Prolonged loss of leg myogenic motor evoked potentials during thoracoabdominal aortic aneurysm repair, without postoperative paraplegia

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Abstract
No postoperative paraplegia occurred in a patient whose leg myogenic motor evoked potentials (mMEPs) disappeared during thoracoabdominal aortic aneurysm repair. A 69-year-old man underwent resection and repair of a type III (Crawford classification) thoracoabdominal aneurysm. An epidural catheter was placed into the epidural space for epidural cooling, and a Swan-Ganz catheter was placed into the subarachnoid space for cerebrospinal fluid (CSF) drainage. Continuous CSF pressure and temperature measurement was carried out the day before surgery. The mMEPs gradually disappeared 10 min after proximal double aortic clamping and complete aortic transection. Selective perfusion of intercostal arteries was started about 20 min after the loss of the mMEPs, but the mMEPs were not restored. Possibly, spinal cord hyperemia, induced by selective perfusion of the intercostal vessels, narrowed the subarachnoid space so that CSF could not be satisfactorily drained during surgery. The spinal cord hyperemia may have decreased spinal function and suppressed the leg mMEPs. The persistence of the loss of mMEPs was undeniably due to the influence of the anesthetic agent or a perfusion disorder in the lower-extremity muscles. Of note, moderate spinal cord hypothermia and postoperative CSF drainage probably resulted in improved lower-limb motor function.

Key words Thoracoabdominal aneurysm · Myogenic motor evoked potential · Spinal cord hyperemia · Moderate spinal cord hypothermia · Cerebral spinal drainage

Introduction

Spinal cord ischemia is one of the most severe postoperative complications of thoracoabdominal aortic aneurysm repair. Several strategies for protecting against spinal cord injury have been tried. These include: mod-erate systemic hypothermia with atrio-femoral bypass [1], cerebrospinal drainage [2], regional hypothermia with epidural cooling [3], and revascularization of the segmental artery with profound hypothermia and circulatory arrest [4]. This report describes a patient in whom myogenic motor evoked potentials (mMEPs) disappeared, without the onset of paraplegia, following the elective repair of a thoracoabdominal aneurysm with moderate spinal cord hypothermia and drainage of cerebrospinal fluid (CSF).

Case report

A 69-year-old man presented to the Niigata City General Hospital (Niigata, Japan) with a type III (Crawford classification) thoracoabdominal aneurysm, for resection and repair. His medical history included a Y-type vascular graft to a ruptured abdominal aortic aneurysm and total arch and descending aorta replacement for a type-I descending aortic aneurysm (DeBakey classification).

Under local anesthetic, a 16-gauge epidural catheter (Epidural Minipack; SIMS Poretx, Hythe, UK) was placed 5 cm into the epidural space at the T11–12 interspace, using a 15-gauge thin-walled Tuohy needle for epidural cooling. A 5F Swan-Ganz catheter (Edwards LifeSciences, Tokyo, Japan) was placed 10 cm into the subarachnoid space at the L3–4 interspace, using a 12-gauge Medicut central venous catheter needle (Nippon Sherwood Medical Industries, Tokyo, Japan) for CSF drainage and continuous CSF pressure and temperature measurement. These catheters were placed the day before surgery. General anesthesia was induced with oxygen, midazolam (5 mg), propofol (50 mg), fentanyl citrate (200 µg), and vecuronium bromide (8 mg). A double-lumen endobronchial tube was placed under fiberoptic bronchoscopic guidance. In addition to standard monitoring, catheters were inserted into the right

