Transesophageal versus transcranial motor evoked potentials to monitor spinal cord ischemia

Kazumasa Tsuda, MD,[a] Norihiko Shiiya, MD, PhD,[a] Daisuke Takahashi, MD,[a] Kazuhiro Ohkura, MD, PhD,[a] Katsushi Yamashita, MD, PhD,[a] Yumi Kando, MD,[a] and Yoshifumi Arai, MD, PhD[b]

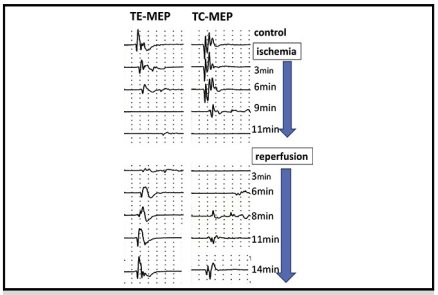
ABSTRACT

Objectives: We have previously reported that transesophageal motor evoked potential is feasible and more stable than transcranial motor evoked potential. This study aimed to investigate the efficacy of transesophageal motor evoked potential to monitor spinal cord ischemia.

Methods: Transesophageal and transcranial motor evoked potentials were recorded in 13 anesthetized dogs at the bilateral forelimbs, anal sphincters, and hindlimbs. Spinal cord ischemia was induced by aortic balloon occlusion at the 8th to 10th thoracic vertebra level. In the 12 animals with motor evoked potential disappearance, occlusion was maintained for 10 minutes (n = 6) or 40 minutes (n = 6) after motor evoked potential disappearance. Neurologic function was evaluated by Tarlov score at 24 and 48 hours postoperatively.

Results: Time to disappearance of bilateral motor evoked potentials was quicker in transesophageal motor evoked potentials than in transcranial motor evoked potentials at anal sphincters (6.9 ± 3.1 minutes vs 8.3 ± 3.4 minutes, $P = .02$) and hindlimbs (5.7 ± 1.9 minutes vs 7.1 ± 2.7 minutes, $P = .008$). Hindlimb function was normal in all dogs in the 10-minute occlusion group, and motor evoked potentials recovery (>75% on both sides) after reperfusion was quicker in transesophageal motor evoked potentials than transcranial motor evoked potentials at hindlimbs (14.8 ± 5.6 minutes vs 24.7 ± 8.2 minutes, $P = .001$). At anal sphincters, transesophageal motor evoked potentials always reappeared (>25%), but transcranial motor evoked potentials did not in 3 of 6 dogs. In the 40-minute occlusion group, hindlimb motor evoked potentials did not reappear in 4 dogs with paraplegia. Among the 2 remaining dogs, 1 with paraparesis (Tarlov 3) showed delayed recovery (>75%) of hindlimb motor evoked potentials without reappearance of anal sphincter motor evoked potentials. In another dog with spastic paraplegia, transesophageal motor evoked potentials from the hindlimbs remained less than 20%, whereas transcranial motor evoked potentials showed recovery (>75%).

Conclusions: Transesophageal motor evoked potentials may be superior to transcranial motor evoked potentials in terms of quicker response to spinal cord ischemia and better prognostic value. (J Thorac Cardiovasc Surg 2016;151:509-17)



Response to ischemia/reperfusion was quicker in TE-MEP than in TC-MEP.

Central Message

TE-MEP is superior to TC-MEP in terms of quicker response to spinal cord ischemia and better prognostic value.

Perspective

This study addresses the low specificity of TC-MEP to monitor spinal cord ischemia during thoracic aortic surgery, which is largely due to its instability. The results suggest that TE-MEP, which we have shown is more stable than TC-MEP, is superior to TC-MEP in terms of quicker response to ischemia and better prognostic value. It will provide technical ease and improved accuracy in the event of clinical application.

See Editorial Commentary page 518.

From the [a]First Department of Surgery, Hamamatsu University School of Medicine, Higashi-ku, Hamamatsu, Shizuoka, Japan; and [b]Department of Pathology, Seirei Hamamatsu Hospital, Naka-ku, Hamamatsu, Shizuoka, Japan.

This work was supported by the Grants-In-Aid for Scientific Research of the Japanese Ministry of Education, Culture, Sports, Science, and Technology (Grant 24592056).

Read at the 95th Annual Meeting of The American Association for Thoracic Surgery, Seattle, Washington, April 25-29, 2015.

Received for publication April 13, 2015; revisions received Aug 5, 2015; accepted for publication Aug 22, 2015; available ahead of print Oct 13, 2015.

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).

