

Assessment of intraoperative motor evoked potentials for predicting postoperative paraplegia in thoracic and thoracoabdominal aortic aneurysm repair

Toshinori Horiuchi · Masahiko Kawaguchi · Satoki Inoue · Hironobu Hayashi · Ryuichi Abe · Nobuoki Tabayashi · Shigeki Taniguchi · Hitoshi Furuya

Received: 2 March 2010 / Accepted: 21 October 2010 / Published online: 27 November 2010
© Japanese Society of Anesthesiologists 2010

Abstract

Purpose Monitoring motor evoked potentials (MEPs) has been recognized as a highly reliable method to detect intraoperative spinal cord ischemia (SCI) in aortic repair. However, the data regarding the sensitivity and specificity of MEPs for predicting postoperative paraplegia are limited. We retrospectively assessed the value of intraoperative MEP amplitudes for predicting postoperative paraplegia.

Methods The medical records of 44 patients were reviewed. A train-of-five stimulation was delivered to C3–C4, and MEPs were recorded from the abductor pollicis brevis and the tibialis anterior muscles. The cutoff point for detecting SCI was set at 75% decrease of the baseline MEP. Receiver operating characteristic curves were applied at various cutoff points.

Results Three patients (6.8%) had postoperative paraplegia. The minimum MEP during surgery had 100% sensitivity and 64.9% specificity in predicting paraplegia, and the MEP at the end of surgery had 66.7% sensitivity and 78.0% specificity in predicting paraplegia: only 1 patient, who had borderline paraplegia (right monoparesis), showed a false-negative result. Receiver operating characteristic curves indicated that adequate cutoff points for the minimum MEP during surgery and for the MEP

amplitude at the end of surgery were a 75–90% decrease and a 64–75% decrease of the baseline MEP, respectively. **Conclusion** Monitoring MEPs had relatively high sensitivity and acceptable specificity, with the cutoff point set at 75% decrease of the baseline MEP, for predicting paraplegia and paraparesis. Because of the small sample in our study, further investigations would be necessary to investigate an adequate cutoff point that could predict postoperative paraplegia.

Keywords Motor evoked potentials · Thoracic and thoracoabdominal aortic aneurysm · Paraplegia · Sensitivity · Specificity

Introduction

Postoperative paraplegia including paraparesis is a devastating complication in descending thoracic aortic aneurysm (DTAA) and thoracoabdominal aortic aneurysm (TAAA) repair. It is thought that postoperative paraplegia arises from spinal cord ischemia (SCI) as a result of spinal cord malperfusion. Although the incidence of postoperative paraplegia has tended to decrease in the past two decades even in the highest risk surgical repair, i.e., Crawford type II TAAA [1, 2], its prevention is still a cardinal challenge. There are many review articles about the management of DTAA and TAAA repair to prevent postoperative paraplegia, and most of them point out the importance of a multimodal approach [3–5], including preoperative spinal cord angiography, reconstruction of the intercostal or lumbar artery, cerebrospinal fluid (CSF) drainage, distal aortic perfusion, keeping a higher arterial blood pressure perioperatively, and intraoperative neurophysiological monitoring.

T. Horiuchi (✉) · M. Kawaguchi · S. Inoue · H. Hayashi · R. Abe · H. Furuya
Department of Anesthesiology, Nara Medical University,
840 Shijo-Cho, Kashihara, Nara 634-8522, Japan
e-mail: thoriuch@naramed-u.ac.jp

N. Tabayashi · S. Taniguchi
Department of Thoracic and Cardiovascular Surgery,
Nara Medical University, Kashihara, Nara, Japan

Recently, the monitoring of motor evoked potentials (MEPs) has been recognized as a highly reliable method to detect intraoperative SCI [6–8], and motor evoked potentials-guided intraoperative management to achieve adequate spinal cord perfusion seems to be essential to reduce the incidence of paraplegia in DTAA and TAAA repair. However, the data regarding the sensitivity and specificity of MEPs for predicting postoperative paraplegia in DTAA and TAAA repair are limited. In the present study, we retrospectively assessed our continuous experience of DTAA and TAAA repair with MEP monitoring and analyzed the value of intraoperative MEP amplitudes for predicting postoperative paraplegia.

Patients, materials, and methods

Patients

From 1999 to 2009, a total of 44 patients who underwent graft replacement of the DTAA or (and) TAAA repair surgery with MEP monitoring were included in this study. Table 1 summarizes the patients' demographic data and clinical risk profile. Aneurysms were classified as DTAA, Crawford type I, II, III, or IV, and the number of patients with each aneurysm type is shown in Fig. 1. Twenty-four patients underwent preoperative magnetic resonance angiography (MRA) to localize the Adamkiewicz artery (AKA) and its supplier.

Anesthesia technique

Anesthesia was induced intravenously with 2–5 µg/kg of fentanyl, 0.5–1 mg/kg of ketamine, 0.5–1.5 mg/kg of propofol, and 0.1 mg/kg of vecuronium or 0.6 mg/kg of rocuronium (from 2008, rocuronium was used instead of vecuronium at our hospital, so the most recent 5 patients received rocuronium, not vecuronium). Lung separation was achieved using a double-lumen endotracheal tube. Anesthesia was maintained by continuously infusing ketamine at 1–1.5 mg/kg/h, propofol at 0.5–1 mg/kg/h, and vecuronium at 0.04 mg/kg/h or rocuronium at 0.1–0.3 mg/kg/h. The level of neuromuscular blockade was assessed by the M-response from the abductor pollicis brevis (APB) muscle in response to electrical stimulation of the median nerve at 50 mA. The twitch height of the M-response (T1) was maintained at a level of around 25% of the baseline to achieve stable MEPs. Because the blood concentration of intravenous anesthetics can increase in the upper body after aortic cross-clamping [9], the continuous infusion of muscular relaxant and propofol was stopped or decreased after aortic cross-clamping. Fentanyl was intermittently administered in 40 patients, but the other 4 patients, with

