor evoked potentials (MEPs)

| | |
|---|---|
| Inhalational anesthetics | |
| Isoflurane | ↓↓↓ |
| Sevoflurane | ↓↓↓ |
| Nitrous oxide | ↓↓ |
| Intravenous anesthetics | |
| Barbiturate | ↓↓↓ |
| Benzodiazepine | ↓↓ |
| Propofol | ↓↓ |
| Ketamine | — |
| Fentanyl | —/↓ |

Degree of suppression of MEPs: ↓↓↓ (severe); ↓↓ (moderate); ↓ (mild); —, no suppression of MEPs

## Effects of anesthetics on myogenic MEPs

Because various anesthetics have been shown to affect myogenic MEPs, the influence of anesthetics on myogenic MEPs has been widely investigated (Table 1).

Although the introduction of multipulse stimulation has made the intraoperative recording of myogenic MEPs possible, myogenic MEPs induced by such stimulation paradigms are still affected by anesthetic agents [28–31]. For anesthetic management during the intraoperative monitoring of myogenic MEPs, proper understanding of the anesthetic-mediated changes in myogenic MEPs is therefore important.

### *Ketamine*

Ketamine has been reported to have little effect on MEPs [32–35]. Ubags et al. [32] reported that 0.5 mg·kg$^{-1}$ ketamine did not significantly change MEPs induced by transcranial electrical stimulation in patients undergoing spine surgery. Kalkman et al. [33] demonstrated that 1 mg·kg$^{-1}$ ketamine did not cause significant alterations of magnetic MEPs in human volunteers. Ghaly et al. [34] investigated the effects of incremental doses of ketamine on magnetic MEPs in monkeys, and found no amplitude depression until a cumulative dose of 15–20 mg·kg$^{-1}$ had been administered. Therefore, ketamine has been used as an anesthetic agent during the monitoring of myogenic MEPs. Especially in patients with preoperative motor dysfunction, the use of anesthetic agents with suppressive effects on the MEPs may make intraoperative MEP monitoring impossible. In these patients, ketamine can be used successfully during the monitoring of MEPs. However, adverse effects related to ketamine, including psychedelic effects, such as unpleasant dreams, hallucinations, and hypertension may limit the use of ketamine. Kawaguchi et al. [31] reported that these adverse effects were noted in 41% of patients anesthetized with ketamine for the monitoring of myogenic MEPs; however, the incidence of these effects was significantly reduced, to 14%, when low-dose (1–3 mg·kg·h$^{-1}$) propofol was added as a supplement.

### *Propofol*

Propofol has been shown to be a potent suppressor of MEPs induced by electrical and magnetic stimulation with a single pulse [20,36–39]. Kalkman et al. [20] reported that sustained reduction of MEP amplitude occurred after a single dose of 2 mg·kg$^{-1}$ propofol. Taniguchi et al. [36] investigated the effects of propofol on MEPs induced by transcranial magnetic stimulation. They documented that the reduction of MEP amplitude was very large and MEPs were completely abolished before adequate anesthesia was achieved. Jellinek et al. [37] performed intraoperative MEP monitoring induced by transcranial magnetic stimulation under propofol anesthesia and demonstrated that propofol anesthesia caused a reduction of MEPs to 7% of baseline.

Although propofol suppresses MEPs in response to single pulse stimulation, MEPs can be recorded when a train of pulses is used for stimulation. Kawaguchi et al. [31] investigated the effects of propofol on myogenic MEPs induced by transcranial electrical stimulation with a single pulse and a train of three and five pulses in patients undergoing spine surgery. The results indicated that, although MEP amplitudes were suppressed by propofol in a dose-dependent manner, regardless of the stimulation paradigm, the application of train pulse stimulation significantly enhanced MEP responses and made MEP monitoring possible. Pechstein et al. [39] compared isoflurane plus nitrous oxide and propofol anesthesia for recording MEPs after multipulse stimulation and demonstrated that propofol anesthesia was superior to isoflurane and nitrous oxide anesthesia for the intraoperative MEP monitoring. Currently, propofol-based anesthesia is therefore considered to be the standard anesthetic regime for intraoperative monitoring of myogenic MEPs.

