Lumbar CSF Drainage for Spinal Cord Protection

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As disease awareness and diagnostic modalities continue to improve, the prevalence of thoracic aortic disease (aneurysm and dissection) is increasing, affecting up to 16.3 individuals per 100,000 per year. However lifesaving surgery may be, paraplegia remains one of the most devastating complications of thoracoabdominal aortic surgery and is associated with a significant increase in both morbidity and mortality.

Historically, an incidence as high as 40% for this complication has been reported, though more recent reports indicate an incidence of less than 20%

Modern aortic repair techniques employ many modalities aimed at reducing the risk of spinal cord ischemia inherent with surgical management. One of these modalities acting via optimizing spinal cord blood flow is drainage of lumbar cerebrospinal fluid (CSF). Either alone or in combination with other interventions, CSF drainage remains one of the most frequently used spinal cord protection techniques. Despite no definitive proof of efficacy for reducing spinal cord injury, there is compelling data supporting its use. However, the potential benefit of CSF drainage must be balanced against the risks associated with its use, including nerve injury during insertion, compressive neuraxial hematoma formation, intracranial hemorrhage due to excessive drainage, and infection.

Pathophysiology of spinal cord ischemia
Spinal cord injury is thought to result from ischemia (as well as subsequent reperfusion) due to decreased distal aortic perfusion pressure, interruption of segmental arteries supplying the spinal cord, and perioperative hypotension. Risk factors for paraplegia following thoracoabdominal aortic aneurysm (TAAA) repair include emergency presentation (with aortic dissection or rupture), prolonged aortic cross-clamp time, more extensive aneurysms (Crawford type I or II), postoperative hypotension, advanced age, previous abdominal aortic aneurysm (AAA) repair, severe atherosclerotic disease, diabetes, and ligation of spinal collateral vessels.

The spinal cord depends on a single longitudinal anterior spinal artery and two, more plexiform, posterolateral spinal arteries for blood flow, all originating from the vertebral arteries. The anterior and posterior spinal arteries receive segmental contribution from radicular (intercostal) arteries for their blood supply; the largest of these is the artery of Adamkiewicz. This large intercostal artery has a variable origin, but generally originates from T8-L1 in the majority of patients. Intraoperative ischemia of the spinal cord is thought to be, in part, related to interruption of blood flow through these intercostal arteries consequent with cross-clamping of the aorta as well as with surgical ligation during aneurysm resection. However, it is unclear as to the significance of any single segment of blood vessel to the integrity of spinal cord blood flow. Indeed, Griep et al suggested that spinal cord blood flow is unlikely to depend on a single artery of Adamkiewicz.

There are significant hemodynamic changes that occur with aortic cross-clamping that dynamically interact with cerebrospinal fluid hydrodynamics. Marked increases in the proximal
aortic pressure, central venous pressure (CVP), and CSF pressure (CSFP) are seen with concomitant decreases in blood flow in areas distal to the cross-clamp.\textsuperscript{6,8} The result of the hemodynamic changes associated with thoracic aortic cross-clamping is a decrease in spinal cord perfusion pressure (SCPP) in the spinal cord that receives its blood supply by vessels distal to the clamp site, according to the formula:\textsuperscript{8}

\[
SCPP = MAP_d - \text{[CSFP or CVP]} \ast
\]

Where: SCPP is spinal cord perfusion pressure
MAP\textsubscript{d} is distal mean aortic pressure
CSFP is cerebrospinal fluid pressure
CVP is central venous pressure
\ast which ever is greater

**Spinal cord protection**

The techniques utilized for spinal cord protection in this clinical setting are principally directed at either reducing spinal cord metabolism, increasing distal aortic pressure (through various bypass techniques), or controlling the neuraxial outflow pressure (i.e. CSF or CVP).\textsuperscript{25} Given the interruption of collateral blood vessel supply during the period of aortic cross-clamping, thoracic spinal cord blood flow may not consistently be augmented with distal bypass techniques. Thus, the modulation of SCPP through the control of CSFP may be critical in the prevention of spinal cord ischemia during this time period.\textsuperscript{8} Following release of the cross-clamp, the spinal cord is at further risk for ischemia secondary to hypercarbia (that can increase CSFP) and hypotension which can result in decreases in SCPP.\textsuperscript{6,8} The metabolic acidosis following the release of the cross-clamp causes an increase in cerebral blood flow, resulting in elevations in intracranial pressure and CSFP. Anaerobic metabolites are also responsible for a decrease in systemic vascular resistance with often profound hypotension. Furthermore, spinal cord edema as a result of reperfusion injury can also increase CSFP.\textsuperscript{12}