Table 1 Patient demographics and clinical risk profile

Variable ^a	Number of patients (%)
Total number of patients	44 (100)
Age (years)	65.6 ± 12.1
Gender (male)	33 (75.0)
Body weight (kg)	62.9 ± 11.5
Height (cm)	162 ± 9.9
BMI	23.9 ± 3.6
Diabetes mellitus	11 (25.0)
Hypertension	41 (93.2)
SCr ≥ 1.5 mg/dL	7 (15.9)
Chronic hemodialysis	3 (6.8)
Coronary artery disease	9 (20.5)
COPD	13 (29.5)
Cerebrovascular disease	6 (13.6)
Hyperlipidemia	18 (40.9)
Marfan syndrome	2 (4.5)
Clinical presentation	
Elective	42 (95.5)
Urgent/emergency	2 (4.5)
Aneurysm pathology	
Degenerative	29 (65.9)
Dissection	13 (29.5)
Infected	2 (4.5)
All previous aortic surgery (<i>n</i> = 11)	
Ascending/arch	4 (36.4)
DTAA	1 (9.1)
AAA	4 (36.4)
Ascending/arch and DTAA	1 (9.1)
DTAA and AAA	1 (9.1)

AAA abdominal aortic aneurysm, BMI body mass index, COPD chronic obstructive pulmonary disease, DTAA descending thoracic aortic aneurysm, SCr serum creatinine

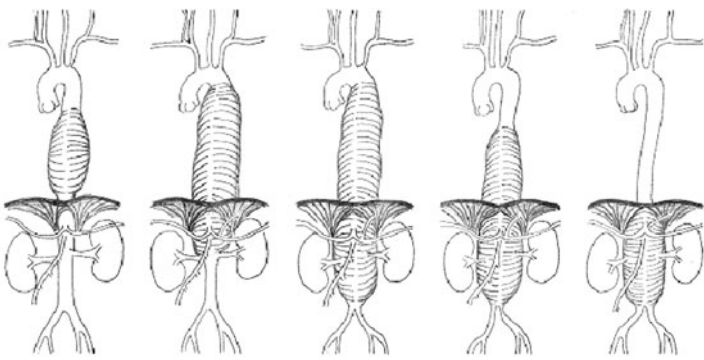
^a Continuous data are presented as means ± standard deviation

continuous infusion of remifentanyl, did not receive fentanyl. In 34 patients an epidural catheter was inserted on the day before surgery for postoperative analgesia.

Surgical technique

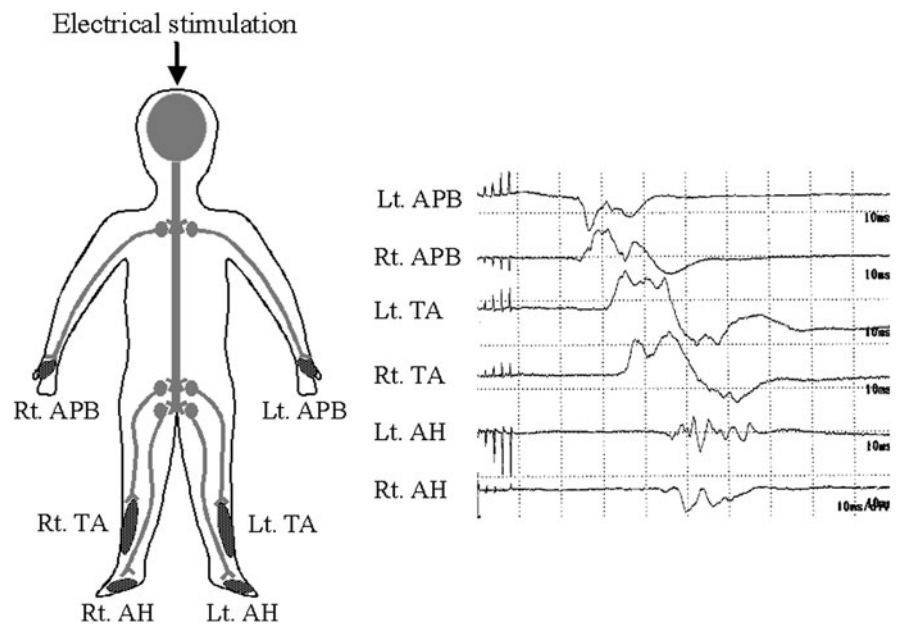
The left or right femoral artery and vein were dissected for cannulation to achieve partial distal aortic perfusion with cardiopulmonary bypass (PCPB), except for 3 patients who received aortic cannulation to the descending thoracic aorta or abdominal aorta at the distal part of the aneurysm for oxygenated blood perfusion, and femoral vein dissection for PCPB. The DTAA and TAAA were exposed and clamped after the establishment of PCPB. The clamps were placed sequentially when the aneurysm involved a long segment. For TAAA including the suprarenal part of the

Fig. 1 Aneurysm types, patient numbers, and incidence of postoperative paraplegia. *DTAA* descending thoracic aortic aneurysm



	DTAA	Crawford I	Crawford II	Crawford III	Crawford IV	Total
Patients	n=30	n=3	n=1	n=7	n=3	n=44
Paraplegia	0 (0%)	0 (0%)	1 (100%)	0 (0%)	0 (0%)	1 (2.3%)
Paraparesis	1 (3.0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (2.3%)
Monoparesis	0 (0%)	0 (0%)	0 (0%)	1 (14.3%)	0 (0%)	1 (2.3%)
Total postoperative paraplegia						3 (6.8%)

Fig. 2 Schematic representation of the monitoring of transcranial motor evoked potentials. *APB* abductor pollicis brevis muscle, *TA* tibialis anterior muscle, *AH* abductor hallucis muscle, *Lt.* left, *Rt.* right



abdominal aorta, visceral and renal protection was performed by selective blood perfusion. In all the TAAA patients (except for one in whom there was difficulty with inserting the catheter) and in 16 of all 30 DTAA patients, a catheter was introduced in the intrathecal space on the day before surgery, and spontaneous CSF drainage was initiated to maintain CSF pressure <10 mm Hg. Spontaneous cooling was permitted to 32°C blood temperature with active rewarming at the end of the procedure. In 3 patients with DTAA, deep hypothermic cardiopulmonary arrest

(DHCA) was applied, and the blood was cooled to between 16 and 20°C.

