

An Approach to Intraoperative Neurophysiologic Monitoring of Thoracoabdominal Aneurysm Surgery

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Summary: Thoracoabdominal aneurysm surgery carries an approximate 10% risk of intraoperative paraplegia. Abrupt cord ischemia and the confounding effects of systemic alterations and limb or cerebral ischemia challenges neurophysiologic spinal cord monitoring. This investigation sought a rapid differential monitoring approach to predict or help prevent paraplegia. Thirty-one patients were monitored with motor evoked potentials (MEPs) and median and tibial somatosensory evoked potentials (SSEPs). MEPs involved single-pulse transcranial electrical stimulation with D wave recording (n = 16), arm and leg muscle MEPs following multiple-pulse transcranial electrical stimulation (n = 12), or both (n = 3). D wave recordings required averaging, invasive epidural electrode insertion, and produced both false positives and false negatives. Muscle MEPs were instantaneous and reliably sensitive and specific for cord ischemia. Cortical and peripheral nerve SSEPs provided rapid detection of systemic alterations and cerebral or limb ischemia. Cord and subcortical SSEPs required excessive averaging time. In conclusion, bilateral arm and leg muscle MEPs with median and tibial peripheral nerve and cortical SSEPs provide sufficiently rapid detection and differentiation of cord ischemia from confounding factors. There were two predicted intraoperative spinal cord infarctions (6.5%) and nine circumstantial examples of possible contributions to deficit prevention. **Key Words:** Intraoperative monitoring—Motor evoked potentials—Somatosensory evoked potentials—Thoracoabdominal aneurysm surgery.

Intraoperative spinal cord infarction is a major complication of thoracoabdominal aneurysm surgery. It causes an anterior spinal cord syndrome with paraplegia and spinothalamic sensory loss but spared posterior column function usually below T6 (Adams et al., 1997). There remains an approximate 10% risk despite preventive surgical strategies (Connolly, 1998; Crawford et al., 1986; Griep et al., 1998; Robertazzi and Cunningham, 1998a). One strategy includes neurophysiologic spinal cord monitoring to detect reversible ischemia, guiding

intervention. Cord ischemia can occur abruptly, and decisions have to be made quickly, so a satisfactory method must provide rapid surgical feedback.

Although somatosensory evoked potentials (SSEPs) assess the clinically spared posterior columns, ischemia may cause temporary transverse cord dysfunction detectable through SSEPs (Galla et al., 1999; Grabitz et al., 1996; Griep et al., 1996, 1998; Robertazzi and Cunningham, 1998a, b; Schepens et al., 1994; Shahin et al., 1996). However, SSEPs do not predict motor function reliably (Guerit et al., 1996; Schepens et al., 1994). The tibial lumbar potential generated in lumbosacral spinal cord gray matter (Emerson and Pedley, 1990) should detect anterior lumbar cord ischemia and has been used to monitor thoracoabdominal aneurysm surgery (Guerit

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et al., 1996), but cannot assess directly the corticospinal system at risk.

Spinal cord stimulation and recording with percutaneous epidural electrodes (Dudra et al., 1997; Matsui et al., 1994, 1997; Yamamoto et al., 1994) cannot assess motor pathways selectively because the dorsal columns are also stimulated antidromically (Toleikis et al., 2000), and risks epidural complications.

Motor evoked potential (MEP) monitoring following transcranial electrical (de Haan et al., 1998; van Dongen et al., 1999) or magnetic (Qayumi et al., 1997; Stinson et al., 1994) brain stimulation represents an important advance in isolating the corticospinal system. Merton and Morton (1980) first described muscle MEPs after transcranial electrical stimulation (TCES), and this method is practical intraoperatively. In awake subjects, spinal epidural recordings after TCES contain a corticospinal "D wave" generated by direct corticomotor neuron depolarization and then a series of "I waves" generated indirectly by cortical synapses. These descending corticospinal volleys summate to depolarize spinal motor neurons, producing muscle responses. Anesthetics suppress cortical and anterior horn synapses, eliminating I waves and impeding spinal motor neuron depolarization, preventing muscle responses after a single TCES stimulus. However, the synaptic epidural D wave is resistant to anesthesia and can be used to monitor corticospinal tract integrity (Deletis, 1993). This method excludes anterior horn cells, which may be disabled more rapidly by ischemia (Qayumi et al., 1997), but low thoracic epidural recordings (below T6) may detect corticospinal tract ischemia during thoracoabdominal aneurysm surgery.

Multiple-pulse TCES (de Haan et al., 1998; Jones et al., 1996; van Dongen et al., 1999) uses a train of three to seven stimuli separated by 1 to 4 msec, producing multiple D waves and the reappearance of I waves, summing to depolarize spinal motor neurons under anesthesia. Omission of neuromuscular blockade then allows high-amplitude muscle responses during single trials. Such rapid and specific assessment of the corticospinal system including spinal motor neurons could impact substantially thoracoabdominal aneurysm surgery.

Several potentially confounding alterations occur during thoracoabdominal aneurysm surgery, including limb or cerebral ischemia, hypothermia, hypotension, anesthesia, and scalp edema. Limb ischemia can be detected by peripheral nerve SSEPs (Gugino et al., 1992). Alterations of cortical SSEPs from both upper and lower extremities can identify systemic alterations (Shahin et al., 1996).

This investigation sought to define a sufficiently rapid monitoring method to detect and to differentiate cord ischemia from confounding alterations during thoracoab-

dominal aneurysm surgery. It also sought to correlate neurophysiologic recordings with patient outcome and to determine whether these methods might predict or help prevent paraplegia.

METHODS

Thirty-one consecutive patients (15 women, 16 men; age range, 37 to 78 years; mean age, 66 years) undergoing thoracoabdominal aneurysm surgery were studied prospectively. Each patient underwent pre- and postoperative neurologic examination (D.M.) and baseline SSEP studies, and gave informed consent for intraoperative monitoring including single- or multiple-pulse TCES and epidural recording electrode insertion if used. No patient had a history of epilepsy.

There were three phases of development searching for an optimal method (Table 1). During the first phase (16 patients) we used single-pulse TCES and recorded the D wave from a bipolar epidural electrode inserted before induction through a 17-gauge Tuohy needle by an anesthesiologist. Chest radiographs documented electrode location. Time-consuming repositioning or replacement was sometimes required to achieve a low thoracic (T7 to T11) location. On two occasions the electrode remained at a higher level than desired (T5 and T6). Stimuli of 250 to 1,000 V (adjusted to provide a sufficient D wave for monitoring) with a 50-microsecond time constant and a 0.4-Hz frequency were applied between the C1 and C2 or FC1 and FC2 scalp sites (American Electroencephalographic Society, 1994b) through spiral needle electrodes inserted after induction. Five to 20 trials were averaged, requiring approximately 12 to 50 seconds. The recording bandpass was between 150 to 500 Hz and 1,000 to 5,000 Hz, adjusted to optimize the response.