### *Nitrous oxide*

Nitrous oxide has been used during MEP monitoring as a supplementary anesthetic, although a number of investigators have shown that it suppresses MEPs. Woodforth et al. [40] recorded myogenic MEPs in response to single pulse stimulation in patients anesthetized with fentanyl and 70% nitrous oxide, although MEP amplitudes were very low, less than 50μV. Zentner et al. [41] investigated the effects of nitrous oxide on MEPs in response to transcranial electrical stimulation with a single pulse, and demonstrated that 66% nitrous oxide reduced MEP amplitudes to an average of 9% of the baseline values in healthy volunteers. Jellinek et al. [42] also reported that increasing concentrations of nitrous oxide caused a significant reduction in MEP amplitude after single transcranial electrical stimulation in patients under propofol anesthesia; they suggested that nitrous oxide should be maintained below 50% if used as an anesthetic adjunct during MEP monitoring.

The effect of nitrous oxide on myogenic MEPs in response to stimulation with paired pulses or a train of pulses is controversial. van Dongen et al. [43] investigated the effects of nitrous oxide on MEPs in response to a six-pulse train of transcranial electrical stimuli and demonstrated that increasing doses of nitrous oxide reduced MEP amplitudes, but even with 60% nitrous oxide, MEPs were recordable. Pechstein et al. [44] reported that 60% nitrous oxide significantly reduced the amplitude of MEPs induced by transcranial stimulation with a train of five pulses in patients anesthetized with alfentanil and propofol. By contrast, in another report by van Dongen et al. [45], it was noted that 50% nitrous

oxide did not affect the amplitude of MEPs induced by transcranial electrical stimulation with paired pulses during fentanyl and low-dose propofol anesthesia in ten patients. Sakamoto et al. [46] investigated the effects of the stimulation paradigm (single pulse stimulation or train pulse stimulation) and propofol dose (absent, low-dose, or high-dose) as background anesthetics on the nitrous oxide-induced suppression of myogenic MEPs in rabbits. They found that the application of a train of five pulses could reverse the nitrous oxide-induced suppression of MEPs, which was noted after single pulse stimulation, in the absence of propofol infusion and during the administration of low-dose propofol. However, during the administration of high-dose propofol, nitrous oxide significantly suppressed MEPs regardless of the stimulation paradigm. These findings suggested that the nitrous oxide-induced suppression of MEPs could be modified by the use of multipulse stimulation and the administration of propofol. When nitrous oxide is used as a supplement, high-dose propofol may be better avoided.

*Inhalational anesthetics*

A number of authors have demonstrated that inhalational anesthetics, including isoflurane, sevoflurane, halothane, enflurane, and desflurane, suppress myogenic MEPs in a dose-dependent manner. Calancie et al. [47] carried out intraoperative monitoring of myogenic MEPs induced by transcranial electrical stimulation with a single pulse in patients anesthetized with nitrous oxide in oxygen and with narcotics, and demonstrated that the addition of 1% isoflurane abolished the MEPs in five of eight patients. Haghighi et al. [48] examined the effect of isoflurane on MEPs induced by a single-shock stimulation of the motor cortex in 14 rats. They demonstrated that an increase in isoflurane concentration from 0.3% to 1.5% resulted in a progressive increase in the MEP latency and a decrease in peak-to-peak amplitudes. Zentner et al. [49] studied the effects of halothane, enflurane, and isoflurane on myogenic MEPs induced by direct electrical stimulation of the motor cortex with a single pulse in 10 rabbits, and demonstrated that MEP responses were suppressed in a dose-dependent manner and were absent at doses greater than 0.5 minimum alveolar concentration (MAC) for all inhaled anesthetics tested. Haghighi et al. [50] reported that desflurane suppressed myogenic MEPs induced by single direct stimulation of the motor cortex in a dose-dependent manner.

Even if paired pulses or a train of pulses are employed for stimulation of the motor cortex, these cannot overcome the suppressive effects of inhalational anesthetics on MEPs [28–30]. Ubags et al. [29] demonstrated that, although isoflurane suppressed MEP responses significantly, the monitoring of myogenic MEPs was feasible in the presence of up to 0.6% isoflurane in the majority of patients anesthetized with nitrous oxide and sufentanil. Kawaguchi et al. [30] investigated the effects of sevoflurane on myogenic MEPs induced by single and paired transcranial electrical stimulation in patients and demonstrated that, although the success rate with MEP recording and MEP amplitudes after paired stimulation were greater than after single stimulation, both the success rate and the MEP amplitudes after paired stimulation decreased dose-dependently during the administration of sevoflurane. Although the precise site at which myogenic MEPs are suppressed by inhaled anesthetics is unknown, synaptic transmission has been regarded as the primary site of this anesthesia. Zentner et al. [49] suggested that the descending impulse elicited by electrical stimulation of the motor cortex during anesthesia with inhaled anesthetics was inhibited mainly at the level of the spinal interneuronal or motoneuronal systems. During the monitoring of myogenic MEPs, the administration of inhalational anesthetics should be avoided, or limited to a very low concentration.

