PROTECTING THE SPINAL CORD IN THORACOABDOMINAL AORTIC PROCEDURES

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Learning objectives:

1. Be able to identify patients undergoing thoracoabdominal aortic procedures who are at high risk of spinal cord ischemia.
2. Learn intraoperative management strategies and techniques to decrease the risk of spinal cord ischemia in patients undergoing thoracoabdominal aortic procedures.
3. Learn the indications for lumbar cerebral spinal fluid drainage in patients undergoing thoracoabdominal aortic procedures.

Introduction

Spinal cord ischemia remains an important complication of open surgical and endovascular stent graft repair of thoracic and thoracoabdominal aortic aneurysm despite advances in operative technique. Identification of risk factors and interventions to prevent and treat spinal cord ischemia have the potential to prevent spinal cord infarction and the morbidity and mortality associated with paraplegia.

Risk factors for spinal cord ischemia are aneurysm extent, open surgical repair, prior distal aortic operations, and perioperative hypotension. Augmenting spinal cord perfusion by increasing arterial pressure, lumbar cerebrospinal fluid drainage, and reattachment of segmental arteries are effective for the treatment of spinal cord ischemia. Early detection of spinal cord ischemia by intraoperative neurophysiologic monitoring and postoperative neurological examination is important to enable immediate treatment to prevent permanent paraplegia.

Permanent paraplegia after thoracic and thoracoabdominal aortic aneurysm repair can be prevented in many high-risk patients by early detection and immediate treatment of spinal cord ischemia before it evolves to infarction. The mortality and morbidity associated with permanent paraplegia justifies the risks and uncertainties associated with established therapeutic interventions.

The spinal cord is at risk for ischemia or infarction in operations involving the descending thoracic and thoracoabdominal aorta making paraplegia or paraparesis an important complication of these procedures. The surgical treatment of aneurysms, dissections, atherosclerotic ulcers, or injuries of the thoracic aorta is to either replace the diseased segments of the aorta with a prosthetic vascular interposition graft or to exclude it with an endovascular stent graft. Either of these surgical procedures require the temporary or permanent interruption of arterial collaterals that supply the spinal cord. As
a consequence, despite advances in spinal cord protection, the risk of spinal cord ischemia or infarction as a consequence of open surgical repair of thoracoabdominal aortic aneurysms (TAAA) remain in the range of 8% to 28%. The risk of spinal cord ischemia or infarction is less after thoracic endovascular aortic repairs (TEVAR), but still occurs with an incidence of approximately 4% to 7%. Risk factors for spinal cord ischemia after open TAAA repair are emergency operation and aneurysm extent, being greatest in Crawford extent II TAAA and least in Crawford extent IV TAAA. Risk factors for spinal cord ischemia after TEVAR are extent of endovascular stent coverage of the thoracic aorta and prior distal aortic repair.

Spinal cord ischemia after thoracic aortic operations is caused by aortic cross-clamping or circulatory arrest during aortic repair followed by the sacrifice or exclusion or segmental arterial branches from the native descending aorta. The arterial supply to the spinal cord from the cephalic end consists of the anterior spinal artery that arises from both vertebral arteries and supplies the anterior portion of the spinal cord. A pair of posterior spinal arteries also arising from the vertebral arteries supplies the posterior spinal cord. From the caudal end, the anterior spinal artery receives arterial collateral supply from the internal iliac artery and its branches, the middle sacral artery, and the inferior mesenteric artery. Along its course, the anterior spinal artery receives collateral supply by paired intercostals and lumbar segmental arteries branching off the descending aorta. The arteria magna or artery of Adamkewicz, is the name applied to a particular large segmental artery between the T5 to L2 vertebral level, existing between the T9 to T12 vertebral levels in 75% of patients. Within the spinal canal, there is an axial network of small arteries that anastomose with one another and the major arteries that supply the spinal cord.

Strategies to prevent and treat spinal cord ischemia after thoracic aortic operations primarily involve techniques to make the spinal cord less susceptible to infarction, minimize the duration of cord ischemia during operation, augment spinal cord blood flow hemodynamics, and early detection of spinal cord ischemia to permit immediate intervention. While it has been long understood that paraplegia causation is ‘anatomic’, prevention of ischemic infarction is ‘non-anatomic’ or ‘physiologic’. Studies support this concept. In models of spinal cord ischemia caused by TAAA repair, interventions such as deliberate hypothermia, spinal cerebrospinal fluid drainage, increasing mean arterial pressure, and minimizing arterial steal were effective for preventing paraplegia even when intercostal vessels were not re-implanted.

