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**Spinal Cord Protection Strategies in the Endovascular
Treatment of Complex Aortic Disease**

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*To my family, for being home throughout every moment of this journey,
To those who have walked beside me throughout this long journey, making every step a little lighter,
To Medicine, which has taught me that healing does not simply mean curing, but, above all, caring.*

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Introduction

Complex aortic disease

Complex aortic disease is an umbrella term used to describe a heterogeneous group of aortic disorders in which the anatomical extent, morphology, location, or involvement of major aortic branches makes conventional surgical or endovascular treatment particularly challenging. Rather than representing a distinct pathological entity, the term encompasses different forms of aortic disease that share the need for advanced treatment strategies to achieve effective exclusion or reconstruction of the diseased aortic segment while preserving perfusion of critical arterial branches.

From an anatomical and therapeutic perspective, complex aortic disease predominantly includes thoracoabdominal aortic aneurysms (TAAAs), complex abdominal aortic aneurysms (AAAs), chronic aortic dissection with aneurysmal degeneration, and selected forms of penetrating atherosclerotic ulcer (PAU) and intramural hematoma (IMH). These conditions differ in their pathophysiology and natural history but may present a common therapeutic challenge when the diseased aorta involves the origins of the renal or visceral arteries, extends across multiple thoracic and abdominal segments, or compromises the landing zones required for conventional endovascular repair.

For the purposes of the present thesis, the concept of complex aortic disease is used primarily in reference to aneurysmal and post-dissection pathology involving the thoracoabdominal or visceral abdominal aorta and potentially amenable to fenestrated or branched endovascular aortic repair (f/bEVAR). This distinction is important because not every condition classified within the broad spectrum of complex aortic disease is an indication for FEVAR or BEVAR. Treatment selection depends on the underlying pathology, anatomical configuration, disease stage, patient characteristics, previous aortic interventions, and the feasibility of achieving durable exclusion while preserving critical branch vessels.(1)

The increasing adoption of advanced endovascular techniques has substantially expanded the treatment options available for these patients. In particular, fenestrated and branched endografts allow extensive portions of diseased aorta to be excluded while maintaining perfusion of the renal and visceral arteries. At the same time, extensive endovascular reconstruction may require coverage of large portions of the thoracic and abdominal aorta, with consequent interruption of multiple intercostal and lumbar arteries. This creates a direct

link between the treatment of complex aortic disease and the risk of spinal cord ischemia, which represents the central focus of the present thesis.(2)

Thoracoabdominal aortic aneurysms

Thoracoabdominal aortic aneurysms represent one of the most extensive and technically demanding forms of aortic aneurysmal disease. They are characterized by aneurysmal dilatation involving both the thoracic and abdominal portions of the aorta and may extend from the descending thoracic aorta to the infrarenal abdominal aorta. Their treatment is particularly challenging because the diseased segment may encompass the origins of multiple visceral and renal arteries as well as numerous segmental arteries contributing to spinal cord perfusion.

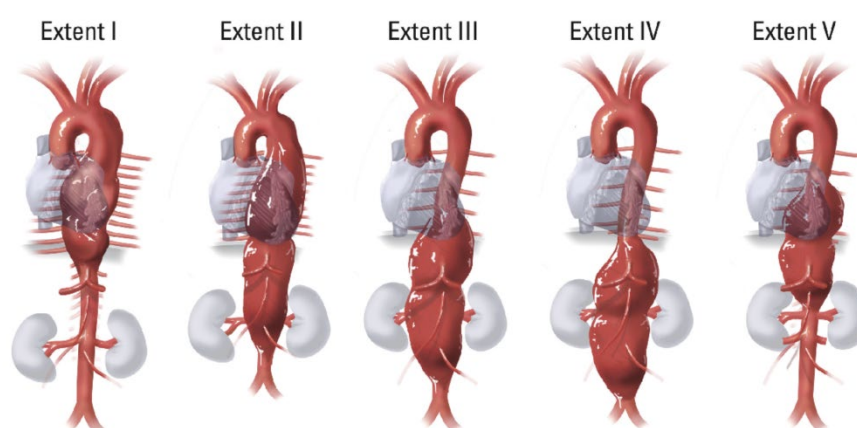
The anatomical extent of TAAA is traditionally described according to the Crawford classification, which categorizes aneurysms according to the longitudinal portion of the thoracoabdominal aorta involved. Type I involves most of the descending thoracic aorta, generally originating from the proximal thoracic aorta distal to the left subclavian artery and extending to the suprarenal abdominal aorta, thereby involving the proximal segment of the abdominal aorta. Type II represents the most extensive anatomical form and involves the entire or nearly the entire thoracoabdominal aorta, extending from the proximal descending thoracic aorta to the infrarenal abdominal aorta and potentially reaching the aortic bifurcation. Type III involves the distal descending thoracic aorta, generally beginning at approximately the level of T6, and extends distally through the thoracoabdominal aorta to the infrarenal abdominal aorta. Compared with type II, therefore, the proximal extent of the aneurysm is more distal. Type IV predominantly involves the abdominal aorta, beginning at the level of the diaphragm, generally around T12, and extending through the visceral aortic segment to the infrarenal abdominal aorta. In this type, thoracic aortic involvement is absent or limited to the short segment immediately above the diaphragm. Type V, introduced by Safi as part of the modified Crawford classification, involves the distal descending thoracic aorta, generally beginning at approximately the level of T6, and extends to the suprarenal aortic segment, thereby encompassing the distal thoracic aorta and visceral abdominal aorta without significant involvement of the infrarenal segment.(3)

This classification is of particular clinical relevance because the anatomical extent of the aneurysm is closely related to the complexity of treatment and the risk of complications,

particularly spinal cord ischemia, owing to the greater extent of aortic coverage and the consequent impairment of segmental arterial blood supply.

