



## UvA-DARE (Digital Academic Repository)

### Efficacy of transcranial motor-evoked myogenic potentials to detect spinal cord ischemia during operations for thoracoabdominal aneurysms

de Haan, P.; Kalkman, C.J.; de Mol, B.A.J.M.; Ubags, L.H.H.M.; Veldman, D.J.; Jacobs, M.J.H.M.

**DOI**

[10.1016/S0022-5223\(97\)70403-3](https://doi.org/10.1016/S0022-5223(97)70403-3)

**Publication date**

1997

**Published in**

The Journal of Thoracic and Cardiovascular Surgery

[Link to publication](#)

**Citation for published version (APA):**

de Haan, P., Kalkman, C. J., de Mol, B. A. J. M., Ubags, L. H. H. M., Veldman, D. J., & Jacobs, M. J. H. M. (1997). Efficacy of transcranial motor-evoked myogenic potentials to detect spinal cord ischemia during operations for thoracoabdominal aneurysms. *The Journal of Thoracic and Cardiovascular Surgery*, 113, 87-101. [https://doi.org/10.1016/S0022-5223\(97\)70403-3](https://doi.org/10.1016/S0022-5223(97)70403-3)

**General rights**

It is not permitted to download or to forward/distribute the text or part of it without the consent of the author(s) and/or copyright holder(s), other than for strictly personal, individual use, unless the work is under an open content license (like Creative Commons).

**Disclaimer/Complaints regulations**

If you believe that digital publication of certain material infringes any of your rights or (privacy) interests, please let the Library know, stating your reasons. In case of a legitimate complaint, the Library will make the material inaccessible and/or remove it from the website. Please Ask the Library: <https://uba.uva.nl/en/contact>, or a letter to: Library of the University of Amsterdam, Secretariat, Singel 425, 1012 WP Amsterdam, The Netherlands. You will be contacted as soon as possible.

*UvA-DARE is a service provided by the library of the University of Amsterdam (<https://dare.uva.nl>)*

# SURGERY FOR ACQUIRED HEART DISEASE

---

## EFFICACY OF TRANSCRANIAL MOTOR-EVOKED MYOGENIC POTENTIALS TO DETECT SPINAL CORD ISCHEMIA DURING OPERATIONS FOR THORACOABDOMINAL ANEURYSMS

Peter de Haan, MD<sup>a</sup>  
Cor J. Kalkman, MD, PhD<sup>a</sup>  
Bas A. de Mol, MD, PhD<sup>b</sup>  
Leon H. Ubags<sup>c</sup>  
Dirk J. Veldman, MD<sup>a</sup>  
Michael J. H. M. Jacobs, MD, PhD<sup>d</sup>

**Objective:** Motor-evoked myogenic potentials after transcranial electrical stimulation monitor the vulnerable motoneuronal system of the spinal cord. This study reports our initial experiences with motor-evoked potentials to assess the adequacy of spinal cord perfusion during operations for thoracoabdominal aneurysms. **Methods:** In 20 patients undergoing thoracoabdominal aneurysm operations, myogenic motor-evoked potentials were recorded. In 18 patients retrograde aortic perfusion was used. When spinal cord ischemia was detected, distal flow or mean arterial pressure was increased in an attempt to restore cord perfusion. By means of sequential crossclamping, motor-evoked potentials were also used to identify intercostal or lumbar arteries that needed to be reimplanted. **Results:** Reproducible motor-evoked potentials could be recorded in all patients. During retrograde perfusion, nine patients showed a rapid decrease in the amplitude of motor-evoked potentials to less than 25% of baseline, indicating spinal cord ischemia. In five patients ischemic changes in motor-evoked potentials could be reversed by increasing distal and proximal blood pressures. In four patients ischemic changes during crossclamping necessitated segmental artery reimplantation. In three of these four patients intercostal or lumbar arteries were reattached. In one patient reimplantation of segmental arteries was not possible; this patient awoke paraplegic. Segmental arteries were ligated after confirmation of intact motor-evoked potentials during aortic clamping in eight patients. None of these patients had a neurologic deficit. The absence of motor-evoked potentials at the end of the procedure always indicated a postoperative motor deficit. **Conclusion:** During operations for thoracoabdominal aneurysms, monitoring of motor-evoked potentials is an effective technique to detect spinal cord ischemia within minutes. This modality can be used to guide the management of distal aortic perfusion techniques and may also help to identify segmental arteries that need to be reattached. (*J Thorac Cardiovasc Surg* 1997;113:87-101)

From the Departments of Anesthesiology,<sup>a</sup> Cardiac Surgery,<sup>b</sup> Anesthesiology,<sup>c</sup> and Vascular Surgery,<sup>d</sup> Academic Hospital, University of Amsterdam, Amsterdam, The Netherlands, and the Delft University of Technology,<sup>b</sup> Delft, The Netherlands.

Received for publication April 30, 1996; revisions requested June 10, 1996; revisions received July 1, 1996; accepted for publication July 8, 1996.

Address for reprints: P. de Haan, MD, Department of Anesthesiology, Academic Hospital, University of Amsterdam, Academic Medical Center, Meibergdreef 9, 1105 AZ Amsterdam, The Netherlands.

Copyright © 1997 by Mosby-Year Book, Inc.

0022-5223/97 \$5.00 + 0 12/1/76349

Paraplegia is a devastating and unpredictable complication of operations on the descending thoracic aorta and thoracoabdominal aorta. The incidence of paraplegia and paraparesis ranges from 0.5% after repair of a coarctation to 41% after resections of extensive thoracoabdominal aneurysms (TAAs) with dissection.<sup>1,2</sup> Neurologic deficits after aortic crossclamping are the result of interruption of spinal cord blood flow of sufficient magnitude and duration to produce irreversible motor neuron injury.<sup>3</sup> Minimizing the degree and duration of spinal cord ischemia by using retrograde aortic

perfusion during crossclamping appears to be protective. However, definitive restoration of spinal cord blood supply after aortic replacement is necessary to prevent postoperative paraplegia. This may necessitate revascularization of critical intercostal or lumbar arteries.<sup>4-6</sup>

Somatosensory evoked potentials (SSEPs) are widely used for intraoperative monitoring of spinal cord function during operations with a risk for postoperative neurologic dysfunction. However, in a prospective study SSEPs and temporary distal aortic perfusion did not reduce the prevalence of early and delayed neurologic complications after operations for thoracic aortic aneurysms.<sup>7</sup> One reason for the failure of SSEPs to identify patients at risk is that the response of the dorsal columns to spinal cord ischemia is slow and does not reflect motor function. There are now several techniques for intraoperative monitoring of motor pathways, but only myogenic responses are entirely specific for the status of the motor neurons in the anterior horn gray matter, which are the least tolerant to ischemia.<sup>8</sup> The use of transcranial motor-evoked myogenic potentials (tc-MEPs) during TAA operations has not yet been described. The purpose of this study was to evaluate the efficacy of this technique in detecting spinal cord ischemia during TAA procedures.

## Methods

**Patient selection and data.** In 20 consecutive patients operated on for lesions of the thoracoabdominal aorta between December 1993 and December 1995 at the Academic Medical Centre of the University of Amsterdam, spinal cord function was monitored with tc-MEPs. The ages of the patients ranged between 22 and 83 years. Perioperative patient data are listed in Table I. The causes of the aortic lesions included atherosclerosis, trauma, and degenerative disease. The TAAs were classified according to the system devised by Crawford and associates.<sup>7</sup> Type I involves the descending thoracic and upper abdominal aorta. Type II includes the entire descending aorta and the abdominal aorta and is the most extensive. Type III includes less than half of the descending thoracic aorta and most of the abdominal aorta. Type IV involves most or all of the abdominal aorta. Eight patients had a TAA of type I, seven patients type II, three patients type III, and two patients type IV. A neurologic examination was performed in all patients on the day before the operation and every day thereafter for 7 days after the operation and before hospital discharge.

**Surgical procedure.** The patients were positioned on a bean bag with the thorax in the lateral position and the abdomen rotated toward the almost horizontal pelvis. In 18 patients distal bypass was used to maintain spinal cord, splanchnic, and renal perfusion and to unload the heart during crossclamping. After exposure of the aorta by a

left-sided thoracophrenic laparotomy, retrograde aortic perfusion was achieved by cannulation of the left atrium and femoral artery and the use of a centrifugal pump (Sarns; 3M Health Care, St. Paul, Minn.) with heparin-coated tubing in 11 patients. In seven patients distal aortic perfusion was performed with the aid of cardiopulmonary bypass with a femoral artery cannula, a long 26F femoral vein cannula up to the right atrium, and systemic heparinization. The latter technique allows hypothermia to be induced, allows the return of spilled blood into the bypass system, and provides better circulatory support. Distal aortic perfusion was started before aortic crossclamping and was adjusted to maintain distal aortic pressure above 60 mm Hg during crossclamping. In two patients no retrograde aortic perfusion was used.

