CME

Lumbar Cerebrospinal Fluid Drainage for Thoracoabdominal Aortic Surgery: Rationale and Practical Considerations for Management

Christine A. Fedorow, MD,* Michael C. Moon, MD, FRCPC,† W. Alan C. Mutch, MD, FRCPC,* and Hilary P. Grocott, MD, FRCPC*†

Paraplegia remains one of the most devastating complications of thoracoabdominal aortic surgery and is associated with a significant increase in both morbidity and mortality. Modern aortic repair techniques use many modalities aimed at reducing the risk of spinal cord ischemia inherent with surgical management. One of these modalities that acts via optimizing spinal cord blood flow is lumbar cerebrospinal fluid (CSF) drainage. 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 are 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. The optimal benefit to risk ratio can be achieved by understanding the rationale for its use and following practical management guidelines. (Anesth Analg 2010;111:46–58)

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.[1,2] 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.[3–5] Historically, an incidence as high as 40% for this complication has been reported,[5–8] although more recent reports indicate an incidence of <20%.[3,9,10] However, it has been suggested that the incidence of paraplegia after thoracic aortic surgery may be increasing because of the expanding complexity of procedures and numbers of medical comorbidities in the patient population undergoing repair.[5]

Modern aortic repair techniques use many modalities aimed at reducing the risk of spinal cord ischemia inherent with surgical management. Distal aortic perfusion using various extracorporeal bypass techniques, intraoperative monitoring with somatosensory and motor evoked potentials, meticulous reimplantation of intercostal blood vessels, mild systemic hypothermia, epidural cooling, deep hypothermic circulatory arrest, and various pharmacologic interventions have all been used. Lumbar cerebrospinal fluid (CSF) drainage, either alone or in combination with other interventions, remains one of the most frequently used techniques.[5,7,11–14] The purpose of this report is to outline the background and rationale for the use of lumbar CSF drainage.

From the Departments of *Anesthesia and †Surgery, University of Manitoba, Winnipeg, Manitoba, Canada.

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Address correspondence and reprint requests to Hilary P. Grocott, MD, FRCPC, Departments of Anesthesia, University of Manitoba, I.H. Asper Institute for Clinical Research, CR3008-369 Tache Ave., Winnipeg, MB, Canada R2H 2A6. Address e-mail to hgrocott@sbgh.mb.ca.

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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 and/or thrombosis of segmental arteries supplying the spinal cord, and perioperative hypotension.[7,11,15–18] Risk factors for paraplegia after 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; Fig. 1), postoperative hypotension, advanced age, previous abdominal aortic aneurysm (AAA) repair, severe atherosclerotic disease, diabetes, and ligation of spinal collateral vessels (Table 1).[4,7,9,16,19–22]

The spinal cord depends on a single longitudinal anterior spinal artery and 2, more plexiform, posterolateral spinal arteries for blood flow, all originating from the vertebral arteries (Fig. 2).[7,23,24] 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, originating from the lower thoracic aorta in the majority of people. This large intercostal artery has a variable origin, but generally originates from T8-L1 in the majority of patients (Fig. 3).[19,25,26] 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 and with surgical ligation during aneurysm resection. However, it is unclear as to the significance of any single segmental blood vessel to the integrity of spinal cord blood flow. Indeed, Griepp et al.[27] suggested that spinal cord

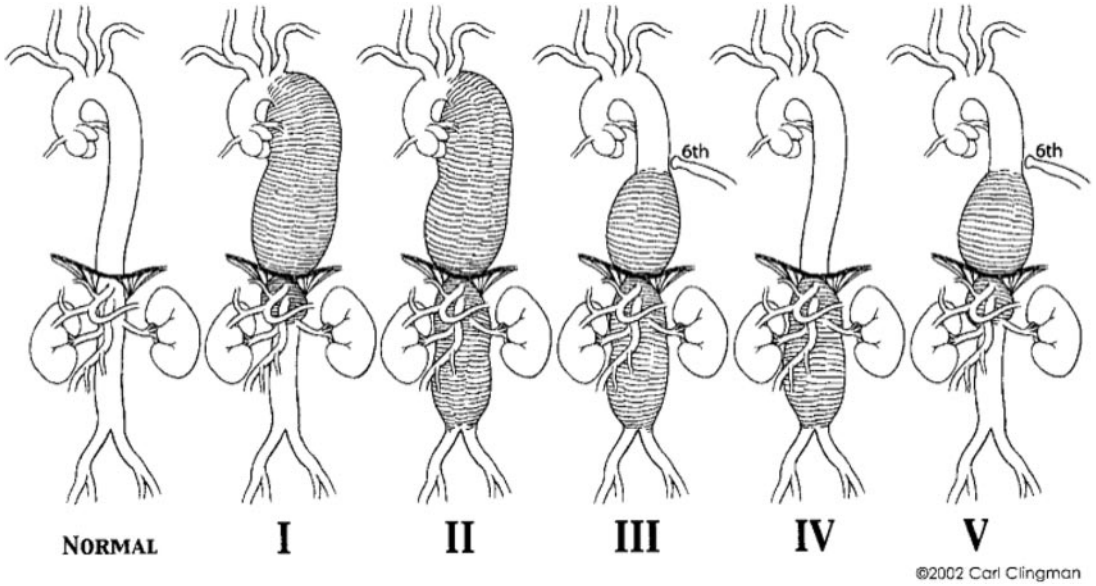

Figure 1. Crawford classification of aortic aneurysm. Extent I, from just distal to the left subclavian artery to above the renal arteries. Extent II, from just distal to the left subclavian artery to below the renal arteries. Extent III, from the sixth intercostal space to below the renal arteries. Extent IV, from the 12th intercostal space to below the renal arteries (total abdominal aortic aneurysm). Extent V, below the sixth intercostal space to just above the renal arteries. (From Crawford et al.,[89] with permission.)

