Novel approaches to spinal cord protection during thoracoabdominal aortic interventions

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INTRODUCTION
Spinal cord protection remains clinically important after thoracoabdominal aortic interventions (TAAIs) because spinal cord ischemia is still common and significantly worsens the perioperative morbidity and mortality \([1,2,3]\). Spinal cord ischemia is manifested in the conscious patient by the development of lower extremity weakness or during surgery by the attenuation of spinal cord signals with monitoring of somatosensory-evoked potentials and motor-evoked potentials \([4,5,6]\). The aortic repair techniques in the contemporary era can be open, endovascular, or hybrid. Total endovascular repair may include the use of fenestrated or branched endovascular stent components to not only exclude the aneurysm, but also to preserve aortic branch perfusion. Hybrid TAAIs typically involve nonfenestrated endovascular repair of diseased aorta combined with open surgical transposition of aortic branches to preserve critical organ perfusion \([7,8]\). This review is focused on the recent approaches to protect the spinal cord during TAAI from an anesthesiology perspective. It will highlight the risk factors for spinal cord ischemia and the principal spinal cord preservation strategies.

Purpose of review
Spinal cord ischemia after thoracoabdominal aortic interventions is a devastating complication because it significantly worsens the perioperative morbidity and mortality. Long-term outcome is also affected because of medical complications which are directly related to the neural deficits. Paraplegia has significant medical, social, and financial aspects. Limited mobility, the need for assistance in activities of daily living, makes paraplegia an important target for prevention. An understanding of spinal cord blood supply, risk factors for spinal ischemia, and strategies for spinal cord rescue in this setting can help minimize the negative outcome effects of this important complication.

Recent findings
The vascular supply of the spinal cord is via an extensive collateral arterial network with multiple auxiliary arterial supplies. Risk factors for spinal cord ischemia include extensive aortic repair, prior aortic repair, spinal cord malperfusion on clinical presentation, systemic hypotension, acute anemia, prolonged aortic clamping, and vascular steal. Spinal rescue strategies include systemic hypothermia, endovascular aortic repair, permissive systemic hypertension, cerebrospinal fluid drainage, pharmacologic neuroprotection, and intensive neuromonitoring.

Summary
The progression of spinal cord ischemia after thoracoabdominal aortic interventions can frequently be arrested before irreversible infarction results. This spinal cord rescue depends on the early detection and immediate multimodal intervention to maximize spinal cord oxygen supply. The devastating outcomes associated with spinal infarction in this setting offset the risks and knowledge gaps currently associated with contemporary interventions.

Keywords
cerebrospinal fluid drainage, collateral arterial network, evoked potentials, hypothermia, spinal cord ischemia
The vascular supply to the spinal cord is via the anterior spinal artery in the mid-to-lower thoracic area (T8 to L1). The caudal arterial supply to the SCAN is from the internal iliac arteries and their branches. The SCAN concept explains that spinal cord perfusion stems from multiple interconnected sources that require adequate perfusion pressure and vascular tone throughout the arterial network.

**RISK FACTORS FOR SPINAL CORD ISCHEMIA: THE IMPORTANCE OF THE SPINAL COLLATERAL ARTERIAL NETWORK**

The cause of spinal cord ischemia after TAAI is a net oxygen debt in the spinal cord, where neural oxygen supply is hampered by the surgical intervention. Spinal cord protection after TAAI depends on an understanding of the risk factors for spinal cord ischemia, summarized in Table 1. An important mechanism underlying these risk factors is the compromise of the spinal collateral arterial network (SCAN), as outlined in the comments in Table 1. The SCAN concept has resulted in large part from the bench and bedside research by Griep and colleagues [9,10].