Dacron tube grafts were anastomosed with running 3-0 Prolene sutures (Ethicon, Inc., Somerville, N.J.). The aorta was crossclamped distal or proximal to the left subclavian artery, depending on the extent of the aneurysm. A second clamp was placed some centimeters more distally. The aorta was completely transected and a Dacron graft anastomosed. Then the aorta was sequentially crossclamped and the aneurysm was opened. Depending on tc-MEPs, segmental arteries were reattached by an aortic island, containing one or several orifices, anastomosed to the graft. If segmental arteries were reattached, duration of spinal cord ischemia was minimized by immediate perfusion of the grafted arteries.

After the abdominal aorta had been opened in patients with type II, III and IV TAAs, the celiac trunk, superior mesenteric artery, and both renal arteries were selectively perfused with 9F Pruitt catheters (Ideas for Medicine, Inc., Clearwater, Fla.) connected to the extracorporeal circulatory system. These arteries were reimplemented in the graft while organ perfusion was maintained.<sup>9</sup>

**Anesthetic technique.** Patients received 2 to 4 mg of lorazepam orally 1 hour before the operation. Anesthesia was induced with etomidate 0.3 mg/kg and sufentanil 5  $\mu$ g/kg, given intravenously, and was maintained with sufentanil 4  $\mu$ g/kg per hour and ketamine 2 mg/kg per hour. Additional ketamine 50 mg intravenously was given at signs of inadequate anesthesia. Muscle relaxation was induced and maintained with vecuronium. The level of neuromuscular blockade can affect tc-MEP amplitude and should be maintained at a level that is compatible with recording of compound muscle action potentials, usually around 20% of baseline amplitude. The level of neuromuscular blockade was assessed with a Relaxograph neuromuscular transmission monitor (Datex, Helsinki, Finland). This device applies a train of four supramaximal stimuli to the ulnar nerve at the wrist every 20 seconds and records the resulting hypothermic compound muscle action potential. The level of neuromuscular blockade was expressed as the amplitude of the response to the first stimulus expressed as a percentage of control, that is, before administration of the muscle relaxant. The alarm of the Relaxograph monitor was set at 20% and was connected to the on/off switch of an infusion pump (Braun, Melsungen, Germany), thus achieving simple on/off closed-loop control. Whenever the level of neuromuscular blockade decreased below the set point, the alarm of the Relaxograph monitor activated the infusion

**Table I.** Perioperative patient data

Patient No.	TAA type	Perfusion	Clamp time (min)	Placement of proximal aortic clamp	Segmental arteries	Operative technique	Tc-MEPs	Neurologic outcome
1	III, CD	None	44	T-8	Ligation with tc-MEP control T-10, L-1, L-2 (5×)	Proximal and distal clamp, reimplantation renal and mesenteric vessels	Preserved for 18 min after x-clamp; thereafter, loss due to peripheral nerve ischemia. Full return after release of x-clamp.	Normal
2	III	None	76	T-9	Ligation with tc-MEP control T12-L2 (6×)	Proximal and distal clamp, reimplantation renal and mesenteric vessels	Preserved for 23 min after x-clamp; thereafter, loss due to peripheral nerve ischemia. Full return after release of x-clamp.	Normal
3	I	LA-A. Fem	40	T-6	None	Proximal and distal clamp	No change.	Normal
4	I	LA-A. Fem	41	T-5	None	Proximal and distal clamp	No change.	Normal
5	I	LA-A. Fem	55	T-4	Ligation with tc-MEP control T-6, T-7 (4×)	Proximal and distal clamp, CSF-drainage	Technical bypass pump failure, ischemic tc-MEP changes within 1 min. Full tc-MEP recovery 2 minutes after restoration of bypass flow.	Normal
6	II	LA-A. Fem	84	T-4	Preservation T-11, T-12 (4×)	Staged clamping, reimplantation renal and mesenteric vessels, octopus, CSF-drainage	No tc-MEP changes.	Normal
7	II, CD	LA-A. Fem	175	Between CCA and LSA	Preservation T6-T11 (8×)	Staged clamping, reimplantation renal and mesenteric vessels, octopus	Ischemic tc-MEP changes after profuse ICA back-bleeding; return to baseline with insertion of Pruitt catheters in ICA orifices. Selective reimplantation of these ICAs. Three episodes with ischemic tc-MEP changes during x-clamp; reversible with increases of retrograde flow. During two episodes, distal aortic pressure was 60 mm Hg.	Normal
8	I, CD	LA-A. Fem	48	Between CCA and LSA	None	Proximal and distal clamp	No tc-MEP changes.	Normal

**Table I.** *Cont'd*

<i>Patient No.</i>	<i>TAA type</i>	<i>Perfusion</i>	<i>Clamp time (min)</i>	<i>Placement of proximal aortic clamp</i>	<i>Segmental arteries</i>	<i>Operative technique</i>	<i>Tc-MEPs</i>	<i>Neurologic outcome</i>
9	I	LA-A. Fem	85	T-4	None	Proximal and distal clamp	Ischemic tc-MEP changes during x-clamp, distal aortic pressure 50-60 mm Hg, return to baseline (pump flow 2.4-4.1 L/min).	Normal
10	I	LA-A. Fem	76	T-6	Ligation with tc-MEP control T-11, T-12 (4×)	Proximal and distal clamp	Ischemic tc-MEP changes during x-clamp, distal aortic pressure 45-60 mm Hg, return to baseline (pump flow 2.7-3.8 L/min).	Normal
11	II, AD	LA-A. Fem	89	T-4	None	Staged clamping, reimplantation renal and mesenteric vessels, octopus, CSF drainage	Tc-MEPs intact during thoracic aortic replacement. Ischemic tc-MEP changes after x-clamp T-12-bifurcation, segmental artery reimplantation not possible and no tc-MEP recovery.	Paraplegia
12	III, CD	LA-A. Fem	83	T-10	Ligation with tc-MEP control T12-L1 (4×)	Staged clamping, reimplantation renal and mesenteric vessels, octopus, CSF drainage	No tc-MEP changes.	Normal
13	I	LA-A. Fem	122	T-5	None	Proximal and distal clamp	No tc-MEP changes.	Normal
14	II, CD	F-F bypass with oxygenator	119	T-4	Preservation T8-T11 (6×)	Staged clamping, reimplantation mesenteric vessels, octopus, CSF drainage	No tc-MEP changes. Elective ICA reimplantation.	Normal
15	II	F-F bypass with oxygenator	127	Between CCA and LSA	Ligation with tc-MEP control (T6-9, 8×) and preservation (L-2, 2×)	Staged clamping, reimplantation renal and mesenteric vessels, octopus, CSF drainage	Tc-MEPs intact during thoracic aortic replacement. Ischemic tc-MEP changes after x-clamp T11-bifurcation. Full tc-MEP recovery after L-2 reimplantation. Thrombosis of reattached arteries second postoperative day.	Delayed paraplegia
16	IV	F-F bypass with oxygenator	96	T-11	Ligation with tc-MEP control T-12 (2×)	Staged clamping, reimplantation renal and mesenteric vessels, octopus, CSF drainage	No tc-MEP changes.	Normal

**Table I.** *Cont'd*

<i>Patient No.</i>	<i>TAA type</i>	<i>Perfusion</i>	<i>Clamp time (min)</i>	<i>Placement of proximal aortic clamp</i>	<i>Segmental arteries</i>	<i>Operative technique</i>	<i>Tc-MEPs</i>	<i>Neurologic outcome</i>
17	IV	F-F bypass with oxygenator	94	T-10	Preservation L-1, L-2 (4×)	Staged clamping, reimplantation renal and mesenteric vessels, octopus, CSF drainage	Ischemic tc-MEP changes after the start of distal aortic perfusion, before x-clamp. Despite corrective measures, no tc-MEP recovery during the procedure.	Paraplegia
18	II	F-F bypass with oxygenator	173	LSA	Preservation T4-6 (6×)	Staged clamping, reimplantation renal and mesenteric vessels, octopus, CSF drainage	Ischemic tc-MEP changes after clamping the aorta from the LSA to T-8. Reimplantation of six ICAs. Tc-MEP recovery of the left leg after 353 min.	Temporary paresis of the right leg
19	II	F-F bypass with oxygenator	100	T-5	Ligation with tc-MEP control T-7, T-8, L-2 (5×)	Proximal and distal clamp, reimplantation renal and mesenteric vessels, octopus, CSF drainage	No tc-MEP changes.	Normal
20	I	F-F bypass with oxygenator	96	T-5	Ligation with tc-MEP control T7-9 (4×)	Proximal and distal clamp	Ischemic tc-MEP changes during x-clamp, distal aortic pressure 55-70 mm Hg, return to baseline. Ischemic tc-MEP changes during reperfusion MAP 65-80 mm Hg, return to baseline.	Normal

