Motor and Somatosensory Evoked Potentials

Their Role in Predicting Spinal Cord Ischemia in Patients Undergoing Thoracoabdominal Aortic Aneurysm Repair with Regional Lumbar Epidural Cooling

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Background: Paraplegia is a devastating complication for patients undergoing repair of thoracoabdominal aortic aneurysms. A monitor to detect spinal cord ischemia is necessary if anesthesiologists are to intervene to protect the spinal cord during aortic aneurysm clamping.

Methods: The medical records of 60 patients who underwent thoracoabdominal aortic aneurysm repair with regional lumbar epidural cooling with evoked potential monitoring were reviewed. The authors analyzed latency and amplitude of motor evoked potentials, somatosensory evoked potentials, and H reflexes before cooling and clamping, after cooling and before clamping, during clamping, and after release of aortic cross clamp.

Results: Twenty minutes after the aortic cross clamp was placed, motor evoked potentials had 88% sensitivity and 65% specificity in predicting spinal cord ischemia. The negative predictive value of motor evoked potentials at 20 min after aortic cross clamping was 96%.

Conclusions: Rapid loss of motor evoked potentials or H reflexes after application of the aortic cross clamp identifies a subgroup of patients who are at high risk of developing spinal cord ischemia and in whom aggressive anesthetic and surgical interventions may be justified.

THE main indication for surgical resection of thoracoabdominal aortic aneurysms (TAAAs) is their potential to rupture.[1] However, surgical repair of TAAAs is complicated by significant morbidity and mortality.[2] The incidence of paraplegia can be as low as 6% or as high as 40% if all manifestations of spinal cord ischemia (SCI) are included.[3,4]

Surgical techniques to decrease the duration of ischemia include the Crawford aortic inlay technique, the single-clamp repair technique, and the sequential aortic clamping technique.[5] Intrathecal papaverine combined with cerebrospinal fluid (CSF) drainage can decrease the incidence of SCI.[6] Drainage of CSF during aortic cross clamping is a physiologic technique that maintains cord perfusion pressure and decreases the incidence of SCI.[7,8] Mild systemic hypothermia (33°–34°C) protects neural tissue during ischemia,[9] but even mild hypothermia is associated with an increase in bleeding and infectious complications.[10]

Some advocate monitoring the function of the spinal cord with evoked potentials (EPs)—either somatosensory evoked potentials (SSEPs) or motor evoked potentials (MEPs)—to detect SCI and the efficacy of interventions to treat spinal cord ischemia.[11] MEPs as a monitor of SCI are preferred because they monitor the activity of the anterior spinal cord where the cell bodies of the motor neurons lie.[12]

A recent review of TAAA surgical repair found that regional lumbar epidural cooling (RELEC) decreased the incidence of SCI compared with historic controls.[13] However, the authors did not use EPs to monitor for SCI. The aim of our study in patients undergoing TAAA surgical resection using RELEC was to determine the utility of EPs in predicting the development of postoperative paraparesis and paralysis.

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Materials and Methods

The Mayo Clinic institutional review board (Rochester, Minnesota) approved this retrospective study of all patients with a TAAA who underwent surgical repair using RELEC between 1998 and 2000, who had EP monitoring, and who survived the surgical procedure. Patients who had not given previous consent for a retrospective research chart review were excluded from the study. Demographic data, age, and sex were recorded. Aneurysms were classified as Crawford type I, II, III, or IV and V (descending thoracic aneurysm).[14]

Anesthesia Technique

The anesthesia technique was standardized. Intravenous fentanyl, 50–200 μg, was used for sedation preoperatively. Anesthesia was induced intravenously with fentanyl, up to 10 μg/kg, followed by intravenous sodium thiopentone, 2–5 mg/kg titrated to produce unconsciousness. Atracurium, 0.5 mg/kg, was then administered intravenously. Lung separation was achieved using a bronchial blocker. Either a Univent® (Vitaid Ltd., Toronto, Ontario, Canada) endotracheal tube or an endotracheal tube with an Arndt® Bronchial Blocker (Cook Critical Care, Bloomington, IN) was used. Correct positioning of the bronchial blocker was confirmed using a fiberoptic bronchoscope. Anesthesia was maintained by infusing a solution of 60 ml (50 μg/ml) of fentanyl and 1.6 mg scopolamine intravenously at a rate of 0.3 ml · kg^{-1} · h^{-1} for the duration of the case. Neither drug interferes with MEPs, and scopolamine, importantly, has an amnestic effect. Atracurium at 0.2 μg · kg^{-1} · min^{-1} was infused to maintain the compound muscle action potential at 20% of baseline.

