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Use of the Electrospinogram for Predicting Harmful Spinal Cord Ischemia during Repair of Thoracic or Thoracoabdominal Aortic Aneurysms

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Background: **To reduce the incidence of misleading assessments, and to derive criteria for critical spinal cord ischemia during thoracic or thoracoabdominal aortic aneurysm repair, the authors epidurally stimulated and recorded somatosensory evoked potentials (ESEP) below and above, respectively, the spinal segment at risk (electrospinogram).**

Methods: **Epidural somatosensory evoked potentials were analyzed in 100 consecutive patients undergoing resection of aortic aneurysms using two bipolar catheters (stimulation at the L2 level and recording at the T3 level) for the following criteria: 1) the time until ESEP disappeared completely after cross clamping, 2) the duration of complete ESEP loss during and after cross clamping, and 3) the time until ESEP recovered after declamping. Postoperatively, neurologic deficits were evaluated by a neurologist who was unaware of the ESEP recordings.**

Results: **Three types of patients could be identified. First, thirty-one patients neither showed ESEP loss nor neurologic deficits. Second, ESEP loss occurring later than 15 min after cross clamping was associated with a neurologic deficit in 2 of 29 patients (6.9%). And, third, 12 of 40 patients (30%) presented a neurologic deficit when ESEP loss occurred within 15 min after cross clamping. Further indicators of an impending risk were a total ESEP loss greater than 40 min (sensitivity 100%, specificity 68%, positive predictive value [PPV] 35%, and negative predictive value [NPV] 100%), and a recovery of ESEP later than 20 min after declamping (sensitivity 93%, specificity 86%, PPV 52%, and NPV 99%).**

Conclusions: **Epidural somatosensory evoked potentials appeared to be a reasonable intraoperative predictor of postoperative neurologic outcome, and informs surgeons and anesthesiologists about the impending danger at an early state of the operation. (Key words: Complications: neurologic deficit; spinal cord injury. Monitoring: epidural somatosensory evoked potentials; spinal cord function. Surgery: thoracic.)**

This article is accompanied by a Highlight. Please see this issue of ANESTHESIOLOGY, page 27A.

PARAPLEGIA and paraparesis are widely feared, but somewhat unpredictable complications associated with the repair of thoracic or thoracoabdominal aortic aneurysms. Based on the extent of the repair, the incidence of such neurologic deficits ranges from 3 to 40%.[1] A monitoring technique that improves the timeliness and degree of neuroresuscitative measures may be clinically useful in view of the failure of prophylactic surgical interventions,[2–5] and the adjunctive use of drugs like naloxone[6] and papaverine[7] alone, to eliminate neurologic sequelae.

Somatosensory evoked potential (SEP) monitoring elicited by posterior tibial nerve stimulation has been reported to yield a large number of false positive responses, *i.e.*, absent responses but no neurologic deficit.[8,9] Loss of the physiologic function indicated by SEP loss does not necessarily imply inadequate spinal blood flow, because this technique cannot distinguish between peripheral (n. tibialis) and central (spinal cord or cerebral) ischemia.

We, therefore, used the electrospinogram, a technique developed by Tamaki *et al.*[10] to monitor spinal cord function during spine surgery, in patients undergoing resection of a thoracic or thoracoabdominal aneurysm. This technique involves recording of real-time epidural somatosensory evoked potentials (ESEP) from the T3–T4 level of the spinal cord after epidural stimulation of

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the lumbar spinal cord (L2–L3). We aimed at deriving criteria for ischemic spinal cord injuries during repair of thoracic and thoracoabdominal aortic aneurysms by monitoring spinal cord function before, during, and after cross clamping of the thoracic aorta.

Materials and Methods

After obtaining approval from our institutional board, and informed consent from each patient, we studied 100 consecutive patients. During the last 5[fr1/2] yr (March, 1987, to November, 1992), 30 women and 70 men, aged 28–82 yr (mean age 63.5 ± 12 yr) were scheduled for repair of thoracic or thoracoabdominal aortic aneurysm (type I–IV, according to Crawford's classification[11]). Preoperative patient demographics and the distribution of the types of aneurysm are listed in table 1. Two staff anesthesiologists (K. D. S. and B. M.) and one staff surgeon (W. S.) participated in this study as part of their regular duty.