radial artery and the left dorsalis pedis artery for continuous pressure monitoring and blood sampling. A Swan-Ganz oxymetry continuous cardiac output (CCO) thermodilution catheter (Edwards LifeSciences, Irvine, CA, USA) and a quad-lumen central venous catheter (Arrow Japan, Tokyo, Japan) were placed through the right internal jugular vein, for hemodynamic measurements and to provide infusion ports for anesthetic and vasoactive medications. mMEPs were evoked with a multipulse transcranial electrical stimulator (Digitimer D185 cortical stimulator; Digitimer, Welwyn Garden City, UK), applied through 1.5-cm diameter circular electrodes placed at C1 and C2 scalp sites (International 10 to 20 System), and recorded from circular electrodes (Vitrodes; Nihon-Kohden, Tokyo, Japan) attached to limb muscles, including the deltoid and rectus femoris muscles. The mMEPs were recorded at a stimulus intensity of about 10% above the level that produced maximal mMEP responses, typically 800V. The stimuli consisted of four pulses, with an inter-stimulus interval of 1ms and a square waveform of 0.1ms width. Maintenance of anesthesia included the use of oxygen, propofol ($5 mg \cdot kg^{-1} \cdot h^{-1}$), and fentanyl citrate ($2800 \mu g$). The bispectral index (BIS) was maintained between 22 and 45. Neuromuscular function was monitored by measuring the acceleromyographic responses of the adductor pollicis muscle to stimulation of the right ulnar nerve by the train-of-four (TOF) technique (TOF-Guard; Organon Teknika, Boxtel, Netherlands). The TOF ratio (T4/T1) was maintained at 50%–70%, with a continuous infusion of vecuronium bromide ($0.1–1.0 mg \cdot h^{-1}$). The extracorporeal circulation system (CompoIII; Tonokura Ika Kogyo, Tokyo, Japan) consisted of two cannulae: a left femoral venous cannula for venous drainage and a left femoral arterial cannula for arterial return. The first phase of the aortic reconstruction consisted of clamping the proximal and distal sides of the aneurysm. The mMEPs elicited from the rectus femoris muscles disappeared after about 3min of cross-clamping, and were restored after unclamping. The second phase consisted of proximal double aortic clamping and complete transection of the aorta. The mMEPs remained normal for up to 5min of cross-clamping but gradually disappeared and were undetectable at about 10min. The mean radial arterial pressure was about 90mmHg, the mean dorsalis pedis arterial pressure was about 40mmHg, and the CSF pressure was about 20mmHg during the aortic clamping. One pair of intercostal vessels was recognized in the aneurysmal sac and was sacrificed because they were very small and did not exhibit free backflow of blood. The distal clamp was moved down and the entire aneurysmal sac was continuously exposed after proximal aortic anastomosis of the homograft. Two pairs of large intercostal vessels were recognized at the T11 and T12 levels in the aneu-

rysmal sac, and these did exhibit free backflow. Selective perfusion of intercostal arteries was started at about 20min after the loss of blood flow maps, but the maps remained unavailable. The celiac artery and superior mesenteric artery were perfused, using extracorporeal shunting. Distal aortic anastomosis was performed and separate intercostal inclusion grafts were reconstructed. The mMEPs elicited from the lower extremities were undetectable by the end of surgery (Fig. 1).

A modified infusion pump was used to deliver iced normal saline solution (1000ml begun at a rate of 5–$12 ml \cdot min^{-1}$ through the epidural catheter), with the goal of decreasing the CSF temperature to 25°C, before and during aortic cross-clamping. When this method failed to lower CSF temperature to the target range, systemic moderate hypothermia (31°C–32°C) was induced, and the CSF temperature dropped to 29.4°C–32.7°C (Fig. 2).

The difference between the mean dorsalis pedis arterial pressure and the CSF pressure was maintained at about 20mmHg by adjustment of the aortic cross-clamp, to ensure flow to the intercostal vessels. Once selective perfusion to the intercostal vessels had commenced, the CSF pressure was gradually increased to 40mmHg, which was in excess of the mean dorsalis pedis arterial pressure. Though we were not able to aspirate CSF to decrease the CSF pressure, aortic declamping after the distal anastomosis temporarily decreased the CSF pressure, which then gradually increased to 40mmHg. Finally, two intercostal arteries were anastomosed to the aortic tube graft in an end-to-side fashion. As soon as the selective perfusion to the intercostal vessels was completed, the CSF pressure rapidly decreased, to 10mmHg (Fig. 3). The mMEPs in both lower extremities remained undetectable at the end of the operation, which lasted for 473min.

CSF was allowed to drain freely by gravity whenever the CSF pressure exceeded $10 cmH_2O$. Mean arterial pressure was maintained at 70–100mmHg. CSF pressure exceeded $10 cmH_2O$ at 9h after operation, and the total CSF drainage volume was 60ml over 15h. The patient had slight movement of the lower limbs 4h postoperatively and was discharged, clinically normal, 16 days postoperatively.