Techniques of MEP recording and monitoring and subsequent operative strategy

A schematic representation of MEP monitoring is shown in Fig. 2. Multipulse transcranial electric stimulation was performed using a multipulse stimulator (D-185; Digitimer, Welwyn Garden City, UK). A train-of-five pulse stimulation

was delivered at 2 ms interstimulus intervals (500 Hz). The stimulating electrodes consisted of a pair of 14.5 mm silver-plated disk electrodes at C3 (cathode) and C4 (anode) (international 10–20 System) affixed with conductive paste. The stimulus intensity of transcranial stimulation was determined at the beginning of MEP recording as supra-maximal (approximately 500 V). The compound muscle action potentials were bilaterally recorded from the skin over the APB and tibialis anterior (TA) muscles. In the more recent patients, the abductor hallucis (AH) muscle was added for action potential recording. A ground electrode was placed on the left or right arm proximal to the elbow. Evoked myographic responses were amplified with a 0.3- to 3-kHz bandpass filter. An intraoperative MEP measurement system (Neuropack MEB-5508 or MEB-2208; Nihon Koden, Tokyo, Japan) was used for MEP monitoring. The MEP was defined as “peak-to-peak” amplitude of the waveform. The baseline MEP was measured before the aorta was cross-clamped. The cutoff point for detecting SCI was set at 75% decrease of the baseline MEP in the TA muscle on the side contralateral to the femoral artery cannulation, to exclude the attenuation of MEPs from the TA muscle caused by leg ischemia with the cannulation. Spontaneous fluctuations of MEPs are common, although the intensity of neuromuscular blockade is unchanged [6]; therefore, a decrease of MEPs was considered significant only when the MEP values were decreased by >75% on several consecutive stimulations. Eleven patients underwent a “test-clamp” of the intercostal or lumbar arteries for about 10 min before aortic cross-clamping, and the MEP was measured every 1 min during the “test-clamp”. After the first aortic cross-clamping, the MEP was measured every 1 min for 15 min. Even if there were no significant MEP decreases, the MEP was measured every 5–10 min until the end of surgery. The clamps were placed sequentially when the aneurysm involved a long segment, thus allowing stepwise exclusion of aortic segments and the assessment of changes in MEP amplitudes. If the MEP decreased after placement of the aortic clamp, the mean proximal and distal aortic perfusion pressures were increased, patent intercostal and lumbar arteries were reattached, and the patient was actively cooled to 32°C. When the origin of the intercostal or lumbar artery supplying the AKA was inside the cross-clamped aneurysm, the intercostal or lumbar artery was selectively perfused and reattached if it was patent.

Outcome measurements

The primary outcome measure was the absence or presence of postoperative paraplegia, including paraparesis. Neurologic diagnoses were made by neurologists who were not blinded to the MEP values during surgery.

Statistical analysis

Numerical data are shown as means \pm standard deviation (SD) or medians [ranges]. Sensitivity and specificity were calculated and a receiver operating characteristic curve was applied to determine the appropriate cutoff point for the minimum MEP during surgery and for the MEP at the end of surgery, using 100, 90, 75, 64, and 50% decrease of MEP as a diagnostic measure for postoperative paraplegia.

Results

Clinical outcome

Intraoperative and postoperative data are shown in Table 2. No intraoperative deaths occurred. A total of 3 patients (6.8%) died in the hospital, and 2 of them were diagnosed with acute-type paraplegia. A total of 3 patients (6.8%) were diagnosed with acute-type paraplegia (Fig. 1): 1 patient with a type II TAAA was diagnosed with paraplegia, 1 patient with a DTAA was diagnosed with paraparesis, and 1 patient with a type III TAAA was diagnosed with monoparesis (motor function in only the right leg was incomplete). No delayed paraplegia occurred.

Results of MEP monitoring

Motor evoked potentials (MEPs) were measured successfully in all patients, and the results are shown in Fig. 3. In 24 patients, MEPs were adequate, i.e., the MEP decrease was <75% of the baseline, throughout the surgery. In the remaining 20 patients, significant MEP decreases, i.e., MEP decrease of >75% of the baseline, occurred. In 9 of these 20 patients, MEPs were restored after increasing the proximal and distal aortic pressure or/and reattaching intercostal or/and lumbar artery(ies), and in the other 11 patients, significant MEP decrease was sustained during the surgery; however, no SCI occurred in 9 of these 11 patients.

The minimum MEP during surgery had 100% sensitivity and 64.9% specificity in predicting paraplegia, excluding 4 patients undergoing DHCA whose MEPs completely faded away under DHCA. The MEP at the end of surgery had 66.7% sensitivity and 78.0% specificity in predicting paraplegia: only 1 patient, who had borderline paraplegia (right monoparesis), showed a false-negative result.

Table 3 summarizes the sensitivity and specificity of MEPs at various cutoff points for the minimum MEP during surgery and for the MEP at the end of surgery, with regard to the development of paraplegia. Receiver operating characteristic curves are shown in Fig. 4 and they indicate that adequate cutoff points for the minimum MEP

Table 2 Intraoperative and postoperative data

Variable ^a	Number of patients (%)
Total number of patients	44 (100)
Operative time (min)	434 [212–692]
CPB time (min)	114.5 [50–308]
Total cross-clamp time (min)	101.0 [50–289]
Perfusion strategy	
PCPB with mild to moderate hypothermia	40 (90.9)
DHCA	4 (9.1)
Minimum rectal temperature during CPB (°C)	34.8 [16.0–37.7]
Number of reimplanted intercostal/lumbar arteries	
0	29 (65.9)
1	12 (27.3)
2	1 (2.3)
3	2 (4.5)
Result of preoperatively detected AKA supplier with MRA (<i>n</i> = 24)	
Preserved	16 (66.7)
Reimplanted	5 (20.8)
Ligated	3 (12.5)
CSFD catheter insertion	29 (65.9)
Epidural catheter insertion	34 (77.3)
Intravenous anesthetic	
Total propofol infusion (mg)	1,035 [100–4,910]
Total ketamine infusion (mg)	722.5 [0–1,400]
Opioid	
Total fentanyl infusion (μg)	1,200 [0–2,800]
Total remifentanyl infusion ^b (μg)	3,300 [1,580–4,500]
NMB agent	
Total vecuronium infusion ^c , median [range] (mg)	18 [12–43]
Total rocuronium infusion ^d , median [range] (mg)	128 [75–240]
ICU stay (days)	6 [3–289]
Hospital stay (days)	58 [20–438]
Operative mortality	3 (6.8)
Due to intestinal ischemic necrosis (with paraplegia)	1
Due to respiratory failure (with paraparesis)	1
Due to cardiac tamponade with AAD (without paraplegia)	1

AAD acute aortic dissection, AKA Adamkiewicz artery, CPB cardiopulmonary bypass, CSFD cerebrospinal fluid drainage, DHCA deep hypothermic cardiopulmonary arrest, MRA magnetic resonance angiography, NMB neuromuscular blockade, PCPB partial distal aortic perfusion with cardiopulmonary bypass, ICU intensive care unit

^a Continuous data are presented as medians [ranges]

^b Utilized in 5 patients

^c Utilized in 38 patients

^d Utilized in 6 patients

during surgery and for the MEP amplitude at the end of surgery were 75–90 and 64–75% decrease of the baseline MEP, respectively.