Spinal Cord Function Monitoring, Anesthetic and Surgical Management

Two 18-G polyethylene bipolar epidural catheters[12] (NeuroStim; Vygon Medical, Aachen, Germany) were used for stimulating and recording ESEP. Under local anesthesia and sterile conditions, both catheters were inserted percutaneously and advanced 5 cm into the epidural space *via* 16-G Tuohy needles. The stimulating catheter was placed at the L2–L3 spinal level (*via* puncture at the L4–L5 intervertebral level) and the recording catheter at the T3–T4 spinal level (*via* puncture at the T5–T6 intervertebral level). Postoperatively, the thoracic epidural catheter was used to administer morphine for pain relief.

The correct positioning of the catheters was tested in the awake, but premedicated, patient (1–2 mg flunitrazepam orally 90 min before arrival), and preoperatively, with the patient in the right decubitus position after induction of anesthesia (reference recordings). If inadequate or no responses were recorded, both catheters were used as stimulation catheters, and recordings were done from a scalp electrode to detect the malpositioned catheter. The malpositioned catheter was reinserted.

Stimulation variables were adjusted to an amplitude that produced comfortable (painless) muscle reactions or paresthesia in the premedicated patients. The signal generation and averaging (sample size: 64 sweeps) of the ESEP was done using the Medelec ER 94a evoked potential system (Vickers Healthcare, Surrey, Great Britain). Stimulation was delivered in squarewave pulses 0.2 ms wide at a rate of 30–50 pulses s^{-1}, with an intensity of 15–20 mA. The recording time was 20 ms, and bandpass filters were used at 30–3,000 Hz. Recordings were taken immediately before cross clamping, every 3–5 min during cross clamping, and after cross-clamping release until the end of the case. Each recording was displayed on a screen after averaging for use during the operation, and plotted immediately for later data analysis.

Table 1. Characteristics and Outcome in 100 Patients Undergoing Resection of Thoracic Aortic Aneurysms

	No ESEP Loss	ESEP Loss
n	31	69
Age (mean ± SD)	63 ± 15	64 ± 10
Gender (%) female	27	32
Type of aneurysm*		
Type I	28	38 (ND = 12%)
Type II	31	38 (ND = 31%)
Type III	28	24 (ND = 18%)
Type IV	13	0
Aortic cross-clamping time (min) (mean ± SD, 95% confidence interval)	62 ± 30 (51–73)	59 ± 27 (53–65)
ESEP findings (mean ± SD, 95% confidence interval)		
Disappearance time (min)	0	18 ± 9 (15.9–20.1)
Duration of ESEP loss (min)	0	64 ± 50 (52.2–75.8)
Recovery time (min)	0	26 ± 40 (16.6–35.4)
ND (number of patients)	0	14

ND = Neurologic deficit.

* Classification of aneurysms according to reference 11.

Anesthesia was induced with 2–4 mg/kg intravenous thiopental and 0.2–0.4 mg fentanyl, and neuromuscular block was produced with 0.1 mg/kg vecuronium. Anesthesia was maintained with isoflurane, 0.3–0.5 mg/h fentanyl infusion, and nitrous oxide in oxygen depending on surgical requirements. Eucapnia was produced as assessed by capnography and blood gas analyses.

To keep mean arterial blood pressure within narrow limits during aortic cross clamping, sodium nitroprusside or nifedipine was infused when blood pressure increased above 110 mmHg. If mean arterial blood pressure decreased to less than 90 mmHg, dopamine or dobutamine was administered.

The operative technique consisted of cross clamping of the thoracic aorta and graft inclusion[8] in 83 cases without adjuncts. In 17 patients, a temporary axillofemoral shunt was inserted at the discretion of the surgeon, but primarily for cardiac reasons (*i.e.*, history of congestive heart failure or aortic valvular regurgitation).

Neurologic deficits included paraplegia and paraparesis. The latter term was defined as weakness with preservation of motion against gravity and resistance. Neurologic examinations were performed in all patients preoperatively and before hospital discharge by a neurologist who was unaware of the ESEP recordings.

