Bipolar transesophageal thoracic spinal cord stimulation: A novel clinically relevant method for motor-evoked potentials

Ken Yamanaka, MD, Kazumasa Tsuda, MD, PhD, Daisuke Takahashi, MD, Naoki Washiyama, MD, PhD, Katsushi Yamashita, MD, PhD, and Norihiko Shiiya, MD, PhD

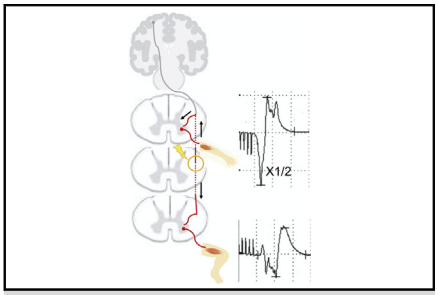
ABSTRACT

Objective: Although transesophageal motor-evoked potential elicited by monopolar cervical cord stimulation is more stable and rapid in response to ischemia than transcranial motor-evoked potential in canine experiments, direct cervical alpha motor neuron stimulation precludes clinical application. We evaluated a novel stimulation method using a bipolar esophageal electrode to enable thoracic cord stimulation.

Methods: Twenty dogs were anesthetized. For bipolar transesophageal stimulation, the interelectric pole distance was set at 4 cm. Changes in amplitude in response to incremental stimulation intensity (100-600 V) were measured to evaluate stability. Spinal cord ischemia was induced by aortic balloon occlusion at the T8 to T10 level for 10 minutes to evaluate response time or at the T3 to T5 level for 25 minutes to evaluate prognostic value. Neurological function was evaluated using the Tarlov score at 24 and 48 hours postoperatively.

Results: Bipolar transesophageal stimulation was successful in all animals and their forelimb waveforms were identical to those after transcranial stimulation. The minimum stimulation intensity to produce >90% of the maximum amplitude was significantly lower in both monopolar and bipolar transesophageal stimulation than in transcranial stimulation ($n = 5$). Time to disappearance and recovery (>75%) of the hindlimb potentials were significantly shorter by both monopolar and bipolar transesophageal stimulation than by transcranial stimulation ($n = 5$). Correlation with neurological outcomes was comparable among all stimulation methods ($n = 10$).

Conclusions: Motor-evoked potential can be elicited by bipolar transesophageal thoracic cord stimulation without direct cervical alpha motor neuron stimulation, and its stability and response time are comparable to those elicited by monopolar stimulation. (JTCVS Techniques 2020;4:28-35)



Forelimb potentials by thoracic cord stimulation are evoked through synaptic transmission.

CENTRAL MESSAGE

Bipolar transesophageal thoracic cord stimulation can elicit motor evoked potentials without direct cervical alpha motor neuron stimulation. It retains the advantages of transesophageal stimulation.

PERSPECTIVE

Transesophageal motor evoked potentials elicited by bipolar thoracic cord stimulation is more stable and rapid in response to ischemia than transcranial motor evoked potentials. The upper limb potentials can be used as a real-time control because the cervical alpha motor neurons are not stimulated directly. It may improve the utility of spinal cord monitoring during aortic surgery.

See Commentaries on pages 36 and 38.

From the First Department of Surgery, Hamamatsu University School of Medicine, Hamamatsu, Japan.

Supported by the Grants-In-Aid for Scientific Research of the Japanese Ministry of Education, Culture, Sports, Science and Technology (grant No. 16K10656).

Partly presented at the Resident Poster Competition at the 97th Annual Meeting of The American Association for Thoracic Surgery, Boston, Massachusetts, April 29-May 3, 2017.

Received for publication Aug 3, 2020; revisions received Aug 3, 2020; accepted for publication Aug 10, 2020; available ahead of print Aug 15, 2020.

Address for reprints: Norihiko Shiiya, MD, PhD, First Department of Surgery, Hamamatsu University School of Medicine, 1-20-1 Handayama, Higashi-ku, Hamamatsu, Shizuoka 431-3192, Japan (E-mail: shiyanor@hama-med.ac.jp).

2666-2507

https://doi.org/10.1016/j.xjtc.2020.08.006

Abbreviations and Acronyms

biTE = bipolar transesophageal
MEP = motor evoked potential
monoTE = monopolar transesophageal
TC = transcranial
TE = transesophageal

▶ Video clip is available online.

Ischemic spinal cord injury remains the most devastating complication after distal thoracic aortic operations. Surgeons have been concerned about spinal cord perfusion pressure since the 1950s and 1960s, and Miyamoto and colleagues[1] have already reported the use of cerebrospinal fluid drainage in 1960. Since the introduction of the collateral network concept by Griepp and colleagues,[2] the importance of maintaining collateral blood flow to the spinal cord has widely been recognized. However, during aortic surgery, surgeons cannot directly measure the blood flow to the spinal cord. Neurophysiological monitoring of spinal cord function, which has been used to detect spinal cord ischemia during aortic surgery,[3-5] may thus serve as an essential tool to adjust spinal cord perfusion pressure.

Among the several techniques used for monitoring, transcranial motor-evoked potential (TC-MEP) has widely been accepted as the method of choice,[5,6] because of its convenience and high sensitivity.[7] Loss of TC-MEP has been shown to be associated with increased risk of spinal cord injury.[5,8,9] However, it is not stable enough, and spontaneous fluctuations of amplitudes up to 50% of baseline are common.[4] Although Kawanishi and colleagues[10] have reported high sensitivity and specificity using 75% of baseline as a cutoff level, many others use much lower levels (25%-50% of baseline) because of the instability.[5,7] In addition, recovery of TC-MEP does not necessarily mean that the patient is neurologically intact.[8,9]

Several facilitative techniques have been employed, including the multitrain stimulation technique, to improve the reproducibility and reliability of TC-MEP.[11] However, brain stimulation for a few seconds at a frequency of 50 to 60 Hz has been reported to easily induce seizures or neural injury,[12,13] which precludes the use of the optimal frequency (25-100 Hz) for multitrain brain stimulation. Because repetitive measurements in relatively short intervals (5-10 minutes) are usually employed for several hours during aortic surgery, multitrain brain stimulation may increase the risk of seizures and neural injuries.