During the second phase (three patients) we monitored the D wave and muscle MEPs from closely spaced (approximately 1 to 2 cm) and braided, sterile, single-use needle electrodes inserted after induction by the clinical neurophysiologist in the first dorsal interosseous or thenar, tibialis anterior, and abductor hallucis muscles of each side. Muscle recording bandpass was 20 to 20,000 Hz. Because the hemisphere under the anode is stimulated preferentially, right muscle MEPs were recorded after left-hemisphere multiple-pulse anodal stimulation, and then vice versa. Each recording began using three pulses at a 1-msec interstimulus interval. Stimulus voltage was increased until both arm and leg muscle responses contralateral to the anode were obtained. Then, adjustments of pulse number and interstimulus interval were made until a visually optimal muscle response was achieved (usually three to five pulses at a 1 to 4-msec

TABLE 1. Evolution of neurophysiologic monitoring methods

| Phase | Patient no. | SSEP | | | | MEP |
|-------|-------------|-------------------|-------------------|------------------|------------------|------|
| | | Right Median | Left Median | Right Tibial | Left Tibial | |
| 1 | 1 | N20 | N20 | PF, P37 | PF, P37 | ‡D |
| | 2 | N20 | N20 | P37 | P37 | D |
| | 3 | EP, N20 | EP, N20 | P37 | P37 | D |
| | 4 | N20 | N20 | PF, P37 | PF, P37 | D |
| | 5 | N20 | N20 | PF, P37 | PF, P37 | D |
| | 6 | N20 | N20 | PF, P37 | PF, P37 | D |
| | 7 | EP, N13, N20 | EP, N13, N20 | PF, LP, P37 | PF, LP, P37 | D |
| | 8* | EP, P14, N20 | Absent | PF, LP, P37 | Absent | D |
| | 9 | EP, N20 | EP, N20 | PF, LP, P37 | PF, LP, P37 | D |
| | 10 | EP, N20 | EP, N20 | PF, LP, P37 | PF, LP, P37 | D |
| | 11 | EP, N13, N20 | EP, N13, N20 | PF, LP, P37 | PF, LP, P37 | D |
| | 12 | EP, N20 | EP, N20 | PF, P37 | PF, P37 | D |
| | 13 | EP, P14, N20 | EP, P14, N20 | PF, LP, P31, P37 | PF, LP, P31, P37 | D |
| | 14 | EP, P14, N20 | EP, P14, N20 | PF, LP, P31, P37 | PF, LP, P31, P37 | D |
| | 15 | EP, P14, N20 | EP, P14, N20 | PF, P31, P37 | PF, P31, P37 | D |
| | 16 | EP, N13, P14, N20 | EP, N13, P14, N20 | PF, LP, P31, P37 | PF, LP, P31, P37 | D |
| 2 | 17 | EP, N13, P14, N20 | EP, N13, P14, N20 | PF, LP, P31, P37 | PF, LP, P31, P37 | DPM |
| | 18 | EP, N13, P14, N20 | EP, N13, P14, N20 | PF, LP, P31, P37 | PF, LP, P31, P37 | DPM‡ |
| | 19 | EP, N13, P14, N20 | EP, N13, P14, N20 | PF, LP, P31, P37 | PF, LP, P31, P37 | DPM |
| 3 | 20 | EP, N13, P14, N20 | EP, N13, P14, N20 | PF, P31, P37 | PF, P31, P37 | PM |
| | 21 | EP, N20 | EP, N20 | PF, P37 | PF, P37 | PM |
| | 22 | EP, N13, P14, N20 | EP, N13, P14, N20 | PF, P31, P37 | PF, P31, P37 | PM |
| | 23 | EP, N13, P14, N20 | EP, N13, P14, N20 | PF, P31, P37 | PF, P31, P37 | PM |
| Final | 24 | Br, N20 | Br, N20 | PF, P37 | PF, P37 | PM |
| | 25 | Br, N20 | Br, N20 | PF, P37 | PF, P37 | PM |
| | 26† | Br, N20 | Br, N20 | PF, P37 | Absent | PM |
| | 27 | Br, N20 | Br, N20 | PF, P37 | PF, P37 | PM |
| | 28 | Br, N20 | Br, N20 | PF, P37 | PF, P37 | PM |
| | 29 | Br, N20 | Br, N20 | PF, P37 | PF, P37 | PM |
| | 30 | Br, N20 | Br, N20 | PF, P37 | PF, P37 | PM |
| | 31 | Br, N20 | Br, N20 | PF, P37 | PF, P37 | PM |

* Patient no. 8 had an antecedent right-hemisphere stroke and absent left SSEP.

† Patient no. 26 had an antecedent left-leg compartment syndrome and absent left tibial SSEP; however, gastrocnemius MEP were obtained.

‡ Incorrect instrument settings caused a technical failure to record the D wave in patient no. 18.

SSEP, somatosensory evoked potential; MEP, motor evoked potential; D, D wave; PM, peripheral muscle; DPM, D wave and peripheral muscle; Br, brachial potential; EP, Erb's point; PF, popliteal fossa; LP, lumbar potential.

Other SSEP nomenclature and recording generally followed guidelines of the American Electroencephalographic Society (1994a).

interstimulus interval). Monitoring proceeded with these settings, but incremental adjustments of pulse number or voltage were sometimes required later during surgery.

During the third phase (12 patients), we omitted D wave recordings.

During all three phases, bilateral median and tibial SSEPs were obtained, and the methodology evolved concurrently (see Table 1). The stimulus was a duration of 0.2 msec, with a 25-mA intensity for median stimulation and a 50-mA intensity for tibial stimulation. An averaging delay of 2 to 5 msec for median SSEPs and 5 msec for tibial SSEPs was used. Stimulus frequency was 5.1 or 7.1 Hz for tibial nerves and 5.1, 7.1, or 9.1 Hz for median nerves. The bandpass was generally 30 to 300 Hz but 150 to 1,000 Hz was often used for peripheral SSEPs. Recording derivations and nomenclature followed guidelines of the American Electroencephalographic Society

(1994a). An exception to this was the tibial P37 cortical response, which was often recorded using an individually optimized scalp derivation for each side after partially mapping its scalp distribution bilaterally after induction (MacDonald, 2001). SSEP surface recording electrodes were applied with collodion at measured sites by a registered EEG technologist experienced in intraoperative monitoring. Relevant leads were braided tightly to reduce noise. Impedances were maintained at 1 to 2 kOhm. An optically isolated adhesive plate ground electrode was applied to the left shoulder. Initially we monitored either cortical potentials (median N20 and tibial P37) alone, or with peripheral responses (Erb's point and popliteal fossa [PF]). We then added spinal cord (median N13 and tibial lumbar potential) and subcortical (median P14 and tibial P31) potentials (American Electroencephalographic Society, 1994a). The final approach used

only a peripheral and cortical response for each nerve and replaced the median Erb's point potential with a brachial (Br) peripheral nerve potential recorded from two closely spaced (approximately 2 cm) and braided adhesive disk electrodes on the medial surface of each arm at the midhumeral level or just above the cubital fossa.