Data Collection and Analysis

Sensitivity, specificity, and negative (NPV) and positive predictive values (PPV) of the following criteria were calculated and analyzed statistically using McNemar's test: 1) the time until ESEP disappeared completely after aortic cross clamping (disappearance of ESEP), 2) the duration of complete ESEP loss during and after aortic cross clamping (duration of ESEP loss), and 3) the time until ESEP recovered after cross-clamping release (recovery of ESEP).

The ESEP data were collected, stored, and analyzed on an Intel 80386-based microcomputer using the Statistical Package for Social Science (SPSS/PC+, SPSS-Software, Munich, Germany). Quantitative data were presented as mean ± SD with 95% confidence interval. Comparisons of quantitative data (age, gender, cross-clamping time, and type of aneurysm) were made using the Mann-Whitney U test. The chi-square analysis with continuity correction (or Fisher's exact test) was applied for categorical data. Possible relations between dynamic time criteria (disappearance of ESEP, duration of ESEP loss, and recovery of ESEP) were analyzed by linear regression using the least-squares method. $P < 0.05$ was considered statistically significant.

Results

Epidural somatosensory evoked potential monitoring was possible in all 100 patients with thoracic and thoracoabdominal aortic aneurysms divided according to Crawford's classification (table 1).[11]

Disappearance of ESEP

Thirty-one patients did not lose ESEP during aortic cross clamping, and had no neurologic deficit postoperatively (no false negative). Cross-clamping time and type of aneurysm did not differ between these patients and those 69 patients in whom ESEP loss occurred (tables 1 and 2).

In 29 patients, ESEP disappeared more than 15 min after cross clamping (26 ± 8 min, with a 95% confidence interval of 23.1–28.9 min), and, in two of these patients (6.9%), a neurologic deficit was present postoperatively. Forty patients lost ESEP within 15 min (12.6 ± 2.9 min after aortic cross clamping, 95% confidence interval 11.7–13.5 min; fig. 1a), and, in this group, 12 patients (30%) presented neurologic deficits postoperatively. Thus, early intraoperative loss of ESEP marked patients at high risk to develop neurologic deficits postoperatively (sensitivity 86%, specificity 67%, NPV 97%, and PPV 30%; table 2, fig. 1a), but patients without ESEP loss during aortic cross clamping were not at risk.

Duration of ESEP Loss

Loss of ESEP of less than 40 min duration (28 patients; fig. 1b) was not related to neurologic deficits (no false positive), irrespective of the onset of ESEP loss (17 ± 9 min, 95% confidence interval 14.3–19.7 min in patients without neurologic deficit *vs.* 13 ± 6 min, 95% confidence interval 10.7–15.3 min in patients with neurologic deficits, P = NS) and the cross-clamping time (72 ± 26 min, 95% confidence interval 64.1–79.9 min in patients without neurologic deficit *vs.* 72 ± 30 min, 95% confidence interval 60.7–83.3 min in patients with neurologic deficit; P = NS). Only patients with a total loss of ESEP for greater than 40 min (14 of 41 patients) developed neurologic deficits (no false negative, sensitivity 100%, specificity 68%, NPV 100%, and PPV 35%; table 2, fig. 1b). There was no significant correlation ($r = -0.287$) between duration of ESEP loss (>40 min) and disappearance of ESEP (<15 min).

Table 2. Sensitivity, Specificity, and Predictive Values of ESEP Findings to Predict Neurologic Deficits

Criterion	I Global ESEP Loss	II Disappearance of ESEP <15 min after Cross-clamping	III Duration of ESEP Loss >40 min	IV Recovery of ESEP >20 min after Declamping
TP	14	12	14	13
TN	31	58	59	74
FP	55	28	27	12
FN	0	2	0	1
Predictive values (%)				
Sensitivity	100	86	100	93
Specificity	36	67[a]	68[a]	86[b,c]
PPV	20	30	35	52[b]
NPV	100	97	100	99

TP = true positive; TN = true negative; FP = false positive; FN = false negative; PPV = positive predictive value; NPV = negative predictive value.
Sensitivity = TP/(TP + FN); specificity = TN/(TN + FP); PPV = TP/(TP + FP); NPV = TN/(TN + FN).
[a]criterion I, [b]criterion II, or [c]criterion III, $P < 0.01$ (McNemar's test).