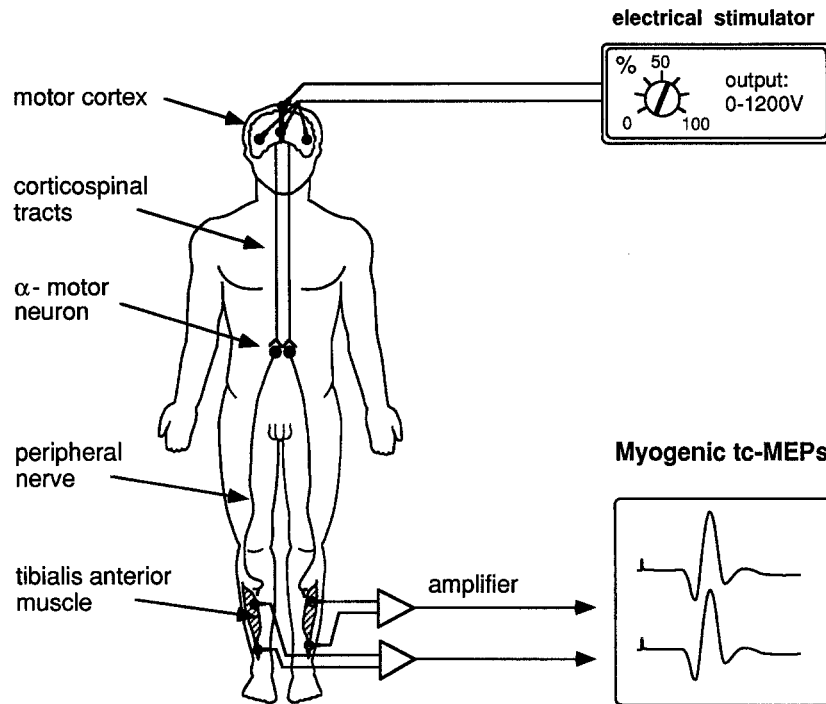
*TAA*, Thoracoabdominal aneurysm; *Tc-MEPs*, transcranial motor-evoked potentials; *CD*, chronic dissection; *T*, level of thoracic vertebral body; *L*, level of lumbar vertebral body; *x-clamp*, aortic crossclamping; *LA-A, Fem*, left atrium-femoral artery bypass; *CSF*, cerebrospinal fluid; *octopus*, selective organ perfusion; *CCA*, common carotid artery; *LSA*, left subclavian artery; *ICA*, intercostal artery; *AD*, acute dissection; *F-F*, femoral vein-femoral artery bypass with oxygenator; *MAP*, mean arterial pressure.

pump, which delivered an infusion of vecuronium at a rate of 2 µg/kg per minute. If one-lung ventilation was planned, the patient was intubated with a double-lumen endotracheal tube. Five-lead electrocardiograms including ST segments, end-tidal carbon dioxide, rectal and blood temperatures, urine output, blood pressure (via radial and femoral arterial catheters), and central venous and pulmonary artery pressures were monitored continuously. Mean arterial pressure (MAP) was maintained between 60 and 100 mm Hg. During the clamp period, blood pressure was mainly controlled by adjusting bypass flow of the centrifugal pump or by controlling preload of the heart via the heart-lung machine. Short-acting β-adrenergic blockade and intravenous nitroglycerin were

used if preload management could not control high blood pressure. Shed blood was collected, processed in a cell salvage device, and reinfused during the operation. Temperature was allowed to decrease spontaneously, reaching rectal temperatures between 31° and 34° C during the crossclamp period. Starting in 1994, cerebrospinal fluid pressure was monitored routinely until the third postoperative day (10 patients) and fluid was allowed to drain spontaneously if cerebrospinal fluid pressure increased above 15 mm Hg.

#### Technique of MEP recording

*Transcranial stimulation.* Tc-MEPs were evoked by applying paired stimuli to the scalp via four 9 mm silver electroencephalographic disk electrodes. The anode was



**Fig. 1.** Schematic representation of the technique used to elicit and record myogenic tc-MEPs. The motor cortex is stimulated with a short electrical pulse, and compound muscle action potentials are recorded from the left and right anterior tibial muscles.

placed at Cz and three interconnected cathodes were placed at Fpz, A<sub>1</sub> and A<sub>2</sub> (International 10-20 system).<sup>\*</sup> Each individual stimulus lasted 50  $\mu$ sec, and the interval between the paired stimuli was 3 msec.<sup>10</sup> So that paired stimulation could be achieved, two identical transcranial electrical stimulators (D 180A, Digitimer, Welwyn Garden City, United Kingdom) were used.

**Muscle action potential recording.** Compound muscle action potentials were recorded from the skin over the left and right anterior tibialis muscle and from the skin over the left and right thenar muscles by means of adhesive gel silver/silver chlorine electrodes (Fig. 1). The signals were amplified 5,000 to 20,000 times (adjusted to obtain maximum vertical resolution) and filtered between 30 and 1500 Hz with the use of a 3T PS-800 biologic amplifier (Twente Technology Transfer, Twente, The Netherlands). Data acquisition, processing, and analysis were performed on a computer with an analog-to-digital converter and software written in the LabVIEW programming environment (National Instruments, Austin, Tex.).<sup>11</sup> After stabilization of the level of neuromuscular blockade, the supramaximal stimulus intensity was assessed. A stimulus intensity that produced maximal tc-MEP amplitude was used. Single-sweep responses of sufficient amplitude were obtained after each paired stimulus, and signal averaging was not necessary. Baseline tc-MEPs were measured every 5 min-

utes until the aorta was crossclamped and every minute during and after crossclamping. Amplitude and onset latency of tc-MEPs were recorded. A reduction of tc-MEP amplitude of the tibialis anterior muscle during or after the aortic crossclamp period to less than 25% of baseline was considered an indication of ischemic spinal cord dysfunction. In case of an amplitude reduction, the MEP signals of the thenar muscles were used to distinguish between spinal cord ischemia and systemic factors or technical problems.

**Interventions after detection of ischemic tc-MEP changes.** When ischemic tc-MEP changes were observed during graft inclusion or reperfusion, attempts were first made to restore spinal cord perfusion. This was achieved by increasing retrograde flow, distal aortic pressure, and MAP. When significant backbleeding from segmental arteries was observed, 3F Pruitt catheters were inserted into the intercostal artery orifices to reduce the "steal" effect from the anterior spinal artery.

Tc-MEPs were also used to identify segmental arteries critical to spinal cord blood supply. When exclusion of an aortic segment resulted in ischemic tc-MEP changes within 5 minutes after placement of the clamps, then the intercostal and lumbar arteries in that aortic segment were considered critical and were reimplemented. If no ischemic tc-MEP changes were observed, the intercostal and lumbar arteries in that segment were ligated.

**Statistical analysis.** Tc-MEP data are expressed as medians and 10th to 90th percentiles. Within-patient variability of tc-MEP amplitudes was calculated as the

\* Jasper HH. The ten-twenty electrode system of the international federation. *Electroencephalogr Clin Neurophysiol* 1958;10:371-5.

coefficient of variation ( $CV = \text{Standard deviation}/\text{Mean} \times 100\%$ ) during 2 hours before aortic clamping.

## Results

The neurologic status of 19 patients was within normal limits before the operation. One patient (patient 18) had an abnormal plantar reflex in the right leg, as a result of a left hemispheric stroke. None of the patients had preoperative motor deficits. Reproducible myogenic tc-MEPs could be recorded in all patients. Baseline amplitude was 607  $\mu\text{V}$  (136 to 1498  $\mu\text{V}$ ), and latency to onset was 31 msec (25 to 35 msec). The voltage that obtained maximal tc-MEP response amplitudes was 700 V (450 to 850 V). During a 2-hour period before proximal aortic crossclamping, the within-patient variability (coefficient of variation) of the baseline tc-MEP amplitude was 26% (11% to 50%) and of baseline tc-MEP latency, 3.3% (1.4% to 8.7%). With the closed-loop neuromuscular controller, the level of neuromuscular blockade was maintained between 17% and 23% of control. Variations in the level of neuromuscular blockade had only a minor influence on the observed within-patient tc-MEP variability. Perioperative patient data are given in Table I.

