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Delayed response of transcranial myogenic motor-evoked potential monitoring to spinal cord ischemia during repair surgery for descending thoracic aortic aneurysm

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Abstract
The efficacy of transcranial myogenic motor-evoked potential (tc-MEP) monitoring during thoracic aortic surgery has been the subject of some reports, because tc-MEP monitoring can rapidly reflect changes in spinal cord blood flow during thoracic aortic cross-clamping. In this article, we present a case in which delayed loss of tc-MEP signals was observed after cross-clamping of the descending thoracic aorta. We must be aware that tc-MEPs recorded from the lower extremities can fail to provide rapid detection of spinal cord ischemia in the upper thoracic level after cross-clamping of the descending thoracic aorta.

Key words tc-MEP · Aortic surgery · Paraplegia · Spinal cord ischemia

Introduction

Ischemic spinal cord injury after aortic surgery remains a devastating complication, because patients with postoperative paraplegia and paraparesis have decreased survival rates [1]. Consideration of the clinical importance of spinal ischemia has led to efforts to characterize the potency of numerous pharmacological, surgical, and physical interventions to reduce spinal neuronal degeneration during periods of transient spinal cord ischemia. The efficacy of transcranial myogenic motor-evoked potential (tc-MEP) monitoring during thoracic aortic surgery has been the subject of some reports, because tc-MEP monitoring can rapidly reflect changes in spinal cord blood flow during thoracic aortic cross-clamping [2–4]. In general, it is reasonable that tc-MEPs are lost almost instantaneously after cross-clamping of the thoracic aorta, probably as a result of synaptic failure in the anterior horn. Here, however, we report a patient who developed paraparesis after surgery in which delayed loss of tc-MEP signals was observed after cross-clamping of the descending thoracic aorta.

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Case report

A 63-year-old, 83-kg man was hospitalized emergently because of the impending rupture of a descending thoracic aneurysm. The patient's medical history was notable for hypertension and diabetes mellitus. Computed tomography revealed the presence of an aneurysm, beginning distal to the left subclavial artery and extending to the level of the diaphragm.

General anesthesia was induced with 1 mg·kg^{-1} propofol, 1 mg·kg^{-1} ketamine, and 1.5 μg·kg^{-1} fentanyl, and maintained with infusions of propofol and ketamine and intermittent injections of fentanyl. The patient was intubated using a double-lumen endotracheal tube, following the intravenous infusion of 1 mg·kg^{-1} suxamethonium; then 0.05 mg·kg^{-1} vecuronium was administered after a compound myogenic action potential evoked by transcranial electrical stimulation was recorded as the control value for tc-MEPs. Anesthesia was initially maintained with the propofol infusion at a rate of 80 μg·kg^{-1}·min^{-1} and ketamine at a rate of 1 mg·kg^{-1}·h^{-1}. The infusion rate of propofol was varied to maintain the bispectral index (BIS) value between 50 and 60 [5]. Transcranial electrical stimulation was performed by using a multipulse stimulator (D-185; Digitimer, Welwyn Garden City, United Kingdom). Stimulation was performed by train-of-five pulses with an interstimulus interval of 2 ms. The outputs were delivered to the scalp by a single pair of 14.5-mm silver disk electrodes applied to C3 (cathode) and C4 (anode) (International 10–20 System). The stimulus intensity of the transcranial stimulation was determined at the beginning of MEP

monitoring and was set just supramaximal to each stimulus. Compound muscle action potentials were recorded from the skin over the left anterior tibial muscle. The patient was placed in the right decubitus position with the lower half of the body rotated posteriorly to facilitate right femoral vessel cannulation, and a contralateral femoral arterial line was used to assess arterial pressure.

Stone's incision was used as the surgical approach to the aneurysm. The entire aneurysm was exposed, with tapes applied for both distal and proximal aortic clamping. A full dose of heparin (300 $IU \cdot kg^{-1}$) was administered. Adjuncts for aortic clamping consisted of normothermic partial femoral vein-femoral artery bypass. The cardiopulmonary bypass circuit was an open system, and included a reservoir, oxygenator, and heat exchanger. Distal aortic perfusion was started before the aortic cross-clamping was performed. After the start of distal aortic perfusion, the proximal aorta was clamped distal to the left subclavian artery and anastomosed to the Dacron prosthesis. The aortic cross-clamps were then moved distally; one was placed distal to the anastomosis and another above the diaphragm. We then observed changes in tc-MEP amplitudes for 5 min without any surgical procedures. Although there was a maximal decrease in the amplitude of tc-MEPs to 45.8% of the preclamping value, the decrease was not judged as significant. The aneurysm was opened 7 min after the aortic cross-clamping. After the opening of the aneurysm, the tc-MEP amplitudes varied between 50% and 92% of the preclamping value until 20 min after the aortic cross-clamping. The amplitude of the tc-MEPs started to decrease gradually 20 min after the aortic cross-clamping and, in spite of the mean pump flow being changed (maximum, 3.2 $l \cdot min^{-1}$) to increase the distal blood pressure (maximum mean arterial pressure in the femoral artery, 109 mmHg), the waveform disappeared completely 25 min after the aortic cross-clamping (Fig. 1). No additional vecuronium was administered before the aortic cross-clamping, and the amplitude of an action potential recorded from the left anterior tibial muscle by percutaneous electrical stimulation of the common peroneal nerve was the same as that of the control level. Although two pairs of intercostal arteries (seventh and ninth) were reconstructed, we did not observe any tc-MEP waveform until the end of the surgery.