Table 1. Risk Factors for Paraplegia After TAAA

Risk factors	Mechanism of injury
Risk factors for paraplegia after open TAAA repair	
Emergency presentation (aortic dissection or rupture)[7,20]	Decreased perfusion pressure
Postoperative hypotension[17]	
More extensive aneurysms (Crawford type I or II)[4,7,9,20]	Acute disruption of collateral circulation
Ligation of spinal collateral vessels[10,27]	
Prolonged aortic cross-clamp time[7]	Worse ischemia-reperfusion injury
Previous abdominal aortic aneurysm repair[21]	Decreased collateral circulation
Severe atherosclerotic disease[10,22]	
Diabetes[4]	More medical comorbidities
Advanced age[4]	
Risk factors for paraplegia after endovascular TAAA repair (TEVAR)	
Previous abdominal aortic aneurysm repair[19,42,44,51]	Decreased collateral circulation
Severe atherosclerosis of the thoracic aorta[19]	
Hypotension[19,47]	Decreased perfusion pressure
Injury to the external iliac artery[19,47]	Acute disruption of collateral circulation
Occlusion of the left subclavian artery or hypogastric arteries[42,43]	
More extensive coverage of the thoracic aorta by graft[44,48,51]	

TAAA = thoracoabdominal aortic aneurysm; TEVAR = thoracic endovascular aortic repair.

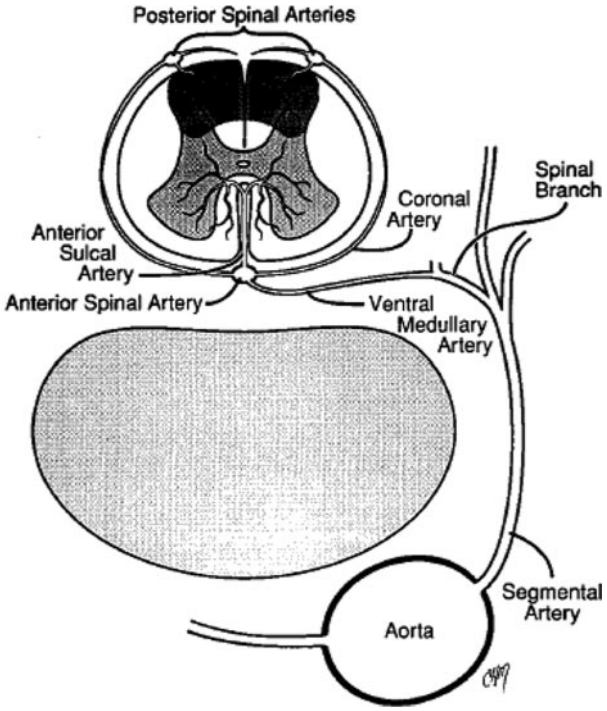

Figure 2. Intrinsic and extrinsic blood supply of the spinal cord. (From Plecha et al.,[24] with permission.)

blood flow is unlikely to depend on a single artery of Adamkiewicz. In addition to the more direct contributions to spinal cord blood flow, 1 study demonstrated (through the use of somatosensory evoked potentials) that in patients with significant thoracic aortic disease, spinal cord integrity may also be maintained by an extensive network of collateral vessels, including contributions from lumbar arteries and the pelvic circulation.[10]

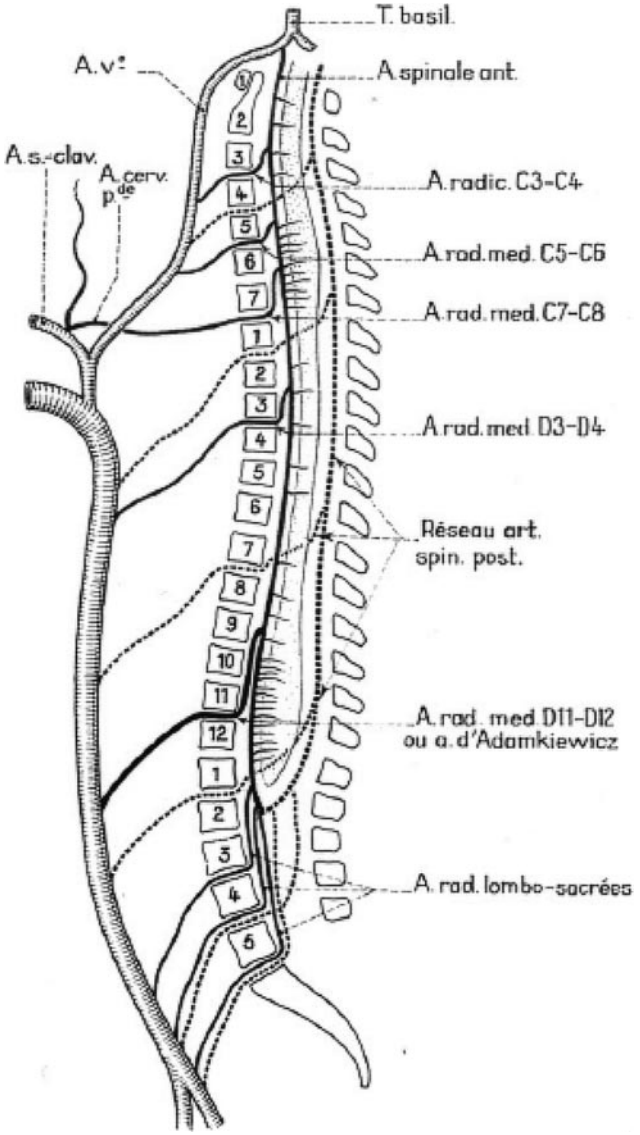

Figure 3. Spinal cord blood supply. Note radicular arteries supplying both the anterior and posterior spinal arteries. Also note the large distal radicular artery and Adamkiewicz joining the anterior spinal artery between T9 and T11. (From Djindjian,[26] reprinted with permission from the American Journal of Roentgenology.)