Case report: patient with paraplegia (Fig. 5)

A 78-old-woman was scheduled for open surgical repair of a Crawford type II TAAA. Preoperative MRA was not performed. On the day before surgery, a CSF drainage catheter was inserted. The surgery was performed with PCPB using the left femoral artery and vein for cannulation. The MEPs from the left TA muscle disappeared 32 min after left femoral artery cannulation, probably because of left leg ischemia. Two minutes after the

proximal aortic clamping, MEPs from the right TA muscle significantly decreased, and 2.5 min after the proximal clamping, they completely disappeared. The patent L1 lumbar artery was promptly reattached and perfused, but MEPs from the right TA muscle never appeared again. She showed flaccid paraplegia after surgery.

Case report: patient with paraparesis (Fig. 6)

A 75-year-old woman was scheduled for open surgical repair of a DTAA that extended from just below the left subclavian artery to the level of the diaphragm. Preoperative MRA detected an AKA arising from the right L1 lumbar artery. On the day before the surgery, an epidural

Fig. 3 Flow chart showing the classification of the patients according to significant intraoperative change in motor evoked potentials (MEPs) and postoperative paraplegia. *Normal*, patients with normal lower-limb motor function after surgery

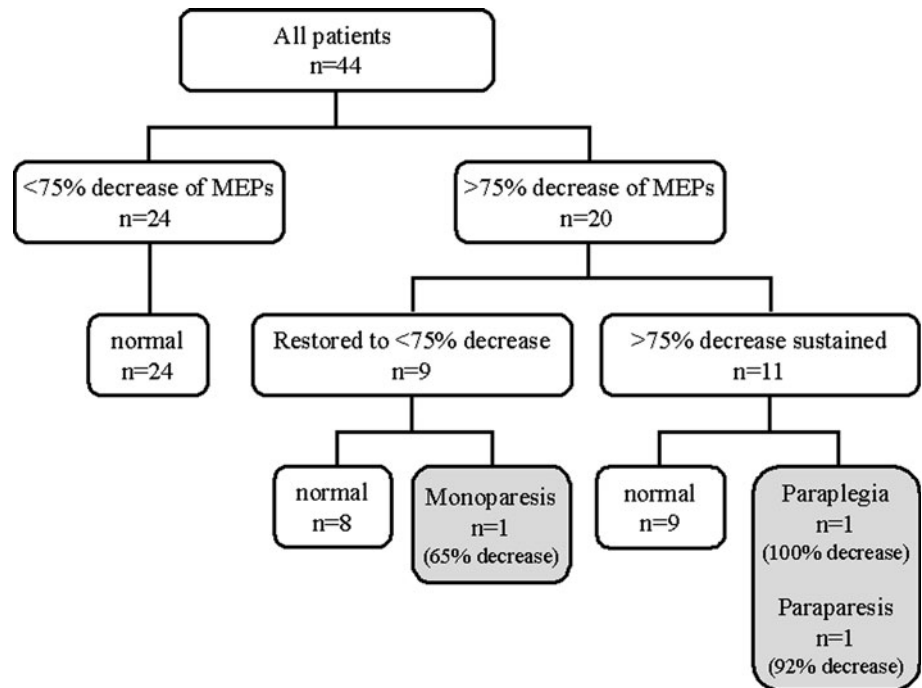


Table 3 Sensitivity, specificity, and predictive values at various cutoff points of MEP decrease for predicting postoperative paraplegia

Cutoff point	Sensitivity, %	Specificity, %	PPV, %	NPV, %
Minimum MEPs during surgery (n = 40) ^a				
100% decrease of MEPs (no waveform)	66.7 (2/3)	83.8 (31/37)	25.0 (2/8)	96.9 (31/32)
>90% decrease of MEPs	66.7 (2/3)	78.4 (29/37)	20.0 (2/10)	96.7 (29/30)
>75% decrease of MEPs	100 (3/3)	64.9 (24/37)	18.9 (3/16)	100 (24/24)
>64% decrease of MEPs	100 (3/3)	56.8 (21/37)	15.8 (3/19)	100 (21/21)
>50% decrease of MEPs	100 (3/3)	48.6 (18/37)	13.6 (3/22)	100 (18/18)
At the end of surgery (n = 44)				
100% decrease of MEPs (no waveform)	33.3 (1/3)	87.8 (36/41)	16.7 (1/6)	94.7 (36/38)
>90% decrease of MEPs	66.7 (2/3)	85.4 (35/41)	25.0 (2/8)	97.2 (35/36)
>75% decrease of MEPs	66.7 (2/3)	78.0 (32/41)	18.2 (2/11)	97.0 (32/33)
>64% decrease of MEPs	100 (3/3)	75.6 (31/41)	23.1 (3/13)	100 (31/31)
>50% decrease of MEPs	100 (3/3)	70.7 (29/41)	20.0 (3/15)	100 (29/29)

MEPs motor evoked potentials, NPV negative predictive value, PPV positive predictive value

^a Four cases with deep hypothermic cardiopulmonary arrest were excluded

catheter and a CSF drainage catheter were inserted. The aneurysm was successfully repaired and the right L1 lumbar artery was preserved with no significant decrease of MEPs, so no intercostal arteries were reattached. After the termination of PCPB, the MEP from the right TA muscle was significantly decreased, and hypoperfusion of the spinal cord was suspected. The mean arterial pressure was increased, but at the end of the operation, the MEP from the right TA muscle was only 8% of the baseline; however, the MEPs from the bilateral AH muscles were intact. She

showed paraparesis after surgery: she could move her ankles but could not draw up her knees.