In 1882, Adamkiewicz described the spinal cord vascular plexus of the anterior and posterior spinal arteries with augmentation from cephalic, central, and caudal input [5,9,10]. Griep et al. showed that perfusion in these spinal arteries is supported by complex and intertwined collateral network, with cephalic contribution to the SCAN from the brachiocephalic arteries and with significant contribution from the vertebral arteries. The central arterial input to the SCAN is from multiple segmental aortic branches such as the intercostal and lumbar arteries. This central arterial supply to the SCAN is scarcely dispersed in the lower thoracic area, but frequently includes a large arterial branch at the level of the lower thoracic called the artery of Adamkiewicz or the arteria radicularis magna. This augmented supply from this large segmental artery is vital, given the small diameter of the anterior spinal artery in the mid-to-lower thoracic area (T8 to L1). The caudal arterial supply to the SCAN is from the internal iliac arteries and their branches. The SCAN concept explains that spinal cord perfusion stems from multiple interconnected sources that require adequate perfusion pressure and vascular tone throughout the arterial network.

**STRATEGIES FOR SPINAL CORD PROTECTION**

The contemporary strategies for perioperative spinal cord protection can frequently be understood as therapeutic consequences of the SCAN concept. Although they are summarized in Table 2, they will now be more fully explored in this section.

**Deep hypothermia**

Deep hypothermia for spinal cord protection is an established technique in TAAI as it minimizes the neural oxygen demand during aortic reconstruction when oxygen delivery is compromised [11**,12**]. The definition of deep hypothermia by the recent expert consensus is a systemic temperature range of 14.1–20.0 degrees Celsius, ideally measured at the nasopharynx [13†]. In addition to spinal cord protection, deep hypothermic circulatory arrest (DHCA) offers multiple advantages in open TAAI such as minimal dissection of periaortic tissues, elimination of the requirement for sequential aortic clamping, easy access to the aortic arch, and excellent visceral organ protection [11**,12**]. In a recent single-center evaluation of open thoracoabdominal aneurysm repair (n = 243: Crawford extent I 26%; Crawford extent II 40%; Crawford extent III 34%) utilizing DHCA, the incidence of spinal cord ischemia was 5.3% (13 patients: nine with paraplegia and four with paraparesis) [14]. Emergency surgery was significantly associated with spinal cord ischemia compared with elective surgery (16.7 vs. 3.9%; P = 0.04) [14]. Fehrenbacher and colleagues have also recently demonstrated in a similar large contemporary series (n = 343) with DHCA that the incidence of spinal cord ischemia was 1.1–3.2%, depending on the aortic disease [15,16]. These data demonstrate that experienced perioperative teams can achieve excellent spinal cord protection in extensive open TAAI with DHCA.
Thoracic endovascular aortic repair

Extensive meta-analysis has suggested that across diverse aortic diseases, thoracic endovascular aortic repair (TEVAR), per se, compared with open repair significantly decreases the risk of spinal cord ischemia in TAAI [5,7,8]. A recent analysis of TEVAR vs. open repair in DeBakey Type III chronic dissections (n = 89) from two experienced medical centers demonstrated that TEVAR significantly reduced the risks of perioperative mortality (0 vs. 10.3%) and spinal cord ischemia (0 vs. 12.1%) [17]. Contemporary meta-analysis of hybrid TAAI has demonstrated in very high-risk patients (n = 660: 19 studies) that hybrid techniques may reduce mortality and spinal cord ischemia, although the investigators concluded that further high-quality trials are indicated to clarify the extent of these outcome advantages [18].

A large single-center analysis of TAAI (n = 406) over 11 years demonstrated an unexpectedly low incidence of spinal cord ischemia, namely 2.7% with a permanent neurological deficit of only 1.5% [19]]. Independent risk factors for spinal cord ischemia in this series were consistent with Table 1: previous abdominal aortic aneurysm repair [odds ratio 4.8 (OR); 95% confidence interval (CI) 1.4–17.2; P = 0.026], extensive coverage of the descending thoracic aorta (OR 3.6; 95% CI 1.1–12.3; P = 0.038), and implantation of thoracoabdominal branched or fenestrated grafts (OR 9.5; 95% CI 1.2–50.6; P = 0.032) [19**]. The investigators postulated that the extensive arterial network, the SCAN, as explained by Griepp and colleagues, is the likely explanation for the low incidence of spinal cord ischemia in their reported TEVAR experience. Although TEVAR results in coverage of central spinal segmental arterial supply, back bleeding is prevented, and in this fashion prevents vascular steal in that part of the vascular network. Furthermore, the investigators pointed out that collateral perfusion from the paravertebral muscle compartment will also augment spinal segmental perfusion in the