Among these anatomical patterns, Crawford extents I–III are particularly relevant to spinal cord protection, as treatment generally requires extensive thoracic or thoracoabdominal aortic coverage. The exclusion of large numbers of intercostal and lumbar arteries may reduce the direct arterial inflow to the spinal cord and increase its dependence on the collateral vascular network. Consequently, extensive TAAA repair represents one of the clinical settings in which spinal cord ischemia is of greatest concern.

FIGURE I. —*Crawford's Classification of complex abdominal aortic aneurysms.*



Historically, open surgical repair represented the principal treatment strategy for patients with extensive TAAA. Open repair, however, requires major thoracoabdominal surgery and may involve aortic cross-clamping, substantial blood loss, visceral ischemia, and prolonged cardiopulmonary and physiological stress. These factors may be particularly relevant in the typically elderly population affected by degenerative aortic disease and in patients with significant cardiovascular, pulmonary, or renal comorbidities. The development of endovascular techniques has progressively expanded the possibility of treating TAAAs without conventional open replacement. In patients with suitable anatomy, fenestrated and branched endovascular repair allows exclusion of the aneurysmal aortic segment while maintaining perfusion of the celiac artery, superior mesenteric artery, and renal arteries. Current European guidance recognizes fenestrated and branched technologies as important

treatment options for appropriately selected patients with complex thoracoabdominal aortic disease. However, the endovascular treatment of TAAA introduces a fundamental physiological trade-off. The same extensive aortic coverage required to exclude the aneurysm may simultaneously eliminate multiple segmental arterial branches supplying the spinal cord. The neurological consequences therefore depend not only on the technical success of aneurysm exclusion but also on the patient's residual collateral circulation and the ability to maintain adequate spinal cord perfusion throughout the perioperative period.

This relationship is particularly relevant for Crawford extent I–III aneurysms, in which the length of aortic coverage is substantial and the number of potentially sacrificed intercostal and lumbar arteries is high. Previous aortic surgery, occlusion or intentional coverage of the left subclavian or hypogastric arteries, and perioperative hypotension may further compromise the collateral network. These mechanisms provide the anatomical and physiological basis for the high priority given to spinal cord protection during extensive thoracoabdominal endovascular repair.

Complex abdominal aortic aneurysms

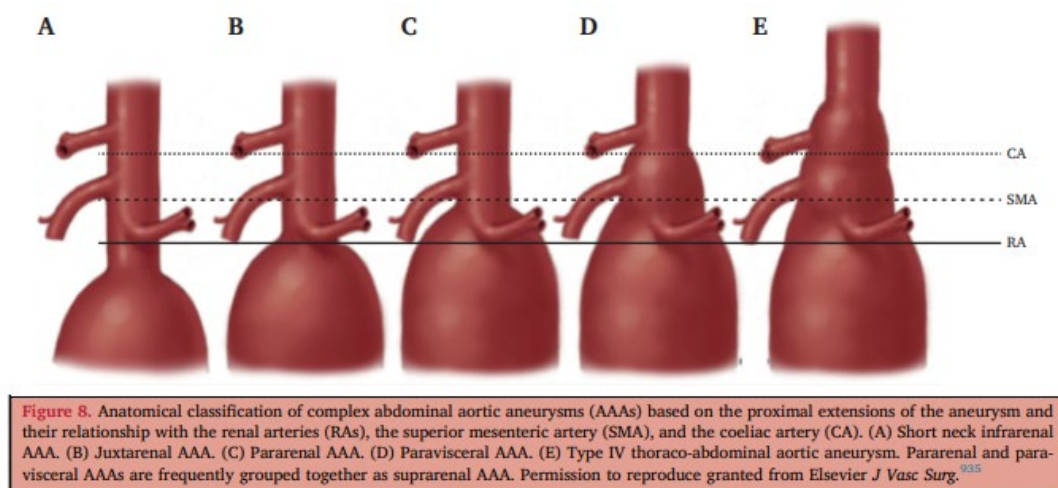
Complex abdominal aortic aneurysms comprise aneurysms in which the proximal aortic segment required for conventional endovascular sealing and fixation is absent or inadequate because of involvement of the renal or visceral arteries.

