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Spinal Cord Injury after Thoracic Endovascular Aortic Aneurysm Repair

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Abstract

Purpose—Thoracic endovascular aortic aneurysm repair (TEVAR) has become a mainstay of therapy for aneurysmal and other disorders of the thoracic aorta. The purpose of this narrative review article is to summarize the current literature on the risk factors, pathophysiology of spinal cord injury (SCI) following TEVAR, and to discuss various intraoperative monitoring and treatment strategies.

Source—Articles considered in this review were identified through PubMed using the following search terms: thoracic aortic aneurysm, TEVAR, paralysis+TEVAR, risk factors+TEVAR, spinal cord ischemia+TEVAR, neuromonitoring+thoracic aortic aneurysm, spinal drain, cerebrospinal fluid drainage, treatment of spinal cord ischemia.

Principal findings—Spinal cord injury continues to be a challenging complication after TEVAR. Its incidence after TEVAR is not significantly reduced compared to open TAAA repair. However, compared to open procedures, delayed paralysis/paresis is the predominant presentation of SCI after TEVAR. The pathophysiology of SCI is complex and remains not fully understood though the evolving concept of the importance of the spinal cord's collateral blood supply network and its imbalance after TEVAR is emerging as a leading factor in the development of SCI. Cerebrospinal fluid drainage, optimal blood pressure management, and newer surgical techniques are important components of the most current spinal cord protection strategies.

Conclusion—Further experimental and clinical research is needed to aid in the discovery of novel neuroprotective strategies for protection and treatment of SCI following TEVAR.

Introduction

Over the last decade, thoracic endovascular aortic repair (TEVAR) has increasingly become a mainstay of therapy for thoracic aortic diseases, such as aneurysm and acute aortic syndromes. Despite being less invasive than open surgical repair, TEVAR procedures are still associated with significant risk of spinal cord injury (SCI). Crawford was the first to describe a thoracoabdominal aortic aneurysm (TAAA) classification system in 1986. The classification system, developed when only open repair was being performed, was based on

the extent of the aorta involved, but also showed a relationship to the relative incidence of SCI. Subsequently, the incidence of SCI after TEVAR has generally **demonstrated** a similar incidence to that after open TAAA repair when compared to a similar Crawford class (Figure 1).[1] The overall incidence of SCI (i.e., paresis or paralysis) after TEVAR ranges from 2–10%. [2, 3] For example, in 2011, Ullery *et al.*[4] reported 2.8% incidence of SCI in a study of 424 TEVAR procedures. DeSart *et al.*[5] reported 9.4% of SCI in 607 TEVAR procedures in 2013, and in 2014, Scali *et al.*[6] showed an incidence of 9.2% in 741 TEVAR procedures.

The pathogenesis of SCI after TEVAR is multifactorial and is not fully understood. There are two suggested theories behind the mechanism of SCI. The first relates to inadequate remodeling of the collateral blood supply network to maintain spinal cord viability. The second implicates atheroembolism **of** aortic plaque material into the segmental arteries supplying the spinal cord. Although there is considerable debate circulating in the literature on the optimal preventive and definitive treatment measures for SCI after TEVAR, the main strategies are aimed at increasing mean arterial blood pressure and draining cerebrospinal fluid (CSF) in order to optimize spinal cord perfusion pressure (and blood flow). The ACCF/AHA guidelines **(though not updated since 2010)** recommend CSF drainage for spinal cord protection in open TAAA repair and TEVAR for patients at high risk of SCI determined by the presence of specific risk factors (**e.g., prior abdominal aortic surgery**).[7] **In** 2015, the European Association for Cardiothoracic Surgery **similarly** recommended CSF drainage for TAAA repair **for** patients at high risk for SCI.[8] Although randomized controlled studies showed benefit for CSF drainage in open TAAA repair,[9] its routine use in TEVAR procedures remains **uncertain and a topic of considerable debate**.[8, 10, 11]

The aim of this narrative review is to provide an overview of the pathophysiology, risk factors, and clinical presentation of SCI following TEVAR, as well as to highlight the relevant anesthetic and newer surgical techniques for its prevention and treatment.

Risk of SCI in TEVAR

The advantages of the relatively lesser invasive TEVAR over TAAA open repair are consistent with what one might expect from the avoidance of the extensive surgical trauma **consequent** with open TAAA repair. They include reductions in intensive care unit length of stay, hospital length of stay, organ dysfunction, postoperative pain, and overall costs.[12, 13] However, a similar reduction has not been seen in the incidence of SCI. To estimate the risk of SCI in TEVAR, Uchida reviewed 59 studies with a total number of 7309 TEVAR patients. [14] He demonstrated a 0–10.3% SCI range, with an average of 4.5%. Recently, Etz *et al.*[3] analyzed the risk of paralysis reported by “aortic centers of excellence” from 6 clinical trials of TEVAR that included 1050 patients (between 2001 and 2008) and 10 clinical trials with open TAAA repair in 6034 patients (between 2005 and 2011). They showed similar rates of paralysis in open and TEVAR procedures, especially for type II aortic aneurysm (0–22% in open repair vs 19% in TEVAR) (Figure 1). However, there was only one study that mentioned the risk of SCI in TEVAR in relation to the Crawford classification. In this retrospective study, the authors reviewed a total of 724 thoracic or TAAA repair procedures (352 TEVAR patients and 372 open repair patients); they did not show a significant

difference in the incidence of SCI between both procedures (4.3% for TEVAR vs 7.5% for open repair, $P = 0.08$).[15] Indeed, when compared to open repair, not only is the reduction of SCI and paralysis less than expected, delayed (i.e., greater than 24 hours following the procedure) paralysis occurred more frequently than acute paralysis [4, 10, 16]

The limitation of the current literature in estimating the risk of SCI after TEVAR compared to that after open repair is due to the lack of direct comparative prospective studies. In addition, the indications for TEVAR are expanding to include patients at lower risk for the development of perioperative complications, which may bias recent results when compared to previous studies. Furthermore, potentially important patient demographic and surgical details are inconsistently reported in the clinical trials. Finally, the continued refinement of both the surgical techniques and the development of newer techniques (e.g., minimally invasive segmental artery coil embolization [MISACE] and temporary aneurysm sac perfusion [TASP]) may have an impact on the risk of SCI, which makes direct comparisons even more difficult. In the future, comprehensive systematic reviews and meta-analysis that account for the previously mentioned limitations may be able to more accurately estimate the relative risk of SCI in TEVAR patients.

Pathophysiology of Spinal Cord Injury after TEVAR

The blood supply to the spinal cord is arguably more complex than the blood supply to any other vital organ.[17] Its main blood supply comes from one anterior spinal artery and two posterior spinal arteries that run longitudinally along the cord. These anterior and posterior spinal vessels, originating from the vertebral arteries, are fed by additional collaterals from paired segmental arteries arising directly from the aorta with 2–3 in the cervical region, 2–3 in the thoracic region and 0–1 in the lumbosacral region.[18] In the thoracic region, these segmental arteries arise from the aorta or the subclavian artery as intercostal and lumbar arteries. The segmental arteries divide into muscular and spinal branches; the spinal braches give rise to anterior and posterior radicular arteries that join the anterior and posterior spinal arteries. The most dominant anterior radiculomedullary artery in the thoracolumbar area is known as the artery of Adamkiewicz (Figure 2). Although historically it was considered the most important vessel supplying the cord, this has largely been discounted in recent years.[19]