**Table 1. Risk factors for spinal cord ischemia during thoracoabdominal aortic interventions**

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<th>Risk factors</th>
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<td>Complicated aortic dissection with visceral malperfusion, e.g., acute type B dissection</td>
<td>In acute complicated aortic dissection, there may be spinal cord ischemia, renal and intestinal ischemia, because of regional malperfusion due to aortic branch occlusion or exclusion from the dissection process.</td>
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<tr>
<td>Total extent of thoracoabdominal repair, e.g., Crawford Extent II aneurysm repair has a very high risk of spinal cord compromise</td>
<td>The risk of spinal cord ischemia increases with the extent of aortic repair because of greater loss of segmental arterial spinal cord supply.</td>
</tr>
<tr>
<td>Prior thoracoabdominal aortic segment repair, e.g., open repair of abdominal aortic aneurysm; endovascular repair of the descending thoracic aorta</td>
<td>Prior aortic repair aggravates the risk of spinal cord ischemia because of the loss of spinal cord arterial segmental supply from the prior procedure. In this setting, the spinal cord has compromised reserve of its collateral arterial network.</td>
</tr>
<tr>
<td>Extent of preservation of spinal segmental arterial supply, e.g., intercostals arteries; lumbar arteries</td>
<td>The risk of spinal cord ischemia may be reduced if fenestrated endograft is used, or in open aortic repair if intercostal arteries are reimplanted into the aortic graft.</td>
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<tr>
<td>Duration of aortic clamping</td>
<td>In the absence of distal aortic perfusion, the duration of aortic clamping to allow open aortic replacement often correlated with the duration of spinal cord hypoperfusion and hence ischemia. Distal aortic perfusion techniques will not completely mitigate the risk of spinal cord ischemia.</td>
</tr>
<tr>
<td>Acute anemia</td>
<td>Significant acute anemia is typically associated with severe bleeding. Acute anemia aggravates spinal cord ischemia by impairing oxygen delivery as a consequence of low hemoglobin mass.</td>
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<tr>
<td>Systemic hypotension</td>
<td>Hypotension or circulatory collapse significantly compromises spinal cord perfusion and thus may precipitate spinal cord ischemia from decreased oxygen delivery.</td>
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<tr>
<td>Systemic vasodilation with vascular steal</td>
<td>Vasodilators such as sodium nitroprusside have been utilized for control of hypertension associated with aortic clamping. The systemic vasodilation resulted in vascular steal, compromising spinal perfusion pressure to precipitate spinal cord ischemia. Back bleeding from open branches into the surgical field in open repair will also cause the steal phenomenon.</td>
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Spinal cord perforation strategies during thoracoabdominal aortic interventions et al.

Spinal cord perfusion pressure is defined as the difference between systemic mean arterial pressure and right atrial pressure whichever is greater [2,3,4,5]. Consequently, any increase in right atrial pressure to pressures higher than CSF pressure will decrease the spinal cord perfusion pressure. Raised right atrial pressure may result from increased preload and increases in intrathoracic pressure because of positive end-expiratory pressure. Furthermore, if mean arterial pressure is elevated, there will be a higher spinal cord perfusion pressure and enhanced perfusion of the spinal cord through the SCAN. This principle is central in the perioperative management of patients undergoing TAAI to preserve spinal cord perfusion and protect against spinal cord ischemia. An important tenet of modern spinal cord protection during TAAI (whether open or endovascular) includes intentionally maintaining the perioperative mean arterial blood pressure in the high physiologic range, namely 80–100 mmHg [21,22].