Two extensive reviews have emphasized the relative paucity of human randomized controlled trials examining lumbar CSF drainage in TAAA surgery.\textsuperscript{22,26} They both concluded that this treatment must be more extensively studied and that CSF drainage alone, without other spinal cord protection adjuncts, may have limited benefit. Despite the limitations of past studies, there is continued interest in utilizing CSF drainage for spinal cord protection. Evidence does suggest that CSF drainage is an effective rescue maneuver for patients that develop delayed-onset paraplegia.\textsuperscript{3} Furthermore, there has been renewed interest in CSF drainage in studies that have demonstrated improved measures of functional spinal cord integrity as measured by recovery of evoked potentials.\textsuperscript{25}

The largest and most recent study by Coselli et al\textsuperscript{27} was a randomized controlled trial of 145 patients undergoing extent I or II TAAA repairs. Standard techniques of mild hypothermia, left heart bypass, and reattachment of intercostal arteries were used in all groups. The interventional group in the study utilized lumbar CSF drainage to maintain a CSFP less than 10 mmHg. The authors were able to demonstrate an 80% reduction in the incidence of postoperative neurologic deficits (13.0% vs 2.6%, p=0.03), albeit with no difference in mortality between groups (p=0.68). Safi et al performed a retrospective analysis of 1004 TAAA repairs performed between 1991 and 2003.\textsuperscript{28} Cerebrospinal fluid drainage in combination with distal aortic perfusion was employed in 741 patients. Their analysis demonstrated that when this combined approach was used, a significant decrease in postoperative neurologic deficits was detected, along with an increased long-term survival rate. Based on their data, it was estimated
that the number needed to treat to reduce one neurologic deficit was 5 for type II aneurysms and 20 for less extensive aneurysms.

The most recent meta-analysis by Cina et al. \textsuperscript{11} concluded that CSF drainage may be a useful adjunct in the prevention of paraplegia in Type I and II aneurysms in experienced centers. \textsuperscript{11} This report emphasized the benefits of a large volume surgical practice on outcome. \textsuperscript{29} A pooling of 3 randomized controlled trials examining 289 patients showed a significant decrease in the incidence of postoperative paraplegia when CSF drainage was used (number needed to treat 9, 95\% CI 5-50; odds ratio 0.35, 95\% CI 0.12-0.99). When these data were combined with cohort studies, the odds ratio for paraplegia post TAAA surgery with CSF drainage for spinal cord protection became 0.3 (95\% CI 0.17-0.54). \textsuperscript{11}

**TEVAR**

Endovascular repair (TEVAR) is an increasingly employed technique for the treatment of TAAA. This method minimizes the hemodynamic consequences of clamping and unclamping the aorta, obviates the need for CPB, large thoracotomy incisions, and one-lung ventilation, and as a result can significantly reduce morbidity and mortality compared with open repair. \textsuperscript{30,31} Although TEVAR has significantly decreased the overall incidence of neurologic complications with TAAA repair, \textsuperscript{30,32,33} the risk of paraplegia with TEVAR is reported to be as high as 8\%. \textsuperscript{30,31,33,34} The precise pathophysiology of spinal cord injury with TEVAR is not clear but may be related to disruption of radicular artery blood flow to the spinal cord through occlusion of segmental collateral vessels by the endovascular graft or through disruption of other collaterals from the pelvic, lumbar, and hypogastric vessels.\textsuperscript{35-37} Consistent with hypoperfusion as a primary etiology of spinal cord injury are data suggesting that collateral perfusion is of particular importance in the cause of spinal cord injury during TEVAR. Indeed, an increased incidence of paraplegia following TEVAR was seen in patients with previous AAA repair, prolonged hypotension, severe atherosclerosis of the thoracic aorta, injury to the external iliac artery, occlusion of the left subclavian artery or hypogastric arteries, and more extensive coverage of the thoracic aorta by the graft, all of which may impede collateral blood flow. \textsuperscript{30,31,33,36,38,39}

Promising data from Hnath et al. in a prospective observational study utilizing historical controls outlined a significantly decreased incidence of postoperative spinal cord injury with TEVAR when CSF drainage was utilized. \textsuperscript{40} They studied 121 patients undergoing elective or emergent endovascular thoracic aortic stent graft placement. None of the patients in the CSF drainage group had spinal cord injury, whereas 5 (8\%) of the individuals without spinal fluid drainage developed neurologic deficit within 24 hours of their procedure (p<0.05). They were able to demonstrate a significant benefit despite the CSF drainage group having a larger number of patients with previous AAA repair, more extensive aneurysm coverage, and more frequent left subclavian artery coverage, all of these contributing to a higher risk for spinal cord injury.