Regional Lumbar Epidural Cooling, Spinal Cord Drainage, and Temperature Measurement

An epidural catheter was inserted at T12, for infusion of saline that was cooled by passing the saline through multiple coils of tubing submerged in ice water. At the T12 epidural site, a thermistor was inserted into the epidural catheter, and the temperature of the infusate was maintained at 10.8°C. Another epidural catheter was inserted at L4–L5 to drain any excess fluid from the epidural space. To measure the temperature of the intrathecal space, a 4-French pediatric pulmonary artery catheter with thermistor tip was inserted intrathecally *via* a 15-gauge epidural needle at L3–L4 or L2–L3 (fig. 1). After placement of all catheters, a cooling test was undertaken with the T12 epidural catheter infused with cooled saline to achieve and document that we could decrease the CSF temperature to 28°C. The infusion of cooled saline was then stopped. Thirty minutes before aortic cross clamping, the cooled saline infusion was restarted at 600 ml/h to cool the CSF to approximately 26°–28°C. After this was achieved, the infusion rate of cooled saline was decreased and maintained at 300 ml/h. Cooling was then maintained until the aortic cross clamp was released. The patient's CSF temperature was measured continuously and recorded every 5 min by the thermistor on the 4-French catheter.

To attempt to optimize the spinal cord perfusion pressure, the 4-French pulmonary artery catheter was used to drain CSF to gravity throughout the case, with monitoring of CSF pressure every 5 min during aortic cross clamping and every 15 min after release of the aortic cross clamp.

Surgical Technique

After epidural catheters and invasive monitors were placed, the anesthetized patient was placed in the right lateral decubitus position, and a left thoracoabdominal incision was made. The right lung was ventilated with collapse of the left lung to allow easier dissection of the TAAA. Before aortic cross clamping, cannula were inserted in the left atrium and left internal iliac artery, allowing for a centrifugal pump to shunt blood from the left atrium to the left internal iliac artery. During resection of the TAAA, intercostal arteries were identified and reimplanted when possible. After aortic cross clamp release, RELEC was terminated, and the spinal cord was allowed to passively rewarm.

Evoked Potential Technique

The patient was prepared for EP monitoring by a certified clinical neurophysiologic technician who assisted in monitoring EPs throughout the duration of the operation. A clinical neurophysiologist assured proper meth-

Fig. 1. Regional lumbar epidural cooling. CSF = cerebrospinal fluid; Fr = French; G = gauge; NS = normal saline.
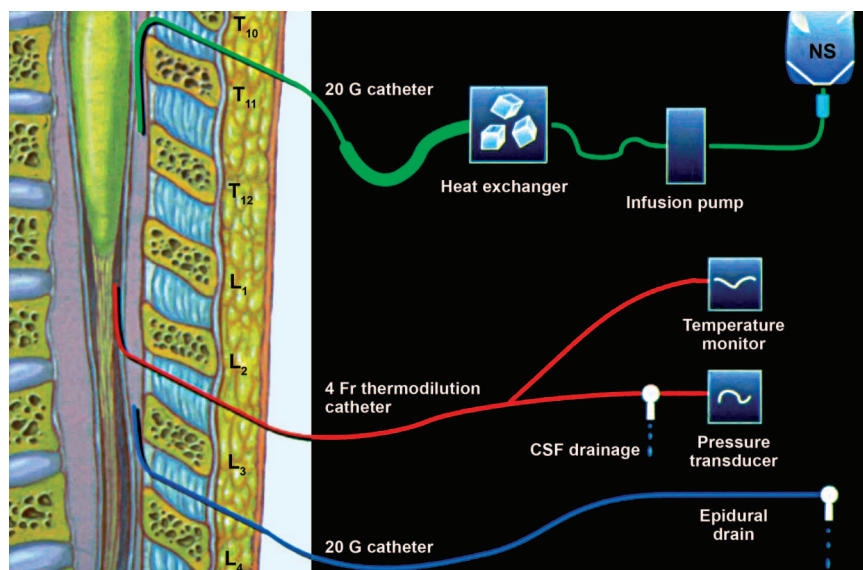
odology throughout the surgery. Each patient had SSEPs, MEPs, and the H reflex monitored using a Nicolet Viking IVP machine (Nicolet, Madison, WI). SSEPs were elicited by percutaneous stimulation of each tibial nerve at the ankle sequentially. Bilateral tibial stimulation was used if responses were not obtained with unilateral stimulation. Tibial SSEPs were recorded from Cz to Fz with Disa 10-mm needle electrodes. MEPs were elicited by percutaneous stimulation of the cervical spinal cord with a supramaximal stimulus to needle electrodes on the left and right C7 lamina and a nasopharyngeal electrode. After induction of anesthesia and before surgical incision, the amplitude of the MEPs at a fixed stimulus intensity was compared using each of the three electrodes as anode and as cathode to identify the optimal electrode pair. Responses were then obtained with supramaximal stimulation. MEPs were recorded from surface electrodes over the anterior tibial, rectus femoris, and abductor hallucis muscles. The H reflex was elicited with submaximal stimuli through a needle electrode in the popliteal fossa. H reflexes were recorded with surface electrodes on the soleus muscle.