Consequently, in these patients whose radicular vessels may already be significantly diseased, spinal cord ischemia may be less related to interruption of radicular artery blood flow during surgery, and more related to decreases in collateral perfusion as a result of the hemodynamic responses to aortic cross-clamping. Collateral perfusion can also be reduced if excessive reductions in proximal pressure occur.

There are significant hemodynamic changes that occur with aortic cross-clamping that dynamically interact with CSF 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.[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:[8]

$$SCPP = MAP_d - (CSFP \text{ or } CVP \text{ [whichever is greater]})$$

where SCPP = spinal cord perfusion pressure; MAP_d = distal mean aortic pressure; CSFP = cerebrospinal fluid pressure; and CVP = central venous pressure.

The techniques used for spinal cord protection in this clinical setting are principally directed at reducing spinal cord metabolism, increasing distal aortic pressure (through various bypass techniques), or controlling the neuraxial outflow pressure (i.e., CSF or CVP).[28] 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. The modulation of SCPP through the control of CSFP may be critical in the prevention of spinal cord ischemia during this time period.[8] After 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.[6,8] The metabolic acidosis after the release of the cross-clamp causes an increase in cerebral blood flow, resulting in increases in intracranial pressure (ICP) 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.[15]

BENEFITS OF LUMBAR CSF DRAINAGE

Based on the concept that decreasing CSFP will increase SCPP, experimental studies of spinal cord injury induced by aortic cross-clamping have shown promising results with the use of CSF drainage to reduce the incidence of paraplegia.[29–32] Dasmahapatra et al.,[31] using a dog model of spinal cord ischemia induced by aortic cross-clamping, demonstrated a significant relationship between CSF drainage and reductions in the degree of spinal cord ischemia. Bower et al.[30] measured gray matter spinal cord blood flow in a similar model. They demonstrated both increased blood flow in animals having CSF drainage during thoracic aortic occlusion and less reperfusion hyperemia. This was also associated with improved functional outcomes.

The clinical evidence, however, is somewhat mixed with the interpretation of the available clinical studies requiring consideration of the nuances of overall study size and design (Table 2).[11,18,25,33–38] Some studies did not control CSFP to a sufficient degree to demonstrate significant effect, or may have been inadequately powered to detect a decrease in the incidence of paraplegia. For example, one small randomized controlled trial (n = 98) of TAAA repair conducted by Crawford et al.[39] failed to show any benefit of CSF drainage for improving neurologic outcomes. Although this study was well designed, the volume of CSF drained intraoperatively was limited to 50 mL, possibly an inadequate volume to have a meaningful treatment effect in most patients. In addition, the CSF drainage was discontinued at the end of the operation and CSFP was neither measured nor

Table 2. Benefits of Lumbar CSF Drainage in Open and Endovascular Thoracoabdominal Aortic Aneurysm Repairs

Study type and reference	No. of patients	Surgery type	Study design	Other adjuncts for spinal cord protection	Results (paraplegia incidence)	*P* value
RCT						
Crawford et al.,[39] 1991	98	High risk open TAAA (Crawford I and II)	<50 mL CSF, no postoperative drainage	±DAP, ±reattachment intercostal/lumbar arteries	30% (14 of 46) CSF drainage vs 33% (17 of 52) control	0.8
Svensson et al.,[35] 1998	33	High risk type I and II TAAA	CSFP <10 mm Hg	IP, ±DAP, ±reattachment intercostal/lumbar arteries, ±active cooling	11.7% (2 of 17) CSF drainage + IP vs 43.8% (7 of 16) control	0.0392
Coselli et al.,[40] 2002	145	Type I or II TAAA repair	CSFP <10 mm Hg up to 48 h postoperatively	Moderate heparinization, DAP, mild permissive hypothermia, reattachment intercostal/lumbar arteries	2.6% (2 of 82) CSF drainage vs 13% (9 of 74) control	0.03
NRHC						
Svensson et al.[33] 1988	19	TAA or TAAA surgery	CSFP 5–15 mm Hg	IP, ±DAP	9% (1 of 11) CSF drainage + intrathecal papaverine vs 42.1% (8 of 19) control	0.058
Acher et al.,[84] 1990	47	TAA and TAAA	CSFP <14 mm Hg	Naloxone	4% (1 of 23) CSF drainage IV naloxone vs 29% (7 of 24) no CSF drainage or naloxone	<0.03
Hollier et al.,[36] 1992	150	TAAA replacement	CSFP <10 mm Hg	Avoid glucose solutions, passive hypothermia, STP, mannitol, nimodipine, reattachment intercostal arteries	0% (0 of 42) intervention vs 6% (6 of 108) control	<0.01
Murray et al.,[37] 1993	99	Descending thoracic and TAAA repair	CSFP 15 mm Hg	±DAP	8.5% (4 of 47) CSF drainage vs 8.9% (4 of 45) no CSF drainage	NS
Acher et al.,[85] 1994	110	Thoracic and TAAA (acute or Crawford I or II)	CSFP <10 mm Hg	Naloxone, moderate hypothermia	1.6% (1 of 61) CSF drainage + IV naloxone vs 22.4% (11 of 49) control	0.001
Safi et al.,[38] 1998	271	Descending thoracic and TAAA	CSFP <10 mm Hg continued for 3–4 d postoperatively	±DAP, moderate permissive hypothermia	4.4% (7 of 159) CSF drainage + DAP vs 14.2% (16 of 112) no adjunct	0.004
Acher et al.,[20] 1998	217	Thoracic and TAAA	CSFP <10 mm Hg	Naloxone, moderate hypothermia	3.4% (5 of 147) CSF drainage + IV naloxone vs 21% (12 of 58) control	<0.001
Safi et al.,[14] 2003	1004	Descending thoracic and TAAA repairs	CSFP ≤10 mm Hg up to 3 d postoperatively	SSEP, DAP, reattachment intercostal/lumbar arteries, mild hypothermia	2.4% (18 of 741) CSF drainage + DAP vs 6.8% (18 of 263) no adjunct	<0.0009
Leyvi et al.,[13] 2005	91	Descending thoracic and TAAA	CSFP <10 mm Hg	DAP, mild permissive hypothermia	5.5% (3 of 54) CSF drainage vs 0% (0 of 37) control	NR
Estrera et al.,[21] 2005	300	Descending thoracic aortic aneurysms	CSFP <10 mm Hg continued for 3 d postoperatively	±DAP, ±reattachment intercostal arteries	1.3% (3 of 238) CSF drainage + DAP vs 6.5% (4 of 62) no adjunct or single adjunct only	<0.02