**CSF drainage complications**

Complications of lumbar CSF drainage include those related to the lumbar puncture, the presence of an indwelling catheter, and the drainage of CSF. Direct spinal cord or nerve root injuries from needle placement or subsequent neuraxial hematoma have been reported. \textsuperscript{41-44} Symptomatic intracranial hypotension presenting as headache, abducens nerve palsy, as well as intracranial hemorrhage (ICH). Infection due to the presence of the catheter can lead to local infection or meningitis. Catheter fracture can occur during removal causing local irritation or infection. Intracranial hemorrhage (IH) following CSF drainage may be the most devastating of
these complications. Draining too large a volume of CSF over a short period of time is a documented risk factor for a subdural ICH. In general, 10 mL/hr has been recommended, in the absence of any paraparesis, when more aggressive (up to 20 mL/hr) drainage may be necessary. The mechanism relates to intracranial hypotension which, in turn, causes stretching and tearing of bridging dural veins.

Wynn et al published a retrospective analysis of 486 lumbar drains placed between 1987 and 2008. Symptomatic ICH following spinal drainage was seen in 1% of patients (5 out of 482), with a 2.9% incidence of asymptomatic intracranial bleed. They showed an overall mortality with spinal drain complications of 0.6% but the patients with symptomatic ICH had a very high mortality with death in 3 out of 5 patients as a result. Estrera et al, in the largest series of lumbar CSF drains reported (n=1,107), observed a 40% mortality rate when ICH occurred. Presence of blood in the draining CSF has been thought to be a sensitive indicator of ICH even without neurologic symptoms. Therefore, if blood is detected in the CSF, urgent imaging of the brain should be considered.

Neuraxial hematoma can be a disastrous complication of lumbar drain placement. Although the risk for this catastrophic injury appears to be relatively low, spinal cord injury as a direct complication of lumbar CSF drainage itself may resemble the signs of spinal cord injury secondary to ischemia resulting in a delay of diagnosis. One retrospective study of 65 patients undergoing TAAA repair found intraspinal hemorrhagic complications in 2 of their patients (3.2%). A database review of 162 CSF drains in patients undergoing TAAA repair (with concomitant partial left heart bypass and systemic anticoagulation) revealed no intraspinal hemorrhagic complications. Nonetheless, a high index of suspicion for neuraxial hematoma must be maintained when any patient with a lumbar drain presents with lower extremity neurologic deficit. The treatment of this injury is surgical and delay in therapy may result in permanent injury. One study suggests reserving imaging to the population of patients that do not respond to prompt CSF drainage and blood pressure augmentation, considering that spinal cord ischemia is far more common than neuraxial hematoma formation.

Central to the increased risk of neuraxial hematoma is the systemic anticoagulation that is usually employed with these procedures. The relatively large needle (usually a 14 gauge Tuohy) used to place the CSF drainage catheter may also increase the bleeding risk. The American Society of Regional Anesthesia and Pain Medicine (ASRA) guidelines for neuraxial anesthesia in the anticoagulated patient recommend that instrumentation of the neuraxis be avoided in patients with preexisting coagulopathy, that the time from the procedure and anticoagulation should exceed 60 minutes. Although Cheung et al reported no spinal hematomas in their case series of 162 patients having lumbar drain placement followed by extracorporeal circulation (with systemic anticoagulation), when a traumatic or bloody tap is encountered, it raises several management issues. Traumatic instrumentation of the neuraxis has been described as a risk factor in up to 50% of neuraxial hematomas. One review has suggested delaying the case for 24 hours in the event of a bloody tap when subsequent anticoagulation for CPB is planned. However this has not been studied extensively, nor has the safe length of waiting time needed prior to anticoagulation been investigated. Some centers have elected to place the drains 24 hours preoperatively to avoid this situation. However this may place the patient at increased risk of infection, and it requires that the patient be hospitalized preoperatively which increases cost. It may be reasonable to continue with surgery if a bloody tap occurs during same day lumbar drain placement as the length of time between catheter placement and administration of
anticoagulation can be several hours. If postoperative neurologic deficits are detected in this situation, more urgent imaging of the spine should be considered to rule out hematoma.

Other complications of lumbar drain placement such as infection and retained catheters are exceedingly rare. The database analysis by Cheung et al. reported retained catheter fragments in 3 of 162 patients (1.8%), one of whom subsequently developed meningitis. It was suggested that in order to minimize catheter fracture risk, that removal of the catheter should be done by a practitioner familiar with the catheters (and their intrinsic tensile strength) and that the patient should be positioned in the lateral decubitus position with both hips and back flexed. This maneuver increases the space between the vertebrae and spinous processes thereby preventing catheter entrapment which could result in an increased amount of force required to remove the catheter and subsequent fracture.

References