Case report: patient with monoparesis (Fig. 7)

A 65-year-old man was scheduled for open surgical repair of a Crawford type III TAAA. Preoperative MRA detected an AKA arising from both the right Th12 intercostal and L1 lumbar arteries. On the day before the surgery, an epidural catheter and a CSF drainage catheter were inserted. The surgery was performed with PCPB using the

Fig. 4 Receiver operating characteristic curves; **a** for the minimum motor evoked potential (MEP) amplitudes during surgery and **b** for the MEP amplitudes at the end of surgery. The numbers in each circle show the cutoff points of percent decrease of the baseline MEP amplitude

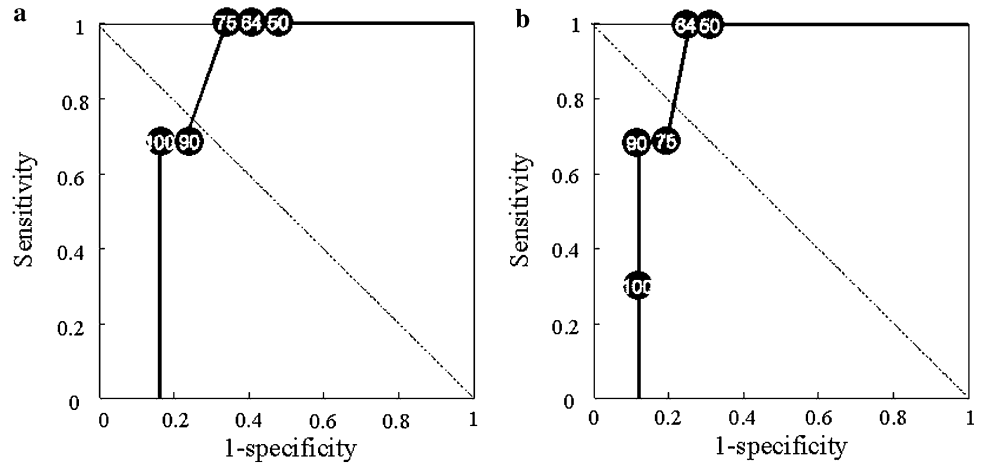
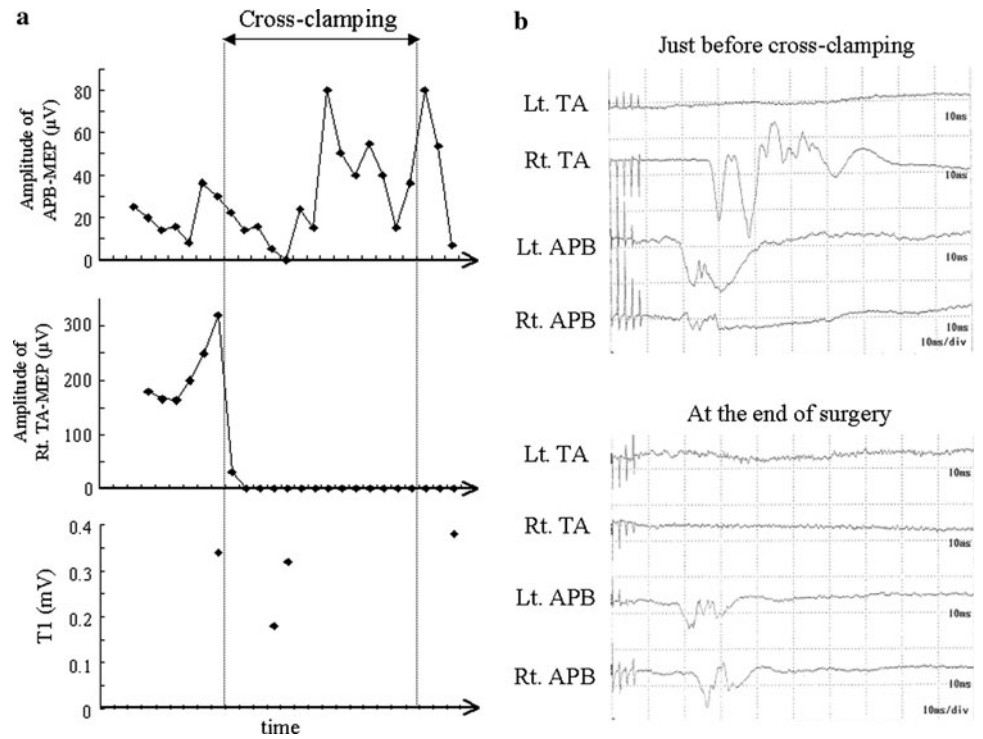


Fig. 5 The motor evoked potential (MEP) data from the patient with postoperative paraplegia. **a** Mean MEP amplitudes of the bilateral abductor pollicis brevis muscles (APB), MEP amplitudes of the right tibialis anterior muscle (TA), and twitch height of the M-response (TI). The time course includes aortic cross-clamping and at the end of surgery. **b** Representative MEP waveforms just before aortic cross-clamping and at the end of surgery



abdominal aorta at the level of the celiac trunk for aortic perfusion and the left femoral vein for blood withdrawal. After infrarenal clamping, MEPs from the bilateral TA muscles disappeared, probably because of ischemia in the legs, and the MEPs were quickly restored after reperfusion in the legs. After the reattaching of all visceral branches and the Th10, Th11, and Th12 intercostal arteries, the PCPB was terminated. Thirty-three minutes after PCPB termination, MEPs from the right TA were significantly decreased, but at the end of surgery, they were restored to 35% of the baseline. He showed monoparesis in the right leg after surgery: he could move his right ankle but could not draw up his right knee. His monoparesis gradually improved, and his walking ability was fully regained.

Discussion

In the present study, we assessed intraoperative MEP monitoring during DTAA and TAAA surgery. The results showed that with the cutoff point at 75% decrease of the baseline MEP, the MEP at the end of surgery had 66.7% sensitivity and 78.0% specificity in predicting SCI: only 1 patient, who had borderline paraplegia (right monoparesis), showed a false-negative result.