Discussion

The time from the onset of ischemia to spinal cord injury is unknown [5]. The mMEPs and somatosensory evoked potentials provide an effective technique for assessing spinal cord ischemia. Jacobs et al. [6] reported that critical spinal cord ischemia was detectable within 2min after cross-clamping, by decreased mMEP amplitude. Grabitz et al. [7] have reported that loss of spinal
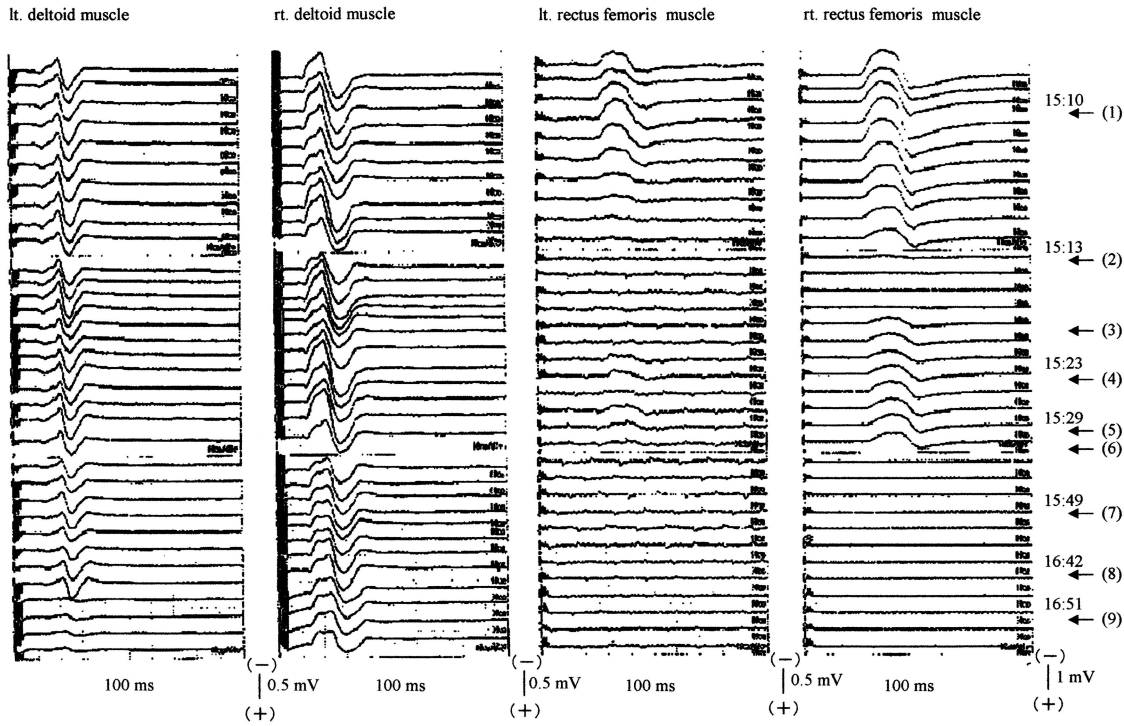
Fig. 1. Recording of myogenic motor evoked potentials (mMEPs) in response to multipulse transcranial electrical stimulation. (*1*) Cross-clamping of proximal and distal sides of the aneurysm. (*2*) Decrease of mMEPs in the bilateral rectus femoris muscles. (*3*) Restoration of mMEPs. (*4*) Double cross-clamping of the proximal side of the aneurysm. (*5*) Complete transection of the distal side of the aneurysm after about 5 min of double cross-clamping. (*6*) Decrease of mMEP in the bilateral rectus femoris muscles, despite increasing the distal perfusion pressure, after about 10 min of cross-clamping. (*7*) Selective perfusion of intercostal arteries at the T11 and T12 levels. (*8*) Aortic declamping after proximal anastomosis. (*9*) Reattachment of intercostal arteries at the T11 and T12 levels. *lt.*, left; *vt.*, right
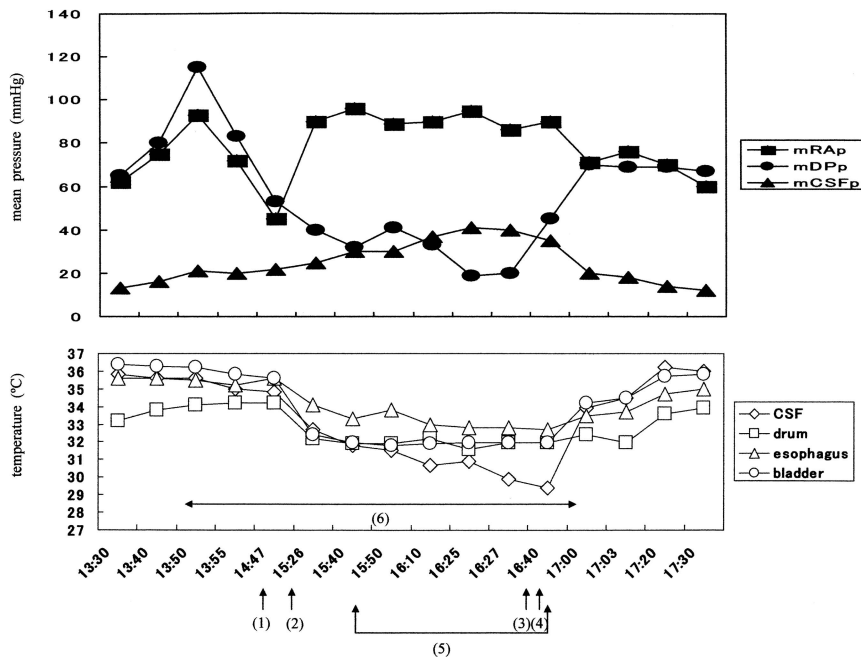