Maintenance of high pressure in the collateral spinal network for at least 24–48 h postoperatively (or longer if there are apparent neurological deficits) is important to the success of a spinal cord protection strategy, as progressive postoperative remodeling of the SCAN has been demonstrated within 48 h [9,20]. These acute adaptations in the SCAN include increases in the diameter of the anterior spinal artery and dilation of the epidural arterial network within 24 h, and a realignment of the arterioles parallel to the spinal cord by 5 days postoperatively [9,20]. These adaptations in the SCAN allow acute

### Table 2. Spinal cord protection strategies during thoracoabdominal aortic interventions

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<td>Systemic hypothermia, e.g., deep hypothermic circulatory arrest; moderate hypothermia with left heart bypass; mild hypothermia with native circulation</td>
<td>Hypothermia reduces spinal oxygen demand and thus extends its tolerance of limited oxygen supply. The aortic repair can be conducted under deep hypothermia with circulatory arrest, moderate hypothermia with circulatory support or mild hypothermia with native circulation. Permissive mild hypothermia should be induced with caution to avoid the development of arrhythmias. Deep hypothermia maximizes spinal cord protection from cooling because of maximal suppression of neural metabolism and hence minimizes oxygen demand.</td>
</tr>
<tr>
<td>Thoracoabdominal endovascular repair, including hybrid techniques, e.g., endovascular stenting with fenestrated or branched grafts; endovascular stenting with open debranching procedures</td>
<td>Endovascular aortic repair lowers the risk of spinal cord ischemia because of beneficial effects on the spinal collateral arterial network. These benefits may be further enhanced if the extensive aortic procedure can be staged.</td>
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<tr>
<td>Systemic hypertension, e.g., maintaining the mean arterial pressure between 80 and 100 mmHg with titrated intravenous norepinephrine infusion</td>
<td>Permissive systemic hypertension increases spinal cord perfusion pressure. The technique should be applied both, during surgery and on arrival to the intensive care unit. It is important during this therapy to monitor for adequate cardiac output and bleeding from disrupted suture lines.</td>
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<tr>
<td>Cerebrospinal fluid drainage (CSF), e.g., lumbar subarachnoid catheter</td>
<td>Drainage of CSF increases spinal cord perfusion pressure. The safe conduct of this intervention is essential to minimize serious complications.</td>
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<tr>
<td>Pharmacologic neuroprotection</td>
<td>These drugs increase neural tolerance of ischemia. The supporting evidence is weak at best. Options include steroids, papaverine, lidocaine, magnesium and minocycline.</td>
</tr>
<tr>
<td>Intensive neuromonitoring e.g., neurologic examination; somatosensory-evoked potentials; motor-evoked potentials</td>
<td>Early detection of spinal cord ischemia is a clinical emergency. A spinal rescue protocol should be urgently implemented in order to recover spinal cord function.</td>
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expansion of its capacity for enhanced spinal cord perfusion. Permissive perioperative systemic hypertension augments spinal cord perfusion after TAAI, giving the SCAN the critical time to expand capacity to compensate for the loss of segmental arterial input resulting from the aortic repair [19**,20,21,22**,23]. This strategy acts as part of a bridge to recovery of the SCAN in the perioperative period.

**Drainage of cerebrospinal fluid**

Perfusion pressurization of the SCAN is often insufficient to fully protect the spinal cord and prevent paraplegia. Perioperative drainage of CSF to maximize spinal cord perfusion pressure (by minimizing the resistance to afferent spinal cord blood supply) is typical in modern TAAI. The 2010 American Heart Association guidelines strongly recommend CSF drainage (Class I, Level B evidence) in TAAI with high risk of spinal cord ischemic injury [23].

In practice, although CSF drainage is primarily utilized in high-risk patients, the final decision depends on the team discussion and institutional practice. Proactive CSF drainage as a routine part of a perioperative protocol in TAAI with TEVAR has recently been demonstrated in a single-center study (n = 94, 2005–2012) to be very effective with a 1.1% incidence of spinal cord ischemia [24]. A second approach, namely selective CSF drainage for postoperative symptomatic spinal cord ischemia after TEVAR, was associated with a 1.4% rate of permanent spinal cord ischemia [25]. A third approach, namely selective preoperative CSF drainage in high-risk TEVAR patients, was evaluated in a recent large single-center series (n = 381, 2002–2012) [26]. The incidences of permanent and temporary spinal cord ischemia in this series were 1.8 and 4.7%, respectively [26]. A single-center analysis of spinal cord ischemia after TEVAR (n = 424, 2002–2010) again confirmed the low incidence of 2.8% with a 75% rate of complete recovery, with early diagnosis and prompt institution of permissive hypertension alone or with CSF drainage [26]. In multivariate analysis, chronic renal insufficiency was the only independent predictor of spinal cord ischemia (OR 4.39; 95% CI 1.2–16; \( P = 0.029 \)) [26]. Further trials are required to confirm renal dysfunction as a predictor for spinal cord ischemia after endovascular TAAI.

