Neurophysiologic Monitoring during Thoracoabdominal Aortic Aneurysm Surgery

To the Editor:
Vaughn et al. are to be commended for their excellent educational case report on anesthesia for thoracoabdominal aortic aneurysm repair. However, there is one area that we believe merits clarification, because it does not bring “completeness” to a full discussion of the subject. In their report, they tend to minimize the relative value and utility of neurophysiologic monitoring during thoracoabdominal aortic aneurysm (TAAA) surgery, while citing a small sample of studies and citing “drawbacks and limitations for the use of somatosensory-evoked potentials (SSEPs) and motor-evoked potentials (MEPs).” One is immediately reminded of the debate on cerebrospinal fluid drainage that raged for many years. As early as 1988, Hollier’s group had provided evidence of the neuroprotective action of cerebrospinal fluid drainage in canine studies. However, skepticism prevailed among surgeons for many years. Crawford himself was highly skeptical of the modality, stating in 1991, “Thus cerebrospinal fluid drainage as we used it, was not beneficial in preventing paraplegia.” But, only in 1994, when Safi et al. demonstrated the association between cerebrospinal fluid drainage and reduced postoperative neurologic deficits in TAAA patients, did the surgical community relent.

Several published reports have identified neurophysiologic monitoring as highly useful in the prediction and amelioration of neurologic complications occurring after TAAA surgery. In a study of 233 patients undergoing TAAA surgery, in which SSEP and MEP monitoring was conducted, our group demonstrated that SSEPs and MEPs were highly correlated when intraoperative changes were irreversible. These irreversible changes were significantly associated with immediate neurologic deficits. In contrast, normal SSEP and MEP findings had a strong negative predictive value for neurologic deficits. These findings confirm the results of Shine et al., who reported a negative predictive value of 96% for MEPs, in the absence of any changes for 20 min after aortic cross-clamp application. In a further refinement, our group examined 108 patients undergoing TAAA surgery, citing loss of SSEPs and MEPs, in 26% and 50% of patients, respectively. Active intraoperative measures, which included intercostal artery reattachment in 85% of patients and judicious management of distal aortic and cerebrospinal fluid pressure, produced a return of SSEPs in all patients and a return of MEPs in all but one patient, (who awoke with an immediate neurologic deficit). MEPs can be highly reactive to spinal cord perfusion, as reported by Koeppel et al., who demonstrated rapid temporal changes of MEP loss and recovery, with interruption and restoration of distal aortic perfusion.

Why do Vaughn et al. and ostensibly other surgical groups not embrace neurophysiologic monitoring during TAAA surgery? Well, they require an experienced team of a surgeon, neurologist, anesthesiologist, and perfusionist to ensure that they are effective and accurate. Coselli and Tsi recently questioned the validity of MEPs, citing interference by anesthesia agents and other potential factors. Our experiential track record has been quite the opposite. Indeed, we limit our use of inhalation agents to approximately 0.5 minimum alveolar concentration, but this is not a novel technique in cardiac anesthesia. We have transitioned to the use of shorter acting muscle relaxants such as rocuronium and cisatracurium. However, in some instances, we have found it necessary to suppress MEPs with a small dose of muscle relaxant to minimize MEP interference of the SSEP signal. Because MEPs actually produce muscle contraction and subtle patient movement, electrode detachment can occur at any time during the procedure and must be detected rapidly by the neurophysiology technician.

What was not borne out in Vaughn’s article was the synergy of effort that is needed when cortical sensory evoked potentials, and in particular, when MEPs degrade during TAAA surgery, consisting of improving distal aortic perfusion (perfusionist), reducing cerebrospinal fluid pressure (anesthesiologist), and intercostal artery reattachment (surgeon). Likened to the aviation industry’s “Crew Resource Management,” the team caring for the TAAA patient must all work aggressively and rapidly in an attempt to reverse these changes and mitigate neurologic complications. Based on the lessons learned from cerebrospinal fluid drainage, and in the interest of patient safety, we should view neurophysiologic monitoring during TAAA surgery not as an obscure modality as Vaughn et al. impugn, but as a “standard-of-care.”

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References

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In Reply:
Drs. Loubser and Sheinbaum purport in their letter that “Based on the lessons learned from cerebrospinal fluid drainage, and in the interests of patient safety, we should view neurophysiologic monitoring during TAAA [thoracoabdominal aortic aneurysm] surgery not as an obscure modality as Vaughn et al. impugn, but as a standard-of-care.” Although it is fair to acknowledge that some centers have successfully adopted these techniques in the interests of patient safety, we disagree that these techniques should be considered “standard of care” (which has major medical–legal connotations).

The most recent American College of Cardiology; American Heart Association; American Association for Thoracic Surgery; American College of Radiology; American Stroke Association; Society of Cardiovascular Anesthesiologists; Society for Cardiovascular Angiography and Interventions; Society of Interventional Radiology; Society of Thoracic Surgeons; and Society for Vascular Medicine guidelines for spinal cord protection during descending aortic open surgical and endovascular repairs specifically state that “neurophysiologic monitoring of the spinal cord (somatosensory evoked potentials or motor evoked potentials) may be considered as a strategy to detect spinal cord ischemia and to guide reimplantation of intercostal arteries and/or hemodynamic optimization to prevent or treat spinal cord ischemia (Class IIb Indication).”2 In point of fact, the only Class I recommendation at present for spinal cord protection in patients at high risk of spinal cord ischemic injury undergoing open or endovascular thoracic aortic repair is cerebrospinal fluid drainage.2

Respectfully, we also disagree that we “impugned” neurophysiologic monitoring as an obscure technique. Rather, after having presented the supporting evidence for neurophysiologic monitoring,3 we simply and correctly stated that “there are limitations and drawbacks for the use of somatosensory evoked potentials and motor evoked potentials for these procedures, and are not standard practice at all institutions.”4 Thus, in our ongoing effort to decrease morbidity and mortality during open and endovascular repair of the descending and thoracoabdominal aorta, we fully support and advocate the use of any of the recommended strategies for spinal cord protection.2

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### References


### Confirmation of Nonanesthetic-induced Malignant Hyperthermia

To the Editor:
We read with interest the important study by Groom et al., Identical de novo Mutation in the type 1 Ryanodine Receptor Gene Associated with Fatal, Stress-induced Malignant Hy-

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This letter was sent to the author of the original article by Groom et al., who chose not to reply.—James C. Eisenach, M.D., Editor-in-Chief.