In the intensive care unit, the patient awoke, was alert, and followed our commands. However, he was unable to move his legs the day after surgery. Neurological findings determined by a neurologist revealed spastic paraplegia, indicating ischemic spinal cord injury. Spinal magnetic resonance imaging 10 days after the surgery showed a signal change in the cord between Th4 and 6, suggesting ischemic spinal cord injury.

Discussion

In the present patient, tc-MEPs recorded from the anterior tibial muscle were lost, for more than 20 min after the clamping of the thoracic aorta, with the result that the patient had paraparesis after surgery. According to neurological examinations by neurologists, and magnetic resonance imaging of the spinal cord, this paraparesis appeared to be due to ischemic injury in the upper thoracic segment (Th 4–6) of the spinal cord.

According to a previous study [6], sensory evoked potential (SEP) monitoring was used to monitor functions in the posterior columns of the spinal cord, while tc-MEPs were used to assess functions in the anterior and anterolateral columns. With respect to blood supply in the spinal cord, the posterior one-third of the spinal cord is supplied by a pair of continuous posterior spinal arteries, and the anterior two-thirds are supplied by the discontinuous anterior spinal artery [7]. The anterior spinal artery receives its blood supply from radicular branches. The major radicular supply to the anterior spinal artery in the thoracic and upper abdominal region is from three to five anterior radicular arteries. Because these arteries originate from Th9 to L3 in most patients, the occlusion of the lower thoracic and/or upper abdominal aorta can induce ischemia of the anterior spinal cord motor tracts, resulting in paraplegia. In general, it is believed that SEP fails to directly monitor the function of spinal motor tracts, because these are at the anterior of the spinal cord supplied by the anterior spinal artery.

Perioperative monitoring of myogenic MEPs in response to transcranial stimulation of the motor cortex provides a method to monitor the functional integrity of descending motor pathways. The main descending fiber tract is the corticospinal tract, which arises predominantly from the precentral gyrus. The fibers descend to the pyramid of the medulla and terminate, directly or by means of inhibitory interneurons, on the motor neurons from which axons emerge from the spinal cord as the ventral root to the muscles [7–9]. Some experimental data have demonstrated rapid disappearance of tc-MEPs after aortic occlusion and their reappearance after reperfusion, providing rapid assessment of the adequacy of spinal cord blood flow. This rapid assessment of spinal cord blood flow offers several advantages in a surgical approach that includes spinal cord protection; e.g., identification of critical segmental arteries during aortic cross-clamping and assessment of adequate blood supply via the reattachment of segmental arteries [10]. Therefore, it is believed that tc-MEP monitoring can provide useful information to clinicians for making decisions with regard to timely interventions aimed at correcting ischemic conditions and preserving spinal cord blood flow.