Techniques to decrease spinal cord and end-organ ischemic time

The original “clamp and sew” technique for TAAA repair was to temporarily interrupt blood flow in the descending aorta by placing “clamping” the proximal neck of the aneurysm segment while “sewing” in the vascular interposition graft. While the “clamp and sew” technique was successful, the risk of paraplegia from spinal cord ischemia was related to the ischemic time for end organs distal to the site of the cross clamp. The incidence of paraplegia was as high as 27% in patients whose aortic cross clamp time exceeded 60 minutes, and 8% in those with cross clamp time less than 30 minutes. As a
consequence, several techniques were devised to decrease the ischemic time of organs perfused by the distal aorta. The Gott Shunt, is a 9 mm heparin-coated polyvinyl tube to passively shunt blood from the proximal aorta or left ventricular apex to the distal aorta beyond the cross clamp. Limitations of the Gott shunt include the inability to control flow through the conduit and the inability to unload the left ventricle. Partial left heart bypass provides a controlled means of perfusing the distal aorta by directing blood from the left atrium to the distal descending aorta or femoral artery. Distal perfusion pressure can be monitored by a femoral arterial catheter in the contralateral femoral artery and flow can be controlled by the centrifugal pump. Partial left heart bypass also unloads the left ventricle to prevent excessive proximal hypertension. Systemic temperature can also be controlled by adding a heat exchanger to the partial left heart bypass perfusion circuit. During partial left heart bypass with a proximal aortic cross clamp, the distal aortic cross clamp can be advanced sequentially as each segment of the descending aorta is reconstructed to minimize end organ ischemia. Furthermore, systemic anticoagulation and the use of a perfusion circuit permits selective perfusion of mesenteric branch vessels through separate balloon catheters.

**Techniques to increase the spinal cord tolerance to temporary ischemia**

Under normothermic conditions, the brain and central nervous system tolerates ischemia poorly, manifesting neuronal dysfunction and neuronal injury within 5 minutes after the cessation of blood flow. The only intervention in humans that has been proven consistently to be effective for protecting the central nervous system from ischemia in the absence of blood flow is hypothermia. The protective effect of hypothermia is believed to be primarily a consequence of the decreased metabolic demands associated with lower temperatures, but may also protect the cell by stabilizing membranes and attenuating the inflammatory and excitotoxic responses to ischemia during reperfusion.

Mild systemic hypothermia in the range of 32°C to 34°C can be achieved by allowing the nasopharyngeal temperature to decrease gradually from exposure after the induction of general anesthesia. Mild systemic hypothermia is typically employed for spinal cord protection prior to aortic cross clamping for open TAAA repair performed using the “clamp and sew” or partial left heart bypass technique. Re-warming after reperfusion can be accomplished gradually being careful to avoid systemic hyperthermia with a heat exchanger in the perfusion circuit, irrigating the thorax with warmed saline, or a forced-air warming blanket.

Deep or profound systemic hypothermia in the range of 10°C to 18°C requires cardiopulmonary bypass (CPB). Deep hypothermia with temporary circulatory arrest (DHCA) is required for the repair of thoracic or TAAA that extend into the aortic arch requiring the temporary interruption of cerebral blood flow. CPB is typically accomplished by cannulation of the right atrium via the femoral vein and direct cannulation of the aorta or femoral artery. Optimal hypothermic conditions for DHCA include electrocortical silence by electroencephalography, a nasopharyngeal temperature of 12°C to 15°C, or at least 50 minutes of cooling on CPB. In patients with aortic regurgitation, a left ventricular vent via the left upper pulmonary vein may be necessary
to prevent left ventricular distention during hypothermic CPB. The risks associated with DHCA and CPB also include stroke caused by cerebral atheroembolism from retrograde blood flow during CPB in patients with severe disease of the descending thoracic aorta, postoperative encephalopathy, and cerebral hyperthermia during re-warming.