Anesthesia consisted of propofol and narcotic infusion ($n = 27$), propofol and narcotic infusion with low-concentration isoflurane ($n = 3$), or low-concentration isoflurane and narcotic infusion ($n = 1$). Ketamine was administered occasionally. Nitrous oxide was omitted. Neuromuscular blockade was omitted for muscle MEPs.

The monitoring team, consisting of one or two technologists and the responsible clinical neurophysiologist (D.M.), was available continuously to provide immediate troubleshooting, optimization, and clinical interpretation. Monitoring began as soon as possible after induction and continued as rapidly as possible throughout until closure. The monitoring sequence was bilateral median SSEPs (asynchronous parallel averaging), bilateral tibial SSEPs (asynchronous parallel averaging), D wave (when used), right-muscle MEPs (left scalp anode), and left-muscle MEPs (right scalp anode), when used.

RESULTS

We learned quickly that disconnecting scalp SSEP electrodes from the headbox for MEP recordings prevented large stimulus artifacts that could obscure MEPs. Similarly, hand muscle leads were usually disconnected temporarily from the headbox during median SSEP recordings to prevent large stimulus artifacts. We encountered electrical interference at the moment of aortic cross-clamping, frequently interfering with monitoring when it was most needed. This was eventually identified as a dissimilar metal artifact from aortic clamps contacting the large thoracic retractor and was solved by preventing contact with sterile cloth pads.

Of 19 D wave recordings, there was one technical failure resulting from inadvertently incorrect instrument settings. Fortunately, this patient had successful muscle MEPs. Epidural electrode locations were T5, 1; T6, 1; T7, 1; T8, 7; T9, 2; T10, 5; and T11, 2. D wave latency increased and voltage decreased (Fig. 1) toward lower placements.

Of 15 muscle MEP recordings, there were no technical failures. In one patient with an antecedent left-leg compartment syndrome, the tibialis anterior muscle was unavailable and we used the gastrocnemius muscle instead. Responses were obtained in single trials, although occasionally repeating a few trials facilitated the response.

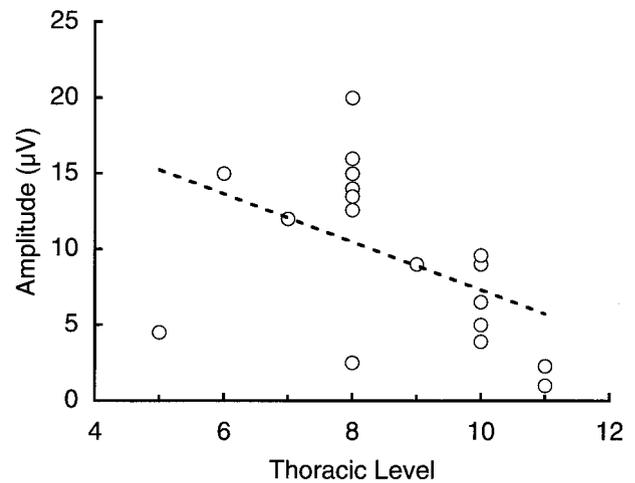


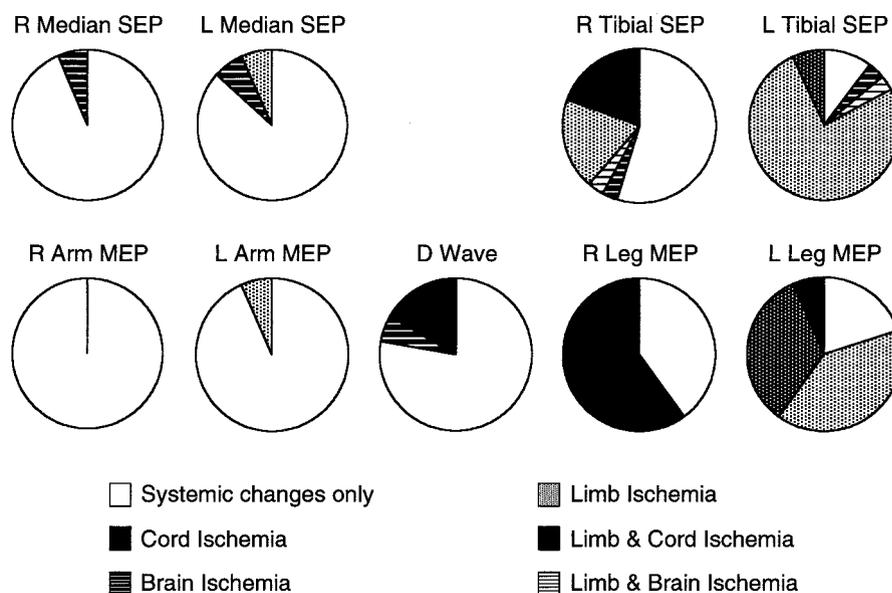
FIG. 1. D wave amplitude decreases toward lower thoracic levels.

Hand muscle MEPs had consistently lower thresholds than leg muscles. Because of random trial-to-trial variability, responses were classified as present or absent. Measurements were unhelpful and eventually abandoned to speed monitoring. Muscle MEPs were more rapid than D wave recordings, which required some averaging and measurement.

We learned that scalp edema resulting from fluid administration reduced TCES effectiveness, presumably because of shunting through the edematous scalp, but could be detected by demonstrating pitting scalp edema and could be overcome by increasing intensity. This produced two early D wave false positives. It was also sometimes necessary to increase stimulus intensity in other cases, probably as a result of cumulative anesthetic effects. These phenomena affected both hand and leg MEPs, and when increasing stimulus intensity restored fading muscle responses in upper and lower extremities, changes were not attributed to cord ischemia. Loss of leg with preserved arm MEPs indicated either leg or cord ischemia, clearly differentiated by SSEP (discussed later). Left-leg ischemia from the occlusive femoral artery bypass cannula regularly produced left-leg MEP loss. In the final two patients, this was prevented using a left femoral side graft rather than a cannula for the bypass (so that the left leg remained perfused and available for monitoring cord function).