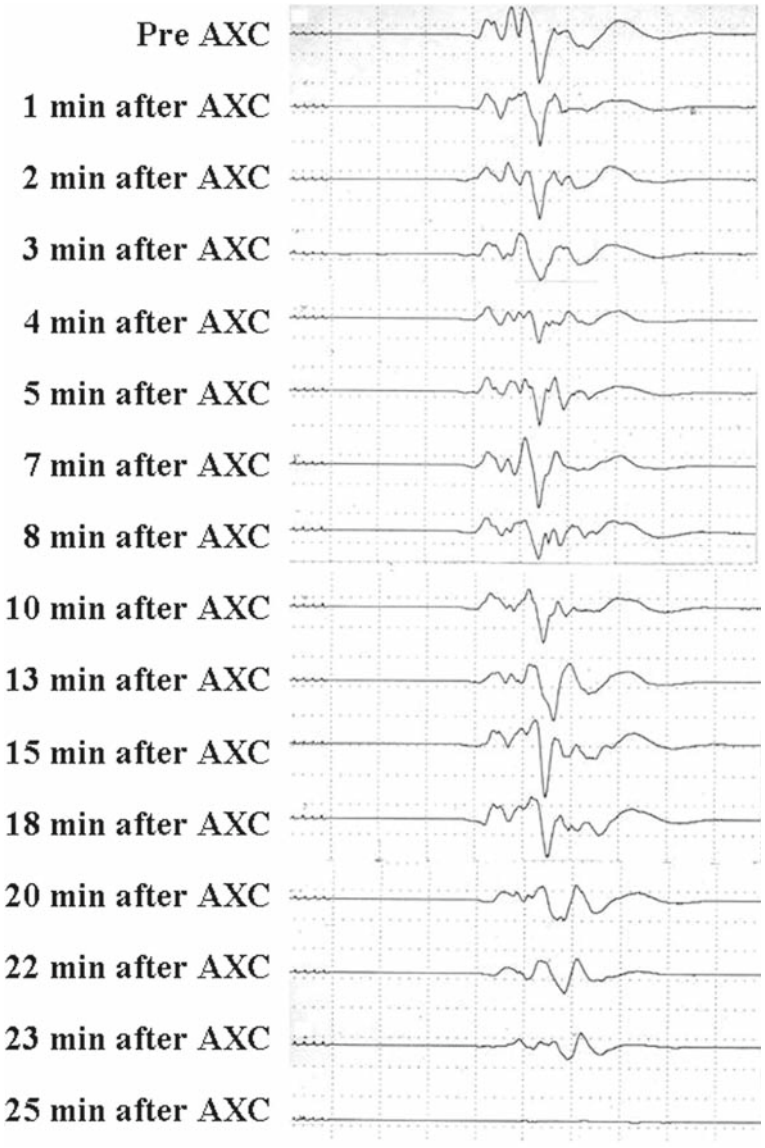

Fig. 1. Motor-evoked potentials (MEPs) recorded from the left anterior tibial muscle. The amplitude of the MEPs started to decrease gradually 20 min after aortic cross-clamping (*AXC*) and the waveform disappeared completely 25 min after the AXC

Although, in general, the criterion for spinal cord ischemia during sensory-evoked potential monitoring is a decrease in amplitude to less than 50% of baseline [11], the rationale for the criterion of a decrease in amplitude to less than 25% of baseline, which is used clinically to detect spinal cord ischemia, is based on theoretical considerations and has been confirmed empirically [2]. Experimental data using a porcine spinal cord ischemia model supported the validity of the empirical criterion (tc-MEP amplitude less than 25% of baseline) for initiating interventions to maintain spinal cord perfusion pressure during surgical procedures involving a thoracoabdominal aneurysm [12]. In our patient, therefore, we did not judge a decrease of tc-MEP amplitude to 45.8% of the preclamping value as significant.

It is known that the thoracic portion of the anterior spinal artery is supplied by the radicular arteries (two or three thoracic radicular arteries, and one or two lumbar radicular arteries) [13]. Hence, normally, two small radicular arteries supply the spinal cord between C8 and Th9; the remainder of the spinal cord is supplied largely by the artery of Adamkiewicz, with minor contribution from an infrarenal radicular artery [14,15]. However, it appears that spinal cord perfusion depends to a great extent on a collateral circulation, rather than being dependent on several segmental arteries [16], and this can explain the clinical observation that the more extensive the aortic involvement, the higher is the rate of paraplegia [17]. In our patient, we speculated that a relatively long segmental clamp of the descending thoracic aorta had induced the ischemia in the upper thoracic level of the spinal cord.

As in the present patient, Meylaerts et al. [2] observed a delayed loss of tc-MEP signals after the clamping of a thoracic segment in three patients. With tc-MEP

monitoring, the signals traverse the corticospinal tract arising predominantly from the precentral gyrus, spinal motor neurons, and neuromuscular junctions. The corticospinal tract and spinal motor neurons are connected by synapses. It is known that synaptic transmission is much more sensitive to ischemia than axonal transmission [18,19]. In tc-MEPs recorded from the anterior tibial muscles, the synapses a located between the L4 and S1 segments of the spinal cord. In our patient, with ischemia in the upper thoracic level of the spinal cord, this ischemic insult may have affected the descending axons in the corticospinal pathway of the tc-MEPs, but not the synaptic connections between the descending axons and the spinal motor neurons. Lips et al. [20] performed an experiment in pigs, in which spinal cord blood flow in either the thoracic or lumbar segments was reduced selectively, as evidenced by laser-Doppler flowmetry, and myogenic tc-MEPs were recorded from the lower limbs. This experiment demonstrated that a severe reduction of lumbar spinal cord blood flow resulted in the rapid loss of myogenic tc-MEPs, whereas a similar blood flow reduction in the thoracic spinal cord resulted in a relatively slow loss of MEPs. According to these results, it is likely that tc-MEPs recorded from the lower extremities would fail to provide rapid detection of ischemia in the upper thoracic level after cross-clamping of the descending thoracic aorta, consistent with the findings in the present patient.