**Prevalence of ischemic tc-MEP changes.** In nine patients tc-MEP changes indicative of spinal cord ischemia were observed either during crossclamping with retrograde perfusion or after aortic repair and release of the crossclamp. Tc-MEPs were unchanged after institution of distal perfusion in 17 of 18 patients. In one patient (patient 17) ischemic tc-MEP changes were seen 14 minutes after the start of retrograde perfusion, but before the aorta was crossclamped. Responses disappeared completely after 18 minutes, and acute dissection with obstruction of critical intercostal arteries was suspected. Despite cessation of bypass flow and pressure manipulations, tc-MEPs did not return during the remainder of the operation, and the patient awoke paraplegic. A possible explanation for the spinal ischemia before crossclamping is that flow reversal caused an intimal flap, atheroma, or air to obstruct an intercostal artery critical to spinal cord blood supply. During the aortic replacement no evidence of an obstruction of a segmental artery was found.

In eight patients the segmental arteries were oversewn after clamping of an aortic segment and confirmation of unchanged tc-MEPs, which suggested that these vessels were not critical to spinal cord blood supply (patients 1, 2, 5, 10, 12, 16, 19, and

20). None of these patients awoke with postoperative paraplegia.

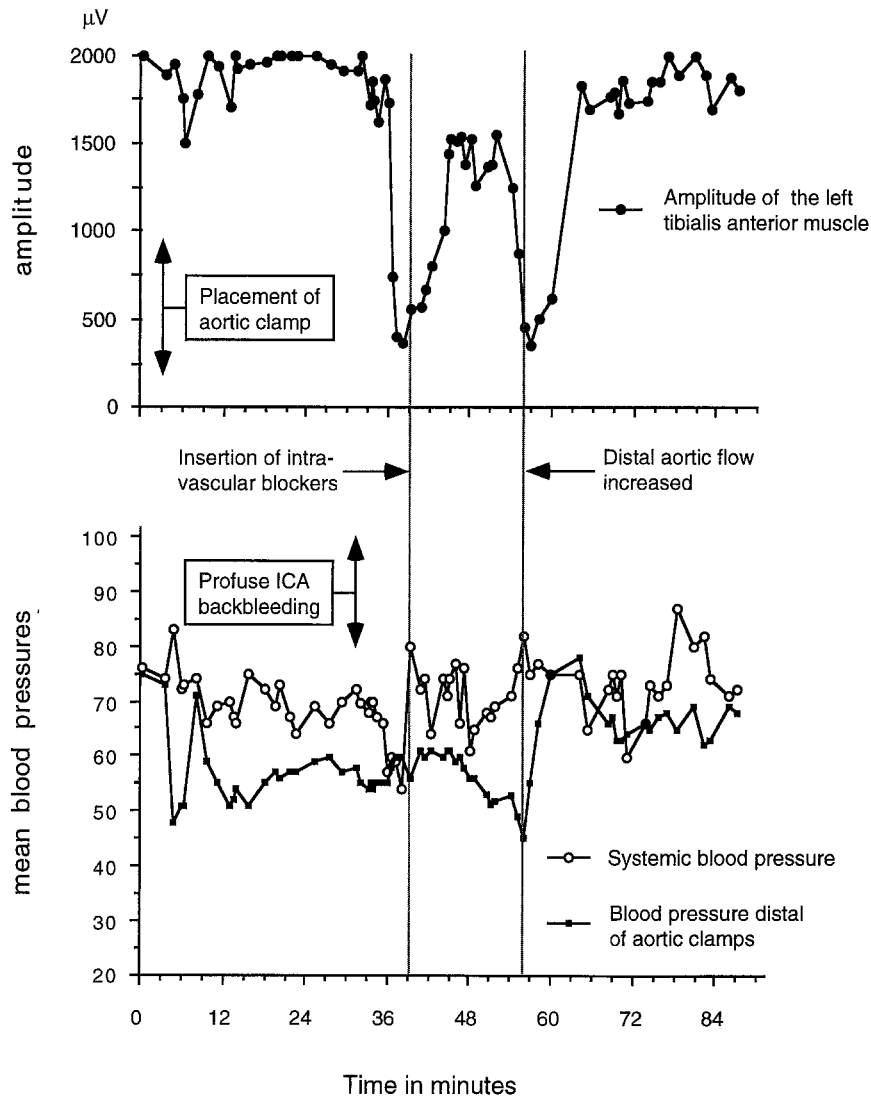
In patients 11, 15, and 18 ischemic tc-MEP changes occurred within the first 5 minutes after exclusion of an aortic segment; accordingly, preservation of segmental arteries was warranted. These patients are described in detail in the case histories that follow.

In five patients (patients 5, 7, 9, 10, and 20) a total of nine ischemic changes in tc-MEP amplitude occurred during crossclamping, which were reversed after interventions to increase spinal cord perfusion. Six interventions consisted of an increase in distal aortic bypass flow, resulting in an increase in distal aortic pressure and a return of MEP amplitude to baseline values. In patient 7 MEP amplitudes were unchanged after initiation of bypass and crossclamping, but decreased after the aneurysm was opened at the thoracic level. Profuse intercostal backbleeding at the T6-12 level was observed. Tc-MEP amplitude recovered after Pruitt blocking catheters had been inserted in the orifices of the intercostal arteries (Fig. 2). We postulated that these intercostal arteries supplied the anterior spinal artery and that the backbleeding produced a pressure gradient along the anterior spinal artery with insufficient spinal cord blood supply. These intercostal arteries were reattached to the graft. The patient had no postoperative neurologic deficits. In patient 5 a technical failure caused a temporary shutoff of the bypass pump. This resulted in loss of the tc-MEP signal within 1 minute. Retrograde perfusion was restored after 2 minutes, and the responses immediately recovered (Fig. 3). In patient 20 tc-MEPs were lost after aortic replacement and cessation of distal bypass, during closure of the surgical wound, but recovered when MAP was increased from 65 to 80 mm Hg.

Compared with amplitude, latency to onset was not a reliable predictor of spinal cord ischemia. There was no correlation between amplitude and onset latency during periods of spinal cord ischemia. When tc-MEP amplitude was reduced to 25% of baseline, no changes in onset latency were observed. Before MEPs disappeared completely, the onset latency increased slightly (1.1 to 2.6 msec) in three patients.

**Clinical results.** Postoperative paraplegia developed in three patients (15%). In patients 11 and 17 tc-MEPs were absent at the end of the operation, and they both awoke paraplegic. In the third patient (patient 15) tc-MEPs were intact at the end of the





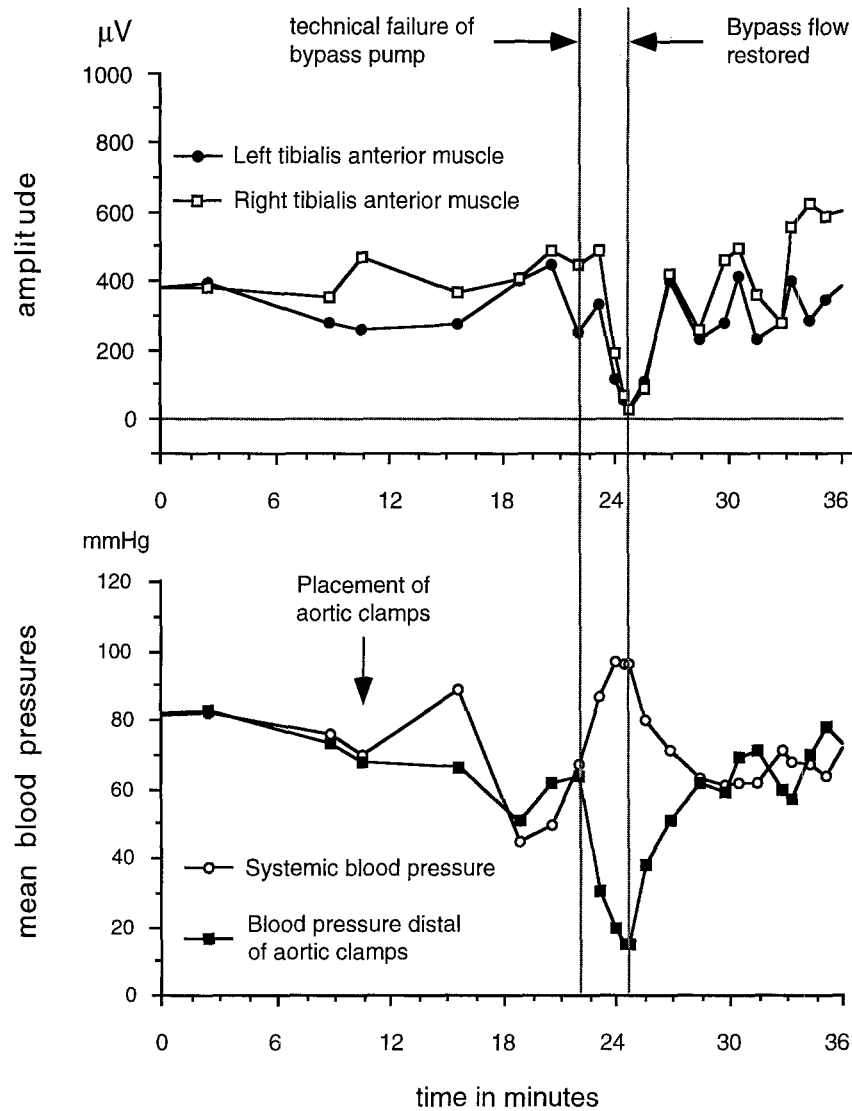
**Fig. 2.** Ischemic tc-MEP changes during an operation for TAA type II (patient 7). The amplitude of the tc-MEP of the left anterior tibial muscle, the systemic blood pressure, and the pressure distal to the clamp are shown versus time. Only tc-MEP amplitude from the left anterior tibial muscle is shown because of peripheral ischemia of the right leg. Profuse intercostal backbleeding at the T6-12 level was stopped by inserting intravascular blocking catheters in the orifices of the intercostal arteries (ICAs) on which tc-MEP amplitude recovered. A second episode of spinal cord ischemia was reversed with increasing distal aortic pressure.