We have previously shown that transesophageal MEP (TE-MEP), which we call monopolar TE-MEP (monoTE-MEP) in this study, is feasible, safe, and superior to TC-MEP in terms of stability, response time to ischemia/reperfusion, and prognostic value in canine experiments.[14,15] We think that it is because supramaximal intensity stimulation can be applied safely and easily to the spinal cord. However, cervical cord stimulation results in the direct stimulation of the cervical alpha motor neurons with strenuous forelimb movement, which may be dangerous in clinical settings, and precludes forelimb potentials to be used as a real-time control.

Assuming that thoracic spinal cord stimulation does not result in the direct stimulation of the cervical alpha motor neurons but excites them through retrograde spinal tract conduction and synaptic transmission, we developed a novel stimulation method with a bipolar esophageal surface electrode (biTE-MEP) to facilitate thoracic cord stimulation. In monoTE-MEP, which stimulates the spinal cord between an esophageal surface electrode and a subcutaneous electrode, the vertebral level of spinal cord stimulation is determined by the position of the subcutaneous electrode, and therefore it cannot be set freely. This study aimed to determine whether biTE-MEP is feasible, whether it can elicit forelimb potentials that can be used as real-time control, and whether biTE-MEP is as useful as monoTE-MEP in terms of stability, response time to ischemia, and prognostic value in canine experiments.

MATERIALS AND METHODS

Twenty adult beagle dogs (weight, 11.2-19.6 kg) were used in this study. All animals received humane care in compliance with standard guidelines as recommended by the Science Council of Japan and the Guide for the Care and Use of Laboratory Animals (National Institutes of Health publication No. 85-23, revised 1985). The protocol was approved by the Institutional Ethics Committee on the Use and Care of Animals (protocol No. 2013014).

Experimental Settings

Anesthesia and instrumentation procedures were performed according to previously reported methods.[14,15] Briefly, the animals were anesthetized with intravenous infusion of propofol (12-24 mg/kg/h) and remifentanil (12-24 μg/kg/h) and were maintained on mechanical ventilation. For MEP recording, a Neuropak MEB-2200 system (Nihon Kohden, Tokyo, Japan) was used for data acquisition, processing, and analysis, with SEN-4100 equipment for electrical stimulation (Nihon Kohden, Tokyo, Japan). For transcranial stimulation of the brain motor area, a cathode was placed at the C4 position and an anode at the C3 position of the International 10-20 system. For monoTE-MEP, spinal cord stimulation was performed between a handmade esophageal luminal surface electrode (cathode) and a nuchal subcutaneous needle electrode (anode) at the first to second thoracic spine (T1-T2) level. For biTE-MEP, spinal cord stimulation was performed through a handmade esophageal luminal surface electrode. A train of 5 pulses was used with a 2.0-ms interstimulus interval and a 0.05-ms pulse width for stimulation. MEPs were recorded from both sides at the forelimb and hindlimb muscles. The amplitudes of the MEPs were measured.

Experimental Protocols

Evaluation of the feasibility, forelimb waveforms, and optimal interelectric pole distance. For biTE-MEP, the esophageal electrode was positioned at the third to fifth thoracic vertebral (T3-T5) level to avoid stimulation of the alpha motor neurons innervating the forelimb muscles (C5-T1). The electrode position was determined under fluoroscopic guidance. Hindlimb MEP amplitudes were compared among 3 distances (2, 4, and 6 cm) to determine the optimal distance between the 2 electric poles. The stimulation intensity was set constant at 500 V for all MEPs.

In the following protocols, this distance was set at 4 cm based on the results of the optimal interelectric pole distance study.

Evaluation of stability. Based on the concept that supramaximal intensity stimulation of the spinal cord contributes to better stability of TE-MEPs, we measured MEPs with the stimulation intensity varying from 100 to 600 V to determine the lowest intensity producing maximum amplitude at the hindlimbs in the same settings as those used in the first protocol. Measurements were repeated thrice for each stimulation condition, and the data were averaged. Practically, the amplitude elicited by 600 V stimulation was considered maximum, and the stimulation intensity that produced more than 90% of this amplitude on both sides was determined.

Evaluation of response time to spinal cord ischemia and reperfusion. We used a model of reversible spinal cord ischemia in this protocol; 10-minutes of aortic occlusion at the T8-T10 level that resulted in complete neurological recovery in our previous experiment.[15] We placed an aortic balloon occlusion catheter (Reliant, Medtronic, Minneapolis, Minn) in the descending aorta at the T8-T10 level, which was introduced through the right femoral artery under fluoroscopic guidance, after administration of 100 U/kg heparin. We measured MEPs at 1-minute intervals during descending aortic balloon occlusion, and every 2 minutes, up to 60 minutes, after reperfusion. Arterial blood pressure was continuously recorded in the left femoral artery and the left common carotid artery to confirm aortic occlusion. Proximal blood pressure was not controlled. Aortic balloon occlusion was maintained for 10 minutes after the hindlimb MEPs had disappeared. Time to MEP disappearance and time to recovery were compared among the 3 MEP methods.