The concept of what constitutes the main blood supply to the spinal cord has evolved in recent years and has a direct impact on the understanding of the relationship between the blood supply of the spinal cord and SCI. In the past, there was a wide belief that the single artery of Adamkiewicz was the most important contributor to the development of SCI after aortic aneurysm repair.[20] Currently, however, there is growing experimental and clinical evidence suggesting that impairment in the collateral network of blood vessels in the spinal cord is more important to SCI than any single artery.[3, 21] This collateral network is an interconnected network of blood vessels between the anterior spinal artery (i.e., the intraspinous network) and the blood supply of the adjacent muscles of the back (i.e., the paraspinous network). There also exist multilevel connections within the intraspinous network with the adjacent - i.e., above and below any particular spinal cord segment. The arterial system of the spinal cord itself is divided into a central centrifugal system supplied by the sulcal arteries and a peripheral centripetal system supplied by the pial network

surrounding the spinal cord.[18] This interconnected network acts as an important alternate source of blood supply when a principal input is excluded, such as with segmental artery exclusion after thoracic stent deployment.[3, 21]

Aortic cross clamping during open TAAA repair results in proximal (to the clamp) hypertension, increased central venous pressure (CVP), increased intracranial pressure, and distal hypotension.[22, 23] The latter causes renal and/or intestinal ischemia as well as lower limb ischemia. The distal hypotension also directly impacts the blood supply to the spinal cord and reduces the spinal cord perfusion pressure, leading to ischemia. [23–25] Additionally, after aortic un-clamping, reperfusion can contribute to injury of the spinal cord through an associated inflammatory response.[26] Reimplantation of the segmental arteries arising from the excised aortic aneurysmal sac has been suggested to reduce SCI. However, there is considerable disagreement among vascular surgeons regarding the management of the segmental arteries in open TAAA repair. For example, many opt to re-implant all intercostal arteries into the aortic graft, while others only selectively re-implant critical segmental arteries or do not re-implant any of them.[27]

Because TEVAR does not involve the physiologic perturbations associated with aortic cross clamping or associated reperfusion injury to the spinal cord, it led to the expectation of significantly lower rates of paralysis. However, it does permanently exclude many of the segmental arteries that are occluded by a covered thoracic stent, which may have longer lasting effects **(than relatively brief periods of aortic cross-clamping during open repair)** on compromising spinal cord blood flow.[28]

Delayed paralysis is a more common presentation of SCI after TEVAR compared to immediate paralysis following open TAAA repair. A possible explanation for these differing clinical presentations is that the aortic cross clamp-mediated spinal cord ischemia (and direct ligation of intercostal vessels) and reperfusion injury may contribute more to immediate ischemia. The fact that the aneurysm sac (with its intercostal tributaries) is left intact during TEVAR may contribute to the development of delayed SCI following the eventual cessation of stent endoleak. However, evidence on the exact pathophysiologic differences is lacking.

With deployment of a covered stent during TEVAR, *collateral network remodeling* is thought to play an important role in maintaining spinal cord blood flow by changing the distribution of blood flow in the intraspinous and paraspinous collateral network after segmental artery exclusion.[29] Rerouting of blood flow from the paraspinous to the intraspinous network helps to maintain spinal cord viability and function (Figure 2). This is achieved by enlargement in the diameter of the intraspinous network, and a change in the orientation of the vessels of the paraspinous network from relatively perpendicular to a parallel alignment to the spinal cord, which results in a greater percentage of the mean aortic pressure being transmitted to the spinal cord. Etz *et al.*[29] described these changes in an experimental porcine model, and demonstrated that this remodeling occurs over **the** two to five days **after** surgery. In addition, Colman *et al.*[19] suggested that perfused non-occluded segmental arteries (either proximal or distal to the covering stent) can maintain blood flow to the ischemic spinal cord segment by reversing the direction of blood flow within the anterior spinal artery, through an antegrade direction from the proximal arteries or retrograde

direction from the distal segmental arteries. If all these compensatory mechanisms are insufficient or fail secondary to thrombus formation in the intraspinous or paraspinous network, embolism or cessation of stent endoleak, spinal cord ischemia **(i.e., frequently delayed)** can occur (Figure 2).[30] The onset and severity of injury after TEVAR depends on the ability of the collateral network to supply the marginally vascularized area in the critical zone of the spinal cord known as the watershed area in the grey matter of the spinal cord.[21, 28, 31, 32]

Another emerging factor involved in the pathogenesis of SCI after TEVAR is atheroembolism from aortic plaque that can be dislodged during the surgical procedure (from manipulation of guidewires and stent deployment) occluding segmental vessels.[33] Indeed, Zipfel *et al.*[34] described 11 patients with SCI in their cohort of 406 TEVAR patients attributing the cause of paralysis in 3 of these patients to atheroembolism after analysis of the computed tomographic scan of the aorta revealed presence of a mobile atheroma in one of these patients. The other two patients had a circular thrombus at the distal landing site, in addition to the unilateral clinical presentation of their paralysis, which further supported atheroembolism as the cause of paralysis. Tanaka *et al.*[35] examined in more detail the role of embolization in causing paralysis in 266 TEVAR patients, and classified spinal cord MRI findings into 3 types: *focal* (asymmetrical focal high intensity on axial T2-weighted images involving 2 or fewer segments of the spinal cord), *sporadic* (asymmetrical multiple high intensity on axial T2-weighted images involving more than 3 segments of the spinal cord, shown on Figure 3) and *diffuse* (symmetrical high intensity on axial T2-weighted images). In their series, based on microscopic examination of the anterior spinal artery and its branches from autopsy of 4 patients with SCI, they attributed sporadic and focal MRI findings to atheroembolism, while diffuse findings were attributed to hemodynamic instability.

Risk Factors for Spinal Cord Injury after TEVAR

Multiple risk factors for the development of SCI after TEVAR have been identified and can be divided into patient and surgical procedure risk factors (Table 1). Patient risk factors include advanced age (> 70 years old),[6] perioperative hypotension (e.g., mean arterial blood pressure <70 mmHg),[36–38] renal insufficiency (creatinine >132 umol/L), [4, 6, 39] COPD,[6] hypertension,[6] and degenerative aneurysms compared to non-aneurysm pathologies[40]. Renal insufficiency has been postulated as a marker of widespread peripheral atherosclerotic disease, which suggests that such patients may preoperatively have a compromised collateral network of blood supply to the spinal cord. On the other hand, the exact underlying mechanism of COPD as a risk factor for SCI is not fully understood.

Surgical risk factors include increased urgency of the procedure,[41] large extent of aortic coverage,[6, 38, 42–44] left subclavian artery coverage,[39] coverage of hypogastric arteries,[36, 40] the use of 3 or more stents,[39] extended duration of the procedure[36], and increased blood loss.[40] The extent of aortic coverage is a particularly important surgical risk factor.[6, 38, 42–44] Indeed, after reviewing 326 TEVAR patients, Feezor *et al.*[44] concluded that the risk of SCI is increased by 30% for every 2 cm of additional thoracic aortic coverage. Amabile *et al.*[43] showed that not only aortic coverage of greater than 205 mm is associated with increased

risk of SCI, but also the length of the thoracic coverage distal to the celiac artery is an important risk factor.

The vascular territories providing blood supply to the spinal cord are the left subclavian, intercostal, lumbar, and hypogastric territories. In a prospective observational single center study of 63 TEVAR patients, Czerny *et al.* [45] demonstrated a direct correlation between coverage of 2 or more of these vascular territories (positive predictive value, 0.67; 95% confidence interval [CI], 0.24 to 0.91; $P = 0.005$) and development of symptomatic SCI, especially when combined with prolonged intraoperative hypotension. They also showed strong agreement between the derived results when they applied them to the data from the multicenter European Registry on Endovascular Aortic Repair Complications (EuREC) cohort.[45] In addition, Martin *et al.*[46] retrospectively reviewed 261 TEVAR patients and reported that 18 of the 27 (67%) SCI patients in their cohort had history of either repaired or unrepaired abdominal aortic aneurysm (AAA) (odds ratio [OR], 3.57; 95% CI, 1.56 to 8.33; $P = 0.003$). This finding has also been shown in other studies.[38, 39, 47] The reason for the increased risk with prior repair AAA is likely due to the compromise of the pelvic and hypogastric collateral blood supply to the spinal cord.