operation, but a delayed paraplegia developed, as confirmed by bilateral absence of tc-MEPs. Seventeen patients had intact myogenic responses and awoke without neurologic deficits at the end of the procedure.

Patient 18 had a postoperative paraparesis in one leg only, which resolved over the course of several days. The neurologic examination revealed a lower motor neuron involvement. At the end of the oper-

ation, tc-MEPs in this patient were absent in the paretic leg and showed no changes in the normal leg.

None of the patients with selective organ perfusion had renal failure or required dialysis. Patients 2 and 3, who did not have selective organ perfusion, died of multiorgan failure 27 and 47 days, respectively, after the operation. Patient 17, who had severe renal failure in the preoperative period, died



**Fig. 3.** In patient 5 a technical failure caused temporary interruption of left atrial–femoral bypass flow. The amplitude of the tc-MEP of the left and right anterior tibial muscle, the systemic blood pressure, and the pressure distal to the clamp are shown versus time. Within 1 minute after retrograde perfusion pressure decreased to zero, myogenic tc-MEPs demonstrated ischemic changes. Immediately after restoration of bypass flow the responses returned to baseline values.

of multiorgan failure 14 days after the operation. Another patient (patient 11) died 2 days after the operation as a result of uncontrollable pulmonary bleeding. Two of the four patients who died were paraplegic.

**Case histories.** Patient 11 was a vital 83-year-old man with extensive atheromatous disease and imminent rupture of a type II TAA. We suspected that a collateral circulation supplied the spinal cord. To test this hypothesis, we temporarily stopped the centrifugal pump after the thoracic aorta was cross-

clamped. Within 2 minutes of retrograde flow reaching zero, the tc-MEPs disappeared. The signal returned to baseline immediately after restoration of retrograde flow. We concluded that collateral flow was insufficient to supply the spinal cord and that reimplantation of intercostal arteries in the graft was warranted. However, no segmental arteries were visible at the level of T8-L1. During the abdominal part of the operation (L1-bifurcation), tc-MEPs disappeared 2 minutes after placement of the clamp, but no lumbar arteries were found.

Tc-MEPs did not return during the operation. The patient awoke paraplegic and died 2 days later. Postmortem examination revealed a spinal cord infarction at the T-4 level.

In patient 15, a 61-year-old man with a type II TAA, staged clamping was used. Clamping the aorta distal of the left carotid artery to the level of T-11 and monitoring of tc-MEPs revealed no intercostal arteries critical to spinal cord blood supply, and eight intercostal arteries were ligated. After clamping of the aorta between the level of T-11 and the bifurcation, tc-MEPs showed ischemic changes suggesting that intercostal arteries critical to spinal cord blood supply originated from this aortic segment. Two large lumbar arteries at the L-2 level were reattached to the graft. Tc-MEPs returned 15 minutes after reperfusion of these intercostal arteries (Fig. 4). The spinal cord was ischemic for 27 minutes, at a blood temperature of 30.6° C. Two days after the operation this patient appeared to have become paraplegic. Myogenic tc-MEPs were absent from both tibialis anterior muscles. Angiographic examination revealed thrombosis of both grafted lumbar arteries.

Patient 18 was a 69-year-old man with a type II TAA. So that the proximal anastomosis of the graft could be constructed, the aorta was clamped between the left subclavian artery and T-8. Immediately thereafter, ischemic tc-MEP changes were observed. The distal clamp was replaced to T-6 and MEPs recovered slightly. After the aorta was opened, tc-MEP amplitude decreased again and did not improve when retrograde flow was increased. It was decided to include four intercostal arteries between the left subclavian artery and T-6 into the graft. Tc-MEPs did not show any recovery after reperfusion of these intercostal arteries. Therefore two large intercostal arteries at the site of the clamp at T-6 were also reattached in the graft. Total spinal cord ischemia time was 53 minutes, and only the tc-MEPs of the left leg recovered (after 6 hours). Postoperative neurologic examination revealed normal function in the left leg and a paraparesis of the right leg. Function in the right leg returned to normal in the course of 1 week.

**Peripheral nerve ischemia.** In 16 patients distal aortic perfusion resulted in temporary occlusion of the left femoral artery at the insertion site of the cannula during the period of retrograde perfusion. In eight patients this resulted in a decrease of tc-MEP amplitude to less than 25% of baseline in the cannulated leg after a median of 37 minutes (30

to 92 minutes), as a result of peripheral nerve and muscle ischemia. Responses returned to baseline values 1 to 6 minutes after flow in the occluded leg was restored. In the remaining eight patients there was no change in tc-MEP amplitude of the cannulated leg.

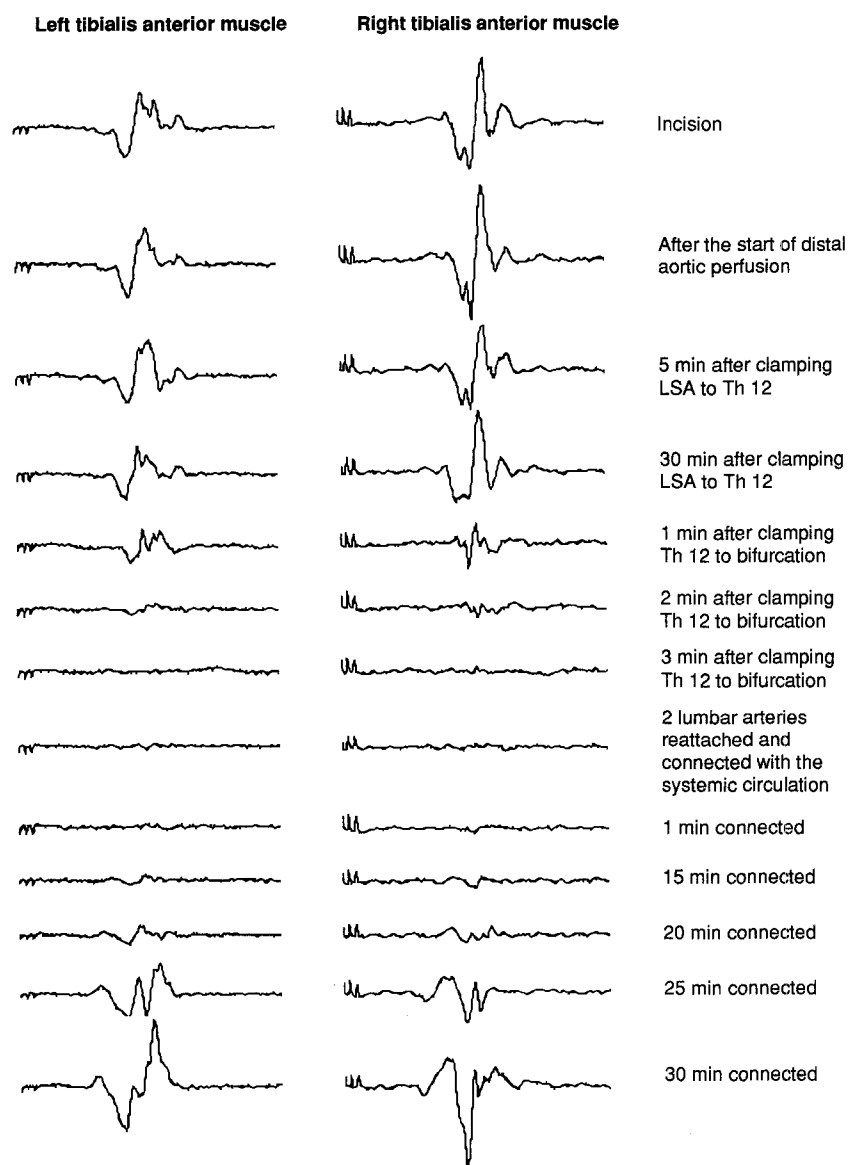
In patients 19 and 20 a separate cannula was inserted in the femoral artery to maintain blood flow in the leg used for retrograde perfusion. In these patients no signs of peripheral ischemia were observed.

