Spinal cord protection in aortic endovascular surgery

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Abstract

A persistent neurological deficit, such as paraplegia or paraparesis, secondary to spinal cord injury remains one of the most feared complications of surgery on the descending thoracic or abdominal aorta. This is despite sophisticated advances in imaging and the use of less invasive endovascular procedures. Extensive fenestrated endovascular aortic graft prostheses still carry a risk of spinal cord injury of up to 10%; thus, this risk should be identified and strategies implemented to protect the spinal cord and maintain perfusion. The patients at highest risk are those undergoing extensive thoracic aortic stenting including thoracic, abdominal, and pelvic vessels. Although many techniques are available, lumbar cerebrospinal fluid drainage remains the most frequent intervention, along with maintenance of perfusion pressure and possibly staged procedures to allow collateral vessel stabilization. Many questions remain regarding other technical aspects, spinal cord monitoring and cooling, pharmacological protection, and the optimal duration of interventions into the postoperative period.

Key words: aortic aneurysm; cerebrospinal fluid; radiography; interventional; spinal cord injuries; vascular surgical procedures

Editor’s key points

• Spinal cord injury occurs in 6.3% of patients undergoing repair of type II aortic aneurysms and 1–10% of patients undergoing endovascular repair of the thoracic aorta.
• The maintenance of an adequate blood pressure both during and after surgery is critical to maintaining spinal cord perfusion.
• Cerebrospinal fluid drainage to avoid pressures above 10 mm Hg is an effective strategy for preventing spinal cord injury but should be reserved for high-risk patients.

A persistent neurological deficit, such as paraplegia or paraparesis, secondary to spinal cord injury (SCI) remains one of the most feared complications of surgery on the descending thoracic or abdominal aorta. This is despite sophisticated advances in diagnostic and interventional strategies, including high-resolution three-dimensional imaging and a transition to less invasive endovascular procedures, including customized fenestrated endovascular aortic graft prostheses. The patients at highest risk are those undergoing extensive thoracic aortic repair for ruptured aneurysm or dissection. The broad class of aneurysm is often described by the Crawford classification, which relates to the origin and distal extent in the thoracoabdominal aorta, with type II being the most extensive (Table 1). In the past, estimates for the incidence of SCI were up to 31% of those undergoing open surgical repair of type II aneurysms, with rates even higher for those with aortic dissection.1,2

Specific strategies to protect the spinal cord focused on minimizing the cross-clamping time and the use of intercostal artery reimplantation. With a greater focus on spinal cord protection, including the use of cerebrospinal fluid (CSF) drainage and increased cardiopulmonary bypass, this rate has decreased to 6.3% for type II aneurysms in open surgery. Although arguably best practice, this figure is still significant, and it was hoped that endovascular techniques might provide some advantage by being less invasive. Ischaemic SCI with permanent dysfunction still occurs in 1–10% of patients published series for thoracic endovascular aortic repair (TEVAR).3 Endovascular aortic procedures are becoming increasingly common, replacing open surgical repair in the majority of instances involving aneurysm resection and thoracic aortic thrombosis surveillance.
or dissection arising distal to the aortic arch. Paraplegia may also follow infrarenal abdominal aortic aneurysm surgery, although for isolated open or endovascular aortic repair (EVAR) it is much less common (<0.25%). Although surgery on the thoracoabdominal aorta is associated with a broad range of significant complications, this paper will focus on perioperative strategies for SCI prevention in TEVAR in particular, using a specific patient as an example.