(*Continued*)

Table 2. (*Continued*)

Study type and reference	No. of patients	Surgery type	Study design	Other adjuncts for spinal cord protection	Results (paraplegia incidence)	*P* value
OCS						
Cheung et al.,[3] 2002	99	Crawford I, II, III delayed onset paraplegia	OCS, CSFP <12 mm Hg in OR	DAP, moderate hypothermia	Delayed onset paraplegia in 8 patients, 5 responded to BP augmentation and CSFP <10 mm Hg	NR
Cheung et al.,[19] 2005	75	TEVAR	PCS, CSFP <12 mm Hg	MAP 75–85 mm Hg, SSEP	Spinal cord ischemia in 5 patients, 3 responded (2 complete, 1 partial) to BP + CSF drainage	NR
Weigang et al.,[28] 2006	31	TEVAR	OCS, CSFP <15 mm Hg	SSEP	Decreased SSEP in 11 patients, 10 responded to CSF drainage and BP/CVP control	NR
NRHS						
Hnath et al.,[53] 2008	121	TEVAR	NRHC, CSFP <15 mm Hg	MAP ≥90 mm Hg	0% (0 of 56) CSF drainage vs 8% (5 of 65) control	<0.05

TAAA = thoracoabdominal aortic aneurysm; CSF = cerebrospinal fluid; IP = intrathecal papaverine; DAP = distal aortic perfusion; SSEP = somatosensory evoked potentials; STP = sodium thiopental; RCT = randomized controlled trial; NRHC = nonrandomized historical cohort; OCS = observational cohort study; TEVAR = thoracic endovascular aortic repair; PCS = prospective cohort study; NS = not significant; NR = not reported; TAA = thoracic aortic aneurysm; CSFP = CSF pressure; BP = arterial blood pressure; CVP = central venous pressure; MAP = mean arterial blood pressure.

controlled postoperatively, eliminating any potential therapeutic effect to patients who may have presented with delayed-onset paraplegia.[3] Removal of CSF in an attempt to decrease CSFP may not improve SCPP if the CVP is unchanged or increases with cross-clamping,[8] thus limiting neuraxial outflow pressure. Lumbar CSF drainage is probably less effective in reversing a neurologic deficit secondary to thrombosis or embolic phenomena in patients with severe atherosclerosis or intraoperative disruption of blood supply,[19] a factor not explored in most studies.

Two extensive reviews have emphasized the relative paucity of human randomized controlled trials examining lumbar CSF drainage in TAAA surgery.[25,34] 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 using CSF drainage for spinal cord protection. Evidence does suggest that CSF drainage is an effective rescue maneuver for patients who develop delayed-onset paraplegia.[3] Furthermore, there has been renewed interest in CSF drainage because of studies that have demonstrated improved measures of functional spinal cord integrity as measured by recovery of evoked potentials.[28]

The largest and most recent study, by Coselli et al.,[40] 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. Lumbar CSF drainage was used in the study on the interventional group to maintain a CSFP of <10 mm Hg. 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.[14] performed a retrospective analysis of 1004 TAAA repairs performed between 1991 and 2003. CSF drainage in combination with distal aortic perfusion was used 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 1 neurologic deficit was 5 for type II aneurysms and 20 for less-extensive aneurysms. The most recent meta-analysis by Cina et al.[11] concluded that CSF drainage may be a useful adjunct in the prevention of paraplegia in type I and II aneurysms at centers with experienced personnel.[11] This report emphasized the benefits of a large-volume surgical practice on outcome.[41] 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% confidence interval [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 after TAAA surgery with CSF drainage for spinal cord protection became 0.3 (95% CI 0.17–0.54).[11]

USE OF LUMBAR CSF DRAINAGE IN ENDOVASCULAR REPAIR OF THORACIC AORTIC ANEURYSMS

Endovascular repair is a technique increasingly used for the treatment of TAAA. This method minimizes the hemodynamic consequences of clamping and unclamping the aorta, obviates the need for cardiopulmonary bypass, large thoracotomy incisions, and one-lung ventilation, and as a result can significantly reduce short-term morbidity and mortality compared with open repair.[42,43] Although thoracic endovascular aortic repair (TEVAR) has significantly

decreased the overall incidence of neurologic complications with TAAA repair,[42,44,45] the risk of paraplegia with TEVAR is reported to be as high as 8%.[42,43,45,46] 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. Most of the literature is in agreement that decreased spinal cord perfusion during TEVAR is likely multifactorial in etiology.[47–49] One study demonstrated angiographically patent radicular arteries despite the development of spinal cord injury after TEVAR, suggesting that perfusion pressure may be critical to maintaining flow through these anatomically preserved segmental vessels.[47]

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 after 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 (Table 1).[42,43,46,48,50,51] Feezor et al.[48] performed a retrospective analysis of 326 TEVAR cases and found that patients with spinal cord injury had a greater length of aortic graft coverage and less native aorta present proximal to the celiac artery after the procedure. On balance, the above commentary supports the contention that where an extensive repair is planned, consideration should be given to methods for spinal cord protection including CSF drainage. This factor is highlighted again by evidence that patients with previous AAA repair may have decreased collateral circulation to the spinal cord, which may put them at increased risk for postoperative paraplegia in both open TAAA repair and TEVAR.[51,52] Schlösser et al.[52] reviewed 72 patients undergoing TEVAR after previous AAA repair, demonstrating a relative risk of 7.2 (95% CI 2.6–19.6, $P < 0.0001$) for developing postoperative spinal cord injury in patients who had previous abdominal aortic surgery.