In patients 1 and 2, who underwent aneurysm resection without distal bypass, tc-MEP amplitude decreased to less than 25% of baseline 18 and 23 minutes after crossclamping, respectively. The most likely explanation is that in this situation progressive peripheral nerve and muscle ischemia prevents recording of myogenic responses. Myogenic responses returned within minutes after placement of the graft and release of the crossclamp. Both patients awoke without neurologic deficits.

## Discussion

The results of this study demonstrate that monitoring myogenic motor responses to transcranial stimulation is clinically feasible during operations for TAA. After the onset of spinal cord ischemia the signal amplitude decreases rapidly. Tc-MEP monitoring has a high sensitivity in predicting neurologic outcome. The potential benefit of this technique during operations on the aorta is that it may guide timely therapeutic decisions regarding protective strategies aimed at preserving spinal cord blood flow and prevention of paraplegia.

Efforts to decrease the prevalence of neurologic complications have been hampered by the lack of a reliable monitor for the adequacy of spinal cord perfusion. If outcome is to be improved, spinal cord ischemia should be detected as early as possible, and protective strategies must be applied before ischemia has produced irreversible neuronal damage. MEPs reflect conduction in the motor tracts of the spinal cord. They can be elicited transcranially or by stimulating the descending motor tracts via epidural electrodes. We have opted to use electrical transcranial stimulation, because magnetic stimulation requires continuous access to the head. Responses can be recorded from the epidural space, the peripheral nerve, or from the muscle. MEPs have been studied in several experimental models of spinal cord ischemia. The results of these investigations consistently confirm that the amplitude of responses recorded from the epidural space decreases more slowly than



**Fig. 4.** Individual tc-MEP waveforms during a type II TAA resection. The patient (patient 15) was receiving retrograde aortic perfusion. During the thoracic part of the operation no ischemic tc-MEP changes were observed and eight intercostal arteries were ligated. During the abdominal part of the operation ischemic tc-MEP changes were observed within 2 minutes after placement of the clamps between T-12 and the bifurcation. Two large lumbar arteries were identified and reattached to the graft. Total spinal cord ischemic time was 23 minutes. MEPs returned 15 minutes after the blood flow in the reattached lumbar arteries was restored.

responses recorded distal to the anterior horn cells after the onset of spinal cord ischemia.<sup>12-14</sup> The results from the present study confirm animal data that myogenic tc-MEPs are extremely sensitive in detecting a reduction in spinal cord blood flow that is insufficient to maintain synaptic impulse transmission.<sup>8</sup> The sensitivity of myogenic motor-evoked

responses to spinal cord ischemia can be explained neurophysiologically. Myogenic responses are dependent on synaptic processes in the spinal motoneuronal system. The anterior horn cells in the gray matter of the spinal cord have a functional resemblance to cortical brain tissue and react with an almost immediate functional loss after the onset

of ischemia. When SSEPs are monitored, a reduction of the amplitude below 50% of baseline is an indication of ischemic spinal cord dysfunction. Because the tc-MEP signal is more variable and more sensitive to ischemia, we opted to select a more restrictive criterion for spinal cord ischemia: an amplitude reduction to 25% of baseline. Because the coefficient of variation for tc-MEP amplitudes in this series was 26%, this criterion is approximately similar to a decrease to more than 3 standard deviations below the average baseline values.

In the present study, tc-MEPs were also used to reach decisions regarding preservation or ligation of specific intercostal or lumbar arteries. In 90% of the cases one of the segmental arteries between T-8 and L-3 supplies the spinal cord.<sup>15</sup> Failure to reattach segmental arteries between T-11 and L-1 increases the risk of paraplegia.<sup>5</sup> However, routine reattachment increases the total aortic crossclamp time, with an attendant higher likelihood of postoperative neurologic deficits.<sup>1</sup> Moreover, with routine reimplantation of segmental arteries between T-11 and L-1, there is some risk of paraplegia when critical intercostal or lumbar arteries originate from a higher or lower level. In the present study we have ligated segmental arteries in eight patients, after tc-MEP confirmation that they were not critical to spinal cord blood supply. None of these patients had postoperative paraplegia. Without monitoring, these vessels would have been reattached, thus prolonging aortic crossclamp time. In three patients (patients 11, 15, and 18), tc-MEP amplitude decreased after exclusion of an aortic segment, suggesting that preservation of segmental arteries was warranted. In two of these patients, intercostal or lumbar arteries were reimplanted and tc-MEPs recovered after restoration of flow in these vessels. The present observations confirm that tc-MEPs can identify an aortic segment from which intercostal or lumbar arteries arise that are critical to spinal cord blood supply; accordingly, a selective segmental revascularization can be performed. This might reduce aortic crossclamp times and avoid the risk that critical segmental arteries from a high thoracic or low lumbar level will not be reattached to the graft.

The short interval between the onset of spinal cord ischemia and a decrease of tc-MEP amplitude allowed us to increase bypass flow and MAP to maintain adequate spinal cord perfusion. In five patients, ischemic tc-MEP changes were reversible after increasing MAP or bypass flow. Cunningham and associates<sup>16</sup> reported that SSEPs were pre-

served in dogs when distal perfusion pressure remained above 60 mm Hg. Three of our patients had ischemic tc-MEP changes when distal aortic pressure was less than 60 mm Hg. However, our data suggest that an MAP or a distal aortic pressure of 60 mm Hg may not always be sufficient to guarantee adequate spinal cord perfusion during or after TAA operations. Tc-MEP monitoring allows the surgical team to distinguish between patients who have adequate spinal cord perfusion and those who will benefit from higher distal flows or pressures (or both). In one of our patients (patient 9), tc-MEP amplitude decreased despite a distal aortic pressure of 60 mm Hg, and the signal recovered only after distal aortic pressure was increased above 70 mm Hg. In patient 20 tc-MEPs showed ischemic changes during the reperfusion phase when MAP was 65 mm Hg and recovered when MAP was increased to 80 mm Hg.

Delayed paraplegia accounts for one third of the cases of postoperative neurologic injury.<sup>17</sup> It may result from a decrease in spinal cord oxygenation during episodes of postoperative hypotension or respiratory failure. The present data suggest that it might be relevant to investigate whether continued tc-MEP monitoring in the postoperative phase is useful in detecting impending spinal cord ischemia, to allow institution of corrective measures to restore spinal cord perfusion.