Evaluation of correlation with neurologic outcomes. The experimental settings and protocols were the same as those used in the response time study protocol, except that aortic balloon occlusion was performed at the T3 to T5 level and was maintained for 25 minutes after the hindlimb MEPs had disappeared. Higher level of aortic occlusion was chosen to evaluate the effects of blood flow interruption to the higher intercostal arteries, because biTE-MEPs of the forelimbs involve retrograde thoracic spinal tract conduction. Duration of ischemia was set at 25 minutes because we wanted to evaluate the relation between the patterns of MEP recovery and neurological outcomes. Using 25-minutes of ischemia, we expected to have a spectrum of neurological outcomes from full recovery to complete paraplegia because 40 minutes of aortic occlusion at T8 to T10 level invariably resulted in paralysis in our previous study.[15] The dogs were allowed to recover with all catheters removed, arteries repaired, and wounds closed. Neurologic function was evaluated by a person who was blinded to the monitoring results according to the modified Tarlov classification (0 = no hindlimb movement, 1 = perceptible movement of the joints of hindlimbs, 2 = good hindlimb movement but unable to stand, 3 = able to stand and walk, and 4 = complete recovery) at the completion of and 24 and 48 hours after the procedure. Animals with paralysis were sacrificed by anesthetic overdose at 48 hours postoperatively, and the spinal cords were explanted for histopathological evaluation.

Statistical Analysis

SPSS software (version 25; IBM-SPSS Inc, Armonk, NY) was used for statistical analysis. The mean of both sides was used for analyses, and each value was expressed as the mean ± standard deviation. For the comparison of stimulation intensity that did not follow the normal distribution, Mann-Whitney *U* test was used. For the comparison of time to change in amplitudes between the 2 stimulation modalities, Student *t* test was used. Bonferroni correction was performed for multiple comparisons.

RESULTS

Feasibility, Forelimb Waveforms, and Optimal Interelectric Pole Distance

We used 5 dogs in this protocol. biTE-MEP was constantly recorded without difficulty in all animals. MEP waveforms were similar among all the stimulation modalities except for that of monoTE-MEP at forelimbs. Distinct latency was observed in the forelimb potentials for biTE-MEP and TC-MEP but not for monoTE-MEP (Figure 1), which indicated that direct cervical alpha motor neuron stimulation could be avoided in biTE-MEPs. We did not observe strenuous movement of the forelimbs in biTE-MEPs (Video 1).

The amplitudes of hindlimb potentials concerning the distance of the interelectric poles in biTE-MEP were 1.46 ± 1.64 mV at 2 cm, 3.53 ± 0.93 mV at 4 cm, and 3.39 ± 0.91 mV at 6 cm. We selected 4-cm distance for subsequent protocols based on this result.

Stability

We used the same 5 dogs as those used in the first protocol. The lowest stimulation intensity to produce more than 90% of the maximum MEP amplitude at the hindlimbs was 540 ± 55 V in TC-MEP, 380 ± 45 V in monoTE-MEP, and 340 ± 55 V in biTE-MEP. These values were significantly higher by transcranial stimulation than by the 2 transesophageal stimulation methods ($P = .008$, significant after Bonferroni correction).

Response Time to Spinal Cord Ischemia and Reperfusion

We used 5 dogs in this protocol. All MEPs from the hindlimb muscles completely disappeared during aortic occlusion in all dogs. After reperfusion, hindlimb MEP showed recovery to more than 75% of baseline in all dogs (Figure 1). No neurologic complications occurred. Data of time to hindlimb amplitude disappearance and time to recovery are shown in Table 1. They were significantly longer in TC-MEP than in the 2 TE-MEPs.

Correlation With Neurologic Outcomes

We used 10 dogs in this protocol. One of these 10 dogs died of refractory hypotension 5 minutes after aortic balloon occlusion was released. In the remaining 9 dogs, all MEPs recorded at hindlimbs disappeared after aortic occlusion, whereas those recorded at forelimbs, including biTE-MEPs, showed no change.

In 5 of 9 surviving dogs, all MEPs recorded at the hindlimbs showed recovery to more than 75% of baseline after