In light of the seriousness of paraplegia, higher sensitivity of monitoring (a lower false-negative rate) is desirable during DTAA and TAAA repair for detecting SCI and thus not missing paraplegia. In the present study, the cutoff point for detecting SCI was set at 75% decrease of the

Fig. 6 The motor evoked potential (MEP) data from the patient with postoperative paraparesis. **a** Mean MEP amplitudes of the bilateral abductor pollicis brevis muscles (APB), MEP amplitudes of the right tibialis anterior muscle (TA), and twitch height of the M-response (TI). The time course includes aortic cross-clamping and at the end of surgery. **b** Representative MEP waveforms just before aortic cross-clamping and at the end of surgery. AH abductor hallucis muscle

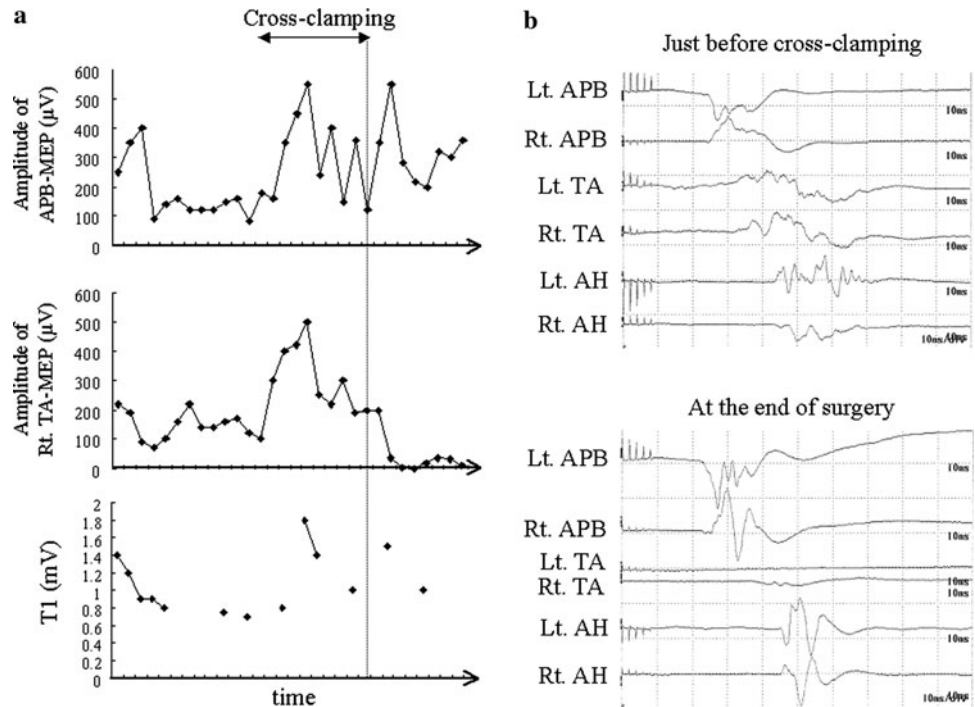
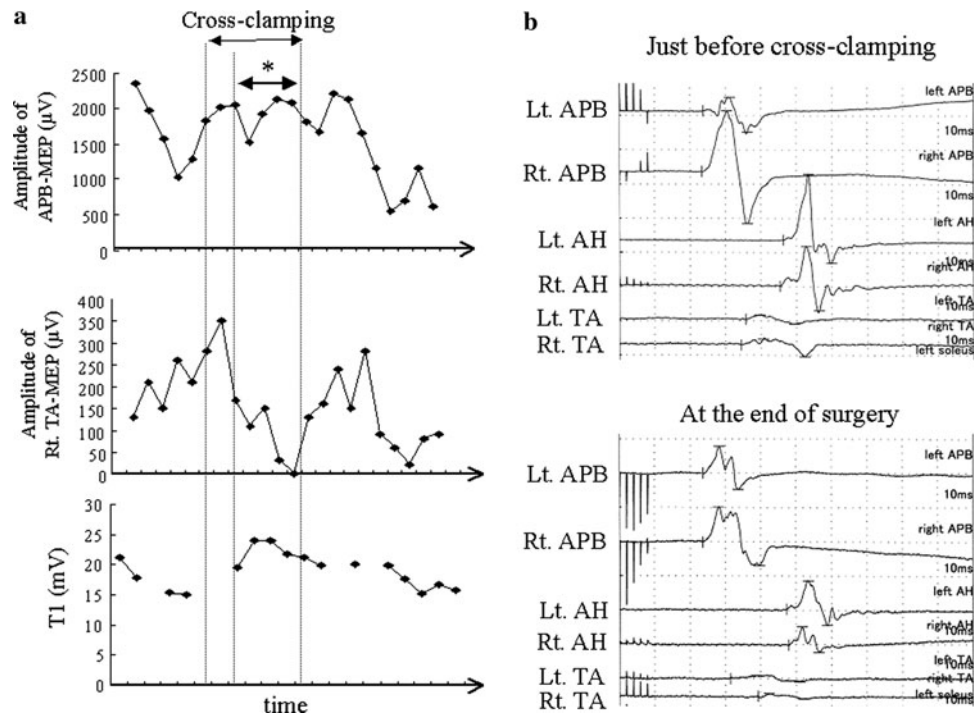


Fig. 7 The motor evoked potential (MEP) data from the patient with postoperative monoparesis. **a** Mean MEP amplitudes of the bilateral abductor pollicis brevis muscles (APB), MEP amplitudes of the right tibialis anterior muscle (TA), and twitch height of the M-response (TI). The time course includes aortic cross-clamping and at the end of surgery. **b** Representative MEP waveforms just before aortic cross-clamping and at the end of surgery. AH abductor hallucis muscle, asterisk infrarenal aortic cross-clamping with ischemia in legs



baseline MEP value in the TA muscle on the side contralateral to femoral artery cannulation, and only one patient, with borderline paraplegia (transient monoparesis), showed a false-negative result at the end of surgery (65% decrease of the baseline MEP). Even in this patient, the minimum MEP value was 88% decrease of the baseline, so we made every effort to increase spinal cord perfusion by reattaching

the intercostal arteries and increasing the proximal and distal aortic pressure, and we applied moderate hypothermia (32°C). These results indicate that MEP monitoring led us toward the maximum intervention for all the patients with paraplegia in our series.