The amplitude (microvolts) and latency (milliseconds) of each EP was recorded approximately 15–20 times at 5-min intervals before aortic cross clamp. The amplitude and latencies of each potential were assessed for variability during this baseline period. A reduction in amplitude was identified as any change beyond that seen during the baseline recording. Each of the EPs was then repeated sequentially as rapidly as possible during and after cross clamp of the aorta (3- to 5-min intervals). An absent EP was defined as an EP amplitude that had decreased to between 0 and 25% of the baseline amplitude measurement. The appendix lists the complete set of guidelines used to extract the voluminous real-time EP data.

A train-of-four median/thenar compound muscle action potential was recorded every 2 min to monitor the level of neuromuscular junction block. Amplitudes of MEPs were corrected for the level of neuromuscular junction block. If there was a change in MEP, the technologist and electrophysiologist evaluated the stimulation and recording systems and the monitoring machine to assure the changes were not due to technical factors. Measurements were then repeated within 5 min, with notification of the operating team when there was further reduction or complete loss of the response.

After aortic cross clamping, if the MEPs were lost, spinal cord perfusion pressure was optimized by increasing mean arterial blood pressure and decreasing cerebrospinal pressure by draining more CSF, and transfusing packed erythrocytes to maintain a hemoglobin of greater than or equal to 10 mg/dl.

Evoked Potential Data Measurements

From the data recorded on each patient, the following factors were extracted:

Before aortic cross clamp: Presence or absence of MEPs, SSEPs, or H reflexes just before aortic cross clamping.

Aortic cross clamping: Duration of aortic cross clamping and surgical time; the time to first absent MEP, SSEP, or H reflex during clamping (only for those with positive EPs just before the cross clamp was placed); the maximum period of time MEP, SSEP, and H reflex were completely absent while clamped; and whether MEPs, SSEPs, and H reflexes were absent just before the cross clamp was removed.

After aortic cross clamp: For patients with absent MEPs, SSEPs, or H reflexes just before the cross clamp was removed, the length of time for the EP to return.

Outcome Measurements

The primary outcome measure was the absence or presence of SCI, defined as paraplegia or paraparesis detected postoperatively.

Statistical Analysis

Numerical data were summarized and the sample median and interquartile range calculated. Patient characteristics were compared with Mann–Whitney and Fisher exact tests to investigate associations with postoperative SCI. Logistic regression was used to determine the relation between the time from clamping to first zero EP and development of SCI, and the maximum time EP was absent and development of SCI. The Kaplan–Meier method was used to estimate the distributions of time to first absent EP during aortic cross clamping and time to return of the EP after cross clamp removal. Log-rank tests were used to examine associations between these times and SCI. Sensitivity and specificity were estimated along with exact binomial 95% confidence intervals, using absent MEP as a diagnostic measure for SCI. Statistical significance was determined at the 5% level.

Results

There were 60 patients who underwent TAAA repair with RELEC using EP monitoring that met the study inclusion criteria; however, 2 patients did not have satisfactory baseline electrophysiologic studies before aortic cross clamping and were excluded. The 2 patients excluded from the analysis did not develop SCI. The median age of the remaining 58 patients was 69 yr, and 55% of the patients were women. The numbers and types of aneurysm and the outcomes are listed in figure 2. Ten patients had postoperative SCI (SCI group), and 48 patients had no evidence of SCI (non-SCI group). There was no evidence of any effect of age or sex on the likelihood of SCI (table 1).

Before Aortic Cross Clamp

Regional lumbar epidural cooling produced moderate spinal cord hypothermia of approximately 28°C before

Fig. 2. Thoracoabdominal aneurysm classification (*A*) and incidence of paraplegia (*B*). These data are from the current study.