They include juxtarenal, pararenal, suprarenal, and paravisceral aneurysms, according to the relationship between the aneurysm and the renal and visceral arterial origins (Figure II).(3)(4) A short-neck infrarenal AAA is characterized by the presence of a segment of healthy aorta between the proximal aneurysm margin and the origin of the renal arteries, with an infrarenal aortic neck length ranging from 4 to 10 mm. Although the renal arteries are not directly involved, the limited length of the proximal neck renders the aneurysm unsuitable for treatment with conventional standard EVAR endografts and may require the use of advanced endovascular techniques. A juxtarenal AAA (JRAA) is characterized by an aneurysm extending to the level of the renal arteries without directly involving their origins, with an infrarenal aortic neck measuring less than 4 mm in length. In this configuration, the segment of aorta immediately below the renal arteries does not provide an adequate landing zone for standard EVAR. The term “juxtarenal” therefore indicates the close proximity of the aneurysm to the renal arteries, which remain anatomically uninvolved. A pararenal AAA (PRAA) is

characterized by involvement of at least one renal artery by the aneurysm, without involvement of the superior mesenteric artery (SMA). In these aneurysms, an adequate infrarenal sealing zone is therefore absent, and treatment requires preservation of perfusion to the involved renal artery, generally through fenestrated or branched endovascular repair techniques or open surgical repair with renal artery reconstruction. A paravisceral AAA (PVAA) has a greater proximal extent, involving the renal arteries and the superior mesenteric artery, while sparing the celiac artery. The aneurysm therefore involves the aortic segment encompassing the origins of the renal arteries and the SMA, requiring preservation of multiple visceral arteries during repair. The term suprarenal AAA is frequently used as a broader category to describe aneurysms extending above the level of the renal arteries. In the ESVS classification, pararenal and paravisceral aneurysms are therefore frequently grouped under the broader category of suprarenal aneurysms. Finally, when the aneurysm involves both renal arteries, the superior mesenteric artery, and the celiac artery, thereby involving the entire visceral segment of the abdominal aorta from the diaphragm to the aortic bifurcation, it is classified as a Crawford type IV thoracoabdominal aortic aneurysm (TAAA) rather than as a complex abdominal aortic aneurysm. This distinction is important because involvement of the celiac artery marks the transition from the classification of complex abdominal aortic aneurysms to thoracoabdominal aortic disease.

In summary, proceeding in a cranio-caudal direction and considering the proximal extent of the disease, the anatomical spectrum ranges from short-neck infrarenal AAA → juxtarenal AAA → pararenal AAA → paravisceral AAA → Crawford type IV TAAA, with progressively greater involvement of the renal arteries, the SMA, and finally the celiac artery.

FIGURE II. —*Anatomical classification of complex abdominal aortic aneurysms.*



Unlike standard infrarenal AAA, these lesions cannot always be treated with conventional EVAR without covering one or more renal or visceral arteries. The principal technical challenge is therefore to achieve a durable proximal seal while preserving perfusion of the target vessels. The 2024 European Society for Vascular Surgery (ESVS) guidelines specifically address complex abdominal aortic aneurysms as a distinct treatment category and include fenestrated and branched endovascular repair among the available treatment strategies. FEVAR was developed to overcome the anatomical limitations of standard EVAR in this setting. Fenestrations incorporated into the proximal endograft are aligned with the origins of the renal and visceral arteries, allowing the main graft to extend across the diseased aorta while maintaining perfusion through the fenestrated target vessels.

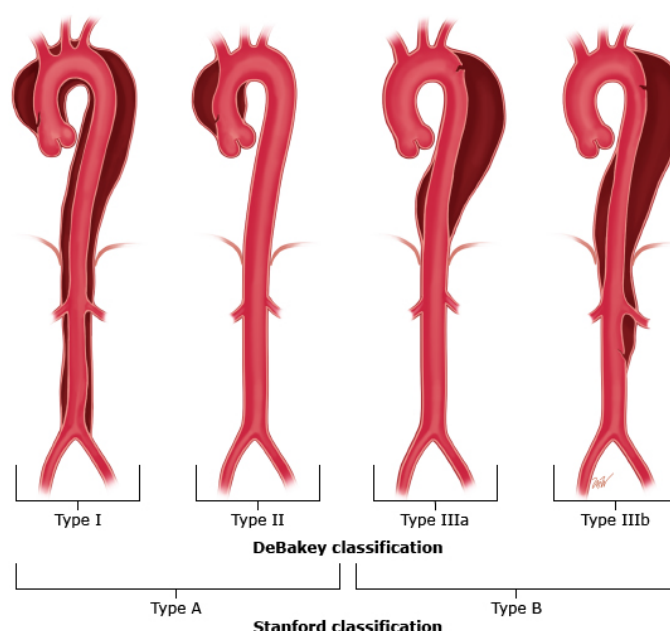
Although the extent of aortic coverage in complex abdominal repair is generally less than that required for extensive thoracoabdominal aneurysms, the procedure may still contribute to spinal cord ischemia in selected patients. The risk becomes particularly relevant when complex abdominal repair is combined with previous thoracic or abdominal aortic interventions, extensive coverage, or compromised collateral vessels. Thus, the neurological risk associated with a given procedure must be interpreted within the context of the patient's cumulative aortic history, rather than considering the current intervention in isolation.⁽¹⁾