There were no SSEP technical failures, but antecedent pathology obliterated SSEPs from one or two limbs in two patients (see Table 1). Systemic changes (hypothermia, hypotension, cumulative anesthetic effects, or scalp edema) occurred in every patient and were identified by parallel tibial and median cortical SSEP alterations, sometimes exceeding the arbitrary "less than 50% of

FIG. 2. Observed causes of evoked potential changes. Each chart indicates the proportion of systemic alterations and cord, limb, or cerebral ischemia causing changes encountered in each evoked potential, when used. Right-leg motor evoked potentials (MEPs) were most specific for cord ischemia. Left-leg MEPs and somatosensory evoked potentials (SSEPs) were often altered by leg ischemia, but this can be prevented by using a femoral side graft rather than a cannula for bypass. No examples of cerebral ischemia happened to occur in the cases with muscle MEPs. These interacting alterations increase the complexity of thoracoabdominal aneurysm monitoring. R, right; L, left.



baseline amplitude" criteria. Control median SSEP recordings prevented false-positive interpretation. Nonparallel changes indicated nonsystemic alterations. Preserved PF potentials with loss of tibial but preserved median cortical SSEPs indicated cord ischemia. Limb ischemia produced a progressive loss of peripheral and proximal SSEPs, and a progressive return after reperfusing the affected limb. This occurred regularly in the left leg because of the occlusive bypass cannula, six times in the right leg during lower body ischemia, and twice in the left arm as a result of aortic cross-clamping proximal to the left subclavian artery. Loss of all cortical but preserved peripheral SSEPs identified cerebral ischemia in two patients. Fig. 2 illustrates the complexity of these interacting factors.

Attempted lumbar and subcortical SSEPs were abandoned eventually because their poor signal-to-noise ratio prolonged averaging time, delaying surgical feedback (see DISCUSSION). Patient positioning distorted supraclavicular anatomy, degrading Erb's point recordings. The median Br potential was unaffected by positioning, and its higher signal-to-noise ratio enhanced recording speed.

The final method consisted of bilateral arm and leg muscle MEPs with median (Br, N20) and tibial (PF, P37) SSEPs, and provided sufficiently comprehensive surgical feedback every 1 to 3 minutes, depending on artifact levels.

There were no adverse effects from TCES or epidural electrode insertion. Specifically, no seizures, scalp burns, tongue biting, epidural infections, or hemorrhages occurred.

Of 16 phase 1 patients with D wave recordings but no muscle MEPs, there was no D wave or SSEP evidence of cord ischemia or cord deficit in 10 patients. However, one of these lost all cortical SSEPs during cardiac arrest, indicating cerebral ischemia even though the D wave was present. SSEP recovery followed resuscitation, but there was postoperative ischemic encephalopathy. In another patient, there was bilateral loss of tibial P37 during lower body ischemia and recovery after reperfusion. Because the D wave at T10 was unaffected, the changes were attributed to leg ischemia, but omission of peripheral tibial SSEPs in this patient prevented a clear neurophysiologic differentiation.

In one patient, loss of right tibial SSEPs above the PF (including the lumbar potential) during hypotension indicated cord ischemia even though the D wave at T8 was unaffected (false negative). Increasing blood pressure reversed the change and there was no deficit.

D wave alterations occurred in five patients. In one, transient D wave loss at 31°C when SSEPs were present returned during rewarming without specific intervention or deficit (Fig. 3). In another, D wave and bilateral median and tibial cortical SSEP loss but preserved peripheral SSEPs during low cardiac output indicated cerebral ischemia, and restoration of cardiac output was followed by a return of all responses. There was mild postoperative ischemic encephalopathy but no cord injury. In one patient there was T10 D wave loss with preserved right tibial SSEPs, suggesting anterior cord ischemia. This prompted anastomoses of multiple segmental arteries to the graft, followed by D wave restoration, and no deficit (Fig. 4). In two patients, D wave

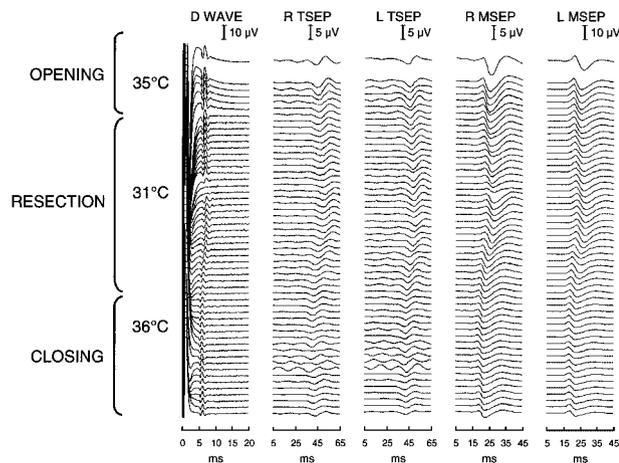


FIG. 3. Transient T10 D wave loss with preserved cortical somatosensory evoked potentials (SSEPs). This occurred at the nadir of hypothermia (31°C) and may have been the result of a greater effect of cooling on the corticospinal tract or brief cord ischemia. The dissociation of SSEPs and motor evoked potentials is of clinical and scientific interest. Note the marked latency shifts from hypothermia and the gradual fall of amplitude to less than 50% of the initial amplitude resulting from systemic changes as identified by median SSEPs. Disconnecting scalp SSEP leads from the headbox can reduce the large stimulus artifact in the D wave traces. TSEP, tibial cortical P37; MSEP, median cortical N20.

amplitude reduction below 50% of initial values raised concern and prompted segmental artery anastomoses to the graft without amplitude improvement or deficit (false positives). In the second of these, scalp examination after intervention disclosed pitting edema, and then increased TCES intensity restored D wave amplitude.

Of three phase 2 patients with both D wave and muscle MEPs, there was no MEP or SSEP evidence of cord ischemia or deficit in one patient. In another patient there was D wave technical failure, transient loss of left-arm and left-leg MEPs and SSEPs resulting from limb ischemia, and transient loss of right-leg MEPs and tibial cortical SSEPs from cord ischemia resulting from brief partial bypass (Fig. 5). Evoked potential evidence of cord ischemia increased the urgency to discontinue partial bypass as soon as possible, followed by restoration of potentials and no immediate postoperative deficit. Unfortunately, postoperative ventricular fibrillation caused ischemic encephalopathy. In the third patient, persistent leg MEP loss despite segmental artery anastomoses predicted paraplegia (Fig. 6). The D wave at T5 was unaffected (false negative), indicating that the infarction occurred below T5. Also, tibial SSEP signs of cord ischemia were delayed compared with muscle MEPs and were recovered, predicting preserved posterior column function but not the paraplegia.