Role of Left Subclavian Artery in SCI

The left subclavian artery provides blood supply to the brain and spinal cord via the left vertebral artery. In doing so, it contributes to the blood supplying the posterior aspect of the circle of Willis via the basilar artery (formed by the union of the left and right vertebral arteries). The left vertebral artery also provides one of the two branches that feed the anterior spinal artery (Figure 4).[48] In addition, the thyrocervical trunk arising from the left subclavian artery, supplies the cervical spinal cord through anterior and posterior radiculomedullary arteries (Figure 4).[49] More than 40% of patients undergoing TEVAR have thoracic aortic pathology in close proximity to the LSA, which requires its intentional coverage.[50] **C**overage **of the left subclavian artery** increases the risk of vertebrobasilar stroke and arm ischemia as shown in a meta-analysis by Rizvi *et al.*[51] (OR, 10.8; CI, 3.2 to 36.7; I^2=0% for stroke) and (OR, 47.7; CI, 9.9 to 229.3; I^2=72% for arm ischemia). Similar results were shown in a more recent meta-analysis by Waterford *et al.*[52] They demonstrated the overall stroke rate after **left subclavian** coverage to be higher compared to TEVAR procedures performed distal to the **subclavian** origin (7.4% vs 4.0%, P < 0.0001).

There are multiple surgical options to maintain flow within the **left subclavian artery** when its coverage is planned **during TEVAR** (Figure 5). Revascularization procedures include either transposition **of the subclavian** to the left common carotid artery or **inserting a** short bypass graft between the **left subclavian** and **the** left common carotid artery. However, these revascularization procedures themselves carry risk of complications (including brachial plexus, vagus nerve, left recurrent laryngeal nerve and thoracic duct injuries, neck hematoma, **subclavian** dissection, and stroke) that range from 10–12%.[53, 54] Newer endovascular techniques for preserving **subclavian** blood flow include the use of fenestrated/branched stent grafts, chimney and periscope grafts, sandwich techniques, or *in situ* fenestration techniques.[55] Fenestrated or branched stents are custom pre-made stents designed to allow perfusion of the **left subclavian artery**. Chimney grafts are inserted in the

subclavian artery parallel to the proximal wall of the aortic stent (Figure 6). The use of a periscope graft is another technical variation of the chimney graft but is inserted through the distal part of the aorta resembling an inverted chimney (Figure 7). Sandwich grafts are inserted between two aortic stents to avoid problems associated with gutters of the chimney grafts (Figure 8). This technique is used with visceral and iliac vessels, but was recently introduced to the aortic arch revascularization. *In situ* fenestration involves intraoperative creation of a fenestration (using a laser)[56] to perfuse the **left subclavian artery** after aortic stent deployment. However, the main concern about this technique is the interruption by the laser of the nitinol ring and the resulting lack of support at the edge of the fenestration; it would also affect the quality and durability of the stent.[55]

The importance of routine preoperative **subclavian** revascularization has been debated and some advocate only performing it in selected high risk patients. In the 2009 practice guidelines from the American Society of Vascular Surgery, routine preoperative **left subclavian artery** revascularization **was recommended** in elective TEVAR patients where subclavian coverage is anticipated to achieve a proximal seal. The guidelines also recommended routine preoperative revascularization in selected patients who have an anatomy that compromises perfusion to critical organs such as the brain and spinal cord.[57] Rizvi *et al.*[51] performed a meta-analysis on 3365 patients from 51 observational studies on TEVAR procedure with or without **left subclavian** coverage. Results from 8 studies showed a non-significant increase in the risk of SCI with LSA coverage (OR 2.69; CI, 0.75 to 9.68; I^2 = 40%). Recently, Patterson *et al.*[58] conducted a meta-analysis on 1002 TEVAR patients from the Medtronic Thoracic Endovascular Registry (MOTHER) data derived from five clinical trials and one institutional series and reported that **subclavian** coverage did not increase the risk of SCI (5%) compared to those without coverage (3%) (P = 0.16). They also showed a non-significant reduction in the incidence of SCI in patients who underwent **left subclavian** revascularization compared with those who did not (4% vs 1.4%, respectively (P = 0.186). Similar results were shown in more recent meta-analysis by Hajibandeh *et al.*[59] on 1161 patients from five observational studies. They did not show a significant difference in the rate of SCI between the **subclavian** revascularization and non-revascularization groups (2.7% vs 4.3%, respectively; P = 0.09). No updated guidelines regarding subclavian coverage have since been published.

Although debate continues on whether to perform *routine* preoperative subclavian revascularization, there are certain situations where *selective* revascularization is recommended.[39, 51, 53, 60–65] Indeed, absolute indications for preoperative revascularization that most authors agree upon include prior left internal mammary artery coronary bypass, presence of a left sided arterio-venous fistula for renal dialysis and left-hand dominant patients. Relative indications remain situations of dominant left vertebral artery, extensive aortic coverage, prior abdominal aortic aneurysm repair, and occluded hypogastric artery. Postoperative indications include upper limb ischemia or vertebrobasilar insufficiency.[51, 53, 66]

Role of Hypogastric Arteries in SCI

The internal iliac artery (also known as the hypogastric artery) provides blood supply to the spinal cord via radicular lumbosacral arteries.[48] Its sacrifice during open AAA repair or during endovascular aneurysm repair (EVAR) is a risk factor for SCI in TEVAR. During EVAR, for example, unilateral coil embolization of the hypogastric artery is done to allow the endograft to anchor in the healthy external iliac artery preventing hypogastric artery endoleak into the aortoiliac aneurysm. This unilateral embolization does not usually lead to complications due to a rich pelvic arterial network. However, bilateral occlusion is associated with an increased risk of pelvic ischemia. Manifestations of pelvic ischemia include buttock claudication, erectile dysfunction, and to a lesser extent, SCI.[67]

Further compromise of hypogastric blood flow can occur during TEVAR when an iliac artery graft is used to serve as a vascular access conduit in patients with severe atherosclerosis of the iliofemoral arteries. This has been associated with a higher risk of SCI where Khoynezhad *et al.*[40] reported 8 of 153 (4.3%) patients with SCI; 5 of the 8 SCI patients required an iliac conduit for the procedure.

Clinical Presentation of Spinal Cord Injury after TEVAR

The clinical manifestations of SCI after TEVAR vary between immediate (at emergence from anesthesia) and delayed onset (>24 hours after surgery), with the majority of the cases (66%) being of delayed onset.[10] SCI is a dynamic process and the clinical presentation varies between paresis of one limb to **full** paraplegia, with or without autonomic dysfunction. These manifestations may be either transient (with partial or full recovery) or permanent (**with** no regression).[5, 68] Mehmedagic *et al.*[69] conducted mid-term follow-up on 29 patients with in-hospital transient neurological deficits and documented full recovery of these deficits before discharge. They reported a permanent, though less severe form of SCI in a subset of these patients, which was overlooked, reflecting the importance of transient paresis on patient long-term quality of life.

Autonomic dysfunction

Three spinal cord elements are involved in cardiovascular function: the descending vasomotor pathways, sympathetic preganglionic neurons, and spinal afferents.[70] Cardiovascular dysfunction is common after SCI. It is known that once SCI occurs, there is damage to the motor and sensory neurons as well as the descending pathways that are involved in the autonomic control of the cardiovascular system; this can lead to hypotension and bradycardia (i.e., spinal shock).[70, 71] Cheung *et al.*[72] demonstrated that in a subset of their delayed paralysis patients, the recovery of neurological function was associated with a decrease in the required dose of vasopressors. This might be attributed to the recovery of the autonomic dysfunction associated with the resolution of the SCI, which explains that not only hypotension can exacerbate SCI, but it can also be a potential indicator of the early onset of SCI when the autonomic system is affected. The relationship between hypotension secondary to ischemic spinal cord autonomic dysfunction and paralysis in TEVAR is a potential opportunity for further investigation.