A systematic review (n = 4936, 46 studies) recently evaluated whether preoperative CSF drainage in TEVAR reduces the risk of spinal cord ischemia [27]. The incidence of spinal cord ischemia in this meta-analysis was 3.89% (95% CI 2.95–4.95%), with no clear reductions evident with routine preoperative drainage, selective preoperative drainage, or no preoperative drainage [27]. An updated Cochrane meta-analysis evaluated CSF drainage in open TAAI for aortic aneurysm (n = 287, three randomized controlled trials before 2005) [28*]. The overall benefit of CSF drainage in this limited dataset was an 80% reduction in the relative risk of spinal cord ischemia. A meta-analysis demonstrated an OR for CSF drainage of 0.48 (95% CI 0.25–0.92) [28*]. In summary, although CSF drainage is indicated in most extensive open TAAI, its role in endovascular TAAI is evolving, given the significantly lower risk of spinal cord ischemia. Current guidelines recommend selective preoperative CSF drainage in patients at high risk for spinal cord compromise after TEVAR, but this will depend on the institutional protocol [22**,23]. CSF drainage is the mainstay of rescue therapy for spinal cord ischemia after TAAI, especially if there is incomplete recovery after several hours of permissive hypertension [22**,23].

**MANAGEMENT OF CEREBROSPINAL FLUID DRAINAGE**

Regardless of the specific protocol, the widely accepted goal is to maintain CSF pressure less than 10–15 mmHg [2,5,29**,30**,31]. Although this CSF pressure goal is widely accepted, it should also be titrated to clinical effect and the overall trend in spinal cord perfusion pressure. For example, if the awake patient has normal lower extremity muscle power, then CSF pressure management becomes less important because at this timepoint the spinal cord is intact.

Once a CSF drainage catheter has been placed, the CSF pressure can be maintained at a defined goal by monitoring the CSF pressure (either continuously or intermittently) and intentionally draining CSF until either the desired pressure is achieved or a specific volume has been drained. This procedure is of particular importance in open TAAI, as application of aortic cross clamp (AXC) may induce a sharp rise in CSF pressure. It occurs in spite of effective distal perfusion via left-sided bypass and effective cardiac preload reduction. A possible explanation for the increase in CSF pressure is that AXC induces a sympathetically mediated vasoconstriction in the systemic and spinal vasculature. This increased tone in the thin-walled spinal veins may decrease the critical closing pressure needed to provoke collapse of the radiculospinal veins as they pass through the dura, with consequent venous engorgement and increase in CSF pressure [32]. Possible explanations for the increase in CSF pressure after stent graft deployment might be attributed to the same cause of sympathetic spinal
stimulation or to the local acidosis-induced venoconstriction, which occurs in the presence of inadequate spinal cord perfusion.

(1) If the CSF pressure remains above goal following drainage, a decision needs to be made if more will be drained or other maneuvers performed to optimize spinal cord perfusion pressure. Though the amount that can ‘safely’ be drained at a given time has not conclusively been determined, many experienced centers are limiting draining to a specified volume of CSF each hour, but limit hourly maximum drainage to less than 25 ml to avoid complications such as intracranial hematoma from torn subdural bridging veins [29**,30**,33,34].