Lumbar CSF drainage has been studied as a method for spinal cord protection during TEVAR procedures (Table 2).[19,28,53] Promising data from Hnath et al.[53] in a prospective observational study using historical controls outlined a significantly decreased incidence of postoperative spinal cord injury with TEVAR when CSF drainage was used. 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 that the CSF drainage group had more patients with previous AAA repair, more extensive aneurysm coverage, and more frequent left subclavian artery coverage, all of which contribute to a higher risk for spinal cord injury. Although this study used a target CSFP <15 mm Hg (higher than what was seen in beneficial studies in open surgery and animal models), SCPP was more than sufficient as these patients also had mean arterial blood pressure targets >90 mm Hg. Furthermore, this study reconfirmed, through post hoc analysis, that the benefit of CSF drainage was greatest in patients at highest risk for injury (i.e., previous AAA repair, more extensive aneurysms, and increased coverage of collateral vessels such as the subclavian, hypogastric, and iliac vessels).[53]

RESCUE THERAPY FOR DELAYED PARAPLEGIA

Although paraplegia may manifest immediately upon recovery from anesthesia, delayed-onset paraplegia is reported both in open[54,55] as well as TEVAR cases.[56–58] In cases in which CSF drainage was not initially used, or had been used but subsequently discontinued, institution of drainage resulted in reversal of the neurologic deficit.[3,16,59,60] This drainage is usually coupled with arterial blood pressure augmentation to maximally benefit SCPP.[3] The delayed-onset paraplegia is most likely an indication that the blood supply to the spinal cord has been compromised, although not irreversibly, and that collateral blood flow, which itself is pressure dependent, is now critical to cord survival. In addition, cord edema (with consequent increases in CSFP) due to ischemia-reperfusion injury[15] may also lead to localized impairment in blood supply. Postoperative hemodynamic instability, thrombosis, embolization, and hematoma formation may all have a role as well. The development of delayed-onset paraplegia should be considered an emergency situation and immediate intervention should be initiated to prevent the ischemic spinal cord from becoming an irreversibly infarcted spinal cord.

INSERTION OF THE LUMBAR CSF DRAIN

The placement of a lumbar CSF drain should ideally be performed in the awake patient, although this policy is very dependent on institutional preferences.[61] This allows for patient feedback in the form of paresthesias or pain, and may serve to minimize the potential for nerve injury related to needle insertion or catheter placement. The patient can be positioned in the lateral decubitus or sitting position; both positions offer certain advantages.[62] Positioning the patient in the lateral position decreases the hydrostatic pressure of the column of CSF, thereby minimizing the amount of inadvertent CSF drained from the relatively large bore needles used to puncture the dura. However, the sitting position may allow for easier location of midline structures, improved ability to position the patient in lumbar flexion, and potentially a decreased risk for bloody tap. The epidural venous plexus is present in the paramedian position, so if the insertion needle drifts from the midline, there may be a higher risk of venous puncture. The lower limit of spinal cord extension should be considered when determining the level of insertion. Ideally, an L4-5 or L3-4 intervertebral space should be closer (i.e., approximately at the level of the iliac crest).[63]

Once the dura has been punctured with the introducer needle, a multiorificed, silastic drainage catheter is inserted. Common methods describe inserting 8 to 10 cm[61,64,65] of

the drain (beyond the tip of the insertion needle) into the subarachnoid space but the insertion of >20 cm has also been described.[28,66] There may be some advantage to inserting the catheter longer distances, because this minimizes the chance of the catheter inadvertently pulling out with patient movement; however, this may put the patient at increased risk for paresthesia or nerve root injury. If a paresthesia is encountered during catheter insertion, the catheter should be slightly withdrawn until the paresthesia subsides and then secured.

After the drain is secured to the patient, it can be connected (using a strict aseptic technique) to the pressure transducer and commercially available drainage bag, and baseline measurements can then be made. There are 2 options for priming the pressure transducer with fluid: with the patient's own CSF, which can be used to prime the system in a retrograde manner, or sterile preservative-free saline before attaching it to the patient's catheter. Both methods minimize the risk of introducing bacteria into the system that could present an extrinsic source for infection. Care should be taken to ensure that the system is not attached to a pressurized flush system typically used with other invasive pressure transducers; in particular, no heparin should be in the fluid priming the transducer system.

WHERE TO ZERO THE TRANSDUCER—THE RATIONALE FOR THE PHLEBOSTATIC AXIS

There are several reasons to support zeroing the lumbar drain pressure transducer used to measure CSFP at the level of the right atrium (i.e., the phlebostatic axis). Although the tragus has been used as a zeroing point when measuring ICP and ICP is often equated with CSFP, the tragus may not be the optimal zeroing point for CSFP as it relates to the spinal cord. The use of the tragus to zero the CSFP, as it pertains to the thoracic and lumbar spinal cord, may result in a lower CSFP reading than that measured using the phlebostatic axis. This is particularly pertinent in the postoperative patient who is receiving care in a 15° to 20° head-up position where the tragus is above the level of the thoracic and lumbar spinal cord. As a result, the SCPP[19] would be calculated to be higher than if the CSFP was zeroed to the phlebostatic axis. This may result in less CSF drainage being instituted than would be used if a phlebostatic axis zeroing point was used. One might also consider that the region of the spinal cord at risk for ischemia is itself approximately at the level of the atrium.