Techniques for producing regional spinal cord hypothermia to as low as 26˚C have been described. The most commonly employed technique is the infusion of 4˚C saline solution into the epidural space via an epidural catheter placed in the T11-T12 vertebral interspace. In this epidural cooling technique, a second, thermistor-tipped catheter is inserted into the subarachnoid space at the L3-L4 vertebral interspace to measure CSF temperature and control CSF pressure by drainage of CSF. Potential risks of epidural cooling include excessive CSF pressure and inability to monitor spinal cord function. Clinical experience with epidural cooling is limited to just a few institutions that routinely use this technique. One series reported that the incidence of postoperative paraplegia after TAAA repair decreased after the introduction of epidural cooling. Regional mesenteric and renal hypothermia can be accomplished by selective perfusion of the mesenteric branch vessels during CPB or partial left heart bypass.

The effectiveness of pharmacologic agents used alone or in combination for neuroprotection to prevent or treat spinal cord ischemia has not been proven. The level of evidence for the efficacy of any pharmacologic neuroprotectant is indeterminate at best. Traditionally, methylprednisolone 1 g i.v. (or 30 mg/kg i.v.), mannitol 12.5g to 25 g i.v., magnesium 1 g to 2 g i.v., lidocaine 100 mg to 200 mg i.v., or thiopental 0.5 g to 1.5 g iv is often administered for spinal cord protection. Small clinical series have also reported favorable results with naloxone 1 mcg/kg/hr iv or intrathecal papaverine at a dose of 30 mg.

*Techniques to augment spinal cord perfusion*

Reports of full or partial recovery from delayed postoperative paraplegia after TEVAR or TAAA repair support the effectiveness of interventions directed at improving spinal cord perfusion for the treatment of spinal cord ischemia. In addition, postoperative events such as hypotension, hemorrhage, or increased CSF pressure that decrease spinal cord perfusion have also been reported to increase the risk of paraplegia after TEVAR and TAAA repair. For these reasons, maintaining spinal cord perfusion by augmenting arterial blood pressure and augmenting cardiac output, together with preventing hypotension, reducing CSF pressure, and reducing central venous pressure is important for the prevention and treatment of spinal cord ischemia.

The physiologic basis for lumbar CSF drainage is that spinal cord perfusion pressure is a function of the mean arterial pressure minus the lumbar CSF pressure. Therefore, increased lumbar CSF pressure has the potential to decrease spinal cord perfusion pressure. Draining CSF by percutaneous insertion of a silastic catheter into the subarachnoid space between lumbar spinal processes has the potential to increase spinal cord perfusion pressure by decreasing the CSF pressure. The technique can be performed prior to operation in high risk patients or after operation in the event of postoperative
spinal cord ischemia. CSF is drained into a sealed reservoir to achieve a lumbar CSF pressure of 10 mm Hg measured by transduce. Two separate meta-analysis on the efficacy of lumbar CSF drainage have been published based on 372 reports that include 3 randomized controlled trials involving 289 patients and 5 cohort studies involving 505 patients. The conclusion from analysis of the pooled data, including an analysis by the Cochrane Collaborative, support the efficacy of lumbar CSF drainage as a component of the multi-modality approach for prevention of neurological injury after TAAA repair. Although the safety of lumbar CSF drainage appears to be acceptable even in patients subjected to full anticoagulation for extracorporeal circulation, complications associated with the technique include subdural hematoma, intraspinal hematoma, remote cerebellar hemorrhage, infection, and even catheter fracture. The most serious complications appear to be associated with intracranial hypotension from rapid drainage of CSF. Precautions such as continuous measurement of CSF pressure, controlled intermittent drainage of CSF, and assessment of coagulation function may decrease the risks associated with lumbar CSF drainage.