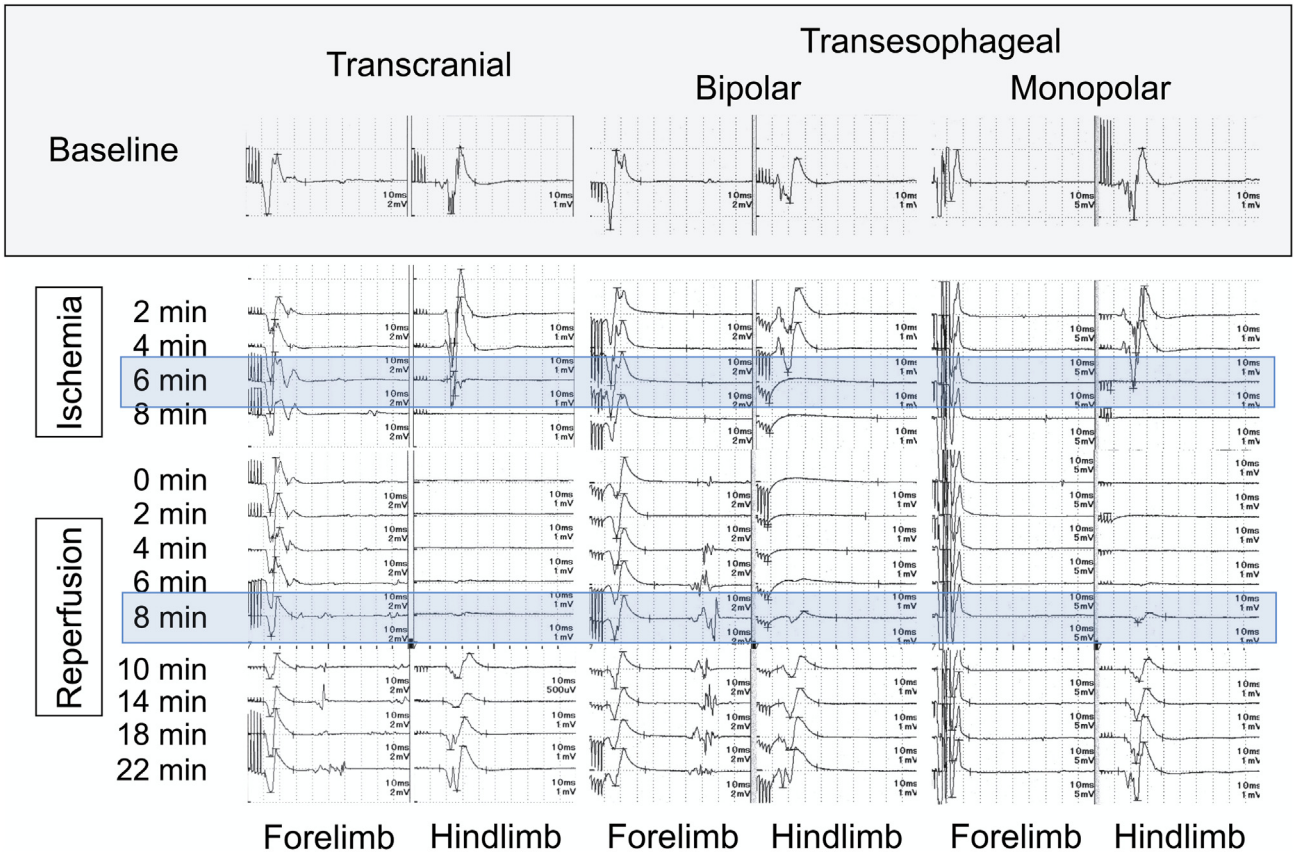
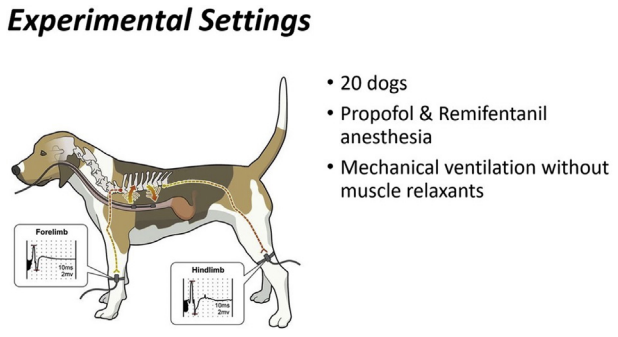


FIGURE 1. Representative waveforms of motor evoked potentials (MEPs) in a dog undergoing 10-minutes of aortic occlusion at the T8 to T10 level. All 5 dogs undergoing this procedure, including this dog, showed full neurological recovery. Both transesophageal MEPs showed a more rapid response to ischemia and reperfusion than the transcranial MEPs.

reperfusion. Compared with the results in the response time study that employed 10-minutes ischemia, longer time periods were required for MEP recovery. However, no significant differences were found among the 3 stimulation modalities (Table 2). Spinal cord dysfunction was not observed (Tarlov 4) in these 5 dogs throughout the postoperative period.

In 3 of 4 other dogs, hindlimb MEPs showed delayed inconsistent recovery to 50% to 75% of baseline in 1 and to 25% to 50% in 2 by all stimulation modalities. These dogs developed immediate paraparesis (2 had Tarlov 3 and 1 had Tarlov 2). Representative MEP waveforms and histopathology of the Tarlov 2 dog are shown in Figure 2.

In the remaining 1 dog, all hindlimb MEPs showed no recovery. This dog developed immediate paraplegia (Tarlov



VIDEO 1. Summary of the study and video clips showing the limb movements during stimulation. Video available at: https://www.jtcvs.org/article/S2666-2507(20)30381-3/fulltext.

TABLE 1. Time to motor-evoked potential (MEP) disappearance and recovery in response to 10-minutes of aortic occlusion (n = 5)

Variable	TC-MEP	biTE-MEP	mono TE-MEP
Time to disappearance (min)	7.2 ± 0.84*	5.4 ± 0.89	5.4 ± 0.89
Time to recovery (min)			
to >25% of baseline	17.6 ± 4.33†	10.8 ± 1.09	10.4 ± 0.89
to >50% of baseline	20.4 ± 5.37†	13.6 ± 3.29	13.2 ± 3.63
to >75% of baseline	27.2 ± 3.63†	18.0 ± 3.46	17.2 ± 3.03

Values are presented as mean ± standard deviation. *TC*, Transcranial; *MEP*, motor-evoked potential; *biTE*, bipolar transesophageal; *monoTE*, monopolar transesophageal. *$P = .015$ versus monoTE and biTE (significant after Bonferroni correction). †$P < .001$ versus monoTE and biTE (significant after Bonferroni correction).