	I	II	III	IV	V
A.	7 (11%)	18 (30%)	17 (28%)	7 (11%)	12 (20%)
B.	0	5 (50%)	5 (50%)	0	0
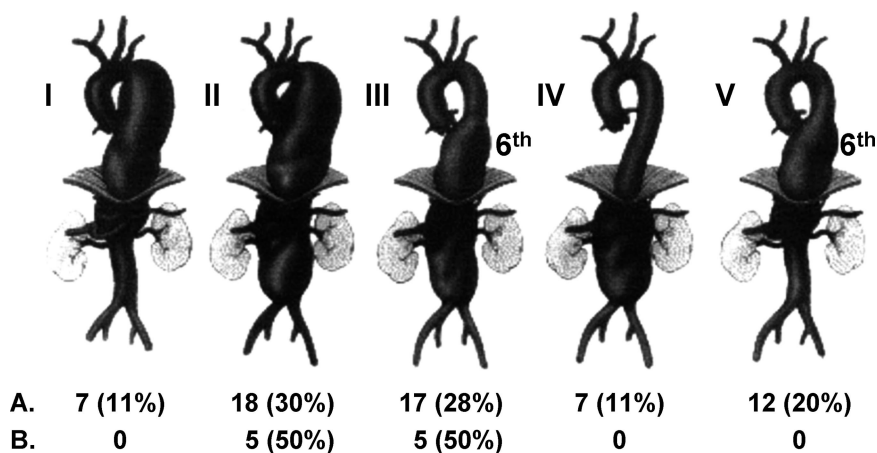
aortic cross clamping. Ninety percent of patients (9 of 10) in the SCI group and 88% of patients (42 of 48) in the non-SCI group had intrathecal temperatures less than or equal to 30°C. Nine of the 58 patients (15%) had absent preclamp MEPs, 2 of the 58 patients (3%) had absent preclamp SSEPs, and 12 of the 58 patients (21%) had absent H reflexes.

Aortic Cross Clamping

The median aortic cross clamp time for the non-SCI group was 60 min *versus* 66 min in the SCI group ($P = 0.23$; fig. 3).

Table 2 and figure 4 summarize EP recording data measured during aortic cross clamping for patients whose MEP reading was present before aortic cross clamp application. For these patients, estimated median time to absent MEPs during aortic cross clamping was 10 min in the SCI group and 31 min in the non-SCI group ($P = 0.004$). Also, there was evidence that a longer absence of MEPs during aortic cross clamping was associated with a greater risk of paralysis ($P = 0.023$). Before aortic cross clamp release, 9 of 9 patients (100%) in the SCI group had no MEP, compared with 28 of 45 patients (62%) in the non-SCI group.

The median time to absent SSEPs during aortic cross clamping was 27 min for SCI patients and 44 min for patients who did not develop SCI ($P = 0.007$). A greater duration of absent SSEPs during aortic cross clamping was associated with a higher risk of paralysis ($P = 0.009$). In the SCI group, there were 8 patients (80%) with absent SSEPs at the time of aortic clamp release, compared with 18 patients (38%) in the non-SCI group.

The estimated median time to lost H reflexes during aortic cross clamping was 12 min in the SCI group and 25 min in the non-SCI group ($P = 0.2$). Patients with a greater duration of absent H reflex during aortic cross clamping seemed to be at a higher risk of paralysis ($P = 0.027$). In the SCI group, 8 of 8 patients (100%) had no H reflexes just before aortic cross clamp release, compared with 32 of 43 (74%) patients in the non-SCI group.

After Release of Aortic Cross Clamp

Regional lumbar epidural cooling was discontinued after release of the aortic cross clamp; it took approximately 15 min for the intrathecal temperature to return to central core temperature ±1°C. Table 3 and figure 5 summarize EP recording data measured after aortic cross clamp release for patients with absent EPs before release of the aortic cross clamp. For these patients, MEPs remained absent longer in the SCI group ($P = 0.006$), with 100% of SCI patients having no MEPs 20 min after aortic

Table 1. Demographics, Spinal Cord Cooling by Outcome

Variable	Non-SCI (n = 48)	SCI (n = 10)	P Value
Age	68 (59–74)	70 (66–74)	0.34
Female sex	25 (52%)	7 (70%)	0.49
CSF temperature*	27.2 ± 2.9	26.2 ± 2.8	
Lowest PA temperature*	34.3 ± 0.8	34.3 ± 1.0	
Percentage cooled	42 (88%)	9 (90%)	1.00
Absent EP before clamping			
MEP	7 (15%)	2 (20%)	0.65
SSEP	1 (2%)	1 (10%)	0.32
H reflex	10 (21%)	2 (22%)	1.00

The sample median and interquartile range are given for numerical variables.

* Mean ± SD.