(2) Setting the drainage unit at a specific height on a bedside pole and allowing the CSF to drain freely as needed. For example, if one levels the drainage unit to 13 cm above the phlebotastic axis, CSF will continue to drain as long as CSF pressure exceeds 10 mmHg (1.3 cm H2O = 1 mmHg). The advantage of this common method is the automatic maintenance of the desired CSF pressure, but profound vigilance is required where this protocol is in use because a serious disadvantage is the potential for excessive drainage (e.g., if the drainage unit falls to the floor), which may predispose to serious complications [30**,34].

Prior to discontinuing and pulling out the CSF drainage, capping off the drain for 24 h prior to the removal of the catheter may allow the CSF pressure to normalize and ascertain whether the patient is free from spinal cord ischemia as spinal cord perfusion pressure subsequently decreases [27]. Although this approach is consistent with our understanding of the SCAN, there are sparse data to support this practice.

**COMPLICATIONS OF CEREBROSPINAL FLUID DRAINAGE**

The complications of CSF catheter insertion and subsequent drainage include catheter fracture, infection, CSF leak, abducens nerve palsy, neuroaxial hematoma, and intracranial hematoma [30**,34]. One of the feared complications of CSF drainage is bleeding, with subsequent potential epidural or subdural hematoma. Clearly, a frankly ‘bloody tap’ during attempted CSF drain placement will necessitate postponement of the aortic repair, but subdural bleeding can also occur as a result of CSF drainage itself because of the tearing of subdural bridging veins. Excessive CSF drainage was associated with intracerebral hematomas and with significant morbidity and mortality [33]. A recent large analysis from a high-volume center (n = 504, 2005–2009) demonstrated a 2.8% incidence of intracranial hematoma after CSF drainage for open TAAI: 72% of these cases were subdural hematomas [30**]. Furthermore, the incidence of postdural puncture headache was 9.7%, of whom 34.6% required epidural blood patch for clinical resolution [30**]. In multivariate analysis, connective tissue disorder was an independent predictor for postdural puncture headache (OR 3.08; 95% CI 1.33–7.13). The investigators concluded that the risk of intracranial hematoma was modest, and that epidural blood patch should be considered early in the management of postdural puncture headache [30**].

In a smaller, recent, Swedish experience (n = 84, 2009–2012), CSF drainage in endovascular TAAI was associated with a 3.6% incidence of serious bleeding complications (one epidural and two subdural hematomas, two of which required neurosurgical intervention) [35]. In summary, CSF drainage has serious but uncommon complications [33–36]. The clinical indication for CSF drainage in TAAI must balance the surgical risks of spinal cord ischemia with the risks of CSF drainage. In endovascular TAAI, the role of this neuroprotective intervention will likely be on a selective basis, given the significantly lower risk of spinal cord ischemia in this setting.

**Pharmacologic neuroprotection**

The search for the ideal neuroprotective agent in the setting of spinal cord ischemia after TAAI has been vigorous and sustained [2,5]. As the onset of spinal ischemia in TAAI is typically known, the neuroprotective agent could be administered intravenously or intrathecally either prophylactically or during ischemia and reperfusion of the spinal cord. Encouraging results from the laboratory experiments have demonstrated promising roles for an array of agents such as allopurinol, activated protein C, adenosine, barbiturates, carbamazepine, lidocaine, magnesium, mannitol, naloxone, papaverine, prostaglandins, steroids, and volatile anesthetics [5]. Despite all these candidate agents, an ideal agent for clinical administration has yet to be found.

A recent clinical trial from a high-volume center (n = 330; 250 exposed to papaverine, 2002–2010) evaluated the neuroprotective effect of intrathecal papaverine as part of a multimodal protocol for spinal cord protection during open TAAI [37**]. The rationale for this intervention is that papaverine, as a vasodilator, will increase spinal cord perfusion, particularly in the narrowed lower thoracic area, when given in the intrathecal space. Exposure to papaverine as part of a multimodal spinal protection protocol in this trial significantly reduced the risk of
permanent paraplegia (3.6 vs. 7.5%; \(P = 0.01\)) and paraparesis (1.6 vs. 6.3%; \(P = 0.01\)) [37*]. The investigators concluded that papaverine likely provides additional neuroprotection in this setting.