0022-5223/$36.00

http://dx.doi.org/10.1016/j.jtcvs.2015.08.120

Abbreviations and Acronyms
MEP = motor evoked potential
TE = transesophageal
TC = transcranial

Myogenic transcranial (TC) motor evoked potentials (MEPs) have been widely used to monitor spinal cord ischemia during aortic surgery as a component of a multidisciplinary spinal cord protection strategy.[1,2] This is because of the quickness in response and high sensitivity compared with other monitoring methods, which enables surgeons to react promptly to spinal cord ischemia during surgery.[3,4] On the other hand, instability and amplitude fluctuation of TC-MEP have been a clinical problem, which results in the low specificity when the cutoff value is set high. This may explain why the cutoff amplitude of the evoked potentials varies considerably from 25% to 75% of baseline among the investigators.[5,6] In addition, recovery of TC-MEP after transient loss does not guarantee prevention from neurologic dysfunction,[7] which makes the prognostic value of TC-MEP low.[7,8]

We have previously reported that transesophageal (TE) electrical stimulation of the spinal cord can safely and easily elicit myogenic MEPs by conventional equipment for TC-MEP.[9] We found that stimulation intensity was always supra-maximal for TE-MEP, which may be desirable to avoid amplitude fading of myogenic potentials, and interindividual variability is small. This made us hypothesize that specificity and accuracy to spinal cord ischemia may be improved by TE stimulation if its response is at least as quick and sensitive as that of TC-MEP.

The concept of monitoring myogenic potentials elicited by direct stimulation of the spinal cord has been proposed by Mochida and colleagues[10] by the use of epidural electrodes for stimulation. Although they have reported that it can promptly detect spinal cord ischemia induced by thoracic aortic crossclamping, it has rarely been used clinically in aortic surgery because of the need for an epidural electrode. Because our technique of TE spinal cord stimulation differs considerably from epidural stimulation, myogenic response may be completely different. The purpose of the present study is to investigate whether or not response of TE-MEP to spinal cord ischemia is as prompt and accurate as that of TC-MEP in a canine model of spinal cord ischemia.

MATERIALS AND METHODS

Thirteen adult beagle dogs (weight, 12.7-20.0 kg) were used. All animals received humane care in compliance with the "Guide for the Care and Use of Laboratory Animals" published by the National Institutes of Health (publication 85-32, revised 1985). The institutional ethics committee on the use and care of animals approved the experimental protocol (No. 2013014).

Experimental Settings

Methods of anesthesia and instrumentation have been reported.[9] Briefly, the animals were anesthetized with intravenous infusion of propofol (12-24 mg/kg/h) and remifentanil (12-24 μg/kg/h) without muscle relaxant, and were intubated and 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 a SEN-4100 equipment for electrical stimulation (Nihon Kohden, Tokyo, Japan). For TC stimulation of the brain motor area, a cathode was placed on the C4 position and an anode on the C3 position of the international 10-20 system. TE stimulation of the spinal cord was performed between the handmade esophageal luminal surface electrode (cathode) and a nuchal subcutaneous needle electrode (anode) that was set at about the fourth to fifth thoracic spine level as a target. A train of 5 rectangular pulses was used with a 2.0-ms interstimulus interval at 500V with a 0.05-ms pulse width. MEPs were recorded at the bilateral abductor pollicis brevis muscles, anterior tibial muscles, and external anal sphincter muscles. Needle electrodes were used to record compound muscle action potentials from the limbs, and a plug-type electrode (a special order product, Nihon Kohden) was used to record external anal sphincter muscle potentials.

Experimental Model

Spinal cord ischemia was induced by inflating an aortic occlusion balloon catheter (Reliant, Medtronic Inc, Minn) at the eighth to tenth thoracic vertebra level. The balloon was introduced through a femoral artery under fluoroscopic control after 100 U/kg heparin was administered (Figure 1). To confirm aortic occlusion, arterial blood pressure was continuously recorded at the contralateral femoral artery and left common carotid artery. No attempt was made to control proximal blood pressure during aortic occlusion.

Experimental Protocol

Before thoracic aortic occlusion, the terminal aorta was balloon-occluded in each animal for 30 minutes to evaluate the influence of lower-limb ischemia on MEPs. Then the balloon was deflated and advanced to the target level of the descending thoracic aorta. After an interval of 15 minutes, the balloon was inflated and the descending thoracic aorta was occluded. TC- and TE-MEP were recorded every 2 or 3 minutes, and time from aortic occlusion to disappearance of waveforms was measured on each recording site. MEP did not disappear by descending aortic occlusion in 1 dog, and this animal was not used for subsequent experiment. In the remaining 12 animals, balloon occlusion was maintained for an additional 10 minutes (n = 6) or 40 minutes (n = 6) after disappearance of all MEPs. Time to MEP disappearance ranged from 4 to 18 minutes, so that total duration of thoracic aortic occlusion was 15 to 21 minutes in the 10-minute occlusion group and 44 to 58 minutes in the 40-minute occlusion group. Then balloon occlusion was released, and time to reappearance of waveform on both MEPs at the anal sphincter and hindlimbs was measured up to 60 minutes after reperfusion.

The animals 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 monitor results according to the modified Tarlov classification (0: no movement of hindlimbs, 1: perceptible movement of the joints of the hindlimbs, 2: good movement but unable to stand, 3: able to stand and walk, 4: complete recovery) at the completion of and 24 and 48 hours after the procedure. Animals with paralysis were