Of 12 phase 3 patients with muscle MEPs but no D

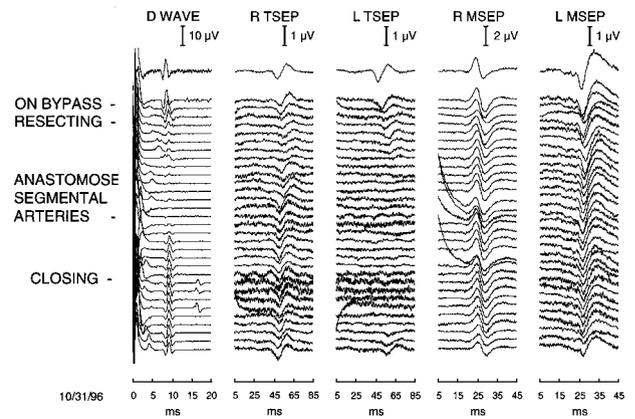


FIG. 4. D wave restoration after intervention. The left tibial somatosensory evoked potential (SSEP) loss was the result of leg ischemia and was recovered after limb reperfusion. The D wave loss during resection suggested cord ischemia. Anastomosing multiple segmental arteries to the graft was followed by D wave restoration and no deficit. The high-frequency artifacts overlaying SSEP traces were introduced by using a traditional 3,000-Hz high-frequency filter, and are preventable by using a 300-Hz setting. The large stimulus artifacts in the D wave traces are preventable by disconnecting scalp SSEP leads from the headbox for motor evoked potential recordings. TSEP, tibial cortical P37; MSEP, median cortical N20.

wave, five had no evoked potential evidence of cord ischemia or deficit. Two patients had MEP and congruent SSEP evidence of cord ischemia. One of these had persistent leg MEP loss despite segmental artery anastomoses but tibial SSEP recovery, and had paraplegia with preserved posterior column function (Fig. 7). The other had leg MEP loss and delayed tibial SSEP alteration

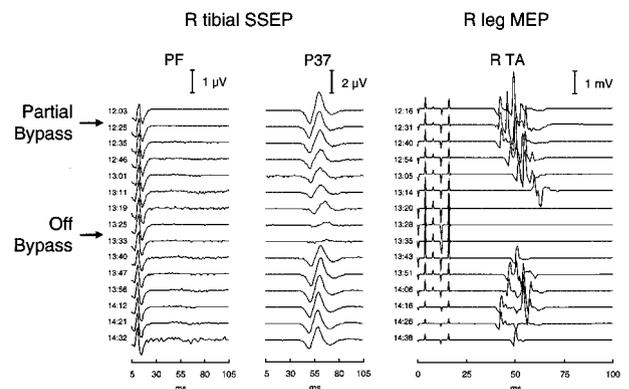


FIG. 5. Congruent motor evoked potential (MEP) and somatosensory evoked potential evidence for cord ischemia, selected traces. During partial bypass, there was a progressive increase in latency and then the loss of right-leg MEPs. Similar but delayed and incomplete right tibial P37 changes occurred. Preservation of the tibial popliteal fossa (PF) response ruled out leg ischemia. All potentials were restored after discontinuing the bypass. The times between each trace were inordinately long as a result of attempted cord and subcortical recordings (not shown). R, right; TA, tibialis anterior.

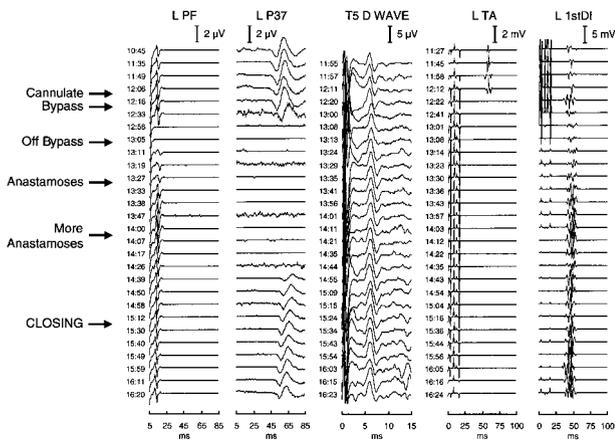


FIG. 6. Intraoperative spinal cord infarction, selected traces. Left tibial somatosensory evoked potential (SSEP) alterations began after left femoral cannulation. Immediately after bypass there was an abrupt, persistent loss of leg motor evoked potentials (MEPs) despite multiple segmental artery anastomoses, predicting paraplegia. The D wave at T5 was unaffected, indicating that the infarction was below that level. After going off bypass and restoring left femoral perfusion, the left popliteal fossa (PF) returned, but not the P37 (tibial cortical response), because of transient transverse effects of cord ischemia. The P37 eventually returned and predicted preserved posterior column function, but not the paraplegia. Preserved arm MEPs ruled out technical MEP failure. Right-leg MEPs (not illustrated) were also lost. The time between each trace is inordinately long because of attempted recordings of cord and subcortical SSEPs, and because of dissimilar metal artifacts from cross-clamps contacting the large thoracic retractor, which was prevented eventually with cloth pads. TA, tibialis anterior; 1stDI, first dorsal interosseous.

restored after increasing bypass flow rate and no deficit. Five patients had leg MEP loss, indicating cord ischemia without congruent SSEP change. Two of these had MEP restoration after increasing bypass flow and awoke without deficit (Fig. 8), but one sustained a delayed postoperative paraplegia. One patient had MEP restoration after increasing blood pressure and no deficit (Fig. 9). One patient had leg MEP loss after aortic cross-clamping restored after clamp release. Choosing a lower aortic level for clamping produced no MEP change and the patient awoke without deficit but died of postoperative cardiac complications (Figs. 10 and 11). One had abrupt leg MEP loss after aortic cross-clamping restored by clamp release. Deep hypothermic arrest and pentothal then obliterated all evoked potentials except markedly delayed peripheral SSEPs, and then the aorta was reclamped and the resection was carried out. There was SSEP and MEP recovery during rewarming and closure, and no deficit.

DISCUSSION

Thoracoabdominal aneurysm surgery is a high-risk procedure. Seven of 31 patients (23%) sustained substan-

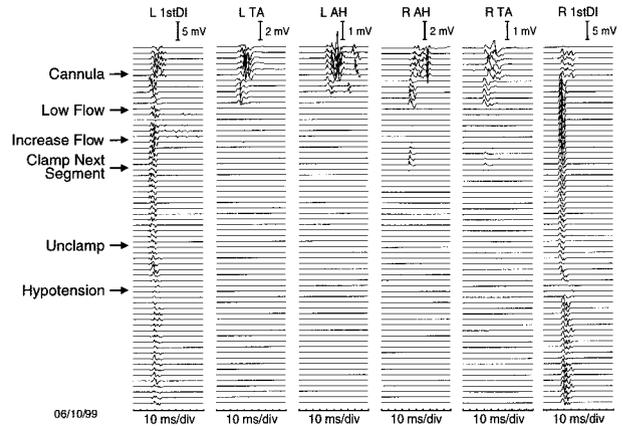


FIG. 7. Intraoperative spinal cord infarction. Only muscle motor evoked potentials (MEPs) are illustrated. Left-leg MEPs were lost because of leg ischemia early. Right-leg MEPs were lost during low bypass flow rates but were restored partially after increasing the flow. Unfortunately, they were lost permanently after clamping the next aortic segment, and neither right- nor left-leg MEPs returned during closure, predicting paraplegia. Similar but delayed tibial somatosensory evoked potential changes occurred but recovered, predicting preserved posterior column function. Arm MEPs showed a transient alteration during brief hypotension. L, left; 1stDI, first dorsal interosseous; TA, tibialis anterior; AH, abductor hallucis; R, right.