In this study, results of tc-MEP monitoring predicted neurologic motor function correctly in all cases. All patients in whom tc-MEPs were absent at the end of the operation awoke paraplegic, and there were no false negative results (postoperative motor deficits despite unchanged motor-evoked responses). One patient who had normal tc-MEPs at the end of the operation became paraplegic as a result of thrombosis of the reattached lumbar arteries, after an episode of low blood pressure on the second postoperative day. Tc-MEPs were absent at the time paraplegia was suspected. In patient 18, who had a temporary paresis of the right leg, only the right-sided tc-MEP showed ischemic changes at the end of the operation. The major limitation of SSEP monitoring during operations on the thoraco-abdominal aorta is the occurrence of motor deficits despite unchanged or recovered SSEPs.<sup>7, 18-22</sup> One explanation might be that ascending sensory conduction in the dorsal columns is preserved longer during ischemia and often recovers after a period of prolonged spinal cord ischemia that has produced infarction in the central gray matter. In addition,

SSEPs may not reflect selective ischemia of the spinal motor system, because the motoneuronal system in the anterior horn receives its blood supply from the anterior spinal artery, whereas the dorsal columns are supplied via the posterior spinal arteries.<sup>23, 24</sup>

Although we were able to record stable myogenic responses to transcranial electrical stimulation during prolonged surgical procedures, careful planning of the anesthetic technique was necessary. Not only are myogenic tc-MEPs sensitive to spinal cord ischemia, but they are readily depressed by many commonly used anesthetics. This imposes limitations on the range of drugs and dosages that can be used. Nitrous oxide,<sup>25</sup> propofol,<sup>26</sup> benzodiazepines,<sup>26</sup> and especially volatile anesthetics<sup>27</sup> depress synaptic conduction and severely decrease the amplitude of the myogenic response. Because tc-MEP amplitude is also influenced by the level of neuromuscular blockade, a stable level of neuromuscular blockade should be maintained.<sup>28, 29</sup> Using the closed-loop vecuronium infusion, we were able to maintain the level of neuromuscular blockade within a narrow range and thus minimize the influence of fluctuations in relaxation level on the variability of the tc-MEP signal. The use of paired stimuli as opposed to a single transcranial stimulus dramatically improves the amplitude and reproducibility of the resulting muscle responses.<sup>10</sup> Fortunately, with the double pulse stimulation paradigm and the anesthetic technique used, we could record tc-MEPs of sufficient reproducibility to allow continuous monitoring of anterior horn function.

Evoked potential techniques that rely on stimulation and recording from nerves and muscles in the leg are limited by the occurrence of lower limb ischemia during TAA operations. When simple aortic crossclamping without retrograde perfusion is used, as in our first two patients, ischemia of the peripheral nerve and muscle will result in loss of myogenic motor responses (and SSEPs), usually after approximately 30 minutes. This implies that once the decision is made to proceed with aortic reconstruction without retrograde perfusion, myogenic tc-MEPs can be used only to monitor spinal cord function after lower limb blood flow is restored. When retrograde aortic perfusion is used, at least one leg will be normally perfused, but peripheral ischemia may occur in the leg used for femoral artery cannulation. In this study tc-MEP amplitude in the occluded leg decreased gradually and was ultimately lost in eight of 16 patients. The practical consequence is that beyond the first 10 minutes after

femoral artery cannulation, only tc-MEPs recorded from muscles in the leg opposite the cannulation site can be expected to reliably reflect spinal cord function. This limitation can be overcome and reliability of the tc-MEPs can be increased if a second cannula is inserted to perfuse the periphery of the femoral artery used for retrograde bypass, as observed in our last two patients.

We conclude from this study that monitoring myogenic motor-evoked responses is effective in detecting spinal cord ischemia during operations for TAA. This technique allows prompt assessment of the adequacy of spinal cord blood flow. When distal perfusion is used, tc-MEPs may guide management of bypass flow and pressures. The present data also suggest that myogenic tc-MEPs may help to identify segmental arteries that are critical to spinal cord blood supply and need to be reimplanted. A larger patient group is needed to confirm whether the availability of tc-MEPs, in support of spinal cord protective techniques, may substantially reduce the prevalence of perioperative and delayed paraplegia.

#### REFERENCES

1. Crawford ES, Crawford JL, Safi HJ, et al. 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.
2. Brewer LA, Fosburg RG, Mulder GA, Verska JJ. Spinal cord complications following surgery for coarctation of the aorta. *J Thorac Cardiovasc Surg* 1972;64:368-79.
3. Svensson LG, Crawford ES, Patel V, McLean TR, Jones JW, DeBakey ME. Spinal oxygenation, blood supply localization, cooling, and function with aortic clamping. *Ann Thorac Surg* 1992;54:74-9.
4. Svensson LG, Patel V, Robinson MF, Ueda T, Roehm JJ, Crawford ES. Influence of preservation or perfusion of intraoperatively identified spinal cord blood supply on spinal motor evoked potentials and paraplegia after aortic surgery. *J Vasc Surg* 1991;13:355-65.
5. Svensson LG, Hess KR, Coselli JS, Safi HJ. Influence of segmental arteries, extent, and aortofemoral bypass on postoperative paraplegia after thoracoabdominal aortic operations. *J Vasc Surg* 1994;20:255-62.
6. Safi HJ, Bartoli S, Hess KR, et al. Neurologic deficit in patients at high risk with thoracoabdominal aortic aneurysms: the role of cerebral spinal fluid drainage and distal aortic perfusion. *J Vasc Surg* 1994;20:434-44.
7. Crawford ES, Mizrahi EM, Hess KR, Coselli JS, Safi HJ, Patel VM. The impact of distal aortic perfusion and somatosensory evoked potential monitoring on prevention of paraplegia after aortic aneurysm operation. *J Thorac Cardiovasc Surg* 1988;95:357-67.
8. de Haan P, Kalkman CJ, Ubachs LH, Drummond JC. Sensitivity of myogenic and epidural transcranial motor evoked potentials for detection of acute spinal cord ischemia [abstract]. *Anesthesiology* 1994;81:A894.
9. Jacobs MJHM, de Mol BAJM, Legemate DA, de Haan P,

- Kalkman CJ. Retrograde and selective organ perfusion during thoraco-abdominal aortic aneurysm repair. *Eur J Vasc Endovasc Surg*. In press.
10. Kalkman CJ, Ubags LH, Been HD, Swaan A, Drummond MD. Improved amplitude of myogenic motor evoked responses after paired transcranial electrical stimulation during sufentanil/nitrous oxide anesthesia. *Anesthesiology* 1995;83:270-6.
  11. Kalkman CJ. Labview: a software system for data acquisition, data analysis, and instrument control. *J Clin Mon* 1995;11:51-8.
  12. Mongan PD, Peterson RE, Williams D. Spinal evoked potentials are predictive of neurologic function in a porcine model of aortic occlusion. *Anesth Analg* 1994;78:257-66.
  13. Reuter DG, Tacker WJ, Badylak SF, Voorhees W, Konrad PE. Correlation of motor-evoked potential response to ischemic spinal cord damage. *J Thorac Cardiovasc Surg* 1992;104:262-72.
  14. Konrad PE, Tacker WA, Levy WJ, Reedy DP, Cook JR, Geddes LA. Motor evoked potentials in the dog: effects of global ischemia on spinal cord and peripheral nerve signals. *Neurosurgery* 1987;20:117-24.
  15. Svensson LG, Klepp P, Hinder RA. Spinal cord anatomy of the baboon: comparison with man and implications of spinal cord blood flow during thoracic aortic cross-clamping. *S Afr J Surg* 1986;24:32-4.
  16. Cunningham JJ, Laschinger JC, Merkin HA, et al. Measurement of spinal cord ischemia during operations upon the thoracic aorta: initial clinical experience. *Ann Surg* 1982;196:285-96.
  17. Crawford ES, Svensson LG, Hess KR, et al. A prospective randomized study of cerebrospinal fluid drainage to prevent paraplegia after high-risk surgery on the thoracoabdominal aorta. *J Vasc Surg* 1991;13:36-45.
  18. Lesser RP, Raudzens P, Luders H, et al. Postoperative neurological deficits may occur despite unchanged intraoperative somatosensory evoked potentials. *Ann Neurol* 1986;19:22-5.
  19. Dawson EG, Sherman JE, Kanim LE, Nuwer MR. Spinal cord monitoring: results of the Scoliosis Research Society and the European Spinal Deformity Society Survey. *Spine* 1991;16:S361-4.
  20. Owen JH, Laschinger J, Bridwell K, et al. Sensitivity and specificity of somatosensory and neurogenic-motor evoked potentials in animals and humans. *Spine* 1988;13:1111-8.
  21. Tsuchida K, Hashimoto A, Seino R, Hirayama T, Aomi S, Koyanagi H. Two case reports of neurologic complications after aortic aneurysm operation. *Nippon Kyobu Geka Gakkai Zasshi* 1989;37:1995-2000.
  22. Schepens MA, Boezeman EHJF, Hamerlijnck RPHM, ter Beek H, Vermeulen FEE. Somatosensory evoked potentials during exclusion and reperfusion of critical aortic segments in thoracoabdominal aortic aneurysm surgery. *J Card Surg* 1994;9:692-702.
  23. Takaki O, Okumura F. Application and limitation of somatosensory evoked potential monitoring during thoracic aortic aneurysm surgery: a case report. *Anesthesiology* 1985;63:700-3.
  24. Zornow M, Grafe M, Tybor C, Swenson M. Preservation of evoked potentials in a case of anterior spinal artery syndrome. *Electroenceph Clin Neurophysiol* 1990;77:137-9.
  25. Ghaly RF, Stone JL, Levy WL, Kartha R, Aldrete JA. The effect of nitrous oxide on transcranial magnetic-induced electromyographic responses in the monkey. *J Neurosurg Anesth* 1990;2:175-81.
  26. Kalkman CJ, Drummond JC, Ribberink AA, Patel PM, Sano T, Bickford RG. Effects of propofol, etomidate, midazolam and fentanyl on motor evoked responses to transcranial electrical or magnetic stimulation in humans. *Anesthesiology* 1992;76:502-9.
  27. Kalkman CJ, Drummond JC, Ribberink AA. Low concentrations of isoflurane abolish motor evoked responses to transcranial electrical stimulation during nitrous oxide/opioid anesthesia in humans. *Anesth Analg* 1991;73:410-5.
  28. Kalkman CJ, Drummond JC, Kennelly NA, Patel PM, Partridge BL. Intraoperative monitoring of tibialis anterior muscle motor evoked responses to transcranial electrical stimulation during partial neuromuscular blockade. *Anesth Analg* 1992;75:584-9.
  29. Stinson LJ, Murray MJ, Jones KA, et al. A computer-controlled, closed-loop infusion system for infusing muscle relaxants: its use during motor-evoked potential monitoring. *J Cardiothorac Vasc Anesth* 1994;8:40-4.