TABLE 2. Time to motor-evoked potential (MEP) recovery after 25-minutes of aortic occlusion (n = 10*)

Variable	TC-MEP	biTE-MEP	monoTE-MEP
Without spinal cord injury (min) (Tarlov 4, n = 5)			
to >25% of baseline	28.4 ± 10.2	26.0 ± 11.6	26.4 ± 11.1
to >50% of baseline	36.0 ± 11.7	33.6 ± 11.5	35.2 ± 12.5
to >75% of baseline	47.6 ± 11.3	46.0 ± 12.6	45.2 ± 13.3
With paraparesis (min) (Tarlov 2 and 3, n = 3)			
to >25% of baseline	50.0 ± 5.3	46.7 ± 6.1	45.3 ± 4.6
to >50% of baseline†	58	56	56
With complete paraplegia (min) (Tarlov 0, n = 1)			
This dog showed no recovery of MEP			

Values are presented as mean ± standard deviation. *TC*, Transcranial; *MEP*, motor-evoked potential; *biTE*, bipolar transesophageal; *monoTE*, monopolar transesophageal. *One dog died 5 minutes after aortic occlusion was released. †Only 1 of the 3 dogs showed recovery to >50% of baseline.

0). Representative MEP waveforms and histopathology of this animal are shown in Figure 3.

DISCUSSION

This study showed the feasibility of biTE-MEP, a practical way of thoracic spinal cord stimulation that could solve the problem of monoTE-MEP, namely, direct stimulation of the cervical alpha motor neurons. Thoracic spinal cord stimulation by biTE-MEP was free of strenuous movement of the forelimbs, and the waveforms were identical to those of TC-MEP; therefore, they could be used as a real-time control. Although cervical alpha motor neurons were excited through retrograde spinal tract conduction, upper thoracic spinal cord ischemia, induced by aortic occlusion at the T3-T5 level, did not affect the forelimb MEPs elicited by T3-T5 thoracic spinal cord stimulation. This study also confirmed the superiority of TE-MEP over TC-MEP regarding the stability and response time to ischemia/reperfusion, which is shared by both monoTE- and biTE-MEPs.

The finding that forelimb MEPs elicited by thoracic bipolar transesophageal stimulation were not affected by proximal descending aortic occlusion was not surprising. The watershed area between the territory of the subclavian artery and the radiculomedullary artery could be mostly

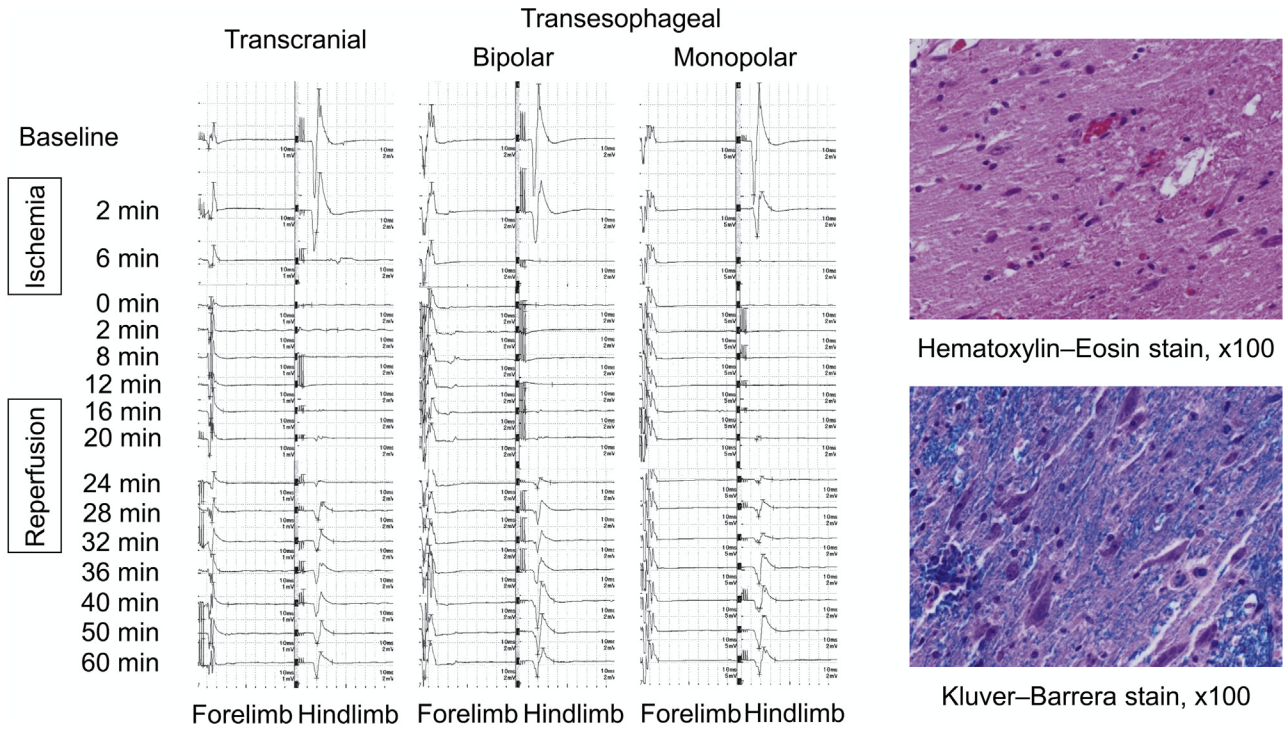


FIGURE 2. Representative waveforms of motor-evoked potentials (MEPs) in the dog with Tarlov 2 paraparesis that underwent 25-minutes of aortic occlusion at the T3 to T5 level, and histopathology of the anterior horn of the proximal lumbar spinal cord. In all the 3 modalities, hindlimb MEPs began to return around 24 minutes after reperfusion, but remained <50% of baseline. Some motor neurons showed degenerative changes, whereas others did not. Cystic cavities and microscopic hemorrhages were observed (hematoxylin eosin stain, ×100). There was no demyelination (Kluver-Barrera stain, ×100).