Recovery of ESEP

When ESEP recovered early (within 20 min after aortic cross-clamping release), the incidence of a neurologic deficit was low (1 of 44 patients; fig. 1c). With this exception, neurologic deficits developed only in patients (13 of 25 patients) in whom ESEP recovered late (58 ± 51 min after cross clamping; sensitivity 93%, specificity 86%, PPV 52%, and NPV 99%; table 2, fig. 1c). This time criterion had a significantly higher specificity ($P < 0.005$) and positive predictive value than all other time criteria ($P < 0.01$). In addition, there was no significant correlation (r = 0.753) between recovery of ESEP (>20 min) after aortic cross clamping and duration of ESEP loss (>40 min).

Eight of the 17 patients in whom a temporary axillofemoral shunt was inserted presented an ESEP loss. In three of these eight patients, a neurologic deficit developed.

Additionally, the incidence of neurologic deficits, which did not differ between the types of aneurysm, is given in table 1.

Discussion

Efforts to detect and prevent neurologic deficits resulting from ischemic damage of the spinal cord during repair of lesions of the descending aorta continue. Based on the extent of the repair, the incidence of neurologic deficits ranges from 3 to 40%.[1] Immediate intraoperative detection of patients at risk, therefore, may be important to alert both surgeons and anesthesiologists, so that timely resuscitative steps can be taken (see below).

Although SEPs have been used in the operating room for years, there is still no consensus on the utility of such monitoring in real-time detection of neurologic deficits during repair of thoracic and thoracoabdominal aortic aneurysms. Previous studies have indicated that somatosensory evoked potentials elicited by peripheral nerve stimulation and recordings from a scalp electrode is the method of choice during resections of thoracic or thoracoabdominal aortic aneurysms,[9,13,14] because the "wake-up test" cannot be used in patients with thoracotomy.[15] Initially, this method was reported to be highly sensitive and reliable in small groups of patients.[13,14] In contrast, subsequent studies reported conflicting results.[8,9,16] For example, Crawford *et al.*,[8] using the same technique, reported 13% false negative and 67% false positive SEP findings in 99 patients.

Because anesthetics, *per se,* may disturb SEP conduction,[17] and latency is altered by temperature changes,[18,19] hematocrit,[20] and limb perfusion,[21] the SEP alterations during aortic cross clamping may, therefore, reflect both, confounding systemic influences and ischemia of either spinal or peripheral nerves.

Drenger *et al.*[9] compared findings from peripheral nerve stimulation with those from spinal evoked potentials, both recorded at the scalp, in a small group of patients with thoracic and thoracoabdominal aortic aneurysms (n = 18). Although peripheral evoked potentials disappeared within 5–30 min after aortic cross clamping in all ten patients without shunt, all seven survivors did well postoperatively. In contrast, both spinal evoked potentials and peripheral evoked SEP, in eight patients with shunt, predicted neurologic out-