CSF = cerebrospinal fluid; EP = evoked potential; MEP = motor evoked potential; PA = pulmonary artery; SCI = spinal cord ischemia; SSEP = somatosensory evoked potential.
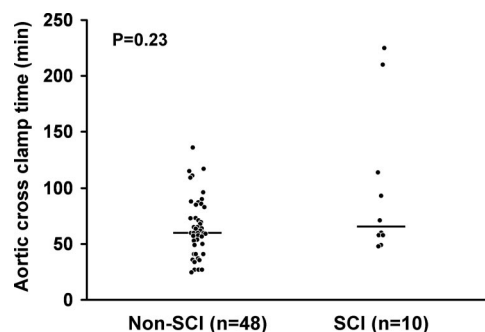

Fig. 3. Total aortic cross clamp time. The *solid horizontal line* represents the sample median. SCI = spinal cord ischemia.

Table 2. Time to Absent EPs after Aortic Cross Clamp

| | | Time to First Absent EP, min | | | |
| | Non-SCI | | SCI | | |
EP	n*	Median (95% CI)	n*	Median (95% CI)	P Value
MEP	41	31 (21–40)	8	10 (0–14)	0.004
SSEP	47	44 (32–76)	9	27 (0.23–49)	0.007
H reflex	38	25 (11–45)	8	12 (5–16)	0.20

* Includes only patients with a positive evoked potential (EP) before cross clamp.

CI = confidence interval; MEP = motor evoked potential; SCI = spinal cord ischemia; SSEP = somatosensory evoked potential.

clamp release, compared with 61% of non-SCI patients. There was no difference in the time to return of SSEPs between the SCI and non-SCI groups ($P = 0.5$). At 20 min after aortic cross clamp release, 50% of SCI patients still had absent SSEPs, compared with 44% of non-SCI patients. The H reflex in the SCI group took longer to return to baseline ($P = 0.038$). In the SCI group, 100% of the patients still had an absent MEP, SSEP, and H reflex 10 min after aortic cross clamp release, compared with 65% of non-SCI patients.

Sensitivity and Specificity of MEPs in Predicting SCI

Table 4 summarizes the sensitivity, specificity, and predictive values of absent MEPs with regard to development of SCI before application of the aortic cross clamp, 20 min after application of the aortic cross clamp, before release of the aortic cross clamp, and 20 min after release of the aortic cross clamp.

Discussion

There were no differences in the distributions of age, sex, or proportion of patients cooled between the SCI
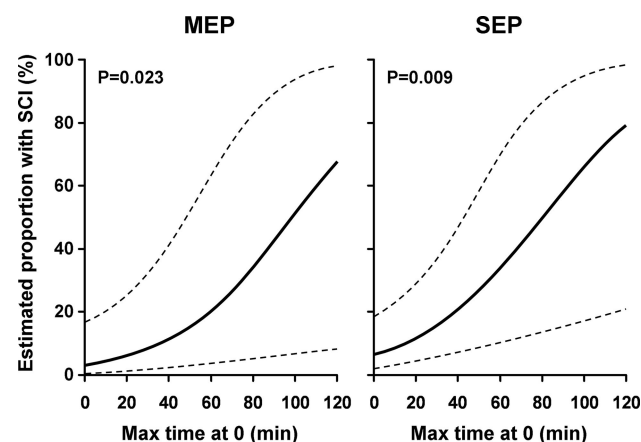

Fig. 4. Estimated probability of paralysis as the maximum time evoked potential is absent during aortic cross clamp. *Dashed lines* denote 95% confidence intervals. MEP = motor evoked potential; SCI = spinal cord ischemia; SEP = somatosensory evoked potential.

Table 3. Estimated Proportion of Patients with Absent EP 20 min after Clamp Release

| | | Estimated Proportion with Absent EP 20 min after Clamp Release | | | |
| | Non-SCI, % | | SCI, % | | |
EP	n*	(95% CI)	n*	(95% CI)	P Value
MEP	28	61 (45–82)	9	100 (66–100)	0.006
SSEP	18	44 (27–75)	8	50 (25–100)	0.55
H reflex	32	65 (50–84)	8	100 (63–100)	0.038

* Includes only patients with an absent evoked potential (EP) just before clamp removal.