Augmenting the arterial pressure alone or in combination with lumbar CSF drainage is another technique for treating spinal cord ischemia. In general, vasopressor agents such as norepinephrine are administered to maintain a mean arterial pressure of 80 mm Hg or greater to ensure a spinal cord perfusion pressure of at least 70 mm Hg. The MAP can be augmented further in increments of 5 mm Hg if spinal cord ischemia persists. During augmentation of the arterial pressure it is also important to assure that cardiac output is satisfactory and that the patient is not anemic in an effort to optimize oxygen delivery. In addition, maintaining a normal or reduced central venous pressure during arterial pressure augmentation may also be important for maximizing spinal cord perfusion pressure. Inconsistent control of the arterial pressure may also explain in part the controversy surrounding the effectiveness of lumbar CSF drainage because decreasing CSF pressure alone without controlling the arterial pressure may limit the ability to improve spinal cord perfusion. Hypotension from bleeding or other causes is often associated with the onset of spinal cord ischemia after TAAA repair, but clinical observations suggest also that spinal cord ischemia may contribute to hypotension as well. In some patients, spinal cord ischemia associated hypotension is caused by neurogenic shock with autonomic dysfunction. In this situation, hypotension may represent an early sign of spinal cord ischemia. Immediate treatment of hypotension associated with spinal cord ischemia is necessary to prevent infarction. Finally, arterial pressure should be monitored carefully when antihypertensive therapy is resumed after successful open TAAA repair or TEVAR to avoid unintentional hypotension that may precipitate spinal cord ischemia. The benefits of arterial pressure augmentation must be weighed against the risk of bleeding and the risks associated with temporary elevation in arterial pressure when implementing this technique in the perioperative period.

Spinal cord perfusion can be augmented surgically by reattachment of intercostals and segmental arteries into the vascular interposition graft during operation. Large segmental arteries with little or no back bleeding may be particularly important for spinal cord perfusion. Alternatively, occlusion or over-sewing of segmental arteries that back-bleed has been advocated to improve spinal cord perfusion by preventing arterial steal.
TEVAR, it is not possible to preserve blood flow in segmental arteries excluded by the stent graft. If the left subclavian artery requires coverage by the endovascular stent graft in order to exclude the aneurysm, subclavian arterial flow can be preserved by prior transposition of the subclavian artery onto the left carotid artery. Another approach to preserving left subclavian artery flow in TEVAR is to perform a left carotid to subclavian bypass graft with subsequent coil embolization of the proximal left subclavian artery stump during TEVAR. Maintaining blood flow in the left subclavian artery may be important for spinal cord perfusion because its branches supply the anterior spinal artery.

**Techniques to detect spinal cord ischemia**

Early detection of spinal cord ischemia is important. Early detection permits early intervention before ischemia evolves to infarction. Difficulty detecting spinal cord ischemia during operation in anesthetized patients may explain in part the poor success in treating patients with immediate postoperative paraplegia after TAAA repair. Successes reported in the treatment of delayed postoperative spinal cord ischemia may be attributed to early diagnosis and immediate interventions to increase spinal cord perfusion. Neurologic assessment of lower extremity motor function can be performed serially as soon as the patient recovers from general anesthesia. For TEVAR, neurologic examination can be performed immediately after operation upon emergence from general anesthesia. Although epidural anesthesia or analgesia can be used for TEVAR or TAAA repairs, it is important to distinguish the effects of central neuroaxial blockade by local anesthetics from spinal cord ischemia. For this reason, some centers choose to avoid the use of regional anesthesia or analgesia with local anesthetics for operations involving the thoracic aorta. Examination should assess strength in the proximal and distal muscle groups of the lower extremities and the presence or absence of sensation. Any neurologic deficit detected should be considered to be spinal cord ischemia until disproved. Delaying the treatment of spinal cord ischemia to obtain imaging studies may decrease the effectiveness of therapeutic interventions.

Detection of spinal cord ischemia during operation in anesthetized patients requires intraoperative monitoring of somatosensory evoked potentials (SSEP) or motor evoked potentials (MEP). The clinical objectives for intraoperative monitoring of spinal cord function during these operations are to ensure adequate spinal cord perfusion throughout the procedure, identify critical vessels for re-implantation, and to establish the mean arterial pressure adequate for spinal cord perfusion. The detection of reversible transient spinal cord ischemic changes by intraoperative monitoring may identify also patients who may be at risk for delayed post-operative paraplegia. Decreased SSEP and MEP amplitude have been shown to correlate with spinal cord ischemia, but the sensitivity and specificity of these techniques for detection of spinal cord ischemia remains to be determined. Intraoperative changes or loss of SSEP or MEP signals are not always caused by spinal cord ischemia. A functioning peripheral nerve is required to generate both SSEP and MEP signals and peripheral nerve ischemia from any cause will affect the associated SSEP or MEP. Vascular malperfusion of a lower extremity can cause loss of peripheral SSEP or MEP in the absence of spinal cord ischemia if blood
flow to the limb is significantly compromised. Malperfusion causes a loss of SSEP or MEP from the ischemic limb. Lower extremity malperfusion may be caused by the aortic dissection itself, atheroembolism, or most commonly from arterial cannulation of the femoral artery for extracorporeal circulation. Similar to malperfusion, operations performed by cross clamping the aorta without distal aortic perfusion will cause SSEP and MEP signals from the lower extremities decay over time after aortic cross clamping. Acute intraoperative stroke will also produce changes in SSEP or MEP. SSEP or MEP changes caused by stroke can be distinguished from changes caused by spinal cord ischemia by comparing signals recorded at different sites along the neural conduction pathway. Stroke is associated with selective loss of cortical signals and typically affects both upper and lower extremity evoked potentials.