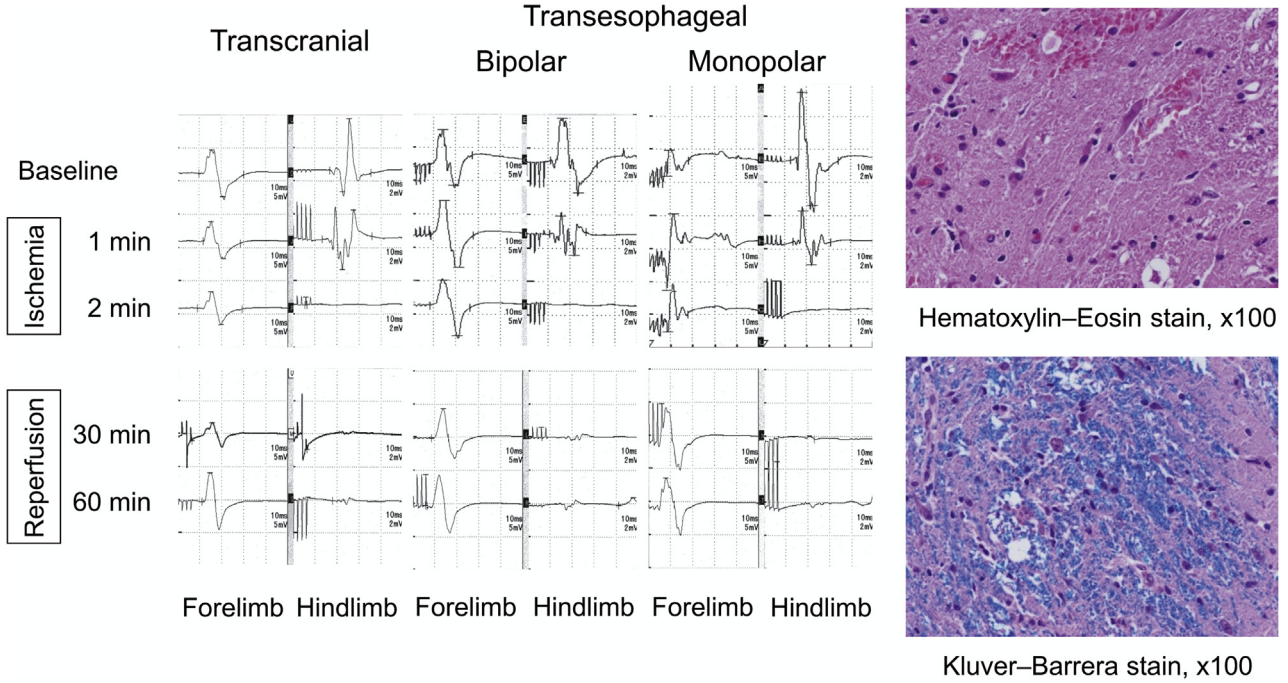


FIGURE 3. Representative waveforms of motor-evoked potentials (MEPs) in the dog with complete paraplegia (Tarlov 0) that underwent 25-minutes of aortic occlusion at the T3 to T5 level, and histopathology of the anterior horn of the proximal lumbar spinal cord. In all the 3 modalities, hindlimb MEPs were lost by 2 minutes after aortic occlusion, and never returned even at 60 minutes of reperfusion. This dog did not regain hindlimb motor function. Many motor neurons were found to have degenerated with eosinophilic or chromatolytic changes. (hematoxylin eosin stain, ×100). Myelin sheath fragmentation, axon swelling, and demyelination were evident (Kluver-Barrera stain, ×100).

located at the T4 level. Because we stimulated the spinal cord at the T3-T5 level, ascending spinal tract conduction to the cervical alpha motor neurons from the stimulation point does not seem to be affected by proximal descending aortic occlusion. In addition, the spinal tract is much more resistant to ischemia than the alpha motor neurons. In this regard, the influence of blood flow interruption on the left subclavian artery needs to be evaluated.

The prognostic value of TE-MEPs was not better than that of TC-MEP in this study, which is not consistent with our previous findings.[15] One reason is that no animal developed spastic paraplegia in the present study, which could be detected only by TE-MEPs.[15] Another reason is that the prognostic value of TC-MEP in this study was better than that in our previous study. This may be due to the difference in the vertebral level of aortic balloon occlusion. In our previous study, the descending aorta was occluded at the T8-T10 level, while the T3-T5 level was used in the present study for 25-minutes of ischemia. Spinal cord ischemia at a higher vertebral level may be more readily detected by TC-MEP because the number of affected motor neurons or fibers is higher, which may have obscured the advantage of TE-MEP.

In clinical applications, the risk of esophageal lesion due to transesophageal electrical stimulation may be a concern. We have previously shown that transesophageal stimulation is free of electrical burn injury.[11] The safety seems to be reconfirmed in this study because all the surviving dogs showed no signs of digestive or inflammatory problems. In addition, using the same stimulation condition, we have never experienced head skin injury in our clinical experiences with TC-MEP. Safety of bipolar transesophageal stimulation was confirmed in our preliminary clinical study.[16]

Another concern in clinical application is the interference with transesophageal echocardiography. Using a specially designed stimulation electrode that allows an echo probe to pass through it, we were successful in performing both examinations concomitantly without any damage to the echo probe in our preliminary clinical study.[16] Further improvements in the electrode design is required to avoid its migration caused by manipulation of the echo probe.