Spinal Cord monitoring during TEVAR

Monitoring spinal cord function during TEVAR can assist in detecting pathologically relevant changes in the functional integrity of neuronal pathways; it has the potential to direct clinicians to the need for intraoperative intervention. That said, few centers report performing intraoperative spinal cord neuromonitoring data during TEVAR. In addition to the costs associated with the necessary equipment, its relatively low rate of use compared to open procedures can be attributed to the lack of aortic cross clamping, the relatively short duration of the procedure, the requirement for expert interpretation, and the often delayed nature of paralysis. Despite evidence-based guidelines suggesting a benefit to intraoperative spinal cord monitoring in spine surgery,[73] the role of neuromonitoring in TEVAR is still evolving. The latest AHA guidelines suggest considering neurophysiological monitoring of the spinal cord during both open TAAA repair and TEVAR for prevention and/or treatment of SCI. However, the level of evidence is only class IIb [i.e., usefulness or efficacy is less well established by evidence/opinion] (Table 3).[7] Additional studies are needed to examine the full potential of neuromonitoring in diagnosis and prevention of SCI and paralysis in TEVAR.

Motor and Sensory Evoked Potentials

Motor evoked potential (MEP) monitoring is used to evaluate the descending spinal pathways, while somatosensory evoked potential (SSEP) monitoring evaluates the ascending pathways. Hence, they can be considered as useful tools in diagnosis and management of SCI during TEVAR.[74] Weigang *et al.*[74] reported loss or changes in the SSEP and MEP in 11 of 31 (35%) patients undergoing TEVAR. They described recovery of these changes in 10 of the 11 (91%) patients after they intervened by increasing the blood pressure and draining CSF. However, the remaining patient did not respond to these intraoperative interventions and interestingly, did not have any postoperative neurological deficits (i.e., false positive). It is worth mentioning that they also reported that one patient with delayed paresis had intraoperative MEPs and SSEPs that were unchanged (i.e., false negative). Similarly, Schurink *et al.*[75] reported >50% reduction in MEPs in two of ten (20%) patients undergoing TEVAR. In those two patients with MEP reductions, the procedure was carried out in stages in order or to minimize the risk if SCI by allowing sufficient time for the collateral blood supply network to adapt. One of the other eight patients without MEPs changes had temporary right lower leg paresis. Recently, Banga *et al.*[76] observed MEP and/or SSEP changes in 31 of 49 (63%) TEVAR patients. The changes were restored to the baseline in 12 patients after intervention (i.e., CSF drainage and blood pressure elevation). In 18 of their other patients, after intervention and restoration of lower limb blood flow, the overall rate of SCI was 6% (3 patients). The remaining patient presented with immediate paralysis, in which intraoperative evoked potential changes were unable to be restored. Two of the patients presented with delayed SCI.

Thus, the use of monitoring evoked potentials during TEVAR is useful in the detection of SCI. However, it is not clear if patients with observed evoked potentials will actually manifest with postoperative SCI if no intervention was performed. Inability of the evoked potentials to differentiate mild from severe SCI is also one of their limitations. In addition,

the vascular access needed for introduction of the stent during TEVAR may result in leg ischemia, limiting the use of SSEP and MEP monitoring from the ipsilateral leg.[77] For these reasons, the current available literature does not appear to support its routine use.

Effect of Anesthesia on Evoked Potentials

During general anesthesia, volatile anesthetics affect the cortical waveforms by increasing cortical latency and decreasing SSEP amplitude. Therefore, for practical purpose, volatile anesthetics should be kept at no more than half the minimum anesthetic concentration (MAC).[78, 79] Isoflurane at concentrations of 0.75 and 1.0 MAC produce good MEP responses in only 61% and 8% of the patients, respectively after multi-pulse transcranial electrical stimulation.[80–82] Similarly, sevoflurane at 0.5 and 0.75 MAC produce adequate responses in only 55% and 10% of the patients, respectively, when using dual-pulse electrical stimuli. Although propofol suppresses the alpha motor neuron in the grey matter of the spinal cord, serum concentrations kept at or below 1 μg/mL generally allow a MEP response in most patients. In addition, opioids used at low-dose in continuous infusions do not generally affect MEP responses. The use of neuromuscular blocking agents should be closely monitored to provide muscle relaxation and an adequate MEP response; single twitch height (T1) of 20% to 50% of the baseline can achieve this. Thus, the preferable anesthetic protocol during evoked potential neuromonitoring should lean heavily towards total intravenous anesthesia (TIVA) –e.g., with propofol and remifentanil – with only low concentrations of volatile agents, if at all.[83]

Near-infrared spectroscopy (NIRS)

The use of near infrared spectroscopy (NIRS) in TEVAR in an attempt to detect SCI was first reported in 2011.[84] In this report, NIRS optodes were placed over the surface of the thoracic and lumbar paraspinous muscles of two patients, providing real time measurement of blood oxygen saturation in the paraspinous collateral network (which shows a strong direct association to spinal cord microcirculation). The first patient demonstrated a decrease in the spinal cord NIRS signal after deployment of thoracic aortic stent, which improved after augmenting MAP and draining CSF. The second patient developed SCI, which was consistent with the reduction in the NIRS signal that did not recover after any intervention. Von Aspern *et al.*[85] compared measurements of paravertebral muscle oxygenation using NIRS to direct measurements of the spinal cord oxygenation and microcirculatory flow by laser Doppler flowmetry in seven juvenile pigs. There was a significant positive correlation ($r = 0.51$ to 0.52; $P < 0.001$) between measurements of both devices and there was no significant difference between lumbar paraspinous muscle oxygenation and direct spinal cord oxygenation ($P = 0.30$). Thus, at least in theory, NIRS monitoring of the paraspinous muscles may indirectly reflect oxygenation in the spinal cord. However, this technique has not yet been validated clinically. [3, 85, 86]

Spinal Cord Neuroprotective strategies in TEVAR

Spinal cord protection during TEVAR requires a team approach during the perioperative period including input from the anesthesiologists, surgeons, and ICU team members. Anesthesiologists are responsible for optimizing spinal cord perfusion throughout the

procedure. Surgeons have to make perioperative decisions regarding staging of the procedure and management of the left subclavian and hypogastric arteries. Other new surgical techniques of minimally invasive segmental artery coil embolization and temporary aneurysm sac perfusion have also been introduced to minimize risk of SCI (Table 2).

Perioperative Anesthetic Management

General Measures

Ensuring adequate oxygenation and perfusion by maintaining a normal cardiac index and adequate hemoglobin - though the precise limits are not well defined - are important measures for spinal cord protection during TEVAR.[10, 87] However, it is important to determine the best method for increasing the cardiac index. The detrimental effects of increasing cardiac index through expansion of blood volume include a higher CVP (potentially detrimental to spinal cord blood flow, as discussed below) and tissue edema (including spinal cord tissues) while cardiac output augmentation with adrenergic drugs may have potential negative effects at the microcirculatory level of the spinal cord.