Intraoperative monitoring of SSEP is performed by placing stimulating electrodes on the skin adjacent to peripheral nerves in the arms or legs. Electrical stimulation of the peripheral nerves in the limbs generate action potentials that can be measured from recording electrodes over the lumbar plexus, brachial plexus, spine, brainstem, thalamus and cerebral cortex. A potential limitation of SSEP monitoring is that spinal cord ischemia confined to the anterior spinal cord may cause a selective motor deficit with intact sensation. In this situation, SSEP monitoring may fail to detect spinal cord ischemia. An advantage of SSEP monitoring is that it is relatively reliable to perform and easy to interpret by comparing the amplitude and latency of SSEP’s recorded from the upper and lower extremities. The fidelity of SSEP’s are improved with neuromuscular blockade under general anesthesia. Although high concentrations of inhaled anesthetics, thiopental, or propofol can attenuate cortical SEP signals, a balanced general anesthetic with inhaled anesthetics maintained at a concentration of 0.5 MAC provide consistent conditions for monitoring intraoperative SSEP.

Motor evoked potentials (MEP) elicited through transcortical electrical stimulation have also been advocated for the detection of intraoperative spinal cord ischemia. To monitor MEP, myogenic potentials are produced in extremity muscle groups by delivering multipulse, electrical stimulation to the scalp, overlying the motor cortex. The evoked potentials elicited from this stimulation travel from the motor cortex, through cortical spinal tracts, anterior horn cell, peripheral nerve, and finally to muscle. An interruption in this pathway will result in the loss or reduction of amplitude in the MEP. In theory, monitoring MEP should be more sensitive and specific than SEP to detect spinal cord ischemia in the territory supplied by the anterior spinal artery. MEP monitoring has been used to identify critical intercostal arteries for reattachment following the acute loss of lower extremity MEP signals during TAAA repair. Intraoperative MEP monitoring can be challenging because the amplitude of MEP are sensitive to neuromuscular blocking agents and many general anesthetic agents. General anesthetic regimens utilizing intravenous infusions of remifentanil, ketamine, propofol, or etomidate without neuromuscular blockade or carefully controlled incomplete neuromuscular blockade are often required to maintain satisfactory MEP signals during operation.

Conclusion

Advanced contemporary surgical and anesthetic techniques that include TEVAR, partial left heart bypass, deliberate hypothermia, DHCA, reattachment of segmental arteries,
lumbar CSF drainage arterial pressure augmentation, and intraoperative neurophysiologic monitoring have improved the safety of thoracic and thoracoabdominal aortic aneurysm repair. Despite these advances and an improved understanding of spinal cord perfusion, spinal cord ischemia and infarction causing postoperative paraplegia or paraparesis remains an important and debilitating complication of thoracic and thoracoabdominal aortic operations. The seriousness and mortality associated with this complication justifies the routine clinical application of techniques to prevent and treat spinal cord ischemia despite inherent risks associated with some of the techniques and the lack of definitive evidence based on randomized controlled trials. Existing clinical experience supports the efficacy of arterial pressure augmentation and lumbar CSF drainage for the treatment of delayed onset paraplegia caused by spinal cord ischemia when applied immediately at the first appearance of neurologic signs in patients undergoing TEVAR or TAAA repairs.

References


Additional references on Lumbar CSF drainage aand spinal cord protection for TEVAR