Delayed onset spinal cord injury is an increasingly recognized problem. Although several mechanisms seem to be involved, reduced spinal cord blood flow reserve by extensive intercostal sacrifice seems to play an important role.[17] This may explain why postoperative decrease in perfusion pressure is associated with delayed onset injury.[18-20] The role of neurophysiological monitoring to prevent delayed onset injury seems limited,[1,8] because it reflects spinal cord function at the time of examination. However,

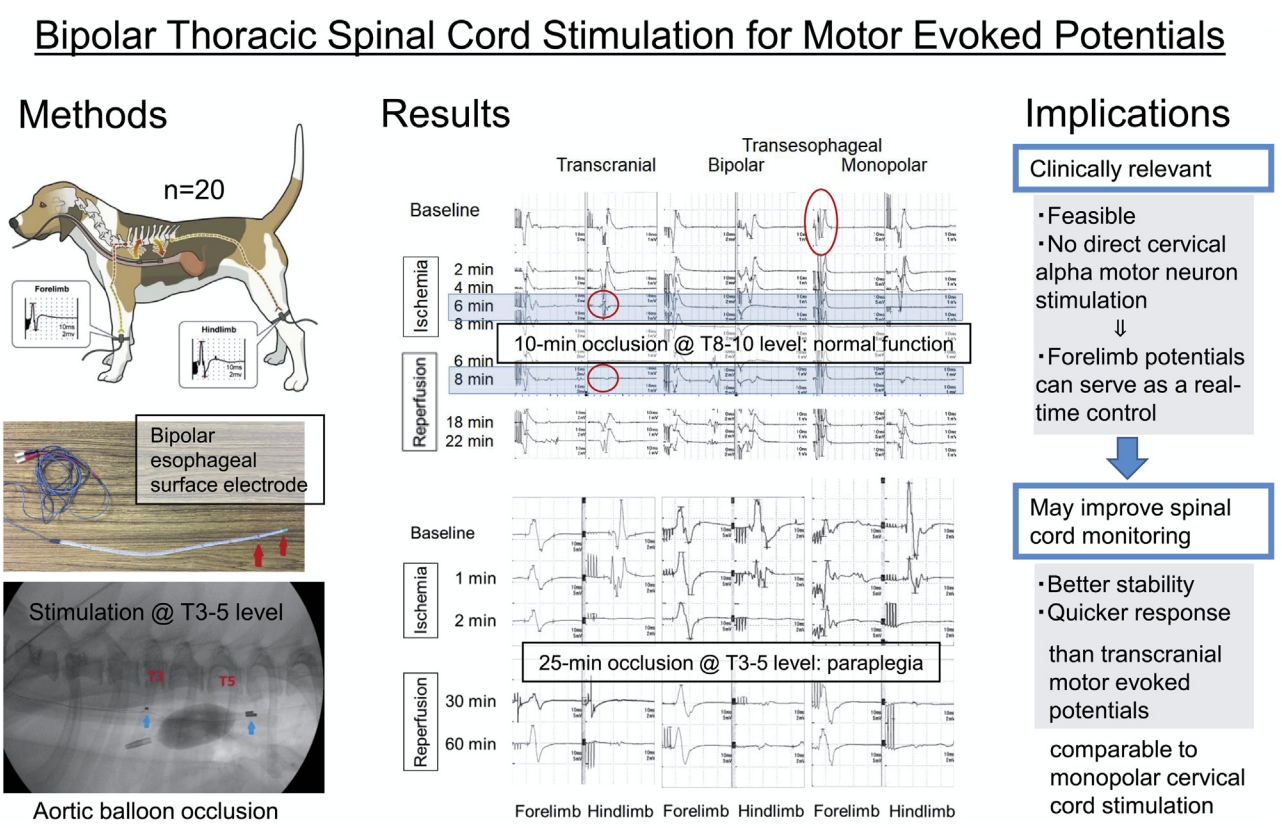


FIGURE 4. In the canine experiments, bipolar transesophageal thoracic cord stimulation can elicit motor-evoked potentials without direct cervical alpha motor neuron stimulation. It is clinically relevant because the upper limb potentials can serve as a real-time control. It may improve spinal cord monitoring because of its stability and quick response.

intraoperative MEP changes that were reverted by raising perfusion pressure without intercostal reconstruction may have a value in detecting patients at risk, because postoperative hypotension may result in spinal cord ischemia in such patients.

Study Limitations

Because the anatomy of dogs is considerably different from that of humans, the results of the present study could not be directly translated into clinical practice. First, the thickness of the human thoracic vertebral body is approximately 3 cm, 3 times thicker than that of dogs. Second, the lower thoracic esophagus of humans moves away from the spinal cord, so that the esophagus-to-spinal cord distance becomes longer when the stimulation electrode is inserted deeper. This is not the case with the canine esophagus. Third, a dilated descending aorta may displace the esophagus away from the spinal cord. All these factors may raise the threshold stimulation intensity to elicit MEPs. Therefore, stimulation at the midthoracic level may not be possible, and better stability may not be reproduced in clinical practice. Indeed, in our preliminary clinical study, we had to modify the stimulation condition to elicit lower limb potentials.[16] However, the waveforms obtained in our clinical study suggested that direct cervical cord stimulation could be avoided. The better stability and more rapid response to ischemia/reperfusion, which also seemed to be reproduced in the preliminary clinical experience, remain to be confirmed in further clinical studies.

CONCLUSIONS

biTE-MEP is comparable to monoTE-MEP regarding its stability, response time to ischemia, and prognostic value, and it is effective in avoiding direct cervical cord stimulation in canine experiments (Figure 4). Whether these results can be directly translated into clinical practice remains to be evaluated, because the anatomical condition in the patients cannot be replicated in the present experimental model.

Conflict of Interest Statement

The authors reported no conflicts of interest.

The *Journal* policy requires editors and reviewers to disclose conflicts of interest and to decline handling or reviewing manuscripts for which they may have a conflict of interest. The editors and reviewers of this article have no conflicts of interest.

The authors thank Toshihiko Hasegawa for providing invaluable technical assistance.