tial perioperative neurologic complications: paraplegia (n = 3), ischemic encephalopathy (n = 3), or death (n = 1). Intraoperative monitoring could not have prevented three postoperative complications (9.7%; one paraplegia, one ischemic encephalopathy, and one

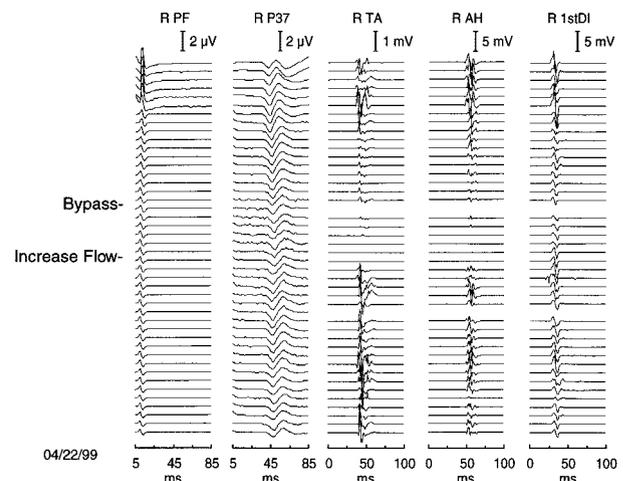


FIG. 8. Transient cord ischemia detected by motor evoked potentials (MEPs) but not somatosensory evoked potentials (SSEPs). The right tibial popliteal fossa (PF) trace changed because we elected to switch from a 30- to a 150-Hz low-frequency filter. Abrupt loss of leg, but not arm, MEPs with preserved tibial SSEPs suggested anterior cord ischemia during low bypass flow. Increasing the bypass flow rate restored leg MEPs and there was no deficit. R, right; TA, tibialis anterior; AH, abductor hallucis; 1stDI, first dorsal interosseous.

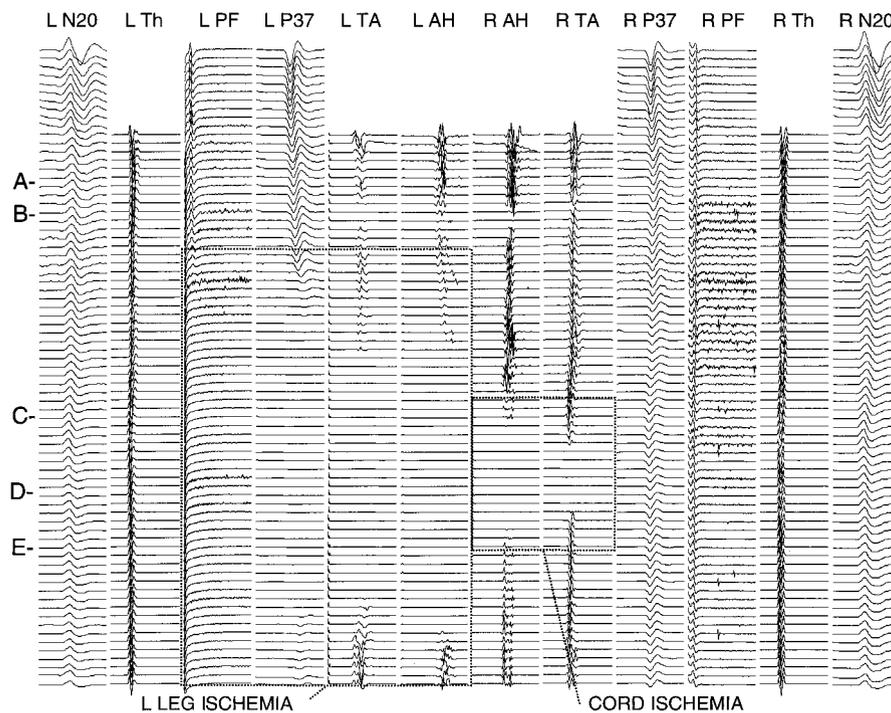


FIG. 9. Cord ischemia detected by motor evoked potentials (MEPs) but not somatosensory evoked potentials (SSEPs). MEPs were omitted initially because neuromuscular blockade had been used for intubation. The median brachial responses are not shown but were stable throughout. **(A)** Cannulate left femoral. There was a delayed progressive loss of peripheral and cortical left tibial SSEPs and then leg muscle MEPs resulting from left-leg ischemia. **(B)** On bypass. Transient, incomplete MEP alterations occurred without intervention. **(C)** Blood pressure was 80/50 mmHg. Loss of right-leg MEPs indicated cord ischemia, and increased the urgency to correct the hypotension. Right-leg SSEPs were unaffected. **(D)** Blood pressure was 110/80 mmHg with restoration of right-leg MEP. **(E)** Repair left femoral artery. After a delay, left-leg MEPs and then SSEPs showed recovery, indicating limb reperfusion. Arm recordings provided valuable controls. Omitting subcortical and lumbar SSEPs greatly reduced the averaging time, improving surgical feedback. L, left; N20, median cortical SSEP; Th, thenar; PF, tibial popliteal fossa SSEP; P37, tibial cortical SSEP; TA, tibialis anterior; AH, abductor hallucis; R, right.

death). Four intraoperative complications (13%; two paraplegias and two ischemic encephalopathies) were potentially preventable.

If neurophysiologic monitoring is to contribute to deficit prevention in these surgeries, it must identify cord ischemia rapidly and reliably, and differentiate this from systemic alterations and limb or cerebral ischemia.

Although SSEPs do not assess directly the corticospinal motor pathway jeopardized by anterior spinal cord ischemia, they do provide important information. Our results confirm that acute cord ischemia sometimes may disturb posterior column function transiently and may be detected with tibial SSEPs (Galla et al., 1999; Grabitz et al., 1996; Griep et al., 1996, 1998; Robertazzi and Cunningham, 1998a, b; Schepens et al., 1994; Shahin et al., 1996). However, SSEPs are clearly less sensitive and specific than muscle MEPs for cord ischemia and cannot be considered sufficient for thoracoabdominal aneurysm monitoring. Tibial SSEPs do predict specifically the expected preservation of postoperative posterior column

function, but cannot predict motor outcome (Guerit et al., 1996; Schepens et al., 1994). Median and tibial cortical SSEPs identify systemic effects and cerebral ischemia. Peripheral nerve SSEPs clearly identify limb ischemia (Gugino et al., 1992), and upper extremity SSEPs provide valuable systemic controls (Shahin et al., 1996).