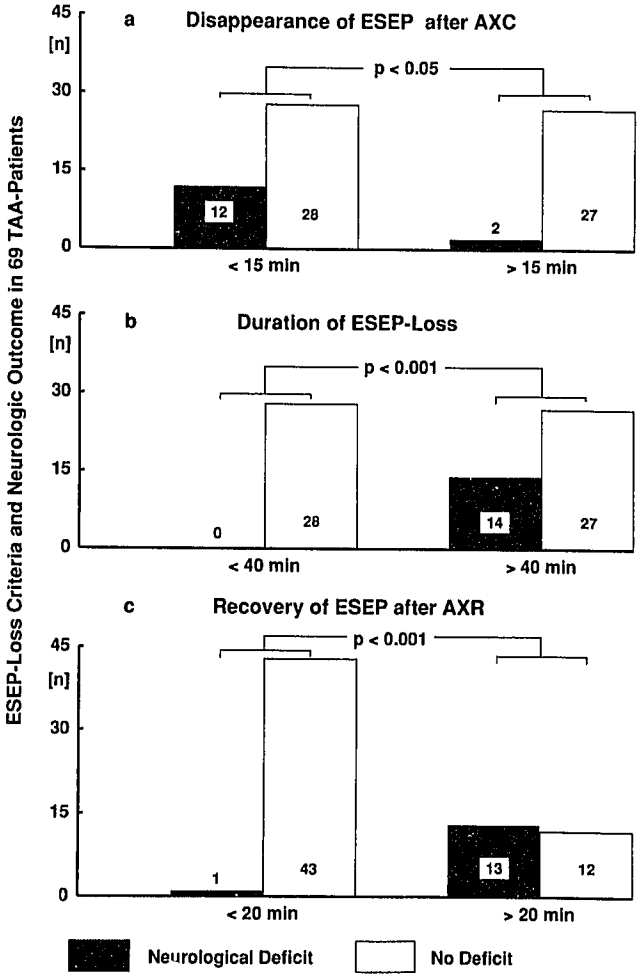

Fig. 1. Relations between intraoperative epidural somatosensory evoked potential (ESEP) findings and postoperative neurologic outcome of 69 consecutive patients with intraoperative ESEP loss during thoracic or thoracoabdominal aortic aneurysm repair. Three criteria were separated: (*a*) disappearance of ESEP (within 15 min) after aortic cross clamping (AXC); (*b*) duration of total ESEP loss; and (*c*) recovery time after aortic declamping (AXR; *P* values refer to chi-square test comparing the proportion of patients with neurologic deficits according to the time criteria shown in *A–C*).

come accurately (no false negative findings). Therefore, only cases in which distal shunt perfusion was established during cross clamping appeared to permit the use of peripheral evoked potentials for real-time detection of ischemic spinal cord injuries. However, it should be considered that: 1) this conclusion is based on findings from a small number of patients, and 2) inadequate distal shunt perfusion may mimic spinal ischemia when the limb and the peripheral nerve are malperfused, as has been demonstrated by Crawford *et al.*[8] in 99 patients. Thus, it appears preferable to use spinal (epidural) electrodes instead of peripheral nerve stimulation during aortic cross clamping.

Somatosensory evoked potentials recording from the scalp is also subject to confounding influences. Because volatile anesthetics, *per se*,[17] and complications such as brain edema[22] (hyperperfusion, in case of aortic cross clamping proximal to the left carotid or subclavian artery) and cerebral air embolism[23] (after cross-clamping release) are known to alter SEP findings, the latter may mimic spinal cord dysfunction without being related to the region of primary interest.

To minimize such misinterpretations of SEP findings, stimulation and recording electrodes were placed in the epidural space sufficiently away from the spinal segment at risk, but in very close proximity to the spinal

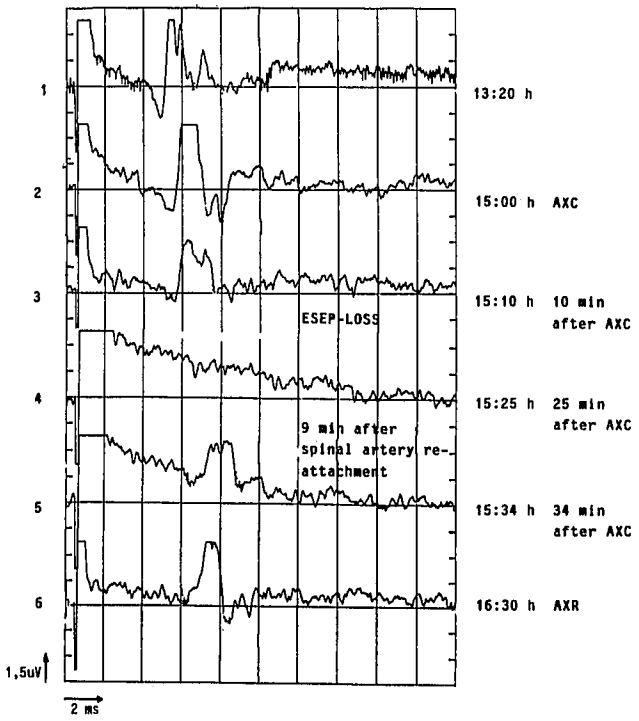

Fig. 2. Spinal cord monitoring using epidural somatosensory evoked potentials (ESEP; stimulation at the L2–L3 and recording at the T3–T4 spinal level) in a 41-yr-old male patient with a penetrating thoracoabdominal aneurysm (Crawford type II). Recording 1: Control recordings. Recording 2: At the time of aortic cross clamping (AXC). Recording 3: Ten minutes after AXC. Recording 4: Loss of ESEP 25 min after AXC. Recording 5: Nine minutes after four spinal cord arteries were reattached. Recording 6: At cross-clamping release (AXR). The patient who underwent aortic repair after completion of the study presented no neurologic deficit postoperatively.