**Spinal cord protection**

Spinal cord ischaemia is clearly the result of a compromise of perfusion, with neurological injury occurring primarily because of profound acute ischaemia or as a consequence of more prolonged insufficiency with or without reperfusion injury. Perfusion insufficiency may be secondary to restriction of segmental arterial inflow during and after surgery, increased tissue pressure attributable to oedema or elevated CSF pressure, or increased venous pressure limiting outflow. The extent of endograft coverage gives an indication of risk, and extensive covered stent placement from the thoracic aorta to the iliac arteries is a risk factor (Fig. 1). Many strategies have been described either to maintain spinal cord perfusion during and after the procedure or to protect the spinal cord against ischaemic or reperfusion injury. Some methods apply to open repair only (e.g. selective intercostal reimplantation) and others are applicable to closed (endovascular) repair. Not all are consistently effective, and most rely on ‘bridging’ a period of ischaemic risk until adequate native perfusion can resume. Staged repairs have been reported to be of benefit in some retrospective series, presumably because this allows time for collateral blood vessels to develop and stabilize over smaller regions of the cord at risk of ischaemia. For detailed recommendations that have recently been published, the reader is directed to a Position Statement by the European Association for Cardio-Thoracic Surgery after a wide review of the literature. Identification of the at-risk patient is an inexact science because of variability in individual anatomy, the extent of endograft coverage, and the location and complexity of endograft placement, in addition to the risk of compromise of spinal cord perfusion by blood pressure variability.

The following sections provide an overview of the various perioperative protective strategies, spinal cord anatomy and physiology. The risks of the individual protective strategies also need to be considered.

**Minimizing the anatomical disruption of blood supply**

It has become evident that the blood supply to the spinal cord is not simply dependent on a few key feeding vessels. Recent reviews and studies have emphasized that there is a rich anatomical network of small vessels surrounding the cord that contributes to the usually single anterior and usually paired posterior spinal arteries. The superior source vessels are branches from the left subclavian and vertebral arteries, which form the anterior and posterior spinal arteries. Throughout its length the anterior spinal arteries receive supply from the paired intercostal and lumbar segmental arteries and then caudally from branches of the inferior mesenteric, internal iliac, and sacral arteries. This rich network will be variably compromised by the anatomical disruption caused by the aortic pathology itself, by the operative ischaemic time, and by the persisting compromise after surgery. An additional concern is that reverse flow from spinal arteries may both contribute to extraprosthesis leaks after the placement of sealed or occlusive stent grafts (type II endoleaks) and ‘shunt’ blood from the spinal circulation by a low-resistance pathway. For these reasons, coiling of branch vessels is sometimes undertaken. Although this decreases the risk of an endoleak, the impact on SCI is less clear.

Preservation of vessels, selective re-anastomosis, or side-branch stenting of these larger supply vessels (including the artery of Adamkiewicz, an often large spinal artery in the lower thoracic to upper lumbar region) has controversial benefit.

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**Table 1** Spinal cord ischaemia outcomes (percentage incidence) related to the Crawford classification of thoracoabdominal aneurysm extent for large series open procedures. *A Type V variant is also described (distal DTA to suprarenal). AA, abdominal aorta; DTA, descending thoracic aorta.

<table>
<thead>
<tr>
<th>Crawford classification</th>
<th>Aneurysm alone (Svensson and colleagues)</th>
<th>Dissection with or without aneurysm (Svensson and colleagues)</th>
<th>Aneurysm (dissection not specified) (Coselli and colleagues)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n=1234)</td>
<td>(n=276)</td>
<td>(n=2286)</td>
</tr>
<tr>
<td>Type I (proximal DTA to suprarenal AA; %)</td>
<td>13</td>
<td>21</td>
<td>3.3</td>
</tr>
<tr>
<td>Type II (proximal DTA to infrarenal AA; %)</td>
<td>31</td>
<td>33</td>
<td>6.3</td>
</tr>
<tr>
<td>Type III (distal DTA to infrarenal AA; %)</td>
<td>6</td>
<td>13</td>
<td>2.6</td>
</tr>
<tr>
<td>Type IV (suprarenal AA to distal AA; %)</td>
<td>4</td>
<td>11</td>
<td>1.4</td>
</tr>
</tbody>
</table>
Maintenance of perfusion pressure gradient

Probably the single most important strategy is the maintenance of an adequate perfusion pressure gradient to the spinal cord. This involves three elements: (i) maintaining an adequate mean systemic pressure; (ii) decreasing systemic venous pressure; and (iii) avoiding increases in CSF pressure surrounding the spinal cord. The patient we describe predominantly demonstrates the influence of these factors. It is important to realize that vulnerability to perfusion inadequacy persists into the post-operative period. Dependence on an adequate systemic perfusion pressure may last for much longer, and well after hospital discharge, depending on the nature of the collateral circulation, although factors other than relative hypotension may be involved. Overall, maintenance of an adequate blood pressure during the procedure and into the recovery period is critically important.