SSEPs provide a neurophysiologic "background" on which to interpret MEP changes, but require time for averaging, introducing a feedback delay. Concentrating on high signal-to-noise ratio bipolar Br, PF, and scalp derivation cortical responses minimizes this. Although cortical SSEPs are well-known to be suppressed by anesthesia, we had no difficulty obtaining sufficiently robust responses under total intravenous anesthesia with propofol and narcotic infusion. Cortical SSEPs fade gradually in amplitude throughout the surgery, probably as a result of cumulative anesthetic effects and scalp edema. These effects are demonstrated easily by parallel changes in upper extremity SSEP controls.

The tibial lumbar potential generated in the ventral

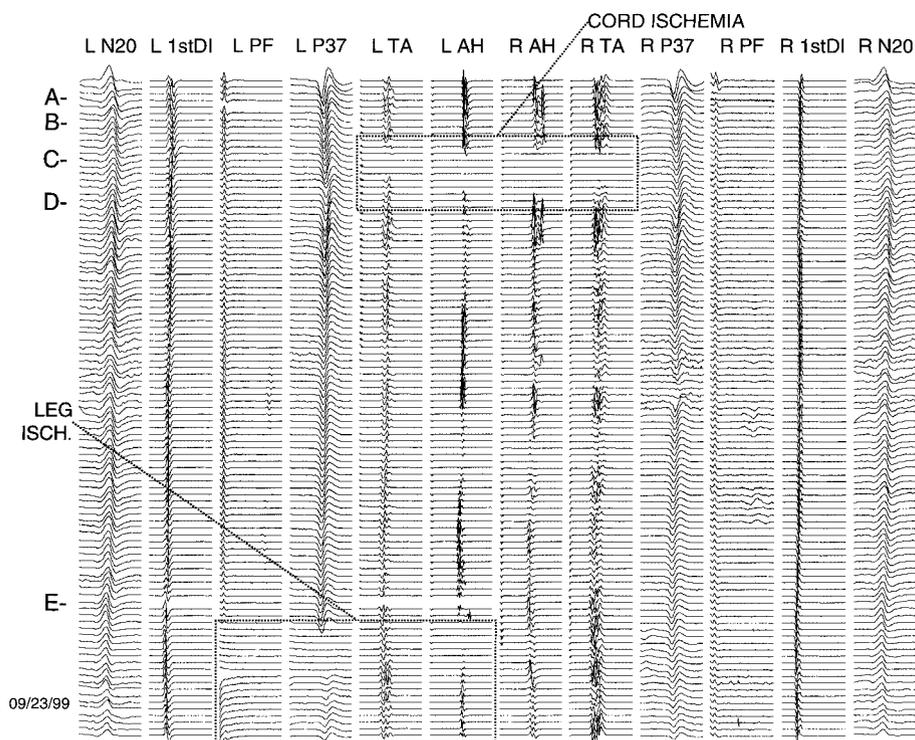


FIG. 10. Surgical guidance provided by leg motor evoked potential (MEP) detection of cord ischemia. The median brachial responses are not shown but were stable throughout. The figure illustrates the final monitoring approach including the prevention of left-leg ischemia during bypass. (A) Left femoral side graft including the limb in the bypass circuit. (B) Cross-clamping of the aorta with abrupt loss of leg MEPs but not somatosensory evoked potentials (SSEPs), indicating cord ischemia. (C) Clamp removal restored leg MEPs. (D) Choosing a lower aortic segment for cross-clamping without producing cord ischemia. Leg MEPs were subsequently present through the resection. A transient right tibial P37 alteration was likely technical. (E) Repairing the left femoral artery produced an unusually abrupt transient limb ischemia affecting SSEPs and MEPs, but at the end of the case, during closure. Arm recordings provided valuable controls. There was no immediate postoperative deficit, but the patient died of postoperative cardiac complications. L, left; N20, median cortical SSEP; 1stDI, first dorsal interosseous; PF, tibial popliteal fossa SSEP; P37, tibial cortical SSEP; TA, tibialis anterior; AH, abductor hallucis; R, right.

lumbosacral cord is resistant to anesthesia and should be sensitive to lumbar anterior cord ischemia (Guerit et al., 1996). This was demonstrated in 1 of 11 attempted lumbar potential recordings in this series. Subcortical SSEPs of brainstem origin offer resistance to anesthesia. Unfortunately, the noise introduced by the long interelectrode distance (e.g., T12 to iliac crest, scalp to noncephalic) and the generally low amplitude of these signals are unfavorable for averaging. In our experience, obtaining reproducible traces consumed inordinate time, unacceptably delaying surgical feedback. Despite their potential benefits, we eventually abandoned these recordings and cannot recommend them for thoracoabdominal aneurysm monitoring when muscle MEPs are available for corticospinal assessment and median SSEPs are available as systemic controls.

This is the first study we know of that evaluates the D wave in thoracoabdominal aneurysm surgery. Despite its theoretical advantages (Deletis, 1993), it cannot be rec-

ommended for thoracoabdominal aneurysm surgery, even though one case provided circumstantial evidence for a contribution to spinal cord infarction prevention (see Fig. 4). There were two false negatives. One occurred when SSEPs indicated cord ischemia but the D wave at T8 did not, probably because ischemia occurred below T8. The other occurred when an inadvertently high T5 electrode failed to detect infarction below that level demonstrated by leg muscle MEP loss and transient tibial cortical SSEP loss. Because of these experiences, placement below T8 is required. However, D wave voltage is quite small at low thoracic levels (see Fig. 1). Also, it was difficult and time-consuming to obtain a low thoracic location, and the invasive insertion carries the risk of epidural complications, although none occurred. Two early false positives involved a substantial fall of D wave amplitude, prompting unnecessary segmental artery anastomoses. In retrospect, probably both were the result of scalp edema, demonstrated in one patient by