Aortic dissection and post-dissection aneurysmal degeneration

Aortic dissection is characterized by a disruption of the aortic intimal layer, allowing blood to penetrate into the media and resulting in the formation of a false lumen. Depending on the location of the primary intimal tear and the extent of dissection propagation, the dissection may involve the ascending aorta, aortic arch, descending thoracic aorta, abdominal aorta, or multiple contiguous aortic segments. Aortic dissections are currently classified primarily according to two classification systems: the DeBakey classification and the Stanford classification. The DeBakey classification divides aortic dissections into three main types according to the site of origin of the dissection and its extent along the aorta. Type I originates in the ascending aorta and extends beyond the aortic arch, frequently involving the descending thoracic aorta and, in more extensive cases, the abdominal aorta. It is therefore characterized by extensive aortic involvement and represents the form with the greatest anatomical extent. Type II originates in the ascending aorta and remains confined to the ascending aorta, without

extending beyond the aortic arch. The dissection therefore involves exclusively the proximal aortic segment. Type III originates in the descending thoracic aorta, distal to the origin of the left subclavian artery, and extends distally along the aorta. It is traditionally further subdivided into type IIIa, when the dissection remains confined to the descending thoracic aorta, and type IIIb, when it extends distally to involve the abdominal aorta. The Stanford classification is simpler and has greater clinical and therapeutic relevance. It divides aortic dissections into type A and type B according to involvement of the ascending aorta, irrespective of the site of origin of the primary intimal tear. Type A dissection includes all dissections involving the ascending aorta, regardless of the site of origin and distal extent of the dissection. Therefore, a dissection originating in the descending aorta but propagating retrogradely to involve the ascending aorta is still classified as Stanford type A. In contrast, type B dissection does not involve the ascending aorta and generally affects the descending thoracic aorta distal to the origin of the left subclavian artery. However, it may extend distally along the thoracic and abdominal aorta, reaching the aortic bifurcation or the iliac arteries.(5,6)

FIGURE III. —*Anatomical classifications of Aortic Dissection.*



The two classification systems can be anatomically correlated, but they are not completely interchangeable. In particular, DeBakey type I and type II dissections are classified as Stanford type A, as both involve the ascending aorta. DeBakey type III generally corresponds to a Stanford type B dissection, as it originates in the descending thoracic aorta and does not involve the ascending aorta. However, it is important to emphasize that the Stanford classification is more directly oriented toward clinical management, as involvement of the ascending aorta identifies a group of patients in whom the risk of potentially life-threatening complications and the therapeutic approach differ substantially from those associated with dissections sparing this segment.

For complex endovascular aortic repair, chronic type B aortic dissection with aneurysmal degeneration is particularly relevant. Following the acute phase, persistent flow within the false lumen may prevent aortic remodeling and lead to progressive enlargement of the dissected aorta. In some patients, aneurysmal degeneration may eventually involve extensive portions of the thoracoabdominal aorta, creating an anatomical scenario requiring incorporation of multiple visceral and renal arteries. The anatomy of chronic dissection differs substantially from that of degenerative aneurysmal disease. The aorta is divided into true and false lumens, and visceral branches may originate from either lumen or receive blood from both. Multiple entry and re-entry tears may maintain communication between the two lumens, while the relationship between the true lumen, false lumen, and visceral branches may change along the length of the aorta. These characteristics can make conventional TEVAR insufficient when the disease extends into the visceral abdominal aorta. In selected patients with chronic type B dissection and thoracoabdominal aneurysmal degeneration, fenestrated and branched endovascular technologies may therefore be used to reconstruct the thoracoabdominal aorta while preserving blood flow to the visceral and renal arteries.(7) The 2026 ESVS Guidelines on Descending Thoracic and Thoraco-Abdominal Aortic Diseases specifically state that endovascular repair using fenestrated and branched technologies should be considered first-line therapy in patients with chronic type B aortic dissection associated with aneurysmal formation involving the thoracoabdominal aorta.

This is particularly important in the context of spinal cord protection because post-dissection thoracoabdominal repair may require extensive aortic coverage comparable to that required for degenerative TAAA. Furthermore, patients with chronic dissection may have undergone previous endovascular or surgical procedures, resulting in cumulative interruption of

segmental and collateral arterial pathways. Thus, previous aortic intervention represents an important component of neurological risk assessment.

Penetrating atherosclerotic ulcer

Penetrating atherosclerotic ulcer (PAU), or penetrating atherosclerotic ulceration, represents one of the main manifestations within the spectrum of acute aortic syndromes (AAS) and consists of ulceration of an atherosclerotic aortic plaque with progressive erosion of the intimal layer and penetration of blood into the aortic media. Unlike classic aortic dissection, in which an intimal tear results in longitudinal separation of the layers of the aortic wall with formation of a false lumen, PAU typically presents as a focal lesion of the aortic wall, whose progression may lead to progressive weakening of the wall and evolution toward intramural hematoma, pseudoaneurysm, dissection, or rupture. PAU predominantly occurs in elderly patients with advanced atherosclerosis and multiple cardiovascular risk factors, including hypertension, smoking, dyslipidemia, coronary artery disease, peripheral arterial disease, and chronic kidney disease. Although the most frequent site is the descending thoracic aorta, the disease may also involve the abdominal and thoracoabdominal aorta, with variable involvement of the major visceral branches. In these locations, PAU may contribute to the development of complex aneurysms or saccular aneurysmal changes, making treatment particularly challenging when the lesion involves or is located in close proximity to the renal arteries, superior mesenteric artery, or celiac artery. Computed tomography angiography (CTA) represents the main diagnostic imaging modality and allows assessment of the location and extent of the lesion, ulcer depth and diameter, aortic wall thickness, and the presence of associated intramural hematoma, pseudoaneurysm, or signs of rupture. Preoperative CTA assessment is also essential in patients undergoing endovascular treatment, as it enables detailed evaluation of aortic and visceral vessel anatomy and facilitates appropriate planning of sealing zones and strategies for preserving the involved arterial branches.