### Commentary

Stimulation of the brain and elicitation of peripheral motor-evoked responses appears to have its origins in early history, but only now is the technique being safely and painlessly performed. In the Hippocratic writings (400 BC), elicitation of contralateral peripheral motor responses from scratching the motor cortex during trepanation or after an open skull fracture is clearly documented. Interest in elicitation of motor responses was revived in 1909 by Cushing<sup>1</sup> when he used local anesthesia for incisions and electrically stimulated the brain directly to obtain motor responses. Curiously, during the interim, there does not appear to have been any interest in the method for spinal cord testing in association with aortic surgery.

Beginning in 1983, we used direct electrical stimulation of the motor cortex of nonhuman primate brains to establish whether lower limb paralysis had occurred after aortic crossclamping in some 70 experiments.<sup>2</sup> To be useful in human beings, however, a less invasive method for testing the spinal cord was needed. During aortic operations, the area of spinal cord that shows earliest evidence of ischemia and thus dysfunction is the lower thoracic and lumbar spinal cord. Of note, the anterior horn motor cells for the lower limbs are situated in the gray matter in this area. The cauda equina and peripheral nerves are somewhat more resistant to ischemia because they consist of white matter and nerve axons. Thus, to test for lower spinal cord function, we placed stimulating stainless steel electrodes alongside the anterior thoracic spinal cord in pigs and measured spinal motor-evoked potentials (MEPs) from needle electrodes in the muscles of the lower limbs.<sup>3,4</sup> This technique was accurate, sensitive, and specific, and the deteriorating function of the spinal cord could be tracked as ischemia worsened. Furthermore, as aortic crossclamps were removed, recovery of the spinal cord, or the lack of recovery, could clearly be followed, enabling the prediction of postoperative paraplegia or paraparesis. Other investigators, including Laschinger<sup>5</sup> and Allen<sup>6</sup> and their colleagues, also found similar MEP methods to be highly accurate. The advan-

tage of using muscle-recording electrodes was that the spinal MEPs were amplified by the muscles, resulting in a myogenic response with greater amplitude. The spinal MEPs were therefore easier to record and allowed subtle changes to be detected. We then used this method in patients, using an intrathecal electrode placed by lumbar puncture, but muscle relaxants could not be used because they eliminated the spinal MEPs recorded from the muscle. Furthermore, without muscle paralysis, the tetanic spasms of the legs were disconcerting. In porcine animal models, we tried to detect the sciatic nerve spinal MEPs and also stimulate the brain with platinum plate electrodes or stimulate the spinal cord via neck needle electrodes to overcome some of these problems. These latter methods met with limited success.

In 1984, Levy and colleagues<sup>7</sup> were able to show that transcranial stimulation of the brain with plate electrodes over the motor cortex resulted in MEPs that could be recorded in high thoracic needle electrodes. In the current article, de Haan and colleagues have significantly advanced the use of MEPs for monitoring spinal cord function in human beings despite the administration of muscle relaxants. As described by Levy, de Haan's group used transcranial stimulation of the motor cortex to elicit the MEPs. The latter potentials, however, were recorded by skin gel electrodes over the anterior tibialis muscles of the leg. To obviate the problem of neurovascular blocking agents eliminating recordable MEPs, they carefully controlled the rate of infusion of vancuronium so that complete blockage was not present. This was done by stimulating the ulnar nerve and testing for hypothermic compound muscle potentials. Using this technique, they were able to show in 20 patients that this method was sensitive and specific for detecting spinal cord ischemia.

Inasmuch as MEP monitoring is highly accurate, and from the study by de Haan and colleagues including patients with muscle relaxants, the question arises whether the method can safely be used? Considering the large number of stimuli applied to each individual patient, their technique appears to be safe with minimal risk. The charge density, however, must not exceed 40 microcoulomb/phase/cm<sup>2</sup>.<sup>8,9</sup> What then are the benefits? Several articles have now been published showing that somatosensory evoked potential (SSEP) monitoring can be used to detect segmental artery blood supply to the spinal cord, to determine whether the distal aortic perfusion pressure is adequate, and also to predict whether the patient will awake with a neurologic deficit. On the contrary, however, other articles have shown that SSEP monitoring has no value in determining perfusion flow rates or in influencing attachment of segmental arteries. They further suggest that SSEP monitoring is inaccurate in determining post-operative neurologic function. This discrepancy is partially due to the fact that SSEP monitors the posterior columns and, thus, sensory neurologic functions, both of which are less affected by spinal cord ischemia. Thus MEP monitoring has potential benefits over SSEP monitoring in detecting spinal cord ischemia resulting from inadequate perfusion or failure to reattach segmental arteries.

Should MEP monitoring be used? If distal perfusion pressures are kept above 70 to 75 mm Hg and all segmental arteries (or even only those between T-7 and L-1, as we advocate) are reattached, it could be argued that MEP monitoring confers no added benefit. Similarly, patients undergoing deep hypothermic circulatory arrest would receive little benefit. From the study of de Haan and colleagues, however, it appears that two patients did benefit from MEP monitoring even though the aforementioned criteria were met.

In summary, de Haan and colleagues have shown this method to be both accurate and safe. Factors militating against it are the multiple crossclampings required for identifying segmental arteries, the extensive equipment needed, and the expertise to detect changes. Nevertheless, clearly it is a most useful research tool, although its application and benefits in the routine clinical setting remain to be established.

Lars G. Svensson, MD, PhD  
Lahey Hitchcock Clinic  
Burlington, MA 01805  
12/1/77320

#### REFERENCES

1. Cushing H. A note upon faradic stimulation of the post-central gyrus in conscious patients. *Brain* 1909;32:44-54.
2. Svensson LG, Richards E, Coull A, et al. Relationship of spinal cord blood flow to vascular anatomy during thoracic aortic crossclamping and shunting. *J Thorac Cardiovasc Surg* 1986;91:71-8.
3. Svensson LG, Patel VP, Robinson MF, Ueda T, Roehm JOF, Crawford ES. Influence of preservation or perfusion of intraoperatively identified spinal cord blood supply on spinal motor-evoked potentials and paraplegia after aortic surgery. *J Vasc Surg* 1991;13:355-65.
4. Svensson LG, Crawford ES, Patel V, McLean TR, Jones JW, DeBaKey ME. Spinal oxygenation, blood supply localization, cooling, and function with aortic clamping. *Ann Thorac Surg* 1992;54:74-9.
5. Laschinger JC, Owen J, Rosenbloom M, et al. Direct noninvasive monitoring of spinal cord motor function during thoracic aortic occlusion: use of motor-evoked potentials. *J Vasc Surg* 1988;7:161-71.
6. Allen BT, Doblas M, Owen J, et al. Cerebrospinal fluid pressure, somatosensory and motor-evoked potentials in spinal cord ischemia after lumbar artery ligation. *Surg Forum* 1991;42:262-5.
7. Levy WJ, York DH, McCaffrey M, et al. Motor-evoked potentials from transcranial stimulation of the motor cortex in humans. *Neurosurgery* 1984;15:287-302.
8. Agnew WF, McCreery DB. Considerations of safety in the use of extra cranial stimulation for motor-evoked potentials. *Neurosurgery* 1987;20:143-7.
9. McCreery DB, Agnew WF, Yeun TGH, 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.