A potential alternative zeroing site that has been suggested is the lumbar insertion site itself. This would result in a higher than expected CSFP compared with that of the phlebostatic axis or the tragus (because its position is lower than the tragus in a head-up position) and an underestimation of the true SCPP. Consequently, the higher CSFP at the lumbar insertion site would result in excessive CSF drainage to target a predetermined SCPP. This excessive CSF drainage could increase the risk of subdural hematoma in patients.[66–68]

CONTINUOUS VERSUS INTERMITTENT CSFP MONITORING

There are practical management guidelines that need to be considered when using lumbar CSF drainage. Important considerations relate to monitoring and drainage techniques. Because the SCPP is an important variable to be monitored in these patients, and with manipulations of its components representing important therapeutic decisions, its continual monitoring is essential.[64] Alternatively, intermittent monitoring allows CSF to drain continuously. However, if the CSF is set to drain at a particular threshold, CSF drainage might be excessive, predisposing the patient to intracranial hypotension. Excessive and uncontrolled CSF drainage could occur, increasing the risk of subdural hematoma formation. As a result, it may be prudent to continuously monitor the CSFP, intermittently drain the CSF in predefined aliquots (typically 10– 15 mL) or predefined upper limit of CSFP (typically 10–12 mm Hg),[61] and thus continuously monitor the SCPP. Furthermore, the continuous monitoring of the pressure waveform would identify the patient who develops an occlusion of the CSF drain (and loss of the waveform). On the contrary, if the drain was set to continuously drain and that drainage stopped, one would never know whether this was caused by occlusion of the lumbar drain or whether it was simply because the predetermined manometer setting had been obtained.

One caveat to the continuous monitoring and intermittent drainage scenario relates to the dynamic setting of the operating room environment where multiple tasks may draw the attention of the anesthesiologist away from the draining CSF. Even though continuous CSFP monitoring (as opposed to continuous drainage) is advocated for all of the previously stated reasons, there will always be some time when drainage is required. To avoid excessive drainage (i.e., >10–15 mL during any 1-hour time period) during this time, the height of the collecting system manometer should be adjusted so that the drainage does not exceed a preset column height (for example, 10 cm, which is equivalent to approximately 6–7 mm Hg). If the column height is set lower than this, and there are circumstances whereby the anesthesiologist is occupied with other operating room tasks, an excessive volume of CSF might be drained. It combines the intuitive rationale of continuous monitoring with the added safety of the manometer column overflow setting that will reduce excessive drainage below a certain pressure. Similarly, when drainage occurs in the intensive care unit, it may also be prudent to set the drainage system so that no more than a preset height of fluid can drain (i.e., 6–7 mm Hg).

COMPLICATIONS OF LUMBAR CSF DRAINAGE

Complications of lumbar CSF drainage (Table 3) include those related to 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.[13,61,65,66] Complications secondary to CSF drainage include symptomatic intracranial hypotension presenting as headache, abducens nerve palsy, and intracranial hemorrhage (ICH). Infection caused by the presence of the catheter can lead to local infection or meningitis. Catheter fracture can occur during removal, causing local irritation or infection.

Table 3. Complications of CSF Drainage in TAAA Surgery

Reference	No.[a]	Catheter fracture	Infection (local/meningitis)	CSF leak[b]	Abducens nerve palsy[c]	Neuraxial hematoma	Asymptomatic ICH	Symptomatic ICH	Mortality attributed to drain complications
Estrera et al.,[61] 2009	1107	0.1% (1 of 1107)	0.2% (2 of 1107)	0.64% (7 of 1107)	0	0	NR	0.5% (5 of 1107)	0.3% (3 of 1107)
Wynn et al.,[70] 2009	482	NR	NR	NR	NR	NR	2.9% (14 of 482)	1% (5 of 482)	0.6% (3 of 482)
Leyvi et al.,[13] 2005	54	NR	NR	NR	NR	NR	NR	5.5% (3 of 54)	11% (6 of 54)
Cheung et al.,[64] 2003	162	1.8% (3 of 162)	1.2% (2 of 162)	0.6% (1 of 162)	0.6% (1 of 162)	0	0	0	0
Dardik et al.,[67] 2002	230	NR	NR	NR	NR	NR	NR	3.5% (8 of 230)	1.7% (4 of 230)
Weaver et al.,[66] 2001	62	0	0	0	0	3.2% (2 of 62)	0	0	NR
Grady et al.,[79] 1999[d]	530	0	0.2% (1 of 530)	2.5% (13 of 530)	0	0	0	0	0

CSF = cerebrospinal fluid; ICH = intracranial hemorrhage; NR = not specifically reported; TAAA = thoracoabdominal aortic aneurysm.
[a] Number of patients with lumbar CSF drains.
[b] CSF leak defined as postdural puncture headache occurring after drain removal.
[c] Caused by cerebellar herniation.
[d] This study was in neurosurgical patients requiring perioperative lumbar CSF drains, not TAAA.

ICH after CSF drainage may be the most devastating of these complications. Draining a volume of CSF that is too large over a short period of time is a documented risk factor for a subdural ICH. In general, 10 mL/h has been recommended, in the absence of any paraparesis, when more aggressive (up to 20 mL/h) drainage may be necessary.[13,61] The mechanism relates to intracranial hypotension which, in turn, causes stretching and tearing of bridging dural veins.[13,67,69,70] High CVP consequent with the application of the aortic cross-clamp may also be a risk factor for intracranial bleeding.[70] This may be caused by a higher transmural cerebral venous pressure (CVP-ICP) at the time of the subsequent decrease in ICP when the CSF is drained, resulting in rupture of intracerebral veins. In addition, patients with cerebral atrophy (i.e., the elderly), arteriovenous malformations, cerebral aneurysms, and previous subdural hematoma may be at higher risk of developing ICH.[70] Wynn et al.[70] published a retrospective analysis of 486 lumbar drains placed between 1987 and 2008. Symptomatic ICH after spinal drainage was seen in 1% of patients (5 of 482), with a 2.9% incidence of asymptomatic intracranial bleed.[70] 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 of 5 patients as a result. Estrera et al.,[61] in the largest series of lumbar CSF drains reported (n = 1107), 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.[70] Therefore, if blood is detected in the CSF, urgent imaging of the brain should be considered.