In this article, we presented a case in which delayed loss of tc-MEP signals was observed after cross-clamping of the descending thoracic aorta. We must be aware that tc-MEPs recorded from the lower extremities can fail to provide rapid detection of ischemia in the upper thoracic level of the spinal cord after aortic cross-clamping.

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References

1. Svensson LG, Crawford ES, Hess KR, Coselli JS, Safi HJ. Experience with 1509 patients undergoing thoracoabdominal aortic operations. J Vasc Surg. 1993;17:357–68.
2. 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.
3. de Haan P, Kalkman CJ, De Mol BA, Ubags LH, Veldman DJ, Jacobs MJ. Efficacy of transcranial motor-evoked myogenic potentials to detect spinal cord ischemia during operations for thoracoabdominal aneurysms. J Thorac Cardiovasc Surg. 1997; 113:87–100.
4. Kakinohana M, Kawabata T, Miyata Y, Sugahara K. Myogenic transcranial motor evoked potentials monitoring cannot always predict neurological outcome after spinal cord ischemia in rats. J Thorac Cardiovasc Surg. 2005;129:46–52.
5. Kakinohana M, Nakamura S, Fuchigami T, Miyata Y, Sugahara K. Influence of the descending thoracic aortic cross clamping on bispectral index value and plasma propofol concentration in humans. Anesthesiology. 2006;104:939–43.
6. Machida M, Weinstein SL, Yamada T, Kimura J, Toriyama S. Dissociation of muscle action potentials and spinal somatosensory evoked potentials after ischemic damage of spinal cord. Spine. 1988;13:1119–24.
7. Gharagozloo F, Neville RF Jr, Cox JL. Spinal cord protection during surgical procedures on the descending thoracic and thoracoabdominal aorta: a critical overview. Semin Thorac Cardiovasc Surg. 1998;10:73–86.
8. Elger CE, Speckmann EJ, Caspers H, Janzen RWC. Corticospinal connections in the rat. I. Monosynaptic and polysynaptic responses of cervical motorneurons to epicortical stimulation. Exp Brain Res. 1977;28:385–404.
9. Fehlings MG, Tator CH, Dean Linden R, Riper IR. Motor evoked potentials recorded from normal and spinal cord-injured rats. Neurosurgery. 1987;20:125–30.
10. Ryder J, Zappulla R, Nieves J. Motor evoked potentials elicited from pyramidal stimulation and recorded from the spinal cord in the rat. Neurosurgery. 1991;28:550–8.
11. de Haan P, Kalkman CJ. Spinal cord monitoring: somatosensory- and motor-evoked potentials. Anesthesiol Clin North America. 2001;19:923–45.
12. Brown RH, Nash CL, Berilla JA, Amaddio MD. Cortical evoked potential monitoring: a system for intraoperative monitoring of spinal cord function. Spine. 1984;9:256–61.
13. Lips J, de Haan P, de Jager SW, Vanicky I, Jacobs MJ, Kalkman CJ. The role of transcranial motor evoked potentials in predicting neurologic and histopathologic outcome after experimental spinal cord ischemia. Anesthesiology. 2002;97:183–91.
14. Urban M. Spinal cord ischemia. Recognition and prevention. In: Black TJJ, editor. Neuroprotection. Baltimore: Williams & Wilkins; 1997. p. 135–62.
15. Charagozloo F, Larson J, Dausmann MJ, Neville RF Jr, Gomes MN. Spinal cord protection during surgical procedures on the descending thoracic and thoracoabdominal aorta: review of current techniques. Chest. 1997;109:799–809.
16. Domisse GF. The blood supply of the spinal cord: a critical vascular zone in spinal surgery. J Bone Joint Surg. 1974;56B: 225–35.
17. Miyairi T, Kotsuka Y, Morota T, Kubota H, Shibata K, Ikeda Y, et al. Paraplegia after open surgery using endovascular stent graft for aortic arch aneurysm. J Thorac Cardiovasc Surg. 2001;121: 1240–3.
18. Crawford ES, Crawford JL, Safi HJ, Coselli JS, Hess KR, Brooks B, Norton HJ, Glaeser DH. Thoracoabdominal aortic aneurysms: preoperative and intraoperative factors determining immediate and long-term results of operations in 605 patients. J Vasc Surg. 1986;3:389–404.
19. Konard PE, Tacker WA, Levy WJ, Reedy DP, Cook JR, Greddes LA. Motor evoked potentials in dog: effects of global ischemia on spinal cord and peripheral nerve signals. Neurosurgery. 1987;20: 117–24.
20. Lips J, de Haan P, Bouma GJ, Jacobs MJ, Kalkman CJ. Delayed detection of motor pathway dysfunction after selective reduction of thoracic spinal cord blood flow in pigs. J Thorac Cardiovasc Surg. 2002;123:531–8.