CI = confidence interval; MEP = motor evoked potential; SSEP = somatosensory evoked potential; SCI = spinal cord ischemia.

and the non-SCI groups, suggesting that SCI was not affected by these demographic factors or the success of RELEC. In our series, the incidences of Crawford type I, II, III, IV, and V TAAA were 11, 30, 28, 11, and 20%, respectively. The incidence of Crawford type I was not as high as has been cited in some reports; however, the incidences of Crawford types II, III, IV, and V fall within the range reported in the literature.[2] Of the 10 cases of SCI, 5 occurred in Crawford type II and 5 occurred in Crawford type III. In the literature, the incidence of SCI is highest in type II, followed by types I, III, and IV.[14] The 17% incidence of SCI observed in this study compares unfavorably with 4.2% and 2.4% incidences of postoperative SCI in two recent series.[5,15] However, a recent review found that the range of SCI varied from 2.4% to 16%.[2] In our patients, 10.5% had paraplegia and 6.5% had paraparesis. Most TAAA series report the incidence of paraplegia only, and less frequently discuss paraparesis. Case series of TAAAs reported in the literature differ significantly with regard to aneurysm type, patient comorbidities, surgical and anesthetic techniques, and adjunct techniques to protect the spinal cord, thus making comparisons between series difficult.
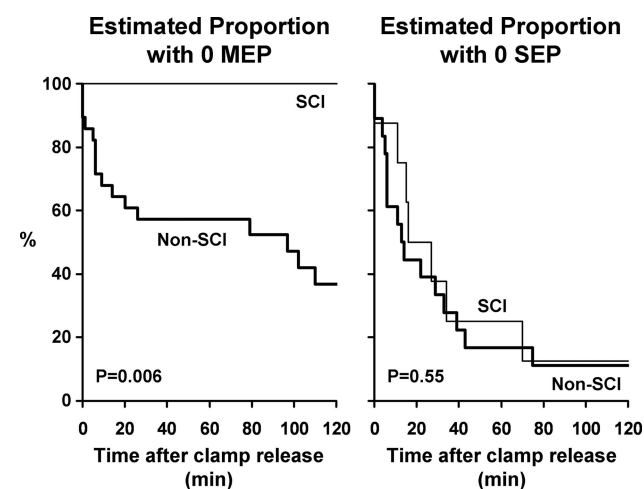

Fig. 5. Estimated probability evoked potential is still absent after surgery. MEP = motor evoked potential; SCI = spinal cord ischemia; SEP = somatosensory evoked potential.

Table 4. Estimated Sensitivity and Specificity of Absent MEP and SCI

Time Point	Sensitivity, % (95% CI)	Specificity, % (95% CI)	PPV, % (95% CI)	NPV, % (95% CI)
Before cross clamp	20 (3–56)	85 (72–94)	22 (3–60)	84 (70–93)
20 min after cross clamp application	88 (81–100)	65 (48–79)	33 (15–57)	96 (81–100)
Immediately after cross clamp release	100 (66–100)	38 (24–53)	24 (12–41)	100 (80–100)
20 min after cross clamp release	100 (66–100)	39 (22–59)	35 (17–56)	100 (72–100)

CI = confidence interval; MEP = motor evoked potential; NPV = negative predictive value; PPV = positive predictive value; SCI = spinal cord ischemia.

The remainder of the discussion will center on the epoch that places the spinal cord at risk, *i.e.*, the aortic cross clamping period (before aortic cross clamp, aortic cross clamping, and after release of the aortic cross clamp) in relation to the MEP, SSEP, and H reflex.

Before Aortic Cross Clamp

After induction of RELEC and before aortic cross clamping, there were absent SSEPs in 2 patients (3%), absent MEPs in 9 patients (15%), and absent H reflexes in 12 patients (21%). There are only a few studies documenting the effect of hypothermia on EPs. Poikilothermic patients who were surface cooled and maintained at $33.5° \pm 0.3°C$ for 4 days developed increased latency of the SSEP. However, the amplitude could not be measured, and only the latencies could readily be identified.[16] Recently, Kottenberg-Assenmacher *et al.*[17] concluded that surface hypothermia to a core temperature of 32°C did not depress the amplitude of median nerve SSEPs but did prolong their latency. In our series, the SSEP seemed to be more resistant to the effects of hypothermia, with only 3% of patients losing their SSEP. Decreasing the core temperature to 28°C in a rabbit model increased MEP latency but did not affect amplitude.[18] In our study, 15% of patients lost their MEP, but there was no difference between the SCI and non-SCI groups. The MEP positive predictive value (PPV) and negative predictive value (NPV) were the lowest before aortic cross clamping.