Fig. 2. Graphic display of cerebrospinal fluid (CSF) pressure and blood pressure, along with CSF, drum, esophagus, and bladder temperatures during the surgical procedure. (*1*) Cross-clamping of the proximal and distal sides of the aneurysm. (*2*) Double cross-clamping of the proximal side of the aneurysm. (*3*) Aortic declamping after proximal anastomosis. (*4*) Reattachment of intercostal arteries at the T11 and T12 levels. (*5*) Selective perfusion of the intercostal arteries. (*6*) Epidural cooling. *Closed squares*, right mean radial arterial pressure; *closed circles*, left mean dorsalis pedis arterial pressure; *closed triangles*, mean CSF pressure; *open diamonds*, CSF; *open squares*, drum; *open triangles*, esophagus; *open circles*, bladder
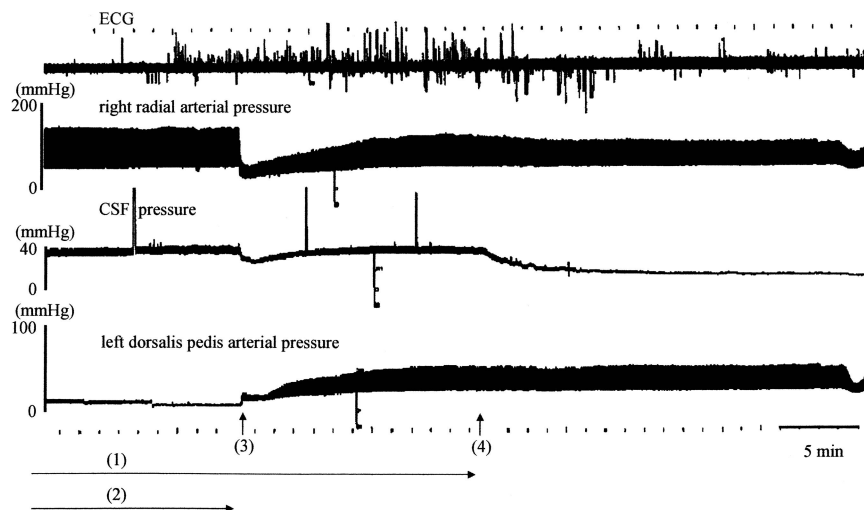
Fig. 3. Recordings of electrocardiogram (*ECG*), right radial arterial pressure, CSF pressure, and left dorsal is pedis arterial pressure from the time of declamping after the distal aortic anastomosis to the end of selective perfusion of the intercostal artery. (*1*) Selective perfusion of the intercostal artery. (*2*) Aortic cross-clamping. (*3*) Aortic declamping after distal aortic anastomosis. (*4*) Reattachment of intercostal arteries at the T11 and T12 levels