The natural history of PAU is variable. Some lesions may remain stable over time, whereas others may undergo progressive morphological changes, with increasing depth or size and development of pseudoaneurysm, intramural hematoma, dissection, or rupture. The risk of progression is higher in the presence of persistent symptoms, lesion enlargement, high-risk morphological features, or concomitant abnormalities of the aortic wall. Asymptomatic PAUs without high-risk features may be managed with optimal medical therapy and radiological surveillance, whereas symptomatic, progressive, or complicated lesions may require

interventional treatment. In the setting of complex abdominal and thoracoabdominal aortic disease, endovascular treatment may require the use of fenestrated endovascular aortic repair (FEVAR) or branched endovascular aortic repair (BEVAR). These techniques allow adequate exclusion of the lesion while maintaining perfusion of the major visceral branches through fenestrations or dedicated branches to the renal arteries, superior mesenteric artery, and celiac artery. The choice between FEVAR and BEVAR primarily depends on the anatomy and location of the lesion relative to the visceral vessels, aortic morphology, and the feasibility of achieving adequate sealing and fixation zones. In patients with PAU involving the juxtarenal, pararenal, or paravisceral aortic segments, the need to extend the proximal sealing zone may result in involvement of the renal arteries and/or visceral vessels, making fenestrated techniques particularly relevant. Conversely, when the disease has a more extensive thoracoabdominal distribution and involves multiple visceral branches, BEVAR may allow simultaneous preservation of several target vessels through dedicated branches. In both settings, preoperative CTA planning and accurate assessment of visceral vessel anatomy are essential for achieving an adequate technical outcome.(8)

Overall, PAU represents a focal and potentially progressive manifestation of atherosclerotic disease of the aortic wall, whose clinical significance depends on its anatomical location, morphological characteristics, and the presence of complications. When involving complex abdominal or thoracoabdominal aortic segments, its management may require advanced endovascular strategies such as FEVAR and BEVAR, which allow exclusion of the lesion while preserving perfusion of the major renal and visceral branches.

Intramural hematoma

Intramural hematoma (IMH), or aortic intramural hematoma, represents one of the main manifestations within the spectrum of acute aortic syndromes (AAS), together with aortic dissection and penetrating atherosclerotic ulcer (PAU). It is characterized by the presence of blood within the media of the aortic wall, in the absence of an evident communication between the aortic lumen and a false lumen through an intimal tear typical of classic aortic dissection. IMH results in thickening of the aortic wall and may cause progressive disruption of its structural integrity, with possible evolution toward dissection, pseudoaneurysm formation, or rupture. IMH is generally associated with elderly patients with atherosclerosis and multiple cardiovascular risk factors, including hypertension, smoking, dyslipidemia, coronary artery disease, and chronic kidney disease. It may involve any segment of the aorta but is most

frequently located in the descending thoracic aorta. From an anatomical perspective, IMH is traditionally classified as type A when it involves the ascending aorta and type B when the ascending aorta is spared and the disease predominantly involves the descending thoracic aorta. This distinction, analogous to that used for aortic dissection in the Stanford classification, has important prognostic and therapeutic implications. The pathogenesis of IMH has traditionally been attributed to spontaneous rupture of the vasa vasorum within the media, resulting in intramural hemorrhage in the absence of an identifiable intimal tear. However, IMH may also be associated with a small intimal lesion or a PAU, making it sometimes difficult to distinguish between the different forms of acute aortic syndrome. Increasing pressure within the aortic wall may lead to progressive expansion of the hematoma, compression of the true lumen, and weakening of the aortic wall. Computed tomography angiography (CTA) represents the primary imaging modality for the diagnosis and characterization of IMH. The typical finding is circumferential or eccentric thickening of the aortic wall, generally hyperattenuating on non-contrast CT and without internal enhancement following contrast administration. CTA also allows assessment of the extent of the hematoma, maximum wall thickness, and the presence of associated PAU, ulcer-like projection, dissection, pleural effusion, periaortic collections, or signs of rupture, as well as its anatomical relationship with the major visceral branches.