*Opioids*

In general, opioids, which are commonly used as anesthetic supplements during intraoperative MEP monitoring, have been considered to have little suppressive effect on myogenic MEPs. Kalkman et al. [20] reported no significant MEP amplitude changes in response to transcranial electrical or magnetic stimulation with a single pulse after an intravenous bolus administration of 3 $\mu g \cdot kg^{-1}$ fentanyl in humans. Schmid et al. [51] also demonstrated that increasing doses of 0–8 $\mu g \cdot kg^{-1}$ fentanyl did not affect MEP amplitudes in response to transcranial magnetic stimulation in humans. By contrast, Thees et al. [52] demonstrated the dose-dependent suppression of MEPs after fentanyl, alfentanil, and sufentanil in rabbits. Scheufler and Zentner [53] also reported that fentanyl, alfentanil, sufentanil, and remifentanil had suppressive effects on myogenic MEPs, but that remifentanil exerted the least suppressive effects in rabbits. In the clinical situation, they also demonstrated that remifentanil suppressed myogenic MEPs in response to multipulse magnetic stimulation, although MEP recordings were feasible. To date, there have been no studies to specifically investigate the effects of opioids on MEPs in response to transcranial electrical stimulation with a train of pulses. However, considering that we routinely use fentanyl without any limitations during the intraoperative monitoring of MEPs in response to multipulse stimulation, even if there is a suppressive effect of fentanyl, it may not be clinically significant.

*Barbiturates*

Barbiturates have been shown to suppress myogenic MEPs in a dose-dependent manner. Taniguchi et al. [36] demonstrated that MEPs elicited by transcranial magnetic stimulation disappeared completely during the continuous infusion of thiopental producing very light anesthesia in 12 of 15 patients. Kawaguchi et al. [54] also reported that 2 mg·kg$^{-1}$ thiopental significantly reduced MEP amplitudes in response to transcranial magnetic stimulation to 42.8% of baseline values in humans. As it is not clear whether multipulse stimulation can overcome the barbiturate-mediated suppression of myogenic MEPs, the use of barbiturates should be avoided during the intraoperative monitoring of MEPs.

*Midazolam*

Midazolam has been shown to have suppressive effects on myogenic MEPs in humans. Kalkman et al. [20] demonstrated that 0.05 mg·kg$^{-1}$ midazolam caused a significant reduction of MEPs in response to transcranial single electrical and magnetic stimulation to 23% and 16%, respectively, of baseline values in humans. By contrast, Scheufler and Zentner [53] demonstrated that midazolam did not suppress myogenic MEPs in response to electrical stimulation with a single pulse in rabbits. So far, there have been no studies to investigate the effects of midazolam on myogenic MEPs in response to multipulse stimulation. Further studies are required.

## Effects of neuromuscular blockade on myogenic MEPs

Myogenic MEPs are affected by the level of neuromuscular blockade. Originally, muscle relaxants were avoided in order to record myogenic MEPs intraoperatively. However, in the absence of neuromuscular blockade, motor stimulation can elicit movement, and this can interfere with surgery. This is especially true with microscopic surgery. Therefore, partial neuromuscular blockade was used for anesthetic management during the monitoring of myogenic MEPs. Adams et al. [55] demonstrated that the intraoperative monitoring of myogenic MEPs was feasible, under conditions of controlled neuromuscular blockade, to maintain the first of four twitches (T1) to 10% of the baseline value. van Dongen et al. [56] investigated the effects of the level of neuromuscular blockade (T1 response, 5%–15% vs 45–55% of baseline) on the within-patient variability and amplitude of myogenic MEPs and demonstrated that, although MEP recording was feasible with a T1 response of 5%–15%, larger and less variable MEPs were recorded at a T1 response of 45%–55% than at a T1 response of 5%–15%. They suggested that a stable neuromuscular blockade aimed at 45%–55% of baseline could provide reliable and recordable muscle responses during the intraoperative recording of myogenic MEPs. In order to maintain the level of neuromuscular blockade within a narrow range and minimize the influence of fluctuations in the level of neuromuscular blockade on MEP variability, the use of a closed-loop continuous infusion of a muscle relaxant is recommended.