Blood Pressure Management and Spinal Cord Perfusion Pressure

Spinal cord perfusion pressure is the difference between mean arterial blood pressure (MAP) and CSF pressure (or CVP, whichever is greater).[31] General guidelines for minimizing SCI include increasing MAP (i.e., > 90 mmHg) and draining CSF (≤10 mmHg) in order to maintain spinal cord perfusion pressure at levels above 80 mmHg.[8, 74] Recently, the European Association for Cardiothoracic Surgeons published a position paper about spinal cord protection in TEVAR recommending increasing of blood pressure in patients with SCI to higher than the individual's preoperative MAP (i.e., and at least 80 mmHg).[8] However, the strength of the provided evidence regarding blood pressure management and CSF drainage in the treatment of SCI is generally limited to expert opinion (i.e., IIaC) (Table 3).[8]

Izumi *et al.*[88] described the effect of maintaining high MAP on spinal cord protection in a rabbit model of aortic cross clamping by comparing high and low blood pressure animal groups to a control group; mean (standard deviation) arterial blood pressure during ischemia were controlled at 121.9 (2.8), 50.8 (4.3), and 82.3 (10.7) mmHg in high blood pressure, low blood pressure, and control groups, respectively. They observed higher spinal cord blood flow, lower markers of oxidative stress, faster recovery of transcranial MEPs, and less histological damage of the motor neurons in the animal group treated with higher MAPs. In addition, Lu *et al.*[89] showed in an experimental rat model of spinal cord ischemia/reperfusion that hypovolemia induced hypotension led to more neurological dysfunction, more extensive neuronal necrosis, but less apoptotic neuronal death when compared to the normotensive control group. Although there is no direct experimental evidence on the effect of blood pressure management in endovascular repair for spinal cord protection, the clinical experience in the literature demonstrates that maintaining spinal cord perfusion pressures above 80 mmHg (by increasing the MAP and draining the CSF) after the deployment of the aortic stent is beneficial in prevention and management of SCI and paralysis. In patients who go on to develop postoperative SCI, a MAP > 90 mmHg should be targeted in an attempt to reverse the weakness. Vasopressors used to attain increased MAP goals should be weaned

slowly over the subsequent 24–48 hours after improvement of the weakness. Such high levels of blood pressures are generally tolerated by TEVAR patients.[10, 87]

Hypothermia

Mild passive hypothermia (32°C to 35°C) may have a role in neuroprotection of the spinal cord though has not been studied in a rigorous fashion.[87, 90] Unlike open TAAA surgical repair, moderate hypothermia is not a current practice in TEVAR as it generally requires a more invasive approach (e.g., intravascular cooling systems) in order to be induced for a minimally invasive surgery.[91] Furthermore, evidence suggests that deeper levels of hypothermia affect the performance of Nitinol-based endovascular grafts *in vitro* by decreasing the radial expansive force and thus the deployed diameter, which could have important clinical impact on graft deformation, migration, and endoleak.[92]

Cerebrospinal fluid drainage

Indications for CSF Drainage

In 2010, the American Heart Association/American College of Cardiology Foundation guidelines recommended CSF drainage for TAAA open and endovascular repair. However, the document only provided references for the open repair.[7] In addition, in 2015, the European Association for Cardiothoracic Surgeons recommended that CSF drainage should be considered in patients undergoing TEVAR for prevention and treatment of SCI, though with only level IIaC evidence (ie.. mostly expert opinion).[8] In a randomized controlled trial for open TAAA repair by Coselli *et al.*,[93] they demonstrated that CSF drainage significantly reduced the incidence of paraplegia/paraparesis [9 out of 69 patients (13%) in control group without CSF drainage versus 2 out of 76 patients (2.6%) receiving CSF drainage; $P = 0.03$]. Similar high-quality data are not available for TEVAR. Hence, the indications and protocols for CSF drain use during TEVAR vary widely between institutions across the US and around the globe.

In a recent systematic review involving 4936 patients by Wong *et al.*[11] addressing SCI and CSF drainage after TEVAR, the authors were unable to clarify the role of prophylactic CSF drainage due to the lack of randomized controlled trials. Uchida also analyzed the literature on paraplegia/paraparesis after endovascular repair of thoracic aortic pathology from 1999 to 2013. Based on his review, Uchida developed indications for CSF drainage in TEVAR, however, these indications need to be validated in prospective randomized controlled trials (Table 3).[14] In the absence of any pharmacological intervention to protect against SCI, the physicians' options are generally limited to CSF drainage and blood pressure management.

There has been recent debate on the value of postoperative institution of CSF drainage in those patients presenting with delayed paralysis. In fact, Kakinohana questioned the role of the CSF drain in prevention of delayed paralysis after TEVAR suggesting that as the mechanism of the delayed paraplegia is not completely understood, its use in this setting cannot be fully justified.[10]

Timing of the CSF Drain Placement

The timing of CSF drain placement also differs among institutions. Some groups recommend placement of the CSF drain the day before surgery so that any complications from drain placement, should they arise, can be recognized early.[94] Other groups recommend insertion on the day of the surgery, in part, to reduce hospital bed utilization and cost.[90] DeSart reviewed a cohort of 607 TEVAR patients where 57 (9.4%) developed some degree of SCI. Of these, 54 (95%) received a CSF drain at some point in their care, with 31 (54%) of them receiving it postoperatively. They concluded that timing of CSF placement did not have a major impact on post-discharge functional impairment or long-term mortality. [5] Thus, the optimal timing of placement of the CSF drain remains uncertain.

In those who go on to develop some degree of SCI, the window for the benefits of adding CSF drain placement to their therapy is also important as the reversal of any deficits likely closes within one to two hours of onset. Bobadilla *et al*[90] performed a retrospective study of 94 TEVAR patients in whom they routinely inserted a CSF drain (both elective and emergent). In their series, only one patient suffered from delayed paralysis. Their proactive protocol reduced the time needed for interventions by allowing for immediate CSF drainage and induced hypertension as soon as paralysis was detected. This routine placement avoids the delay of CSF drain placement in patients with sudden onset of postoperative paralysis. Additional benefits to routine CSF drain placement include increasing the efficacy of induced hypertension for the treatment of postoperative paralysis (to efficiently off-load CSF volume and reduce CSF pressure). Induced hypertension without the aid of a CSF drain has been speculated to be harmful due to potential edema formation in the spinal cord.[95]

Another concern for postoperative drain insertion is the development of post-stent coagulopathy. The aortic stent excludes the aneurysmal sac from the blood flow, causing the blood inside the sac to thrombose. This consumes platelets and coagulation factors **through** activation of coagulation **cascades (with** reduction in fibrinogen and plasminogen levels as well as prothrombin activity) and fibrinolysis (**with** increases in fibrin degradation products and D-dimer levels). These changes were demonstrated in studies by Shimazaki *et al.*[96] and Monaco *et al.*[97] on patients with endovascular thoracic aortic aneurysm and aortic dissection repair. Although both studies concluded that these changes do not have affect patients' outcome, anesthesiologists need to be aware of these mechanisms that may impact the safety of postoperative CSF drain insertion.

The above potential coagulation issues notwithstanding, an alternate approach for timing of CSF drain insertion is the "wait and see" approach, which is much easier to employ in TEVAR (compared to open TAAA repair) as fast-track anesthetic techniques usually allow for early neurologic examination. That said, institutions that implement this approach must have standardized ICU procedures that employ structured and regular neurologic exams, including increased vigilance in the nurse/intensivist along with the avoidance of unnecessary sedation. They should also have clinical infrastructure available to rapidly place a CSF drain should delayed SCI occurs. Acher *et al.*[2] reviewed 22 studies representing the current practice of CSF drainage in TEVAR in the literature. They concluded that despite the wide variation in CSF drainage management among different institutions, rates of SCI

remain in the range of 1% to 10% no matter whether the decision to insert the drain was taken by the surgeon or by defined institutional criteria. In addition, timing of the CSF drainage insertion (intraoperatively vs postoperatively) did not show differences in clinical outcomes.