On the other hand, if the specificity of MEP monitoring were low (higher false-positive rate), unnecessary

interventions would be performed. In the present study, 9 patients with no postoperative paraplegia showed >75% decrease of the baseline MEP value at the end of surgery (specificity was 78.0%, i.e., false-positive rate was 22%). In these 9 patients, 2 patients with a DTAA underwent the surgery with DHCA, and MEPs from all the muscles disappeared after cooling the patients and never appeared after rewarming. In one of these two patients, propofol was continuously infused during DHCA, and in the other patient vecuronium was continuously infused, so the reason why MEPs from both the APB and TA muscles disappeared in the two patients might have been because of the accumulation of these two drugs. The causes of the false-positive results in 7 of these 9 patients were unknown, but it would appear that some technical errors or accumulation of anesthetics may have been responsible for these results. Although the specificity in this study is comparable with other reports [10–12], more efforts to decrease the false-positive rate are necessary.

We set the cutoff point for detecting SCI at 75% decrease of the baseline MEP in the TA muscle on the side contralateral to femoral artery cannulation, in accordance with the report of Jacobs et al. [6]. In an animal experiment, Murakami et al. [13] found a threshold of 75% decrease of the baseline MEP to be indicative of neuronal loss and neurologic deficits in rabbits. On the basis of our results, the cutoff point set at 75% decrease of the baseline MEP at the end of surgery would be acceptable to achieve higher sensitivity and more acceptable specificity. However, because of the small sample in our study, it is difficult to set a precise cutoff point, and further investigations are necessary to investigate an adequate cutoff point that can predict paraplegia.

The data regarding the sensitivity and specificity of MEPs for predicting postoperative paraplegia in DTAA and TAAA repair are limited. In TAAA surgery, Kawanishi et al. [10] calculated the “MEP amplitude ratio” of the flexor hallucis brevis muscle (a plantar muscle), which took into consideration the change of MEPs of the flexor pollicis brevis muscle (a muscle in the hand); this ratio could distinguish between spinal cord ischemia (SCI) and systemic factors or technical failure. Surprisingly, their results showed 100% sensitivity and 100% specificity of MEPs for predicting postoperative paraplegia when the cutoff point was 32–46% decrease of the baseline MEP. We also tested their calculation in our case series; however, our results for sensitivity and specificity did not change so much (data not shown). In TAAA surgery, Shine et al. [11] showed 100% sensitivity and 39% specificity of the MEP at 20 min after cross-clamp release when the cutoff point was 75% decrease of the baseline. A possible explanation for their relatively low specificity was that their results did not reflect MEPs at the end of surgery after attempts were made to restore the decreased MEPs. In addition, they used

regional lumbar epidural cooling during surgery, and this may have affected the results. In DTAA and TAAA surgery, Keyhani et al. [12] demonstrated that sensitivity for permanent MEP change was 62.5%, i.e., the false-negative rate was 37.5%, although specificity was >97%. A possible explanation for their relatively low sensitivity was that the cutoff point was set at 100% decrease (absent) of MEPs. In the light of these reports and our report, it seems that, to achieve higher sensitivity and more acceptable specificity of MEPs with regard to the development of paraplegia, it would be important to measure MEPs at the end of surgery with a cutoff point at 75% decrease of the baseline. In addition, as has been shown by Kawanishi et al. [10], some calculation techniques might help us to exclude systemic factors or technical failures.

In our study, the patient with DTAA who had paraparesis postoperatively showed delayed response of MEPs; she showed significant decrease of MEP amplitudes elicited from the TA muscles after the termination of PCPB. Kakinohana et al. [14] reported a similar case, of delayed response of MEPs to SCI elicited from the TA muscles in a patient with DTAA. In that report, they pointed out that when the SCI occurred in the upper thoracic level, MEPs from the TA muscles might not rapidly respond to the SCI, probably because upper thoracic SCI does not include the synaptic transmission of α -motor neurons responsible for the TA muscles, as shown in an animal study [15]. It may be important to be aware of such a delayed response of MEPs in DTAA repair.

Recently the strategy to prevent postoperative paraplegia in DTAA and TAAA surgery seems to have been shifting from an anatomic to a physiologic (hemodynamic) approach. In 2007, Griep and Griep [16] presented the collateral network concept: i.e., the major declines in paraplegia rates have not occurred from reattaching important radicular arteries but rather from the introduction of strategies that focus on maximizing effective collateral perfusion. Acher and Wynn [17] presented a theory of paraplegia prevention in DTAA and TAAA surgery: paraplegia causation is anatomic but paraplegia prevention is physiologic (non-anatomic). In the present study, we had a patient with paraparesis whose L1 lumbar artery branching AKA was preserved, and this may be a typical episode which points to the importance of the physiologic approach. For the successful management of both the anatomic and the physiologic approaches, MEPs-guided achievement of adequate spinal cord perfusion would be necessary to reduce the incidence of postoperative paraplegia in DTAA and TAAA repair.

There are several limitations in the present study. First, our case series with 44 patients was a relatively small number, and most patients had DTAA. Although we successfully showed the sensitivity and specificity of MEP

monitoring in patients with various degrees of postoperative paraplegia, studies with a larger sample size would be necessary, especially in the high-risk group, i.e., the Crawford type II group, to facilitate the establishment of a more sophisticated strategy to prevent paraplegia. Second, this study was retrospective and did not include all patients undergoing emergency surgery at our institute. The overall results might have been affected by the inclusion of these patients, who would have had more unstable hemodynamics and higher risk of morbidity for paraplegia and a higher mortality risk. Third, mild to moderate hypothermia was applied in 40 of the 44 patients in our study. It is unclear whether the mild to moderate hypothermia affected our results, although it is thought that mild to moderate hypothermia higher than 28°C would not affect MEP amplitude when a train of pulses is used for stimulation [18]. In addition, under DHCA, the value of MEP monitoring during DTAA and TAAA surgery was not determined because the MEPs completely faded away under DHCA in the present setting. Finally, in this study there were no patients with delayed paraplegia, i.e., paraplegia that occurred after confirming normal motor function in the lower extremities postoperatively. The reported incidence of delayed paraplegia overall in patients with SCI is 20–40% [19–22]. The reason that we did not have patients with delayed paraplegia is unclear; however, a possible reason is that the postoperative hemodynamic management was adequate. There are several reports highlighting the importance of postoperative neurophysiological monitoring [23, 24] to prevent delayed paraplegia, further investigation for the application of postoperative spinal cord monitoring would be necessary.