## Effects of hypothermia on myogenic MEPs

Investigations in animals have shown that mild to moderate hypothermia is associated with a substantial decrease in histological damage in models of spinal cord ischemia and injury [57,58]. Hypothermic therapy has been indicated during procedures such as thoracoabdominal aortic replacement, in which the spinal cord is susceptible to ischemia and injury. MEP monitoring may therefore be required under hypothermic conditions during such operations. Although a number of investigators have reported the influence of hypothermia on sensory and auditory evoked potentials, there have been only a few reports dealing with the effect of hypothermia on MEPs [59–63].

Oro and Haghighi [59] investigated the effects of systemic hypothermia on spinal neurogenic MEPs recorded from the epidural space at L1–2 in rats anesthetized with pentobarbital. They demonstrated that the amplitudes of spinal MEPs in response to single pulse stimulation were significantly reduced with a decrease in core temperature, and no spinal MEPs were detectable below 28°C. Meylaerts et al. [60] investigated the influence of regional spinal cord hypothermia on myogenic MEPs in response to transcranial electrical stimulation with a train of five pulses in pigs anesthetized with ketamine, sufentanil, and nitrous oxide. Progressive cooling resulted in an increase in MEP amplitude at 28–30°C, and this was followed by a progressive decrease. In a rabbit model, Sakamoto et al. [63] demonstrated that a reduction of core temperature to 28°C did not significantly influence the amplitude of MEP in response to multipulse stimulation with a train of three or five pulses, whereas MEP amplitude in response to single pulse stimulation was significantly decreased with a reduction of core temperature.

Although the effects of hypothermia on the amplitude of MEPs can vary depending on the degree of hypothermia and the stimulus paradigm, hypothermia-mediated changes in MEP latency seem to be consistent. Oro and Haghighi [59] demonstrated that systemic hypothermia increased the early wave latency and interpeak latencies of spinal MEPs in rats. Meylaerts et al. [60] reported that progressive subdural hypothermia

progressively increased the latency of myogenic MEPs in pigs. Sakamoto et al. [63] also reported that MEP latency was increased linearly with a decrease in core temperature, regardless of the stimulation paradigm, in rabbits. These findings suggested that the monitoring of myogenic MEPs may be feasible under hypothermic conditions of temperatures down to 28°C, as long as a train of pulses is used for stimulation, although MEP latency increases with the reduction of core temperature.

Anesthetic-mediated suppression of myogenic MEPs may be modulated under hypothermic conditions. Kakimoto et al. [61] investigated the effects of hypothermia on the nitrous oxide-induced suppression of myogenic MEPs in rabbits. The results indicated that, although a reduction of core temperature to 35°C or 30°C did not significantly affect MEP amplitude, the nitrous oxide-induced suppression of MEPs was augumented under the hypothermic conditions. These findings suggested that nitrous oxide should be used with care because of its marked suppressive effects on MEPs under hypothermic conditions. Hypothermia may also affect the concentration of anesthetics and neuromuscular blockade, and therefore may affect MEPs. Leslie and colleagues [64] demonstrated that a temperature reduction of 3°C increased blood propofol concentration by 30% during a constant rate infusion. Careful anesthetic management is required for the monitoring of myogenic MEPs under conditions of hypothermia.