CSF Drain Complications

Inherent with all invasive procedures (including CSF drain placement) are their associated complications. A number of studies have outlined the various CSF drain-related complications. Hanna *et al.*[98] reported the complications of CSF drain placement in 81 TEVAR patients. Nine patients (11.1%) experienced minor lumbar drain complications, including spinal headache, puncture-site bleeding, persistent CSF leak, or clinically insignificant subdural hematoma. In a similar study, Arnaoutakis *et al.*[94] reported no complications from spinal drain placement in 48 patients. The reduction of complications in the Arnaoutakis *et al.*[94] series may have been due to the timing of the placement of the catheter (24 hours prior to the TEVAR), as well as the strict anticoagulation management around insertion and removal time. That is, they discontinued clopidogrel 7–10 days before the procedure and subcutaneous heparin 12 hours before drain insertion. The most recent American Society of Regional Anesthesia and Pain Medicine guidelines on insertion of intrathecal catheters recommend stopping clopidogrel at least 7 days before the procedure, stopping IV heparin for at least 4 hours, and stopping subcutaneous heparin for 8–10 hours before the procedure. In individuals using aspirin (for secondary prophylaxis), the guidelines recommend weighing the risk of bleeding against the cardiovascular risks of stopping aspirin.[99] The platelet threshold for drain insertion is recommended to be $\geq 100X10^3/\mu L^3$, international normalized ratio (INR) < 1.3 and a normal activated partial thromboplastin time.[100] In addition to the role of suboptimal coagulation status, clinically significant subdural hematoma can also be attributed to excessive CSF drainage – i.e., at rates > 15–20 ml/hour.[100]

In addition to concerns about CSF drain complications, some physicians believe that CSF drainage prevents early mobilization, requires trained staff, and increases ICU length of stay for at least 48 hours (i.e., the recommended period for postoperative drainage). Zipfel *et al.*[34] reported that 66% of their TEVAR patients did not require ICU care or required ICU care for less than 24 hours, which they were able to achieve because they did not use a CSF drain in their patients.

Newer Surgical Techniques for Prevention of SCI

Several newer surgical techniques are now undergoing evaluation as adjuncts for the prevention of SCI include. These include performing the procedure in stages, the use of minimally invasive segmental artery coil embolization (MISACE), and the allowing of temporary aneurysm sac perfusion (TASP) (Table 2).[101–103]

Staging

In cases with planned coverage of a long segment (i.e., >30cm) of the aorta in extended thoracic and thoracoabdominal aneurysm, a two-stage operation can be considered to

decrease the risk of SCI.[14] Performing the aortic coverage in stages can be intentional or unintentional. Unintentional staging occurs in patients with history of prior open or endovascular aortic aneurysm repair and presenting with extension of their aneurysmal disease that requires TEVAR. Intentional staging repair involves the initial placement of a thoracic aortic endograft covering the aorta from the proximal seal zone to just above the level of the most proximal visceral branch vessels which can then be followed by distal stent placement for distal seal after an interval of 2–3 months.[101]

Minimally Invasive Segmental Artery Coil Embolization (MISACE)

Luehr *et al.*[104] were able to individually embolize the segmental arteries in a porcine model through an endovascular technique known as minimally invasive segmental artery coil embolization (MISACE). Their experimental work showed that this technique essentially "preconditions" the collateral vascular network to ischemia by inducing arteriogenesis and improving vascularity of the spinal cord before TEVAR. The first trial for this technique was done by Etz *et al.*[102] on two patients with large thoracic aortic aneurysms. One of them underwent TEVAR where the segmental arteries were occluded 4–8 weeks before the aneurysm repair; neither of their patients had SCI. However, in an editorial accompanying this publication, the author questioned the reproducibility of this complex technique of endovascular embolization of the segmental arteries on a larger scale of patients, especially in patients with tortuous anatomy or with the presence of thrombus in the aneurysm sac.[105]

A randomized controlled clinical open label trial is starting in 2017 in 20 centers in Europe and 2 centers in the USA for Paraplegia Prevention in Aortic Aneurysm Repair by MISACE (PAPA-ARTiS) [Project ID: 733203, as registered in the Community Research and Development Information Service (CORDIS)]. This trial should shed new light on how many segmental arteries need to be coiled in order to precondition the collateral network and the ideal time interval between the MISACE procedure and TEVAR. However, there are concerns regarding MISACE itself causing SCI, in addition to the concerns arising from artificially augmenting the blood pressure in an unrepaired aortic aneurysm patient to emergently reverse the paralysis. Finally, translating the arteriogenesis concept concluded from studies on juvenile large animals to human with comorbid disease and advanced pathologies is highly speculative.

Temporary Aneurysm Sac Perfusion (TASP)

The TASP surgical technique for prevention of SCI is performed by establishing an endoleak into the covered aneurysm with a branched endograft that has a side branch that will ultimately be used to perfuse a major splanchnic vessel during the first part of a staged procedure. This is followed by second procedure to complete the exclusion of endobranch (with a stent that bridges the TEVAR stent to the intended visceral vessel) feeding the aneurysm sac after a 1–3 month time interval.[103, 106] Use of this technique showed reduction in the rate of permanent paralysis. Kasprzak *et al.*[103] tested the feasibility of this technique in a prospective study on 83 TEVAR patients (40 patients with TASP vs 43 patients without TASP). The incidence of SCI was significantly reduced in the TASP group (5% vs 21%, $P = 0.03$). However, the incidence of subsequent endoleak, potential risk of aortic rupture (occurring in the time between the two stages), and higher rate of temporary paraparesis and

paresthesia (30%) (as reported by Kasprzak *et al.*)[103] necessitate further investigation of this technique.

Implantation sequence rearrangement for branched/fenestrated stents

The final step in branched/fenestrated stents insertion is implantation of the bifurcated body and iliac limbs through large sheaths connected to the femoral/iliac arteries. These sheaths block the blood flow to the pelvis and lower limbs, and subsequently, the collateral circulation to the spinal cord. Some groups have recently changed their implantation sequence to implant the bifurcated body and iliac limbs followed by withdrawal of these large sheaths earlier in the procedure, after deployment of the central stent, in order to restore the blood flow to the femoral and iliac arteries, and hence, reduce the incidence of SCI.[107, 108]

Conclusion

SCI and paralysis continue to be a challenging complication after TEVAR. The incidence of SCI after TEVAR remains relatively high despite recent advances in surgical techniques. Anesthesiologists have a major role in spinal cord protection by careful management of the hemodynamics and institution of protocols for CSF drainage. Newer surgical techniques (i.e., MISACE and TASP) are currently under investigation in an attempt to reduce this dreadful complication. Further preclinical and clinical research is needed to discover novel neuroprotective strategies for protection and treatment of SCI and paralysis following TEVAR.

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Implication Statement

This narrative review article addresses the risk factors for and pathophysiology of spinal cord injury after thoracic endovascular aortic repair (TEVAR). In addition, intraoperative neuromonitoring and treatment strategies for TEVAR-related spinal cord injury are discussed.

Figure 1.

Thoracoabdominal aortic aneurysm repair according to Crawford's Classification and the risk of ischemic spinal cord injury as reported by centers of excellence for aortic repair. (Reprinted by permission of Edizioni Minerva Medica from: J Cardiovasc Sur 2014; 55(suppl 1 to No.2):159–68)[3]

TAAA: Thoracoabdominal aortic aneurysm

NOTE: For Figure 1 please refer to the Figure 1 of the manuscript by Etz et al.:

Etz, D. C., Luehr, M., Aspern, K. V., Misfeld, M., Gudehus, S., Ender, J., . . . Mohr, F. W. (2014). Spinal cord ischemia in open and endovascular thoracoabdominal aortic aneurysm repair: new concepts. J Cardiovasc Surg (Torino), 55(2 Suppl 1), 159-168.