Cerebrospinal fluid drainage is effective because acute changes in the spinal cord in response to ischaemia or reperfusion may result in oedema and increased CSF pressures during the procedure and for 48–72 h (or even longer) afterwards. The effectiveness of CSF drainage in reversing SCI signs and reducing SCI overall has been demonstrated both anecdotally and in a randomized trial. It has been recommended that CSF pressures >10 mm Hg be avoided for at least 48 h after the procedure, that CSF drainage should occur if signs of SCI develop, and that excessive drainage be avoided. It should be remembered that lumbar CSF drainage catheters carry their own risks; therefore, this intervention should be reserved for patients judged to be at high risk or in whom symptoms or signs develop.

Protection of the spinal cord against ischaemia or reperfusion injury

Hypothermia has been advocated to provide some degree of acute tolerance of the spinal cord to interruption of blood flow during surgery, especially during open procedures. Hypothermia may be achieved systemically (e.g. using the cardiopulmonary bypass pump) or by epidural perfusion with cold fluids. Although hypothermia is demonstratively effective in enabling the brain and spinal cord to tolerate ischaemia, its role in the post-operative period is less clear. Systemic hypothermia also carries with it a number of risks, including dysrhythmia, coagulopathy, and metabolic disturbances, which must be balanced against potential benefits, although these effects are minimal with moderate (34°C) systemic hypothermia.

The use of selective spinal cord hypothermia using epidural cooling techniques has also been advocated. Limited published data in TEVAR are available, although case series in open procedures suggest a possible benefit. These studies also often combine other protective techniques. The efficacy of cooling can be monitored by CSF temperature. It is, however, an invasive technique, applicable for a limited duration, and concerns regarding contamination and rebound spinal cord oedema have been raised.

Pharmacological interventions aim to decrease the metabolic requirements of the spinal cord or to decrease the inflammatory or neurochemical responses to ischaemia or reperfusion. Steroids and naloxone have shown benefits in animal studies in reducing the effects of SCI and are reported in some series as components of multimodal therapies. Intrathecal papaverine has been associated with decreased adverse SCI outcomes as part of a multifaceted approach to protection, but awaits prospective trials. Other interventions, such as remote ischaemic preconditioning, may show promise in the future.

Monitoring for spinal cord ischaemia

Monitoring of spinal cord function is of primary relevance in the anaesthetized patient during surgery. Monitoring may be functional (e.g. somatosensory or motor-evoked potentials), metabolic (CSF analysis), or physiological (lumbar CSF pressure and paravertebral muscle oximetry). The purpose of monitoring is to enable an intervention to occur that would improve outcomes. The use of somatosensory and motor-evoked potentials may identify a need during open procedures to reimplant significant spinal arteries, although the sensitivity and specificity of abnormal evoked potential findings is not clearly established because they assess different aspects of spinal cord function and may be affected by lower limb ischaemia. During TEVAR procedures, the options in response to somatosensory or motor-evoked potential changes are more limited and mostly confined to increasing systemic perfusion pressure or drainage of CSF.

The nature of perfusion via an anastomotic network of vessels that also supply the paravertebral muscles has led to an interest in near-infrared spectrometry as a means of monitoring the adequacy of spinal cord perfusion during and after surgery. After surgery, clinical monitoring of lower limb motor and sensory function, in addition to bowel and bladder continence, are important and may indicate a need for blood pressure support or lumbar CSF drainage depending on the circumstances.