The natural history of IMH is variable and may include progression, stabilization, or regression. Some hematomas may undergo complete resolution, whereas others may progress to aortic dissection, pseudoaneurysm formation, or rupture. The risk of progression is higher in the presence of persistent symptoms, increasing hematoma thickness, aortic dilatation, associated PAU, ulcer-like projection, or evidence of progression during follow-up. The presence and anatomical location of IMH should therefore be carefully considered during risk stratification. Clinically, IMH may present with acute chest or back pain, similarly to aortic dissection, or may be identified incidentally on imaging. Management primarily depends on the anatomical location of the IMH, the presence of symptoms, and high-risk radiological features. IMH involving the ascending aorta generally requires urgent evaluation for surgical treatment, whereas uncomplicated IMH of the descending thoracic aorta may initially be managed with optimal medical therapy and close radiological surveillance. In the setting of complex abdominal and thoracoabdominal aortic disease, IMH may extend into the visceral aortic segment and represent a component of the underlying pathology requiring an advanced endovascular strategy. When the disease involves the juxtarenal, pararenal, or paravisceral segments, the presence of IMH may alter the morphology of the sealing zone and necessitate

careful treatment planning. When an endovascular approach is indicated and the major renal and visceral branches are involved, FEVAR and BEVAR may allow exclusion of the diseased aortic segment while preserving perfusion of the target vessels through dedicated fenestrations or branches. High-resolution CTA planning therefore plays a central role in patients undergoing F/BEVAR, allowing assessment of the extent of IMH, aortic morphology, the presence of associated PAUs, and the anatomy of the renal and visceral vessels. Definition of the sealing and fixation zones must take into account the integrity of the aortic wall and the potential progression of the disease, with the aim of achieving adequate endograft anchoring and reducing the risk of complications.(8)

Overall, IMH represents a potentially progressive form of acute aortic syndrome, characterized by a broad spectrum of clinical and radiological presentations. Its management requires an integrated assessment of clinical and imaging findings, with particular attention to its location, extent, hematoma thickness, and the presence of high-risk features. In patients with complex abdominal or thoracoabdominal involvement, any indication for endovascular treatment should be accompanied by meticulous anatomical planning, and FEVAR and BEVAR may represent effective strategies for treating the diseased aortic segment while preserving perfusion of the major renal and visceral branches.

Complex aortic disease amenable to f/bBEVAR

Although the pathological mechanisms underlying TAAA, complex abdominal aneurysms, chronic dissection, PAU, and IMH differ considerably, they may converge on a common anatomical problem: the diseased aortic segment involves or approaches critical branch vessels that cannot simply be covered by a conventional endograft.

In this setting, fenestrated and branched endovascular repair provides a means of combining extensive aortic exclusion with preservation of blood flow to the renal and visceral arteries. FEVAR generally relies on fenestrations or scallops incorporated into the endograft fabric, whereas BEVAR uses directional branches connected to bridging stent-grafts to maintain perfusion of target vessels. The choice between the two approaches depends on the anatomy of the aorta, the number and configuration of target vessels, the location of the proximal and distal landing zones, and the characteristics of the underlying pathology. The application of these techniques has therefore progressively expanded from complex abdominal aneurysms to extensive thoracoabdominal aneurysms and, in selected patients, to chronic post-dissection

thoracoabdominal aneurysmal disease. However, the anatomical advantages of f/bEVAR are accompanied by a specific neurological challenge. Extensive endograft coverage may exclude multiple intercostal and lumbar arteries and thereby reduce direct segmental arterial inflow to the spinal cord. This effect may be amplified by previous aortic interventions, loss of major collateral vessels, extensive thoracic coverage, and perioperative hypotension. Consequently, the treatment of complex aortic disease with f/bEVAR represents a balance between complete and durable exclusion of the diseased aorta and preservation of the vascular pathways required for end-organ and spinal cord perfusion. The greater the extent of aortic coverage, the greater the potential disruption of the collateral network supporting the spinal cord.

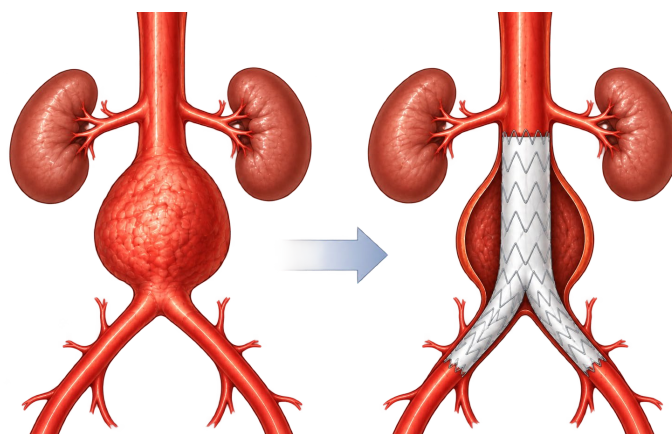
This anatomical relationship provides the rationale for examining spinal cord perfusion before discussing the neurological complications of complex endovascular aortic repair. A detailed understanding of the arterial supply of the spinal cord is therefore essential to explain why extensive thoracoabdominal endovascular procedures may result in spinal cord ischemia and why specific perioperative protective strategies are required.