Aortic Cross Clamp

The aortic cross clamp times were similar between the SCI and non-SCI groups, with median durations of 66 and 60 min, respectively. However, aortic cross clamp times illustrated in figure 3 depict two patients in the SCI group with extremely long aortic cross clamp times. Some studies have found significant increases in the duration of aortic cross clamp time in the group developing SCI, whereas others have not.[19]

There was an early and delayed loss of MEPs and SSEPs after RELEC and aortic cross clamping. In the SCI group, MEPs disappeared at a median of 10 min and the SSEPs disappeared at a median of 27 min, compared with the non-SCI group, in which there was a loss of the MEPs at a median of 31 min and of the SSEPs at a median of 44 min after aortic cross clamping. As soon as MEPs were

lost, attending anesthesiologists attempted to increase distal aortic perfusion pressures, and the surgeon undertook surgical techniques to improve spinal cord blood flow, attaching and perfusing intercostal arteries if possible. Despite these measures, the MEPs did not return to baseline. The PPV for absent MEP was 33%, but the NPV was 96%. The longer the MEPs remained absent, the greater the probability of an SCI was ($P = 0.02$). Similar findings were found for SSEPs; the longer the SSEPs remained absent, the estimated proportion of patients with SCI increased significantly ($P = 0.01$). The earlier the EPs were lost and the longer the EPs remained absent during aortic cross clamping, the more likely the patient was to develop SCI.

A postulated reason for delayed loss of MEPs and SSEPs in the non-SCI group is the effect of prolonged aortic cross clamping together with the hypothermia causing ischemia of the peripheral nerves. Peripheral nerve ischemia is usually reversible. A swine model of thoracoaortic occlusion demonstrated that peripheral nerve ischemia was responsible for delayed EP loss.[20] Peripheral nerve ischemia may also explain why, after release of the aortic clamp, the MEP had a low PPV of 33% but a high NPV of 96%.

With RELEC and the aorta cross clamped, the MEPs were still valuable in detecting SCI, especially when followed by loss of SSEPs. In a swine model of RELEC to 28°C and aortic cross clamping, the time necessary to detect SCI was not affected by cooling, 3.8 min *versus* 3.2 min,[21] supporting the idea that even in the presence of cooling the spinal cord, MEPs still detect SCI. Another series demonstrated that SSEP loss was frequently delayed approximately 15 min compared with the MEP.[22]

Determining the timing of and duration of the loss of EP in predicting SCI is a different strategy from what has been reported for EPs in the past.[23,24] These series describe the number of patients with significant decreased or absent EPs and surgical and anesthesia techniques are implemented in an attempt to reverse the ischemia and restore the amplitude of the EPs. One case series found that 42 of 118 TAAA patients (35.6%) had a significant decrease in their MEPs during cross clamping of critical aortic segments.[23] Of these 42 patients, 25 patients had a persistent decrease in MEPs after perfusion of the critical aortic segment, and after release of the aortic cross clamp, 18 patients had persistent decreases. In this group of 42 patients, 4 developed paraplegia. One pa-

tient developed paraplegia without loss of MEPs but did have a loss of SSEPs. With decreased MEPs, distal and proximal aortic perfusion pressures were increased with CSF drainage and reattachment of intercostal arteries. Another large case series of 184 consecutive patients had a 2.7% incidence of SCI.[24] In this series, proximal double cross clamping of the aorta resulted in inadequate MEPs, with critical MEPs (MEPs defined by the authors as ischemic) in 48 patients (23%); however, all could be corrected by increasing proximal and distal aortic pressure. The authors used MEPs to allow immediate identification of critical spinal cord ischemia. Once it was identified, the authors either reimplanted visible intercostal arteries or used endarterectomy with individual Dacron grafted end-to-end to the orifices of the segmental vessels. Only one patient, who lost MEPs after aortic cross clamping and did not regain MEPs, awoke with paralysis despite revascularization of the segmental arteries.

After Release of Aortic Cross Clamp

Release of the thoracic aortic cross clamp results in reperfusion of the spinal cord and peripheral nerves, hemodynamic instability, and in our series, warming of the intrathecal space. At 20 min after aortic cross clamp release, all MEPs in the SCI group remained absent, whereas in the non-SCI group, 39% of the MEPs returned to baseline. At 20 min, the estimated PPV and NPV were 35% and 100%, respectively. Other large trials of MEPs in TAAA surgery have also found that if MEPs have not returned to baseline, despite increased diastolic aortic pressure and revascularization of the segmental arteries, patients are usually paraplegic.[5,15] There were no differences in the predictive value of SSEPs at 20 min; 100% in the SCI group and 65% in the non-SCI group had no H reflexes present at 20 min after aortic cross clamp release.

H Reflex

The H reflex is a measure of a monosynaptic reflex with input *via* the dorsal root ganglion and dorsal horn and output *via* the anterior horn cell in the sacral spinal cord. Loss of the H reflex is likely due to ischemia to the monosynaptic connections or to the anterior horn cells. In this study, the H reflex was lost rapidly in the SCI group, in contrast to the non-SCI group. The early H reflex loss in the non-SCI group may be a reflection of its greater sensitivity to milder ischemia than for the SSEP. The H reflex also differentiated between non-SCI and SCI patients after the aortic cross clamp was released. The H reflex followed a similar pattern to the MEP, and because the H reflex is a simple method of monitoring spinal cord ischemia, it should be considered whether MEPs are unavailable or not able to be recorded.

