Masters of Cardiothoracic Surgery

Neuromonitoring for descending thoracic and thoracoabdominal aortic aneurysm repair

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Clinical vignette

A forty-four-year-old female presented with an extent II thoracoabdominal aortic aneurysm (TAAA) and lower back pain. Computed tomography demonstrated contained rupture of the 10 cm infrarenal abdominal aorta. The proximal descending aorta (DTA) was 5.5 cm in diameter but the aortic segment T8 to T12 was 3 cm in diameter. Thus, staged repair of the TAAA was planned. The patient successfully underwent first-stage, extent IV TAAA repair, which includes replacement of the aorta from T12 proximally to right iliac and left common femoral distally with bypass to the visceral/renal arteries. There were no motor evoked potentials (MEPs) or somatosensory evoked potentials (SSEPs) changes. All the patent lumbar arteries were ligated. Her postoperative course was uneventful. After ten days of recovery, the patient was taken back to the operating room for the second stage, completion of the extent II TAAA repair.

Surgical techniques

Our surgical techniques for DTA/TAAA repair are described elsewhere (1). Spinal cord ischemia (SCI) is a life-disabling complication after distal aortic repair. We use distal aortic perfusion with left heart bypass, cerebral spinal fluid (CSF) drainage, and moderate hypothermia for organ protection, including the spinal cord.

We routinely use MEPs and SSEPs to monitor spinal cord function (2,3). Briefly, all the neuromonitoring settings are performed simultaneously to the anesthesiologist's line placement. MEPs and SSEPs are evaluated every one to two minutes in the initial fifteen minutes of aortic clamp, then every five minutes.

The SSEP stimulating needle electrodes are placed bilaterally at the level of the malleolus to stimulate the posterior tibial nerves, and the recording electrodes are placed at three levels: posterior fossa; C5 spine; and vertex (*Video 1*). For MEP monitoring, electrodes are place at C3/C4 for stimulation, and recording needles in the abductor digiti minimi, tibialis anterior, and abductor hallucis muscles. A baseline of SSEP/MEP tracings prior to the beginning of the procedure are compared with the subsequent tracing.

When a signal loss occurs, MEPs are lost prior to SSEPs (2,3). We interpret the loss of MEPs as early, reversible SCI, and the loss of SSEP as advanced ischemia that is reaching more irreversible state (3). Thus, when there are changes in MEPs, the following measures are taken to increase the spinal cord perfusion: reattachment of intercostal arteries T8 to T12; reestablish pulsatile flow to the pelvis; increase blood pressure; drain CSF <10 mmHg; optimize hematocrit; and increase distal perfusion pressure >80 mmHg (1,3). After SSEPs are lost, it is crucial to directly restore spinal cord perfusion as soon as possible.

Modification of anesthetic technique is mandatory to not interfere with neuromonitoring. Patients are induced with a loading of fentanyl citrate (2–5 μg/kg), midazolam (30–50 μg/kg), propofol (0.5–1 mg/kg), and rocuronium bromide (0.6–1 mg/kg). Patients are then maintained on a sevoflurane at 0.5 minimal alveolar concentration.

Case continued

The chest was reentered through the previous

thoracoabdominal incision and 6th intercostal space. Left heart bypass with distal perfusion via previous abdominal aortic graft was established and the aorta was cross-clamped distally at the level of T8. A proximal aortic cross clamp was applied just proximal to the left subclavian artery. There was almost immediate loss of MEPs, followed by SSEP loss, despite distal aortic perfusion pressure >80 mmHg. Following removal of the aortic cross clamp, MEPs and SSEPs immediately returned. Due to the MEP and SSEP signal loss, rather than performing a complete replacement with intercostal reattachment, a segment of normal caliber aorta from T8 to T12 was left intact to minimize the spinal cord ischemic time. The patient awoke without neurological deficit and had no evidence of SCI in the postoperative period.

Comments

Clinical results

Our fifteen-year experience with MEP and SSEP during DTAA/TAAA repairs demonstrated that isolated MEP loss and the combination of MEP and SSEP loss were predictive of immediate and delayed onset SCIs (3).

Advantages

Intraoperative MEP and SSEP monitoring provides actionable data that can be critical for intraoperative decision making, such as seen in this case. Loss in MEP/SSEP signals warrant aggressive intervention to improve the spinal perfusion. When comparing our outcomes after DTAA/TAAA repairs to that without MEP/SSEP monitoring (4,5), the incidence of immediate SCIs was less in our study (<2% *vs.* 5–8.0%).

Use of both the MEP and SSEP allows the surgical team to determine if the MEP loss is from ischemic limb or SCI. An MEP loss with SSEP loss at all three levels is considered non-SCI (i.e., ischemic limb). A loss of MEP with a change or loss of SSEP signals in the cervical/cortical channels but preserved normal popliteal signals, is indicative of SCI.

Caveats

Modification of the anesthesia protocols is mandatory not to interfere with MEP and SSEP monitoring. The MEP/SSEP measurements require an experienced neurophysiologist to obtain and interpret the results. In addition, MEP and SSEP monitoring are not stable during deep hypothermia and, thus, not reliable.

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Footnote

Conflicts of Interest: ALE is a consultant for WL Gore, CryoLife, Edwards Lifesciences, and Terumo Aortic. The other authors have no conflicts of interest to declare.

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