## Intraoperative monitoring of myogenic MEPs

### *Aortic surgery*

By the monitoring of myogenic MEPs during thoracoabdominal aortic aneurysm surgery, ischemia of the spinal cord can be detected within minutes (Fig. 3). Early detection of ischemia allows the immediate adjustment of surgical procedures and anesthetic management. In fact, MEP monitoring can be used clinically for the following purposes: (1) to assess whether or not spinal cord perfusion during cross-clamping of the aorta is adequate, (2) to assess whether or not surgical reconstruction is adequate, and (3) to assess the prognosis of motor function, which can promote an early decision in the treatment of spinal cord injury. Jacobs et al. [65,66] reported the results of intraoperative myogenic MEP monitoring in 52 patients with thoracoabdominal aortic surgery. In 14 of the 52 patients (27%), MEP amplitudes decreased to less than 25% of baseline after proximal cross-clamping, indicating critical spinal cord ischemia. However, these changes in MEPs could be corrected by increasing the distal aortic pressure (DAP). The mean DAP required to maintain adequate

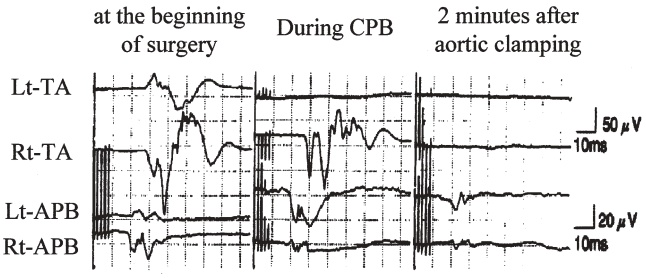


**Fig 3.** Changes in myogenic motor evoked potentials (MEPs) during the repair of a type-II thoracoabdominal aortic aneurysm in a 78-year-old woman. Myogenic MEPs were recorded from the left (*lt*) and right (*rt*) tibialis anterior (*TA*) muscles and the left and right abductor pollicis brevis (*APB*) muscles. During partial cardiopulmonary bypass (*CPB*), MEP responses from the left TA disappeared 30 min after the insertion of a cannula into the left femoral artery for distal perfusion. MEP changes at the site of femoral arterial cannulation, probably due to ischemia of the peripheral nerve, were transient. Two minutes after aortic clamping, MEP responses from the right TA disappeared, but MEPs from the APB remained unchanged, indicating ischemia of the spinal cord. Because only one intercostal artery was detected and reconstructed, MEP did not recover after aortic declamping. Postoperatively, paraplegia developed in this patient

spinal cord perfusion was 66 mmHg, with a wide range between 48 and 110 mmHg. These findings suggest that MEP can be used effectively to determine whether or not spinal cord perfusion is adequate and to determine the degree of DAP that is appropriate to maintain functional integrity of the spinal cord in each patient. Without MEP monitoring, anesthesiologists cannot have such information. In 24 of 52 patients (46%), MEPs disappeared after segmental clamping of the aorta and returned after reattachment of the intercostal arteries. By contrast, in 9 of 52 patients (17%), MEPs disappeared completely, but no intercostal arteries were found. In these patients, aortic endarterectomy was performed and Dacron grafts were anastomosed to the intercostal arteries, resulting in the recovery of MEPs. These data suggest that, with MEP findings, surgeons will know whether or not further reconstruction of the intercostal arteries is required. If surgeons reattach only back bleeding intercostal arteries, without the use of MEP monitoring, they may miss important arteries to be reconstructed in approximately 15% of patients, resulting in the development of postoperative paraplegia and paraparesis. Jacobs et al. [65,66] indicated that an aggressive surgical approach based on MEP findings resulted in a significant reduction of neurologic complications.

### *Spine surgery*

SEPs are still widely used for intraoperative neurophysiological monitoring during spine surgery, especially

scoliosis surgery. In these procedures, derangements of spinal cord functional integrity based on distraction maneuvers can be reflected in dorsal column injury. In fact, Nuwer et al. [8] demonstrated that SEP monitoring reduced neurological deficits after scoliosis surgery. However, because SEPs can only reflect the functional integrity of sensory tracts, they may not reflect the functional integrity of motor pathways. A number of investigators have reported false-negative (postoperative motor dysfunction without SEP changes) results and false-positive results (SEP changes without postoperative motor dysfunction) for postoperative paraplegia [15–18]. In contrast to the use of SEPs, the monitoring of MEPs is now a feasible and reliable technique to assess the functional integrity of motor pathways. Recently, most investigators have recommended the combined use of SEPs and MEPs for the spine surgery, in which the spinal cord can be at risk of injury [10]. This combined use of SEPs and MEPs provides independent verification of spinal cord integrity using two parallel but independent systems, and also allows the detection of occasional insults that may selectively affect either motor or sensory pathways.