Sensitivity and Specificity of MEPs

The sensitivity and specificity of MEPs for monitoring SCI in patients undergoing TAAA repair have not previ-ously been reported. During the operation, attempts were made to restore an absent MEP, especially after aortic cross clamping; this will affect the PPV of absent MEPs. In addition, if other problems (*i.e.*, peripheral nerve ischemia) affect MEPs, this will also influence the PPV of the EPs. Therefore, our PPVs are low at 22–35%. However, our NPV is high, *i.e.*, MEPs present but SCI develops; in our study, there were no patients with a positive MEP after application or release of aortic cross clamp who developed SCI. Although the sensitivity, specificity, PPV, and NPV give insight into the variability of the MEP during TAAA repair, these numbers are derived from a small sample. Although the sensitivity of MEPs in our series is high, the specificity is low, with the result that some patients may be at risk for unnecessary anesthetic and surgical interventions.

Study Limitations

Voluminous EP data were collected in real time throughout the surgical procedures. As with most monitoring in the operating room, there were problems such as missing data points, irregular time intervals, and EPs fluctuating between absent and present. Prospectively, a set of guidelines was developed to extract and analyze the real time EP data (appendix). These guidelines were applied to all the patients' EPs in the study. The EP results pertain only to the loss and return of amplitude of the EP. Latency increases occurred primarily in association with reduction in amplitude and were not a primary finding in any patient. EP latencies did not add to the predictive value of the tests.

Other important limitations of this study were that it was retrospective and had a small sample size resulting in low statistical power. However, all consecutive TAAA patients with EP monitoring during the period were included; no patient was excluded. This study did not involve randomization, but measured the effect of RELEC on MEPs and outcome, in patients undergoing TAAA surgery.

Conclusion

Our study of the utility of EPs to detect paraplegia and paraparesis as evidence of SCI after TAAA repair found that SCI occurred only in Crawford types II and III TAAA. Although RELEC affected EPs (MEP more so than SSEP, more so than H reflex) before aortic cross clamp, there were no differences between the SCI and non-SCI groups. Importantly, RELEC did not prevent the ability of the MEP and the SSEP to discriminate between SCI and non-SCI after application and release of the aortic cross clamp. Our study suggests that rapid loss of MEPs or H reflexes after the application of the aortic cross clamp justifies aggressive anesthetic and surgical techniques to increase spinal cord perfusion. After release of the aortic cross clamp, absent MEPs with rewarming are significant and warrant further study involving agents or techniques

that are neuroprotective and limit ischemia–reperfusion injury, *e.g.*, hypothermia, calcium channel blockers, antioxidants. Validation of this study and evaluation of anesthetic and surgical techniques in patients undergoing TAAA repair require prospective randomized trials. Because of the limited number of patients undergoing TAAA repair and the heterogeneous nature of the populations, combined with different anesthetic and surgical techniques, both standardization and recruitment from multiple centers will be necessary.

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Appendix: Guidelines for Processing Electrophysiologic Data

1. The technician measured and recorded the amplitude (milliamps) and duration (milliseconds) of the MEP, SSEP, and H reflex in real time during the monitoring of the EPs.

2. These raw measurements of each patient were then transferred to a Microsoft Excel (Redmond, WA) spreadsheet. The measurements were divided into baseline, epidural cooling before aortic cross clamp, epidural cooling aortic cross clamp, and after release of aortic cross clamp.

3. For the patient's EP data to be included, there needed to be a minimum of two baseline measurements with amplitude and latency within 10% made within 5 min. These baseline measurements were made before aortic cross clamp and before epidural cooling.

4. An amplitude of less than 20 μV was considered to be inaccurate. Therefore, the amplitude was not recorded, but the latency was still measured.

5. For an EP measurement to be considered to be absent (zero), the following criteria had to be met:
   - The amplitude and latency both had to be absent.
   - Two consistent measurements were to be recorded within 5 min.
   - The time to absent was taken as the time to second absent EP measurement.
   - If only the amplitude or latency was absent, the EP was not defined as zero.

6. For an EP measurement to be considered to be positive after meeting the zero definition, the following criteria had to be met:
   - The amplitude or latency had to be present.
   - Two consistent measurements were to be recorded within 5 min.
   - The time to positive was taken as the time to second present EP measurement.

7. Missing data points in time were not recorded as zero or positive, but were recorded as unknown data.

8. If evoked potential was positive for the entire observation period, 999 was used as a code for "time to first zero."