EVAR, FEVAR and BEVAR: Evolution of Aortic Repair

Endovascular aneurysm repair (EVAR)

EVAR has progressively transformed the management of abdominal aortic aneurysms (AAAs), providing a minimally invasive alternative to conventional open surgical repair. Standard EVAR consists of the deployment of a bifurcated endograft within the aortic lumen, with the proximal and distal sealing zones positioned in healthy segments of the aorta and iliac arteries. The aneurysm is thereby excluded from direct systemic arterial pressure, while blood flow is maintained through the endograft and its iliac limbs.(6)

FIGURE IV. — *Schematic representation of the endovascular treatment of an abdominal aortic aneurysm using EVAR.*



The feasibility of standard EVAR is primarily determined by the morphology of the proximal aortic neck. A sufficient length and adequate diameter of relatively healthy infrarenal aorta are required to achieve effective proximal sealing and fixation. Consequently, standard EVAR is most suitable for infrarenal AAAs in which an adequate landing zone is present below the renal arteries. When the aneurysm extends proximally toward or beyond the renal arteries, the available infrarenal sealing zone may become insufficient, making conventional EVAR unsuitable or associated with an increased risk of proximal type I endoleak and subsequent treatment failure. (3)

The progressive extension of aneurysmal disease into the juxtarenal, pararenal, paravisceral, and thoracoabdominal aorta has therefore represented a major challenge for endovascular

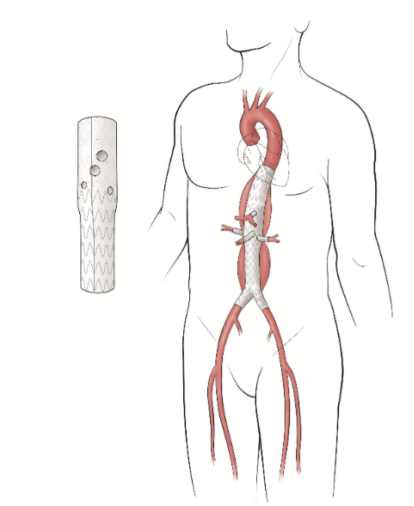
therapy. In these anatomical settings, achieving an adequate proximal seal may require coverage of arteries that provide blood supply to essential organs, particularly the renal, superior mesenteric, and celiac arteries. Covering these vessels with a conventional endograft would result in visceral ischemia and is consequently not acceptable.

The development of fenestrated and branched endovascular technologies has addressed this limitation by allowing the endograft to extend across the visceral segment while maintaining perfusion of the relevant target arteries. FEVAR and BEVAR therefore represent an evolution of standard EVAR rather than completely distinct endovascular concepts. Their defining feature is the incorporation of the visceral and renal arteries into the endovascular reconstruction.(1,9)

Fenestrated endovascular aortic repair (FEVAR)

FEVAR was developed to treat aortic aneurysms in which the proximal sealing zone is located at or immediately adjacent to the renal arteries. The principal characteristic of a fenestrated endograft is the presence of one or more fenestrations, that is, reinforced openings incorporated into the fabric of the main endograft and precisely aligned with the ostia of the target vessels.(3)

FIGURE V. — *Schematic representation of the treatment of a complex abdominal aortic aneurysm using FEVAR.*



Through these fenestrations, bridging stent-grafts are deployed into the renal or visceral arteries, thereby maintaining their patency while allowing the main endograft to obtain a

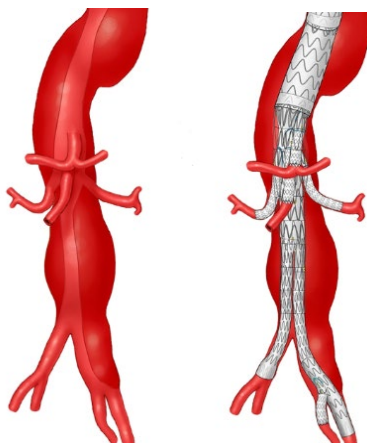
proximal seal in a segment of aorta that would otherwise be unsuitable for conventional EVAR. FEVAR can consequently extend the proximal landing zone above the renal arteries without sacrificing renal or visceral perfusion. The technique is particularly relevant for juxtarenal and pararenal abdominal aortic aneurysms, in which the aneurysm extends to the level of the renal arteries or involves the pararenal aortic segment. Depending on the anatomy, one or more fenestrations may be required for the renal arteries, with additional incorporation of the superior mesenteric artery when the aneurysm extends more proximally. The success of FEVAR depends heavily on accurate preoperative anatomical planning. The position, diameter, orientation, and relative distance of the target vessels must be carefully assessed using high-resolution computed tomography angiography. The configuration of the endograft is then selected or custom-designed to reproduce the patient's individual visceral anatomy.

A major advantage of FEVAR is therefore the possibility of extending the proximal sealing zone while preserving the arterial supply to the kidneys and abdominal viscera. This has substantially expanded the endovascular treatment options for patients with complex abdominal aortic aneurysms who would otherwise require open repair. Contemporary guidelines recognize fenestrated and branched approaches as important treatment options for complex pararenal and thoracoabdominal disease, particularly in patients at increased risk for open surgery. (1)

Branched endovascular aortic repair (BEVAR)

BEVAR extends the same principle to more extensive aortic disease. Instead of fenestrations, the main endograft incorporates one or more directional branches that project into the aneurysmal sac and are subsequently connected to bridging stent-grafts positioned within the target vessels.

