Intraoperative Electrophysiologic Monitoring in Aortic Surgery

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INTRAOPERATIVE MONITORING (IOM) has been used to reduce cerebral and spinal cord injuries during the repair of thoracoabdominal aorta aneurysms (TAAAs). The risks of cerebral injury when the ascending aorta is repaired and of paraplegia when the descending thoracic aorta is involved have prompted the application of numerous techniques to measure blood flow, assess the balance of blood flow and metabolism, and warn of ischemia in order to prompt corrective measures.[1,2] The neurologic risk varies with the portion and length of the aorta that is diseased. The highest risk occurs with Crawford types I and II because both include part of the ascending and the entire descending thoracic aorta.[3]

The most common mechanism of central nervous system (CNS) injury during these procedures is ischemia caused by inadequate arterial perfusion and embolic phenomena. The importance of adequate blood flow to alter the risk of CNS injury is shown in Figure 1. In the brain, as cerebral blood flow falls below an ischemic threshold, the time to infarction decreases.[4-6] For example, cell death may occur after 3 to 4 hours when the perfusion is just below the ischemic threshold with normal metabolism. Infarction occurs at shorter periods as the flow is further lowered and/or metabolism increased. With no perfusion and normal metabolism, cell death occurs in a very short interval (ie, 3-5 minutes). The times and flows in Figure 1 are average values and vary among individuals; however, the general concepts of the loss of electrophysiology being an early warning sign and that time becomes a critical element when flow is reduced remain. These concepts also apply to the spinal cord; however, the values are not well characterized. Hence, during ischemia, an increase in blood flow increases the margin of safety by extending the allowable ischemic time to complete the procedure.

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During surgeries to repair the ascending, transverse, or descending aortic arch, monitoring is used to identify the onset of global or focal hypoperfusion in the brain and spinal cord to allow for correction of the cause (eg, hypotension and mechanical blood flow compromises) and to evaluate the effectiveness of corrective maneuvers (eg, raising blood pressure). Failure to correct blood flow may result in stroke in as many as 12% of patients.[7] Embolic phenomena also may occur, which can contribute to stroke either by interrupting blood flow or aggravating hypoperfusion (which also may reduce the clearance of particulates or gas bubbles).[8,9] When performing surgeries on the descending aorta, the concern for CNS injury shifts to concerns for paraplegia from spinal cord ischemia. The incidence of immediate and late postoperative paraplegia ranges from 2.7% to 5.3%, which is higher in redo surgeries (as high as 10.7%).[10,11] The application of these IOM techniques rests on their anatomic location in the CNS and their sensitivity to ischemia.

The importance of a method to identify spinal cord ischemia with surgery on TAAAs is enhanced because the vascular anatomy is altered by the disease process. With TAAAs, patients frequently develop collateral circulation to supplement blood flow because the arteries are occluded by mural thrombi, arterial plaques, or aortic flaps. The specific vascularity for each patient is not known, and the perfusion of the spinal cord behaves as a meshwork that receives perfusion from proximal (eg, the subclavian artery), distal (eg, the internal iliac [hypogastric] artery), and aortic sources (radicular arteries including the artery of Adamkiewicz).[12] Because patients vary in their dependency on each of these components, monitoring can be helpful to determine the critical contribution of the cephalad aortic radicular arteries and lumbar/pelvic vascularity for each patient. Some patients will be dependent on adequate pressure in a distal bypass perfusion,[13,14] whereas others will require reimplantation of intercostal radicular perforators from the aorta.[13,15-19]

The value of electrophysiologic monitoring also is shown in Figure 1 (ie, the alteration in cortical electric activity accompanies the decline in cerebral blood flow that precedes the risk of neurologic injury). As depicted, cerebral blood flow can be reduced from normal (50 mL/min/100 g) to about 44% (22 mL/min/100 g) while maintaining normal electric activity. Below this level, hypoperfusion occurs, and electric activity is altered. When blood flow reaches about 15 mL/min/100 g, the ischemic threshold is reached, and normal neural synaptic activity ceases (ie, electric activity is lost).[4]

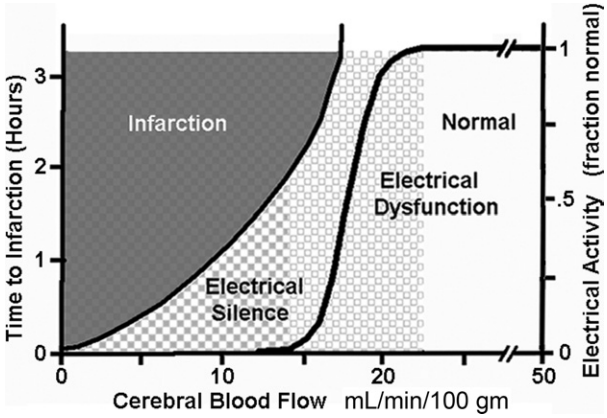

Fig 1. A depiction of the cortical electric activity and the occurrence of neural infarction as the average cerebral blood flow is reduced from normal (50 mL/min/100 g). The electroencephalogram becomes abnormal below 22 mL/min/100 g (ischemic threshold) and absent when blood flow reaches 15 mL/min/100 g. Infarction occurs at 17 to 18 mL/min/100 g after 3 to 4 hours and progressively shorter periods with blood flow below this level. (Reproduced with permission.[6])