Figure 2. Evolution of the dynamic nature of spinal cord ischemia and paralysis after TEVAR

A: Blood flows in the paraspinous and intraspinous network under normal conditions. The arterial system is divided into a *central centrifugal system* supplied by the sulcal arteries and a *peripheral centripetal system* supplied by the pial network surrounding the spinal cord. ***B:*** **Segmental arteries occlusion following aortic stent placement is accompanied by several compensatory mechanisms to maintain integrity of the spinal cord: (1) rerouting of blood flow from the paraspinous to the intraspinous network through dorsal branch of dorsal spinal artery to ventral branch of dorsal spinal artery supplying the radiculomedullary artery (Red arrows), (2) intramedullary intraspinous collateral network between anterior and posterior spinal arteries (green arrows), (3) extramedullary collaterals from the pial plexus, and (4) reversal of blood flow within the anterior spinal artery from nearby non-occluded segmental arteries (blue arrow).** ***C:*** **Failure of one or more of these compensatory mechanisms (black crosses) may lead to spinal cord ischemia: (1) No blood flow from paraspinous to intraspinous network (the blue color represents a thrombus in the segmental artery), (2) Absent intramedullary and extramedullary collaterals, (3) No reversal of blood flow within the anterior spinal artery from nearby non-occluded segmental arteries.** (Used with permission from Nicholas Theodore, M.D. **and publisher**,[48] modified by our group to explain compensatory mechanisms within the spinal cord collateral vascular network and pathophysiology of spinal cord injury in TEVAR).

NOTE: For Figure 2 please refer to the Figure 1 of the manuscript by Martirosyan et al.: Martirosyan, N. L., Feuerstein, J. S., Theodore, N., Cavalcanti, D. D., Spetzler, R. F., & Preul, M. C. (2011). Blood supply and vascular reactivity of the spinal cord under normal and pathological conditions. J Neurosurg Spine, 15(3), 238-251.

Figure 3.

Pattern of spinal cord damage in TEVAR on MRI (sporadic-type findings): asymmetrical multiple high intensity on axial T2-weighted images involving more than 3 segments of vertebra. Infarction is shaded on the illustration. From Tanaka *et al.,* Interact Cardiovasc Thorac Surg 2014 Aug;19(2):205–10. Reprinted with permission [35]

NOTE: For Figure 3 in our review, please refer to the Figure 1 of the manuscript by Tanaka et al.:

Tanaka, H., Minatoya, K., Matsuda, H., Sasaki, H., Iba, Y., Oda, T., & Kobayashi, J. (2014). Embolism is emerging as a major cause of spinal cord injury after descending and thoracoabdominal aortic repair with a contemporary approach: magnetic resonance findings of spinal cord injury. Interact Cardiovasc Thorac Surg, 19(2), 205-210.

Figure 4.

Network of vascular collaterals to the spinal cord: subclavian, hypogastric, intercostal and lumbar arteries. From Amato and Stolf, Jornal Vascular Brasileiro. 2015;14:248–52. Reprinted with permission. [109]

NOTE: For Figure 4 in our review, please refer to the Figure 4 of the manuscript by Amato et al.,

Figure 4. Collateral network: subclavian, hypogastric, intercostal and lumbar arteries

Amato, A. C. M., & Stolf, N. A. G. (2015). Anatomia da circulação medular. Jornal Vascular Brasileiro, 14, 248-252.

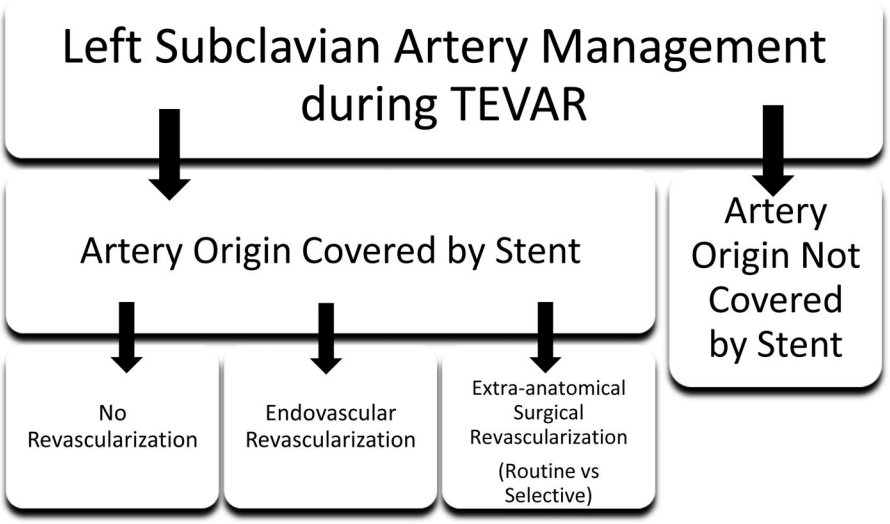

Figure 5. Surgical options for left subclavian artery management during TEVAR

When the operative plan requires covering the origin of the left subclavian artery (LSA), the LSA can be left without revascularization, or can be perfused through extra-anatomical surgical procedures, or more recently, by endovascular techniques. Surgical revascularization is either done routinely or only for selected patients.

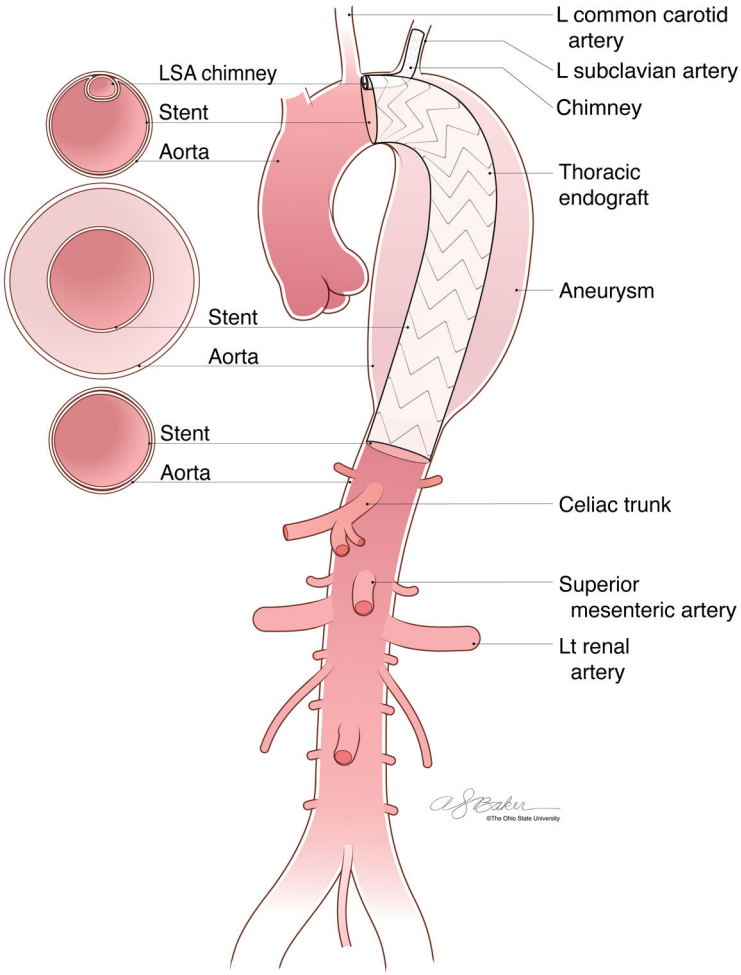

Figure 6. Chimney graft technique for left subclavian artery revascularization

A thoracic aortic endograft is excluding a descending thoracic aneurysm and covering the origin of the left subclavian artery (LSA); a chimney graft is inserted in the LSA parallel to the proximal wall of the aortic stent. The top left figure shows a cross section of the aortic arch with the proximal end of the thoracic aortic stent and the LSA chimney graft in place. The bottom left figure shows a cross section of the distal portion of the aortic stent sealing the descending thoracic aorta.