Case study

Mrs J.D. was 67-yr-old woman with severe vascular disease and new onset back pain presenting for a multibranch TEVAR for a symptomatic Crawford type IV thoracoabdominal aneurysm. Fifteen years earlier, she had undergone an open infrarenal abdominal aortic aneurysm repair with a bifurcated Dacron graft. This was complicated by bowel ischaemia requiring an ileostomy. She had also had a lumbar laminectomy in 1980. She was high cardiovascular risk, having had a myocardial infarction (ST-segment elevation) in 2006 followed by percutaneous coronary intervention with stent and a non-ST segment elevation myocardial infarction 6 yr later followed by another percutaneous coronary intervention with stent. She had ceased smoking in 2003. Her exercise tolerance was limited by dyspnoea at <4 metabolic equivalents, but she had no claudication and was still working. She also had hypertension, hypercholesterolaemia, and primary biliary cirrhosis. Her current medications included aspirin (100 mg day

−1), clopidogrel (75 mg day

−1), ceased 7 days before surgery), metoprolol (100 mg twice a day), irbesartan (300 mg day

−1), and prednisolone (5 mg day

−1).

Investigation revealed a 6.5 cm lower thoracic and suprarenal aortic aneurysm with computed tomography (CT) scan. Cardiac studies showed an ejection fraction of 40%, akinesis of the left ventricle posterior and inferior wall, but no reversible ischaemia with thallium scanning. Liver function was mildly impaired, but renal function and coagulation studies were normal. She had mild emphysematous changes on chest X-ray. On examination, she weighed 50 kg, with multiple abdominal scars and severe kyphoscoliosis. Her abdominal aneurysm was tender to palpation.

The plan was for her to have a two-piece endograft. The first piece lined the distal thoracic aorta and second piece consisted of a four-branch endograft (Cook Medical, Brisbane, Queensland, Australia) with branches to the coeliac axis, superior mesenteric artery, and both renal arteries. The graft also included a reperfusion branch, which was to remain patent for up to a month after surgery to allow perfusion of the aneurysm sac, hence the spinal arteries (especially T11–T12). Coverage was planned to extend from the mid-thoracic aorta to a distal seal zone in the aortic
component of the original infrarenal aortic graft. The postoperative CT result can be seen in Fig. 1.

In addition, Mrs J.D. had diffuse stenosis of her external iliac arteries that rendered the delivery and deployment of the endografts problematic. Therefore, a left iliofemoral Dacron graft was to be placed via open extraperitoneal exposure to facilitate introduction of the devices. Open exposure of the left axillary artery was necessary to allow branch cannulation, and right femoral arterial access by percutaneous sheath was required for preoperative angiography to place the endografts accurately. Owing to the extensive coverage of her mid- and distal thoracoabdominal aorta and coverage of the infrarenal aorta, she was deemed to be at high risk of spinal cord ischaemia; therefore, a lumbar CSF drain was planned.

Preparation for anaesthesia included radial arterial blood pressure monitoring, wide-bore i.v. access, and a central venous catheter. The lumbar CSF drainage catheter was inserted at L3–L4 (presumed) interspace using a 19 gauge multi-hole nylon epidural catheter via a 16 gauge Tuohy needle (Portex®; Smiths Medical Australasia Pty. Ltd, Brisbane, Queensland, Australia). This was placed successfully in one pass, with 10 cm of catheter threaded into the subarachnoid space. This was attached to a Codman drainage kit (EDS 3® CSF External Drainage System; DePuy Synthes, North Ryde, NSW, Australia). Induction of anaesthesia was uneventful; she was intubated and ventilated throughout the procedure, and maintained on sevoflurane with a total of 600 μg of fentanyl during the 7 h procedure.