To reduce the risk of ICH, Leyvi et al.[13] suggested a protocol in which no more than 10 mL of CSF is drained in any 1-hour period. This volume limit suggested by Leyvi et al. should be balanced by the practice of more liberal unlimited volume drainage as outlined in the reports by Coselli et al.[40] and Hnath et al.[53] No direct comparison of restricted versus liberal CSF drainage has been performed. Screening patients for intracranial pathology before placement of the drain is not routinely done, but may be of benefit in those thought to be at particularly high risk. In addition to acute ICH, one report suggests that chronic CSF leak through the dural puncture site may be a possible cause for delayed ICH.[13] Epidural blood patches have been suggested, but this must be done with caution because previous experimental work has shown that introducing fluid into the epidural space can cause a transmitted increase in ICP,[71] especially in individuals with decreased intracranial compliance, as would be seen in an acute subdural hematoma. Similarly, preoperative increased ICP is a contraindication to lumbar CSF drain placement.

Neuraxial hematoma can be a disastrous complication of lumbar drain placement. Although the risk for this catastrophic injury seems 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.[66] One retrospective study found intraspinal hemorrhagic complications in 2 of 65 patients (3.2%) undergoing TAAA repair.[66] 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.[64] Nonetheless, physicians must be alert to the possibility of neuraxial hematoma when any patient with a lumbar drain presents with lower extremity neurologic deficit. This injury is treated by surgery, and delay in therapy may result in permanent injury. One study suggests reserving imaging to the population of patients who do not respond to prompt CSF drainage and arterial blood pressure augmentation, considering that spinal cord ischemia is far more common than neuraxial hematoma formation.[3]

Although blood in the draining CSF has been suggested to be a marker for intracranial bleed, this has been shown to be an insensitive marker for epidural cord hematoma, because the blood may be in the epidural space and

therefore may not communicate with the CSF space.[66] The best imaging modality to differentiate between spinal cord ischemia and hematoma is magnetic resonance imaging (MRI). Increased signal intensity on T2-weighted MRI differentiates hematoma from spinal cord ischemia.[66] Computed tomographic scan of the spine can be performed in situations in which MRI is either not available or not practical; however, this imaging technique may miss a small hematoma and will not provide direct information about cord ischemia. Because time is critical in these situations, it may be helpful to have a contingency plan discussed ahead of time should this complication occur. The input from a neurosurgical and radiologic perspective is essential.

Central to the increased risk of neuraxial hematoma is the systemic anticoagulation that is usually used 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 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, and that the lowest dose of heparin for therapeutic effect should be used.[72] Although Cheung et al.[64] 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.[73] One review has suggested delaying the case for 24 hours in the event of a bloody tap when subsequent anticoagulation for cardiopulmonary bypass is planned.[74] However, this has not been studied extensively, nor has the safe length of waiting time needed before anticoagulation been investigated. Some authors have elected to place the drains 24 hours preoperatively to avoid this situation.[28] 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 because the length of time between catheter placement and administration of anticoagulation can be several hours. However, this delay may not always preclude subsequent hematoma formation because perioperative fibrinolysis[75] could potentially cause later breakdown of a neuraxial hemostatic thrombus and lead to additional bleeding at the site of needle injury. If postoperative neurologic deficits are detected in this situation, more urgent imaging of the spine should be considered to exclude hematoma.

Other complications of lumbar drain placement such as infection and retained catheters are exceedingly rare. The database analysis by Cheung et al.[64] revealed retained catheter fragments in 3 of 162 patients (1.8%), 1 of whom subsequently developed meningitis. It was suggested that to minimize catheter fracture risk, 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.

In nonoperative settings, infection has been associated with the prolonged use of lumbar catheters. One retrospective analysis of lumbar drains used in the management of normal pressure hydrocephalus reported a 0.8% incidence of meningitis when catheters were in place for up to 5 days ($n = 223$).[76] A study investigating complications of lumbar CSF drainage in TAAA repair revealed an incidence of infection of 1.2%.[64] Estrera et al.[61] found a 0.2% incidence of meningitis in the study of 1107 CSF drains. Sources of infectious complications are thought to develop from either hematogenous spread from a remote source of infection or invasion of bacteria through the needle tract.[77] Lumbar drain placement should not proceed through an area of localized skin infection. Evidence for asepsis guidelines during insertion has been extensively reviewed elsewhere.[77] There are good data supporting the use of alcohol-based chlorhexidine antiseptic solutions, thorough handwashing with removal of jewelry, and use of sterile surgical gloves. Full barrier precautions with surgical gowns and masks should also be considered because the critical care literature outlines reasonable evidence of decreased incidence of blood-borne infections with these precautions in central venous catheterization.[78] Handling of the drain during insertion can be cumbersome and wearing a full sterile surgical gown may decrease the risk of catheter contamination from inadvertent contact with nonsterile clothing. Routine culture of the CSF is not indicated in these patients; however, if there is a suspicion of infection, the CSF should be included in the culture samples sent and the drain should be immediately removed because it may present a continual nidus for infection. Postdural puncture headache has also been reported with the use of lumbar CSF drains.[79,80]