In summary, we assessed intraoperative MEP monitoring during DTAA and TAAA surgery. Motor evoked potential monitoring at the end of surgery with a cutoff point of 75% decrease of the baseline had relatively high sensitivity and acceptable specificity with regard to the development of postoperative paraplegia including paraparesis. Because of the small sample size in our study, further investigations are necessary to investigate an adequate cutoff point which can predict postoperative paraplegia in DTAA and TAAA repair.

Acknowledgments The authors thank Ms. Ryuko Imai, Mr. Tsunenori Takatani, Ms. Masami Yamanaka, and the other staff of the Central Clinical Laboratory (Nara Medical University, Kashihara, Nara, Japan) for their invaluable collaboration in monitoring MEPs in this study.

References

1. Svensson LG, Crawford ES, Hess KR, Coselli JS, Safi HJ. Experience with 1509 patients undergoing thoracoabdominal aortic operations. *J Vasc Surg.* 1993;17:357–70.
2. Safi HJ, Estrera AL, Miller CC, Huynh TT, Porat EE, Azizzadeh A, Meada R, Goodrick JS. Evolution of risk for neurologic deficit after descending and thoracoabdominal aortic repair. *Ann Thorac Surg.* 2005;80:2173–9.
3. Bicknell CD, Riga CV, Wolfe JHN. Prevention of paraplegia during thoracoabdominal aortic aneurysm repair. *Eur J Vasc Endovasc Surg.* 2009;37:654–60.
4. Juvonen T, Biancari F, Rimpilainen J, Satta J, Rainio P, Kiviluoma K. Strategies for spinal cord protection during descending thoracic and thoracoabdominal aortic surgery: up-to-date experimental and clinical results- a review. *Scand Cardiovasc J.* 2002;36:136–60.
5. Coselli JS, Conklin LD, LeMaire SA. Thoracoabdominal aortic aneurysm repair: review and update of current strategies. *Ann Thorac Surg.* 2002;74:S1881–4.
6. Jacobs MJ, Mess W, Mochtar B, Nijenhuis EJ, Stadius van Eps RG, Schrink GWH. The value of motor evoked potentials in reducing paraplegia during thoracoabdominal aneurysm repair. *J Vasc Surg.* 2006;43:239–46.
7. Solan TB. Electrophysiologic monitoring during surgery to repair the thoracoabdominal aorta. *Semin Cardiothorac Vasc Anesth.* 2004;8:113–25.
8. MacDonald DB, Janusz M. An approach to intraoperative neurophysiologic monitoring of thoracoabdominal aneurysm surgery. *J Clin Neurophysiol.* 2002;19:43–54.
9. Kakinohana M, Nakamura S, Fuchigami T, Miyata Y, Sugahara K. Influence of the descending thoracic aortic cross clamping on bispectral index values and plasma propofol concentration in humans. *Anesthesiology.* 2006;104:939–43.
10. Kawanishi Y, Munakata H, Matsumori M, Tanaka H, Yamashita T, Nakagiri K, Okada K, Okita Y. Usefulness of transcranial motor evoked potentials during thoracoabdominal aortic surgery. *Ann Thorac Surg.* 2007;83:456–61.
11. Shine TSJ, Harrison BA, De Ruyter ML, Crook JE, Heckman M, Daube JR, Stapelfeldt WH, Cherry KJ, Glociczki P, Bower TC, Murray MJ. Motor and somatosensory evoked potentials. Their role in predicting spinal cord ischemia in patients undergoing thoracoabdominal aortic aneurysm repair with regional lumbar epidural cooling. *Anesthesiology.* 2008;108:580–7.
12. 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.
13. Murakami H, Tsukube T, Kawanishi Y, Okita Y. Transcranial myogenic motor-evoked potentials after transient spinal cord ischemia predicts neurologic outcome in rabbits. *J Vasc Surg.* 2004;39:207–13.
14. Kakinohana M, Abe M, Miyata Y, Oshiro M, Saikawa S, Arakaki K, Kuniyoshi Y, Sugahara K. Delayed response of transcranial myogenic motor-evoked potential monitoring to spinal cord ischemia during repair surgery for descending thoracic aortic aneurysm. *J Anesth.* 2008;22:304–7.
15. 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.
16. Griep RB, Griep EB. Spinal cord perfusion and protection during descending thoracic and thoracoabdominal aortic surgery: the collateral network concept. *Ann Thorac Surg.* 2007;83: S865–9.
17. Acher CW, Wynn M. A modern theory of paraplegia in the treatment of aneurysms of the thoracoabdominal aorta: an analysis of technique specific observed/expected ratios for paralysis. *J Vasc Surg.* 2009;49:1117–24.
18. Sakamoto T, Kawaguchi M, Kakimoto M, Inoue S, Takahashi M, Furuya H. The effect of hypothermia on myogenic motor-evoked

- potentials to electrical stimulation with a single pulse and a train of pulses under propofol/ketamine/fentanyl anesthesia in rabbits. *Anesth Analg*. 2003;96:1692–7.
19. Hollier LH, Money SR, Naslund TC, Proctor CD Sr, Buhman WC, Marino RJ, Harmon DE, Kazmier FJ. Risk of spinal cord dysfunction in patients undergoing thoracoabdominal aortic replacement. *Am J Surg*. 1992;164:210–3.
 20. 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 cases. *J Vasc Surg*. 1986;3:389–404.
 21. 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 aneurysm. *J Thorac Cardiovasc Surg*. 1999; 113:87–100.
 22. Safi HJ, Miller CC, Azizzadeh A, Ilipoulos D. Observations on delayed neurologic deficit after thoracoabdominal aortic aneurysm repair. *J Vasc Surg*. 1997;26:616–22.
 23. Jacobs MJ, Meylaerts SA, de Haan P, de Mol BA, Kalkman CJ. Strategies to prevent neurologic deficit based on motor-evoked potentials in type I and II thoracoabdominal aortic aneurysm repair. *J Vasc Surg*. 1999;29:48–59.
 24. Etz CD, Luehr M, Kari FA, Bodian CA, Smego D, Plestis KA, Griep RB. Paraplegia after extensive thoracic and thoracoabdominal aortic aneurysm repair: does critical spinal cord ischemia occur postoperatively? *J Thorac Cardiovasc Surg*. 2008;135: 324–30.