cord.[10,24] Because amplitude is extremely variable in normal patients,[18] and temperature decreases are often recorded during aortic surgery, we prefer, as do others,[25] to use the complete loss of the signal to indicate spinal cord ischemia. Using this technique, we found that thoracic aortic cross clamping in 100 patients with large aneurysms (type I–IV[11]) is not, *per se,* related to the intraoperative loss of conductive spinal cord function (ESEP loss in 69/100 patients).

In addition, Laschinger *et al.*[13] showed that loss of SEP after aortic cross-clamping may indicate spinal cord malperfusion. However, cross clamping of the aorta (up to 60 min in dogs[26]) does not necessarily mean irreversible ischemic damage, although it demonstrates loss of physiologic function. Accordingly, in our study, no instance of spinal cord damage was observed in patients without complete ESEP loss, and in only 14 out of 69 patients with an ESEP loss was a neurologic deficit present postoperatively. Because ESEP loss alone may be too global a measure to describe patients at risk, dynamic time criteria, such as onset and duration of ESEP loss during aortic cross clamping, and ESEP recovery after aortic cross-clamping release were considered.

All three time criteria were significantly more specific ($P < 0.01$) than the global ESEP loss. Diagnostically, absence of recovery within 20 min after aortic cross-clamping release showed the highest specificity (86%) and a high sensitivity (93%), combined with a reasonable positive predictive value (52%), for indicating patients at risk. Unfortunately, however, the time that must elapse during this process makes it possible for irreversible processes to take place that may make any attempted intervention less effective.

Recent pharmacologic manipulations (*i.e.*, naloxone[6] and papaverine[7]) and currently used surgical adjuncts (shunt and bypass)[2] have failed to eliminate neurologic deficits in patients undergoing thoracic or thoracoabdominal aortic repair. The fact that, in 17 of our patients, a temporary axillofemoral shunt was inserted may have biased the results in some unknown way, but it seems unlikely to have significantly altered the essential results. Future therapeutic strategies may include spinal artery reattachment guided by the results of epidural somatosensory evoked potential monitoring, as presented in figure 2.

We, therefore, conclude that, by recording the real-time epidural somatosensory evoked potentials during aortic cross clamping, this technique may provide investigators and clinicians with a reliable assessment of spinal cord function that may become useful during development and application of resuscitative therapies.

References

1. Crawford ES, Crawford JL, Safi HJ, Coselli JS, Hess KR, Brooks B, Norton HJ, Glaeser DH: Thoracoabdominal aortic aneurysms: Preoperative and intraoperative factors determining immediate and long-term results of operations in 605 patients. J Vasc Surg 3:389–402, 1986

2. Crawford ES, Walker HSJ, Saleh SA, Norman NA: Graft replacement of aneurysms in descending thoracic aorta: Results without bypass or shunting. Surgery 89:73–85, 1981

3. Crawford ES, Coselli JS, Safi HJ: Partial cardiopulmonary bypass, hypothermic circulatory arrest, and posterolateral exposure for thoracic aortic aneurysm operation. J Thorac Cardiovasc Surg 94:824–827, 1987

4. McCoullough JL, Hollier LH, Nugent M: Paraplegia after thoracic aortic occlusion: Influence of cerebrospinal fluid drainage. Experimental and early clinical results. J Vasc Surg 7:153–160, 1988

5. Crawford ES, Svensson LG, Hess KR, Shenaq SS, Coselli JS, Safi HJ, Mohindra PK, Rivera V: A prospective randomized study of cerebrospinal fluid drainage to prevent paraplegia after high-risk surgery on the thoracoabdominal aorta. J Vasc Surg 13:36–45, 1991

6. Dillon PF, Aksoy MO, Driska SP, Murphy RA: Opiate antagonist improves neurologic recovery in spinal injury. Science 211:493–497, 1981