TECHNIQUES OF ELECTROPHYSIOLOGIC MONITORING AND ANESTHESIA IMPLICATIONS

Monitoring for ischemia occurring in the brain during aortic surgery can be performed with electroencephalography (EEG) or evoked potentials. EEG is useful to warn of ischemia because it is produced by the synaptic activity of the cortex, which is very sensitive to ischemia (ie, electroencephalographic loss occurs within 20 to 30 seconds of the cessation of cortical blood flow). As shown in Figure 1, the alteration and loss of electroencephalographic activity correlates with the onset of the risk for neurologic injury and is the basis for its use in carotid endarterectomy. As with all the electrophysiologic techniques, anesthetic agents and physiologic changes (eg, hypothermia) can alter the synaptic function leading to electroencephalographic changes.[20] Fortunately, except for higher doses of most anesthetics that depress electroencephalographic activity (usually 1-1½ minimal alveolar concentration [MAC] of inhalation anesthetics), EEG usually is well maintained with the usual medications for general anesthesia. A few agents (eg, etomidate and ketamine) can increase electroencephalographic activity, but because of this they are not usually used. As such, EEG can be used to warn of brain ischemia when performing aorta surgery when there is a shift to slow frequencies or a significant loss of amplitude.

Somatosensory-evoked potentials (SSEPs) can be used to monitor the brain and spinal cord because their neural pathway traverses these regions. SSEPs are produced in response to the electric stimulation of large, mixed motor and sensory peripheral nerves at the wrist (ie, the median and ulnar nerves) and ankle (ie, the posterior tibial nerve) (Fig 2). The response follows the pathway of proprioception and vibration that enters the spinal cord via the posterior nerve roots and ascends to the ipsilateral dorsal column. It makes its first synapse near the nucleatus cuneatus and nucleatus gracilis and crosses the midline near the cervicomedullary junction, ascending through the brainstem via the contralateral medial lemniscus. It makes a

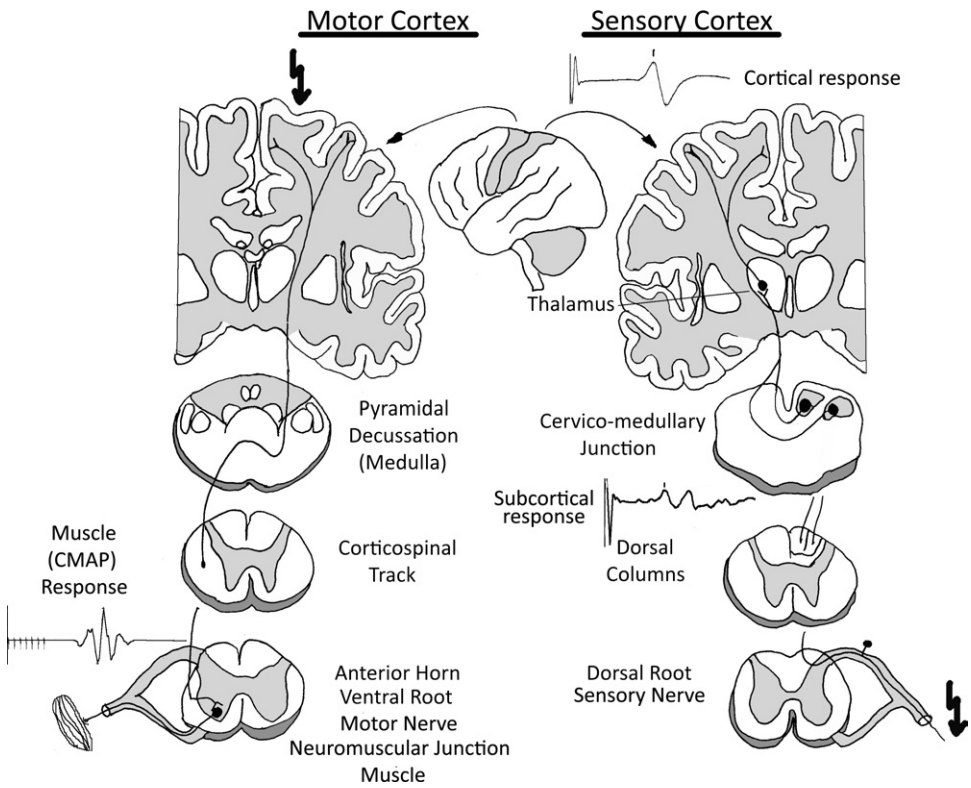

Fig 2. Pathways of the MEP and SSEP are depicted. The muscle MEP (left) is produced by transcranial electric stimulation of the motor cortex (arrow). The response travels down the CST, crossing the midline in the medulla. It continues in the white-matter tracts of the spinal cord and activates the motor nuclei in the anterior horn cell of the spinal cord. The response travels via the ventral root to the neuromuscular junction where the response usually is measured as a compound muscle action potential (CMAP). The cortical SSEP (right) is produced by electric stimulation of a peripheral sensory nerve (arrow). It enters the spinal cord via the dorsal root and ascends the spinal cord in the dorsal columns. It synapses in the cervicomedullary junction, crosses the midline, and synapses in the thalamus before producing a response in the sensory cortex. The response is typically recorded over the sensory cortex (the cortical SSEP) and the cervical spine (the subcortical response).