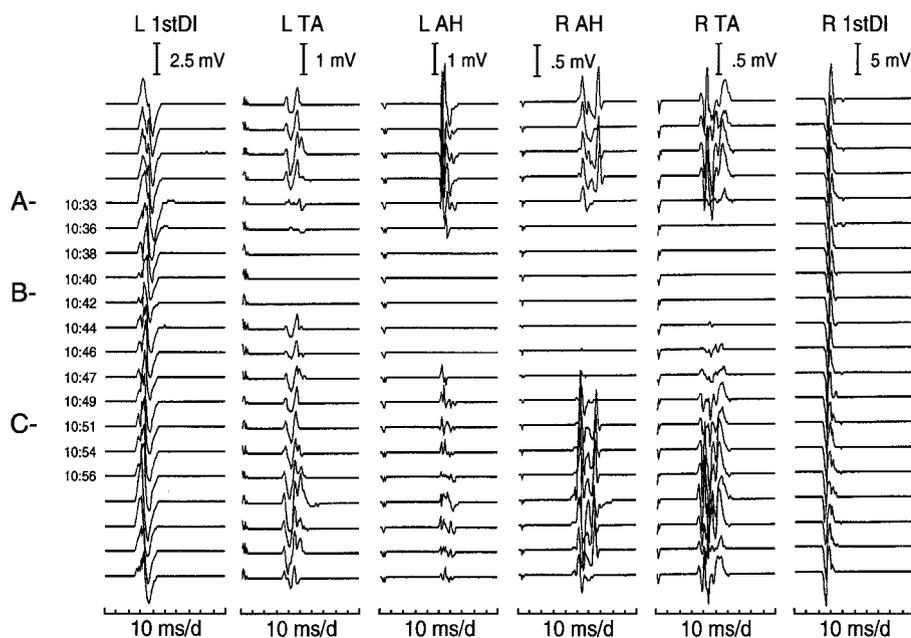


FIG. 11. Same case as Fig. 10, showing motor evoked potential (MEP) detail during aortic cross-clamping. (A) Aortic cross-clamping followed by leg MEP loss, indicating cord ischemia without somatosensory evoked potential (SSEP) alteration. (B) Clamp removal restored MEPs. (C) Choosing a lower aortic segment for clamping allowed the resection to proceed without cord ischemia. Note that full recordings were obtained every 1 to 3 minutes, providing sufficiently rapid surgical feedback. This was possible because subcortical and lumbar SSEPs were omitted and dissimilar metal artifacts were prevented (see text). L, left; 1stDI, first dorsal interosseous; TA, tibialis anterior; AH, abductor hallucis; R, right.

intraoperative scalp examination and corrected by increasing TCES intensity after the unnecessary intervention. Both may have been avoided had we recognized scalp edema earlier. D wave recordings required some averaging and measurement, consuming valuable time. Finally, D wave recordings exclude spinal motor neurons, which should be disabled more rapidly by ischemia than tracts (Qayumi et al., 1997).

Muscle MEPs after multiple-pulse TCES are highly recommended for thoracoabdominal aneurysm surgery monitoring (de Haan et al., 1998; van Dongen et al., 1999). The omission of neuromuscular blockade and resulting patient movement produced no surgical difficulty. There were no adverse effects, technical failures, false positives, or false negatives. Each of 13 patients with present leg muscle MEPs at closure awoke without cord deficit, and two patients with absent leg MEPs but present tibial SSEPs at closure awoke paraplegic with preserved dorsal column function. Right-leg MEPs were most specific for cord ischemia (see Fig. 2), and a minimalist approach could be to monitor this alone, but would impair differentiation from confounding factors. Left-leg MEPs were often lost because of leg ischemia. Femoral side grafting rather than cannulation prevents this, and is highly recommended (see Fig. 10). Arm MEPs provide valuable controls. Muscle MEPs are available in single trials, do not require measurement because presence or absence is interpreted, and thus provide immediate surgical feedback including spinal motor neurons.

Although some investigators are concerned about the safety of TCES, specifically about the possibility of intraoperative seizure induction, this did not occur. Nor has it occurred in more than 120 other cases using TCES in the personal intraoperative experience of the principal author (D.M.) to date. We are not aware of a single reported case of seizure induction among thousands of patients monitored with TCES around the world. Seizure induction by much more intense direct cortical stimulation during cortical mapping is not considered to be a dangerous or catastrophic event and is sometimes even sought during epilepsy surgery. Because neuromuscular blockade is omitted for muscle MEPs, a seizure should be apparent in convulsive motor activity. It seems reasonable to monitor EEGs intermittently to look for electrographic seizure activity, but the principal author has not found this so far when sought. In the occurrence of a seizure during TCES, stimulation could be stopped and appropriate medical intervention undertaken immediately. It may be prudent to avoid TCES for patients with epilepsy. In our view, this theoretically possible but apparently unlikely complication is far outweighed by the highly valuable corticospinal system information provided. At the same time, monitorists, surgeons, and anesthesiologists should remain alert to this possibility, and patients must be aware of this concern for valid informed consent.

After evaluating a variety of approaches, we conclude that a safe, reliable, and sufficiently rapid (see Fig. 11) differential method for spinal cord monitoring during

thoracoabdominal aneurysm surgery consists of arm and leg muscle MEPs after multiple-pulse TCES with median (Br, N20) and tibial (PF, P37) SSEPs. Cloth pads between the thoracic retractor and aortic clamps eliminate dissimilar metal artifacts. Femoral side grafting incorporates the left leg in spinal cord monitoring. Disconnecting scalp leads from the headbox for MEP recordings and disconnecting hand muscle leads for SSEP recordings reduce stimulus artifacts. It would be reasonable to omit SSEPs; however, in our view the benefits of SSEP outlined earlier favor their inclusion.

Our results demonstrate that spinal cord ischemia can be detected rapidly and reliably. Whether monitoring can help prevent paraplegia is another matter. In 11 patients (35%), evoked potential evidence of cord ischemia prompted intervention. In two patients, anastomoses of segmental arteries to the graft did not prevent paraplegia. The other nine patients (29%) provide circumstantial evidence for contributions to deficit prevention. One of these was detected by tibial SSEP changes (but not the D wave at T8), restored after correcting hypotension. One had T10 D wave restoration after segmental artery anastomoses when right tibial SSEPs were present. Two had congruent leg MEP and tibial cortical SSEP loss restored after discontinuing partial bypass in one and segmental artery anastomoses in the other. Five were detected by leg MEPs only, restored after increasing bypass flow or blood pressure, choosing a lower aortic clamp placement, or prompting deep hypothermic cardiac arrest.

Although one patient sustained delayed postoperative paraplegia and another died of postoperative complications, neurophysiologically guided interventions were associated with immediate postoperative success. Interventions to discontinue partial bypass as soon as possible, and correct low blood pressure, cardiac output, or bypass flow rate in five patients (16%) were indicated with or without monitoring. At the same time, it was surprising how often evoked potential evidence of cord ischemia occurred during these circumstances, and this increased intervention urgency. The other four interventions (13%)—segmental artery anastomoses to the graft ($n = 2$), choosing a different aortic clamp site ($n = 1$), and deep hypothermic cardiac arrest ($n = 1$)—provide more compelling circumstantial evidence of possible contributions to deficit prevention.

That intraoperative paraplegia occurred in two patients (6.5%) in this series compares favorably with the expected rate of approximately 10%. However, our results in this small series do not provide clear evidence that neurophysiologic monitoring can improve neurologic outcome of thoracoabdominal aneurysm surgery. Now that a rapid differential monitoring approach has been

defined, it seems likely that improved outcome will become possible. Indeed, our surgeon (M.J.) will no longer undertake thoracoabdominal aneurysm surgery without the neurophysiologic assessment provided by this method.

Acknowledgment: This work would not have been possible without the dedicated efforts of the Vancouver General Hospital intraoperative monitoring technologists Nimira Bapoo, Karen Liddle, and William Gene.

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