The multiplicity of neuroprotective agents points to an inadequate understanding of ischemia and reperfusion in the spinal cord after TAAI from a pharmacologic perspective. Recently, investigators have focused attention on the resident macrophages in the spinal cord (the microglia) that are thought to play a central role in the development of neural death during spinal cord ischemia [38,39,40*]. A laboratory study in rats demonstrated that exposure to the macrolide antibiotic, minocycline, during spinal cord ischemia from thoracic aortic occlusion not only inhibits spinal microglia, but also significantly protects against the clinical development of paraplegia [40*]. This promising study has identified a novel cellular target for further study and possibly a clinical trial in the future.

In summary, although a multiplicity of agents have shown promise in the laboratory, there are to date no robust randomized clinical trials supporting the efficacy of a single agent for spinal cord neuroprotection in TAAI. It appears that steroids are the most frequently administered agents for neuroprotection during open TAAI, but this practice is based on limited evidence [5]. Given the low risk of spinal cord ischemia and expanding clinical applications of endovascular TAAI, it is likely that this practice will gradually disappear unless future clinical trials strongly support an agent.

Intensive neuromonitoring of the spinal cord

In the awake patient after TAAI, serial neurologic assessment will detect the development of spinal cord ischemia. This diagnosis should urgently trigger a protocol for spinal cord rescue, including permissive systemic hypertension with or without CSF drainage [1,2,3*]. In the anesthetized and hence uncooperative patient during TAAI, intraoperative detection of spinal cord ischemia typically relies on neuromonitoring with somatosensory-evoked potentials and motor-evoked potentials [4,5,6**,41,42].

The intraoperative detection of spinal cord ischemia during TAAI is critical because it significantly correlates with the development of paraplegia and poor perioperative outcome [41,42]. The intraoperative diagnosis by transcranial motor-evoked potentials (tcMEPs) gives the team the opportunity to intervene to enhance spinal cord perfusion and save the spinal cord. It is essential to tailor the anesthetic plan so as to maximize the preservation of neural signals both for somatosensory-evoked potentials and tcMEPs [4,5,6**,41,42]. A critical disadvantage of tcMEPs is the high sensitivity to volatile anesthetics and muscle relaxants. Thus, when tcMEP monitoring is planned, intravenous anesthesia should be used with minimal volatile anesthetics or muscle relaxants. This will minimize the interference with the signal profiles from medications and maximize the detection of spinal cord ischemia. Anesthetic interventions to maximize spinal cord oxygen delivery in this setting include systemic hypertension, augmented CSF drainage, and red blood cell transfusion for correction of anemia [22**,23]. Surgical interventions to maximize the spinal protection will depend on the stage and type of TAAI but might include augmentation of distal aortic perfusion, control of bleeding, reimplantation of intercostal arteries, and staging of the TAAI to allow recovery of the SCAN [5,43–45]. In summary, comprehensive evaluation of spinal cord function remains essential during the entire perioperative period in patients undergoing TAAI. In high-risk patients for spinal cord injury, spinal cord monitoring with evoked potentials is indicated. The detection of spinal cord ischemia should urgently trigger interventions for spinal cord rescue, based on institutional protocol, patient factors, and team discussion. The importance of clinical experience, clear communication, and seamless teamwork all contribute significantly to prompt and successful reversal of spinal cord ischemia in this challenging setting.

CONCLUSION

The recent progress in TAAIs has resulted in better freedom from spinal cord ischemia for our patients, although this devastating complication still occurs. The promising areas of investigation in the near future will likely involve refinements in endovascular aortic techniques and a multimodality approach, both for enhanced spinal cord protection in these challenging aortic procedures.

Acknowledgements

Financial support: Institutional.

Conflicts of interest

There are no conflicts of interest.

REFERENCES AND RECOMMENDED READING

Papers of particular interest, published within the annual period of review, have been highlighted as:

- of special interest
- of outstanding interest

Novel approaches to spinal cord protection Augoustides et al.


10. This interesting study highlights in a contemporary series from an experienced center the negative outcome effects of spinal cord ischemia after extensive open aortic repairs.


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