7. Svensson LG, Grum DF, Bednarski M, Cosgrove DM, Loop FD: Appraisal of cerebrospinal fluid alterations during aortic surgery with intrathecal papaverine administration and cerebrospinal fluid drainage. J Vasc Surg 11:423–429, 1990

8. 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 95:357–365, 1988

9. Drenger B, Parker SD, McPherson RW, North RB, Williams GM, Reitz BA, Beattie C: Spinal cord stimulation evoked potentials during thoracoabdominal aortic aneurysm surgery. ANESTHESIOLOGY 76:689–695, 1992

10. Tamaki T, Tsuji H, Inoue S, Kobayashi H: The prevention of iatrogenic spinal cord injury utilizing the evoked spinal cord potentials. Int Orthop 4:313–317, 1981

11. Crawford ES, Schuessler JS: Thoracoabdominal and abdominal aortic aneurysms involving celiac, superior mesenteric, and renal arteries. World J Surg 4:643–652, 1980

12. Kaschner AG, Sandmann W, Larkamp H: Percutaneous flexible bipolar epidural neuroelectrode for spinal cord stimulation: Technical note. J Neurosurg 60:1317–1319, 1984

13. Laschinger JC, Cunningham JN, Catinella FC, Nathan IM, Knoop EA, Spencer FC: Detection and prevention of intraoperative spinal cord ischemia after cross-clamping of the thoracic aorta: Use of somatosensory evoked potentials. Surgery 92:1109–1117, 1982

14. Cunningham JN, Laschinger JC, Merkin HA, Nathan IM, Colvin S, Ransohoff J, Spencer FC: Measurement of spinal cord ischemia during operations upon the thoracic aorta. Ann Surg 196:285–293, 1982

15. Vauzelle C, Stagnara P, Jouvinroux P: Functional monitoring of spinal cord activity during spinal surgery. Clin Orthop 93:173–178, 1973

16. Takaki O, Okumura F: Application and limitation of somatosensory evoked potential monitoring during thoracic aortic aneurysm surgery: A case report. ANESTHESIOLOGY 63:700–703, 1985

17. McPherson RW, Mahla M, Johnson R, Traystman RJ: Effects of enflurane, isoflurane and nitrous oxide on somatosensory evoked potentials during fentanyl anesthesia. ANESTHESIOLOGY 62:626–633, 1985

18. Chiappa KH, Ropper AH: Evoked potentials in clinical medicine (part two). N Engl J Med 306:1205–1211, 1982

19. v.Rheineck Leyssius AT, Kalkman CJ, Bovill JG: Influence of moderate hypothermia on posterior tibial nerve somatosensory evoked potentials. Anesth Analg 65:475–480, 1986

20. Nago S, Roccaforte P, Moody RA: The effects of isovolemic hemodilution and reinfusion of packed erythrocytes on somatosensory and visual evoked potentials. J Surg Res 25:530–537, 1978

21. Benzon HT, Toleikis JR, Meagher LL, Shapiro BA, Ts'ao C, Avram MJ: Changes in venous blood lactate, venous blood gases, and somatosensory evoked potentials after tourniquet application. ANESTHESIOLOGY 69:677–682, 1988

22. Suga S, Sato S, Ishihara N, Togashi O, Yunoki K, Kobari M: Effect of glycerol on ischemic edema evaluated by somatosensory evoked potentials. Adv Neurol 52:185–194, 1990

23. Leitch DR, Hallenbeck JM, Greenbaum LJ: The effects of various gases on cortical and spinal somatosensory evoked potentials at pressures up to 10 bar. Aviat Space Environ Med 54:105–111, 1983

24. Nuwer MR: Spinal cord monitoring, Evoked Potential Monitoring in the Operating Room. Edited by Nuwer MR. New York, Raven Press, 1986, pp 49–101

25. Oka Y, Miyamoto T: Prevention of spinal cord injury after cross-clamping of the thoracic aorta. Jpn J Surg 14:159–162, 1984

26. Grabitz K, Freye E, Prior R, Schrör K, Sandmann W: Does prostaglandine E_1 and superoxide dismutase prevent ischaemic spinal cord injury after thoracic aortic cross-clamping? Eur J Vasc Surg 4: 19–24, 1990