Spinal cord protection involved maintaining the CSF pressure at 12 cm H2O with the catheter on ‘overflow’ at this level. The slow free drainage of CSF could thus be observed, and any increase in CSF drainage would be detected by an increased rate of production, with the pressure self-regulating to the set height of overflow. Cerebrospinal fluid drained at ∼8 ml h−1 throughout the procedure. Blood pressure was supported throughout the procedure using a phenylephrine infusion, aiming for a mean arterial pressure >80 mm Hg. Heparin was administered intermittently to maintain the activated clotting time >200 s.

The procedure itself was technically challenging but achieved a satisfactory result, with all branch targets perfused. A small posterior perfusion branch was part of the graft design, effectively to create an endoleak to allow temporary perfusion of spinal artery branch vessels via the sac. The patient was extubated and transferred to the intensive care unit awake and alert, for ongoing observation and management.

During the next 14 h, she became haemodynamically unstable, requiring norepinephrine (up to 11 μg min−1) and a total of 8 units of packed red cell transfusion. She was neurologically stable throughout. A CT scan identified a retroperitoneal haematoma around the site of the iliofemoral anastomosis, so she was returned to theatre for exploration, drainage, and oversewing of an anastomatic leak under GA. On return to the intensive care unit, she was extubated within 2 h and again neurologically intact. The CSF drain was kept at an overpressure of 12–15 cm H2O and drained <8 ml h−1.

On transfer to the ward the next day, hourly neurological observations continued, and the CSF drain was clamped and released every 6 h for pressure checks. At 36 h after the procedure, her CSF pressure reading was 24 cm H2O, with no neurological signs. Five millilitres of CSF was drained, and the pressure decreased to 16 cm H2O. On review 6 h later, she complained of pain in her left groin and ‘heaviness’ in her left leg, although power and objective sensory assessment was normal. The CSF pressure was 11 cm H2O. The CSF drain was removed uneventfully at 48 h after the procedure. Haemodynamics were normal. An increase in high-sensitivity Troponin-I (hs-TnI) had been noted as 230 ng l−1 at 24 h, which decreased to 118 ng l−1 by 48 h. The ECG was unchanged. She was mildly dyspnoeic, with a haemoglobin of 87 g l−1, but otherwise able to mobilize.

During the next few days, she was assessed as mildly fluid overloaded and diuresed. Her left leg was persistently noted to be mildly weak. The patient was able to weight bear, but pain limited attempts at ambulation. A CT scan on postoperative day 6 showed no endoleak and all branch vessels patent. On recommencing ibersartan she became posturally hypertensive (90/60 mm Hg) and so this was ceased. Haemodynamics then stabilized and ambulation slowly improved. From day 8 to 10 she became progressively hypertensive, and her antihypertensive medications were adjusted accordingly. She also complained of back pain and left flank pain.

At 19.30 h, 11 days after the procedure, she became diaphoretic, light headed, hypertensive (80/40 mm Hg) and bradycardic (45 beats min−1). Shortly after this she lost motor power in both legs and lost sensation below the umbilicus. She was commenced on i.v. epinephrine to support her circulation, and urgent magnetic resonance imaging was ordered to assess the spinal canal (for haematoma), which was not useful because of the metal stent material. A CT scan showed graft patency, with the exception of occlusion to the branch to the left kidney. There was no endoleak. The patient was transferred to the intensive care unit, where her troponin was 20 952 μg l−1. The ECG was suggestive of anterior ischaemia. Discussion occurred between the vascular and cardiology teams regarding the need for anticoagulation, which would preclude the insertion of a new lumbar CSF drain. During this time, her haemodynamics had stabilized on the epinephrine infusion and her sensory and lower limb motor function was returning to normal. It was decided that her spinal cord perfusion was pressure dependent; thus, in light of an acute coronary syndrome, she would be anticoagulated with heparin and tirofiban. She was taken to the cardiac catheter laboratory, where her mid-right coronary stent was found to be thrombosed. This was reopened, and she was commenced on ticagrelor. Her hs-TnI peaked at 47 647 μg ml−1. She was weaned of inotropic support during the next 24 h and discharged to the ward.

She remained in hospital for a further 10 days, which were relatively uneventful, before being discharged home neurologically intact.