References

1. Miyamoto K, Ueno A, Wada T, Kimoto S. A new and simple method of preventing spinal cord damage following temporary occlusion of the thoracic aorta by draining the cerebrospinal fluid. *J Cardiovasc Surg (Torino)*. 1960;1:188-97.
2. Griepp RB, Griepp EB. Spinal cord perfusion and protection during descending thoracic and thoracoabdominal aortic surgery: the collateral network concept. *Ann Thorac Surg*. 2007;83:S865-9.
3. Laschinger JC, Cunningham JN Jr, Baumann FG, Cooper MM, Krieger KH, Spencer FC. Monitoring of somatosensory evoked potentials during surgical procedures on the thoracoabdominal aorta. III. Intraoperative identification of vessels critical to spinal cord blood supply. *J Thorac Cardiovasc Surg*. 1987;94:271-4.
4. Shiiya N, Yasuda K, Matsui Y, Sakuma M, Sasaki S. Spinal cord protection during thoracoabdominal aortic aneurysm repair: results of selective reconstruction of the critical segmental arteries guided by evoked spinal cord potential monitoring. *J Vasc Surg*. 1995;21:970-5.
5. Jacobs MJ, Mess W, Mochtar B, Nijenhuis RJ, Statius van Eps RG, Schurink GW. The value of motor evoked potentials in reducing paraplegia during thoracoabdominal aneurysm repair. *J Vasc Surg*. 2006;43:239-46.
6. Schepens M, Dossche K, Morshuis W, Heijmen R, van Dongen E, Ter Beek H, et al. Introduction of adjuncts and their influence on changing results in 402 consecutive thoracoabdominal aortic aneurysm repairs. *Eur J Cardiothorac Surg*. 2004;25:701-7.
7. Meylaerts SA, Jacobs MJ, van Iterson V, De Haan P, Kalkman CJ. Comparison of transcranial motor evoked potentials and somatosensory evoked potentials during thoracoabdominal aortic aneurysm repair. *Ann Surg*. 1999;230:742-9.
8. Keyhani K, Miller CC III, Estrera AL, Wegryn T, Sheinbaum R, Safi HJ. Analysis of motor and somatosensory evoked potentials during thoracic and thoracoabdominal aortic aneurysm repair. *J Vasc Surg*. 2009;49:36-41.
9. Afifi RO, Sandhu HK, Zaidi ST, Trinh E, Tanaka A, Miller CC III, et al. Intercostal artery management in thoracoabdominal aortic surgery: to reattach or not to reattach? *J Thorac Cardiovasc Surg*. 2018;155:1372-8.
10. Kawanishi Y, Munakata H, Matsumori M, Tanaka H, Yamashita T, Nakagiri K, et al. Usefulness of transcranial motor evoked potentials during thoracoabdominal aortic surgery. *Ann Thorac Surg*. 2007;83:456-61.
11. Tsutsui S, Iwasaki H, Yamada H, Hashizume H, Minamide A, Nakagawa Y, et al. Augmentation of motor evoked potentials using multi-train transcranial electrical stimulation in intraoperative neurophysiologic monitoring during spinal surgery. *J Clin Monit Comput*. 2015;29:35-9.
12. Penfield W, Boldrey E. Somatic motor and sensory representation in the cerebral cortex of man as studied by electrical stimulation. *Brain*. 1937;60:389-443.
13. McCreery DB, Agnew WF, Yuen TG, Bullara L. Charge density and charge per phase as cofactors in neural injury induced by electrical stimulation. *IEEE Trans Biomed Eng*. 1990;37:996-1001.
14. Tsuda K, Shiiya N, Takahashi D, Ohkura K, Yamashita K, Kando Y. Transoesophageal spinal cord stimulation for motor-evoked potentials monitoring: feasibility, safety and stability. *Eur J Cardiothorac Surg*. 2015;48:245-51.
15. Tsuda K, Shiiya N, Takahashi D, Ohkura K, Yamashita K, Kando Y, et al. Transesophageal versus transcranial motor evoked potentials to monitor spinal cord ischemia. *J Thorac Cardiovasc Surg*. 2016;151:509-17.
16. Shiiya N, Tsuda K, Yamanaka K, Takahashi D, Washiyama N, Yamashita K, et al. Clinical feasibility and safety of transoesophageal motor-evoked potential monitoring. *Eur J Cardiothorac Surg*. 2020;57:1076-82.
17. Etz CD, Homann TM, Luehr M, Kari FA, Weisz DJ, Kleinman G, et al. Spinal cord blood flow and ischemic injury after experimental sacrifice of thoracic and abdominal segmental arteries. *Eur J Cardiothorac Surg*. 2008;33:1030-8.
18. Etz CD, Luehr M, Kari FA, Bodian CA, Smego D, Plestis KA, et al. Paraplegia after extensive thoracic and thoracoabdominal aortic aneurysm repair: does critical spinal cord ischemia occur postoperatively? *J Thorac Cardiovasc Surg*. 2008;135:324-30.
19. Wong DR, Coselli JS, Amerman K, Bozinovski J, Carter SA, Vaughn WK, et al. Delayed spinal cord deficits after thoracoabdominal aortic aneurysm repair. *Ann Thorac Surg*. 2007;83:1345-55.
20. Azizzadeh A, Huynh TT, Miller CC III, Estrera AL, Porat EE, Sheinbaum R, et al. Postoperative risk factors for delayed neurologic deficit after thoracic and thoracoabdominal aortic aneurysm repair: a case-control study. *J Vasc Surg*. 2003;37:750-4.

Key Words: spinal cord ischemia, aortic surgery, motor evoked potentials, transesophageal stimulation