somatosensory evoked potential within 15 min after aortic cross-clamping shows poor collateralization and mandates early restoration of the spinal cord blood supply. In the present patient, mMEPs remained normal until about 5 min after proximal double aortic clamping, but were undetectable at about 10 min after this clamping. The mean dorsal is pedis arterial pressure was about 40 mmHg and the CSF pressure was about 20 mmHg when the waveform disappeared. This may imply poor collateralization. Selective extracorporeal perfusion of the intercostal arteries was started at about 20 min after the loss of mMEPs, but the mMEPs were not restored. The CSF pressure reached 40 mmHg after the perfusion was started. The perfusion pressure could not be measured. We speculate that the selective perfusion caused hyperemia with reversible damage in the spinal cord, and thereby decreased spinal cord function. The spinal cord hyperemia may have narrowed the subarachnoid space so that the CSF could not be satisfactorily drained perioperatively. Notably, the cerebrospinal pressure returned to normal immediately upon the termination of intercostal vessel perfusion.

Patients with restored intraoperative mMEPs (unlike patients with persistent loss of mMEPs) are not paraplegic immediately after surgery. Thus, mMEPs are extremely useful in predicting neurological outcome [8]. In the present patient, mMEPs in both upper extremities were recorded, but those in the lower extremities were undetectable at the end of the operation. Loss of mMEPs may not reliably reflect spinal cord function. Blood supply from the extracorporeal circuit to the lower-extremity muscles may have been deficient. It is more difficult to record mMEPs from the lower-extremity muscles than from the upper ones.

Propofol is a potent suppressor of mMEPs induced by single-pulse electrical stimulation [9,10]. Kawaguchi et al. [11] reported that low-dose propofol could be used

effectively as a supplement to ketamine during the intraoperative monitoring of mMEPs to trains of electrical stimuli. In the present patient, propofol anesthesia ($5\,mg\cdot kg^{-1}\cdot h^{-1}$) may have caused the disappearance of mMEPs elicited from the lower extremities. Moreover, the transcranial electrical stimuli responsible for the upper-extremity mMEPs were not transmitted via the cerebrospinal nerve, and they stimulated the muscle directly.

mMEPs can be recorded reliably during moderate subdural hypothermia in pigs. Detection of acute spinal cord ischemia with mMEPs is not delayed when regional CSF temperature is 28°C [12]. The mMEP amplitude in response to a train of pulses did not change at a core temperature of 28°C in rabbits under propofol/ketamine/fentanyl anesthesia [13]. We think that the temperature of 29.4°C did not affect the mMEPs in the present patient. Tsutsumi et al. [14] reported that post-ischemic hypothermia (rectal temperature, 32.5 ± 0.5°C) induced immediately after reperfusion significantly reduced ischemically induced neural damage. Modest hypothermia, for the specific purpose of spinal cord protection during thoracoabdominal aortic aneurysm repair, was achieved by allowing core temperatures to drift into the 32°C–34°C range, which, in animals, has been shown to provide protection [15].

Decompression of the spinal canal by CFS drainage may improve spinal cord circulation during the elective repair of thoracic and thoracoabdominal aneurysms, and may prevent or decrease postoperative neurological injury. Hill et al. [16] reported two patients who had postoperative reversal of delayed-onset paraplegia after postoperative CSF drainage; CSF pressure (10 mmHg) had to be maintained and CSF drainage volume was not restricted. Coselli et al. [17] reported that mean CSF drainage volumes in thoracoabdominal aortic surgery were 261 ± 191 ml (range, 40 to 861 ml). In the present

patient, the CSF drainage volume was 60 ml. Thus, the modest spinal cord hypothermia and postoperative CSF drainage probably resulted in improved lower-limb motor function.

In summary, spinal cord hyperemia induced by the selective perfusion of the intercostal vessels may narrow the subarachnoid space so that CSF cannot be satisfactorily drained perioperatively. The spinal cord hyperemia may decrease spinal function and suppress leg mMEPs. However, without doubt, the persistent loss of mMEPs in our patient was a false-positive effect of the anesthetic agent, or it was due to perfusion disorder in the lower-extremity muscles. Of note, the modest spinal cord hypothermia and postoperative CSF drainage probably resulted in improved lower-limb motor function.

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