SUBARACHNOID OPIATES FOR POSTOPERATIVE ANALGESIA

Neuraxial opioids have been used in numerous settings for perioperative analgesia including in the setting of thoracic aortic surgery.[81] Although this practice is likely underreported in the literature, there is significant reason for caution with this approach in patients at risk for spinal cord ischemia. Indeed, one report discussed the occurrence of paraparesis when an opiate was administered into the CSF. Kakinohana et al.[82] described a patient who had undergone thoracoabdominal surgery and was given 4 mg epidural morphine for pain control. The patient developed postoperative paraparesis that was subsequently reversed with systemic naloxone. In the same report, the authors described an experimental study that replicated this phenomenon of opiate exacerbation of spinal cord ischemia after aortic occlusion in a rat model. Importantly, it seems that this paraparesis risk is in the setting of an already injured (by ischemic mechanisms) spinal cord. The lack of detrimental effect of spinal opiates that is seen in routine anesthetic practice[83] is likely specifically because it is used

Table 4. Best Practices for CSF Drainage

Issue	Recommendation
Preoperative assessment	
Coagulation[72]	No LMWH for 24 h (high dose); 12 h (low dose) No clopidogrel × 7 d No ticlopidine × 10^{-14} d No abciximab × 24–48 h No eptifibatide or tirofiban × 4–8 h Platelets >100 × $10^3/\mu L^3$ INR <1.3, normal aPTT
Localized infection[77]	Avoid placement of drain in an area of localized infection
Intracranial pressure	Avoid placement of drain if patient has evidence of increased intracranial pressure
Insertion	
Asepsis[77]	Alcohol-based chlorhexidine solutions, sterile draping, thorough handwashing with removal of jewelry, sterile surgical gloves, masks, sterile gown
Awake vs asleep[72]	Suggest awake to allow for patient feedback (i.e., pain/paraesthesia)
Timing of insertion[28]	Option to admit to hospital and insert lumbar CSF drain 24 h preoperatively to avoid issues with traumatic tap and systemic anticoagulation
Traumatic/bloody tap[72,74]	Discuss with surgeon, delay anticoagulation at least 60 min, consider delaying surgery 24 h, higher index suspicion postoperative neuraxial hematoma
Intraoperative	
Hemodynamics[8]	Avoid hypotension, MAP and MAP_d to maintain SCPP >60 mm Hg, avoid large increases in CVP
Zero transducer	Phlebostatic axis to ensure accurate calculation of SCPP
CSF drainage[13,61]	CSFP <10 mm Hg or to maintain SCPP >60 mm Hg, no more than 10–15 mL/h CSF drainage, intermittent drainage with continuous monitoring preferred to allow calculation of SCPP and avoid large volumes CSF drainage
Subarachnoid opiates[87,88]	Avoid, may exacerbate spinal cord ischemia
Postoperative	
Hemodynamics	Avoid hypotension
Duration of drainage/monitoring[77]	Avoid prolonged drainage to minimize infection risk, consider keeping drain in place <72 h
Bloody CSF drainage[70]	May indicate ICH, consider imaging brain
New-onset lower extremity neurologic deficit[66]	Worsening spinal cord ischemia vs neuraxial hematoma, increase SCPP (increase MAP, decrease CSFP), consider imaging neuraxis
Coagulation for drain removal[72]	Platelet count >100 × $10^3/\mu L^3$, INR <1.3, normal aPTT delay removal 2–4 h after last heparin dose, hold heparin 1 h after catheter removal

LMWH = low-molecular-weight heparin; INR = international normalized ratio; aPTT = activated partial thromboplastin time; MAP = mean arterial blood pressure; MAP_d = distal mean aortic pressure; SCPP = spinal cord perfusion pressure; CVP = central venous pressure; CSFP = cerebrospinal fluid pressure; ICH = intracranial hemorrhage.

in settings where there is unlikely to be any concomitant spinal cord ischemia.

A potential role of opiates contributing to spinal cord injury has been further explored with studies using opiate antagonists (naloxone) for the prevention of spinal cord ischemia during aortic surgery.[84,85] In these studies, the combination of lumbar CSF drainage and IV naloxone resulted in a lower incidence of paraplegia after TAAA repair. Insights into the mechanisms of an apparent opiate-induced worsening of spinal cord ischemia injury have been the subject of numerous other experimental studies. Kakinohana et al.[86] investigated the effect of various opioids (μ, δ, and κ agonists) on spastic paraparesis after spinal cord ischemia. Using the same rat model of spinal cord ischemia, they found that intrathecal administration of selective μ and δ receptor agonists enhanced the neurologic injury after aortic occlusion. The same effect was not seen with the administration of a κ receptor agonist. In a separate study, the same investigative group also examined an α_2 agonist modulating effect on this opiate-induced injury.[87] IV infusion of dexmedetomidine improved neurologic function in a dose-dependent manner after intrathecal morphine administration after an ischemic injury to rat spinal cords. Adding to the complexity of the pathophysiology is the additional information on K^+ adenosine triphosphate channels and similar-type injuries.[88] Administration of intrathecal nicorandil (a K^+ adenosine triphosphate opener) decreased the minimum dose of morphine necessary to worsen postischemia neurologic injury. Further study is needed to understand the full scope of opiate/spinal cord ischemia interactions.

CONCLUSION

Paraplegia after thoracoabdominal aneurysm surgery remains one of the most devastating postoperative outcomes. The available evidence supports the use of CSF drainage along with aortic perfusion methods distal to the aortic cross-clamp. Furthermore, practical guidelines to aid in minimizing the inherent risk of CSF drainage may allow for more standardized practice when this adjunct is used. By using "Best Practices" for lumbar CSF drainage (Table 4) involving preoperative evaluation, optimal insertion techniques and special considerations for the intraoperative and postoperative use of the technique, the risk of spinal cord injury after thoracoabdominal aortic surgery may be reduced.

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