### *Anesthetic regimens during myogenic MEP monitoring*

Anesthetic regimens during MEP monitoring can vary depending on the type of surgery (Table 2). During spine surgery, propofol and fentanyl anesthesia is usually employed, with or without nitrous oxide, when multipulse stimulation is used. However, in patients with motor dysfunction, MEP recording may be difficult under propofol-based anesthesia. In such patients, in order to reduce the dose of propofol, ketamine may be added to the anesthetic regimen. A neuromuscular blocking agent is administered continuously to keep the T1 level at 25%–50% of control. During thoracoabdominal aortic aneurysm surgery, ketamine-based anesthesia is usually used. The use of propofol may be limited, because cardiopulmonary bypass, hypothermia, and occlusion of the aorta can affect the metabolism and concentration of propofol, resulting in the fluctuation of MEP responses. During such situations, propofol is administered only at a low dose (1–2 mg·kg·h$^{-1}$) or discontinued. Neuromuscular blocking agents are also affected in such situations, so that the monitoring of neuromuscular blockade is mandatory.

**Table 2.** Examples of anesthetic regimens used during the monitoring of myogenic motor evoked potentials (MEPs)

| | |
|---|---|
| Spine surgery | |
| Induction | Propofol (1.5–2.5 mg·kg$^{-1}$) |
| | Fentanyl (1–4 μg·kg$^{-1}$) |
| | Vecuronium (0.1 mg·kg$^{-1}$) |
| Maintenance | Propofol (4–8 mg·kg·h$^{-1}$) |
| | Fentanyl as necessary |
| | 50% Nitrous oxide |
| | Vecuronium; T1 at 25%–50% of control |
| Aortic surgery | |
| Induction | Ketamine (1–2 mg·kg$^{-1}$) |
| | Fentanyl (1–4 μg·kg$^{-1}$) |
| | Propofol (0.5–1 mg·kg$^{-1}$) |
| | Vecuronium (0.1 mg·kg$^{-1}$) |
| Maintenance | Ketamine (1–2 mg·kg·h$^{-1}$) |
| | Fentanyl as necessary |
| | Propofol (1–2 mg·kg·h$^{-1}$) |
| | Vecuronium; T1 at 25%–50% of control |

Anesthetic regimens used at Nara Medical University. Nitrous oxide can be omitted during spine surgery. Target-controlled infusion can be used for the administration of propofol and fentanyl during spine surgery, but such infusion should not be used during aortic surgery

## Future directions in anesthetic management for MEP monitoring

Although the introduction of multipulse stimulation setups has made intraoperative MEP monitoring possible, further improvements in MEP monitoring are required. First, because myogenic MEPs can easily be affected by anesthetic status, specific consideration of the anesthetic used is required, especially during aortic surgery. Cardiopulmonary bypass, hypothermia, and aortic occlusion may significantly affect anesthetic concentrations, resulting in false-positive changes in MEP responses. Therefore, the use of most anesthetic agents, except for ketamine, is limited. However, most anesthesiologists may not prefer ketamine, because of its adverse effects, including psychedelic effects. More suitable anesthetic conditions for MEP monitoring may be required. Second, because complete neuromuscular blockade abolishes myogenic MEPs, partial neuromuscular blockade is used for anesthetic management during the monitoring of myogenic MEPs. van Dongen et al. [56] have suggested that a stable neuromuscular blockade, aimed at 45%–55% of baseline, can provide reliable and recordable muscle responses during the intraoperative monitoring of myogenic MEPs. However, even partial neuromuscular blockade may elicit movement in patients in response to transcranial stimulation. This may interfere with surgery, especially microscopic surgery. In addition, movement-related injuries, including tongue and lip laceration, have been reported [67]. Further improvements to reduce patient movements are required.

## Conclusion

The monitoring of myogenic MEPs has become a feasible and reliable method of monitoring the functional integrity of motor pathways. Especially, the introduc-

tion of multipulse stimulation allows the routine use of the intraoperative monitoring of myogenic MEPs for monitoring the functional integrity of motor pathways. However, myogenic MEPs can be affected by most anesthetic agents and muscle relaxants. Anesthesiologists are therefore required to have a proper understanding of MEPs and to undertake careful management of anesthesia in the light of this knowledge. It is hoped that a team approach by surgeons, neurophysiologists, and anesthesiologists can play an important role in the prevention of postoperative neurological deficits after surgeries in which the spinal cord is at risk of injury.

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