Case study discussion
The management of the patient described highlights several points regarding spinal cord protection in patients undergoing TEVAR procedures.

Assessment of risk
The presence of a previous infrarenal surgical aortic bifurcation graft and iliac vascular disease meant that the distal cord perfusion was already compromised. Extensive coverage of spinal arteries by the proposed branched TEVAR meant that cord perfusion was dependent on proximal vessels and collateral systems distally. However, this was a ‘staged’ procedure; therefore, collateral vessels might have developed or enlarged since the original procedure.6

Plan for protection
A lumbar CSF drain was placed, arterial pressure was supported throughout and into the first 24 h, the left subclavian artery was preserved, and finally, the graft itself contained a patent perfusion branch to maintain perfusion of the sac and intercostals in
the early postoperative phase until development of collaterals. This perfusion branch thrombosed within 2 weeks, so should not be considered as a long-term solution, especially as the overarching surgical aim was to depressurize and thrombose the aneurysm itself.

**Choice of lumbar drain type**

A number of options exist for percutaneous lumbar CSF drains, although the most common choices are between silicone (silastic) catheters and epidural catheters. Either choice should involve a catheter with multiple distal orifices to minimize the risk of obstruction. The insertion depth is typically 8–10 cm. There is no clear advantage to one over the other, with silastic catheters being softer and larger bore but also more prone to kinking, shearing, or breaking, and requiring a larger (e.g. 14 gauge) insertion needle.

**Monitoring environment**

During the procedure, our institution does not use evoked potential monitoring. The aim is for an alert, responsive patient immediately after surgery who can comply with early and frequent neurological assessment in a high-dependency unit environment. These patients should ideally be monitored closely in a high-dependency unit for 48 h or longer, but resource limitations often mean that a stable patient may be discharged to the ward after 24 h. Hourly neurological observations should continue, however.

**Duration of spinal cord drainage**

This was continued for 48 h because she had been asymptomatic, although it could be argued that continued drainage should be provided for a further 24 h, considering the one episode of (asymptomatic) elevated CSF pressure.

The subjective left leg 'heaviness' in the first postoperative week was of concern. However, there was clinically little to find, and the presumption at the time was that this was because of discomfort from the open iliac procedure and residual haematoma from the subsequent anastomotic leak.

**Risk of late cord ischaemia**

This situation was contributed to by hypotension secondary to myocardial infarction and tachyarrhythmia and by thrombosis of the deliberate residual endoleak. An additional concern was late haemorrhage.

There are challenges with imaging the cord because although CT angiography can identify vessel blood flow, the most sensitive technique for spinal cord ischaemia is magnetic resonance imaging, which is made difficult if not impossible by the metallic stent skeleton components of the endograft, even though the device was magnetic resonance imaging 'compatible'.

For this patient, there was an additional difficult decision relating to the potential need for anticoagulation and antiplatelet therapy as after percutaneous coronary intervention for her myocardial infarction. This had to be balanced against the potential benefit of re-inserting a spinal cord drainage catheter. Fortunately, return of motor and sensory function in response to pressure support decreased the imperative for the CSF drain.

Overall, the description of management of this patient emphasizes how marginal the spinal cord blood supply can be, even in the absence of symptoms, and that this vulnerability may persist for some time.

**Conclusion**

In TEVAR procedures, if spinal cord perfusion is severely compromised, recovery may not be possible. Planning is important, and reliance on staged grafts may not be sufficient. However, there is good evidence that short-term strategies that allow for recovery of adequate ongoing perfusion, presumably by the development of collateral blood vessels, are effective. Careful monitoring and assessment and prompt early intervention should symptoms or signs of cord ischaemia develop is critical. Perfusion compromise may occur de novo even weeks after the procedure.

**Authors’ contributions**

Patient procedural management and perioperative care: D.A.S., M.J.D.

Manuscript preparation, editing and review: D.A.S., M.J.D.

**Declaration of interests**

None declared.

**References**


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