LSA: Left subclavian Artery; L: left

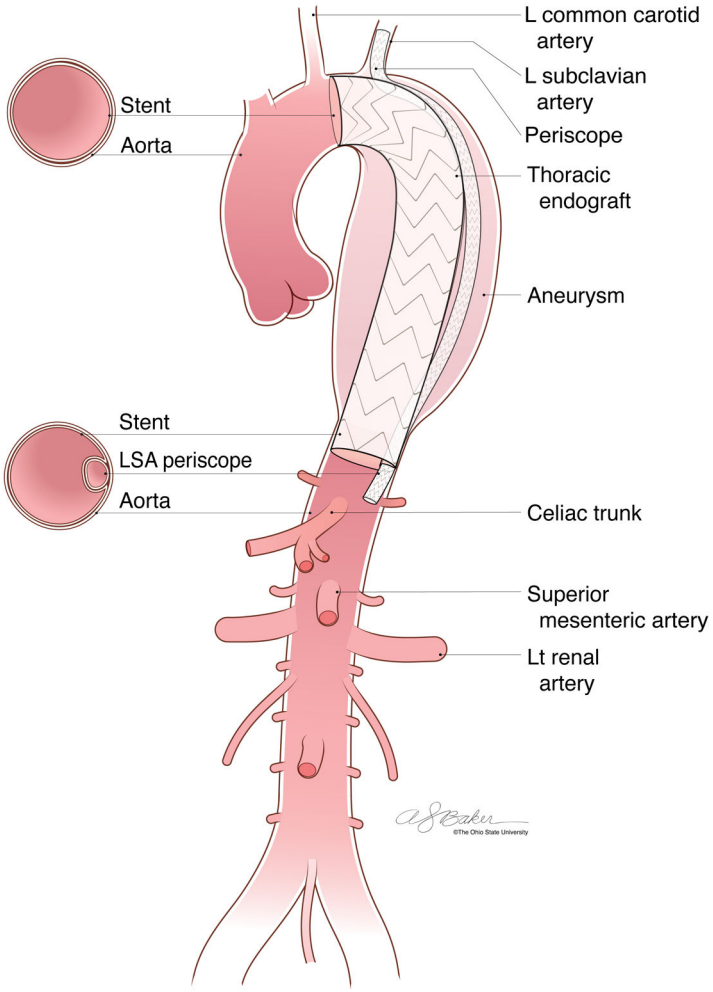

Figure 7. Periscope graft for left subclavian artery revascularization

A thoracic aortic endograft is excluding a descending thoracic aneurysm and covering the origin of the left subclavian artery (LSA); a periscope graft is inserted in the LSA through the distal part of the aorta. The top left figure shows a cross section of the aortic arch sealed with the proximal end of the aortic stent. The bottom left figure shows a cross section of the descending thoracic aorta with the thoracic stent and LSA periscope graft in place.

LSA: Left subclavian Artery; L: left

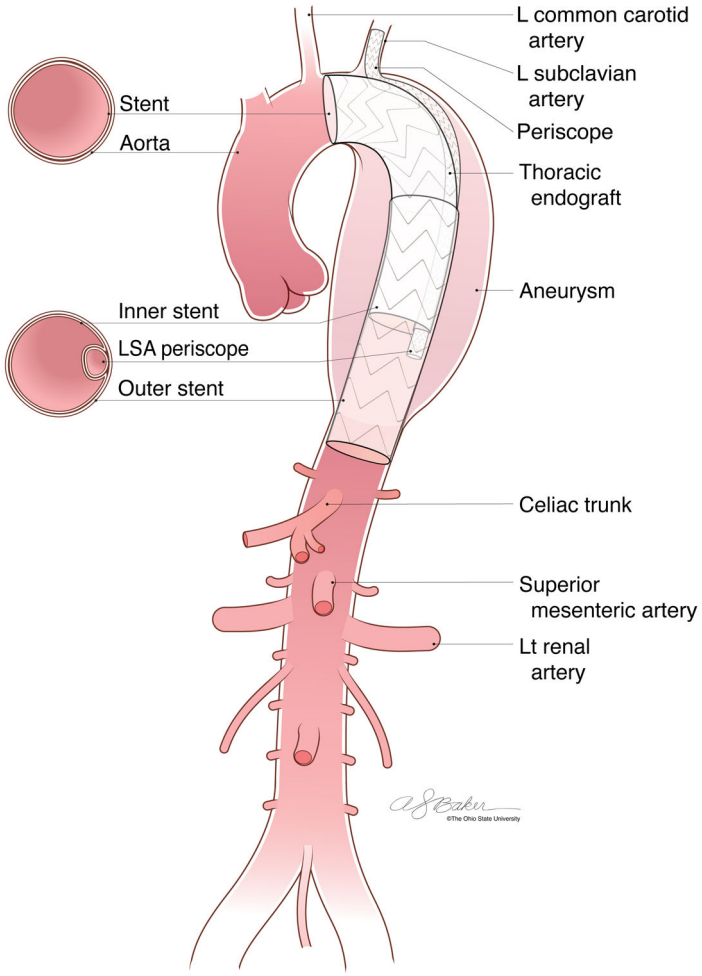

Figure 8. Periscope Sandwich technique for left subclavian artery revascularization

Two thoracic aortic endograft are excluding a descending thoracic aneurysm with the proximal stent covering the origin of the left subclavian artery (LSA); A sandwich graft is inserted between two aortic stents. The top left figure shows a cross section of the aortic arch sealed with the proximal end of the aortic stent. The bottom left figure shows a cross section of the overlapping thoracic stents with the distal end of the sandwich graft between them. LSA: Left subclavian Artery; L: left

Table 1

Risks factors for spinal cord ischemia after TEVAR.

Patient Risk Factors		Surgical Risk Factors	
1	Advanced age (> 70 years)[6]	**1**	Total aortic coverage > 205 mm[6, 38, 42–44]
2	Perioperative hypotension (MAP < 70 mmHg)[36–38]	**2**	Concomitant abdominal aortic aneurysm or prior abdominal aortic aneurysm surgical repair[38, 39, 46, 47]
3	Renal insufficiency (Creatinine >132 umol/L)[4, 6, 39]	**3**	Coverage of 2 or more vascular territories [45]
4	COPD[6]	**4**	Left subclavian artery coverage[39]
5	Hypertension[6]	**5**	Procedure urgency[41]
6	Degenerative aneurysm[40]	**6**	Coverage of hypogastric artery[36, 40]
		7	Adjunct procedure (Iliac conduit)[40]
		8	Use of 3 or more stents[39]
		9	Longer duration of the procedure[36]
		10	Excessive blood loss[40]

MAP: Mean arterial pressure; COPD: Chronic obstructive pulmonary disease

Table 2

Neuroprotective strategies for spinal cord during TEVAR

Anesthetic approaches		Surgical techniques	
1	Maximize oxygen delivery (increase Cardiac index > 2.5 L/min/m^2)[10, 87]	**1**	Staging the procedure[14, 101]
2	Optimize hemoglobin [10, 87]	**2**	Left subclavian artery revascularization[57]
3	Mild hypothermia (32–35°C)[87, 90]	**3**	Minimally invasive segmental artery coil embolization (MISACE)[102, 104]
4	Maintain CSF pressure (≤10 mmHg)[7, 8]	**4**	Temporary aneurysm sac perfusion (TASP)[103, 106]
5	Maintain spinal cord perfusion pressure (> 80 mmHg)[10, 74, 87]	**5**	New implantation sequence for branched/ fenestrated stents[107, 108]
6	Neuromonitoring (e.g., motor/sensory evoked potentials)[7, 8]		

Table 3

Indications for the use of CSF drain in TEVAR.

1	Anticipated endograft coverage of T8–L1 *
2	Coverage of a long segment of thoracic aorta (>30 cm)
3	Compromised collateral pathways; e.g., previous infrarenal aortic aneurysm repair, occluded hypogastric arteries, coverage of the left subclavian artery without revascularization
4	Symptomatic spinal ischemia in a patient who did not have a drain placed preoperatively

* **especially the i**ntercostal/lumber arteries that supply the Adamkiewicz artery identified by preoperative CT/MRI angiography. From Uchida *et al.* General Thoracic Cardiovascular Surgery, 2014 Jul; 62(7):391–7, Reprinted with permission)[14]