FIGURE VI. — *Schematic representation of the treatment of thoraco-abdominal aortic aneurysm using BEVAR.*



This configuration is particularly useful when the distance between the main endograft and the target vessel is too large or anatomically unfavorable for a fenestrated configuration. Branched devices provide a dedicated conduit between the main graft and the target artery and are therefore especially useful in paravisceral and thoracoabdominal aortic aneurysms (TAAAs), where the celiac artery, superior mesenteric artery, and renal arteries may all require incorporation. BEVAR may be performed using custom-made devices designed according to the individual patient's anatomy or, in selected anatomical configurations, off-the-shelf multibranched devices. The latter approach has the potential advantage of reducing manufacturing and waiting times and may therefore be particularly relevant in patients requiring treatment within a shorter time frame.⁽¹⁾ The fundamental difference between FEVAR and BEVAR is therefore not simply the number of target vessels involved, but the mechanical configuration used to incorporate those vessels. In FEVAR, the target artery is aligned with a fenestration in the main graft and connected through a bridging stent-graft. In BEVAR, the target vessel is connected to a dedicated directional branch arising from the main endograft. The choice between the two configurations is determined by the morphology of the aorta, the location and orientation of the target vessels, and the distance between the endograft and the target vessel.

An additional distinction concerns the extent of aortic coverage. FEVAR can often achieve a more distal proximal landing zone than branched devices, potentially preserving a greater number of thoracic intercostal arteries. This anatomical characteristic may be relevant when assessing the risk of spinal cord ischemia.

Indications and anatomical complexity

The transition from standard EVAR to FEVAR or BEVAR is fundamentally driven by the loss of an adequate infrarenal sealing zone and the progressive involvement of visceral or thoracic aortic segments.

A simplified anatomical spectrum can therefore be described as follows:

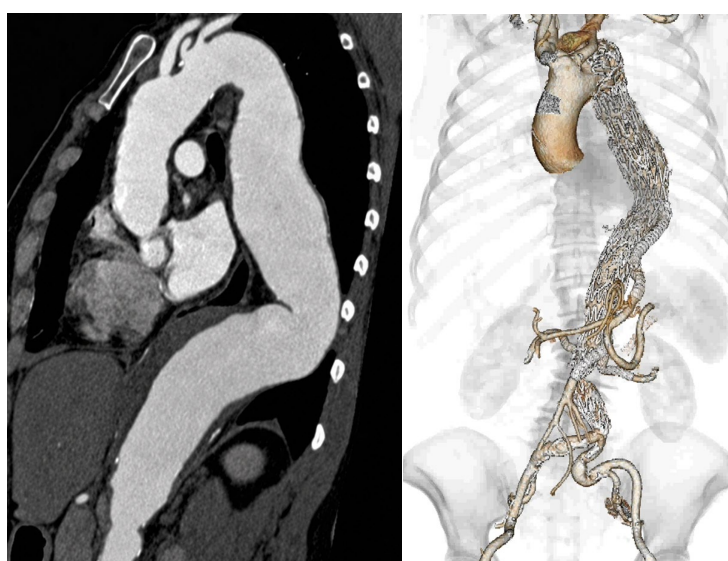
- Standard EVAR: infrarenal AAA with an adequate infrarenal proximal landing zone.
- FEVAR: juxtarenal and selected pararenal or paravisceral aneurysms requiring incorporation of renal and/or visceral arteries.

- BEVAR: more extensive paravisceral and thoracoabdominal aneurysms requiring incorporation of multiple visceral branches and, frequently, treatment over a longer segment of the thoracoabdominal aorta. This distinction is clinically important because increasing proximal and longitudinal extension of the aneurysm generally requires progressively more extensive aortic coverage.

The current EACTS/STS guidelines describe branched/fenestrated repair as an established endovascular treatment for complex pararenal and thoracoabdominal aortic disease.(6) In patients considered unfit for open repair, B/FEVAR is recommended as a first-line treatment option when anatomically feasible. For patients undergoing extensive endovascular repair of high-risk thoracoabdominal aneurysms, a staged approach may be considered to reduce the risk of spinal cord injury. Importantly, not all FEVAR and BEVAR procedures carry the same neurological risk. The extent of aortic coverage is a major determinant of spinal cord ischemia. Repairs confined largely to the abdominal aorta may involve relatively limited interruption of the arterial supply to the spinal cord, whereas treatment of extensive thoracoabdominal aneurysms can require coverage of a large number of intercostal and lumbar arteries. Contemporary clinical data confirm that increasing aneurysm extent is associated with greater rates of spinal cord ischemia and secondary interventions following F/BEVAR.

This distinction is central to understanding why FEVAR and BEVAR should not be considered a single homogeneous procedure. The neurological consequences of a short-segment fenestrated repair of a juxtarenal aneurysm are fundamentally different from those associated with extensive branched repair of a Crawford type I–III thoracoabdominal aneurysm.

FIGURE VII. —*Preoperative vs postoperative thoracoabdominal aortic aneurysm CT scan.*



Advantages and limitations compared with standard EVAR

The principal advantage of FEVAR and BEVAR over standard EVAR is anatomical applicability. Standard EVAR requires an adequate infrarenal landing zone, whereas fenestrated and branched devices allow the proximal sealing zone to be moved into the suprarenal or visceral aorta while preserving perfusion of the target arteries.

This technological evolution has substantially increased the proportion of patients with complex aortic anatomy who can be treated using an endovascular approach. Compared with conventional open repair, contemporary F/BEVAR series generally demonstrate lower perioperative physiological stress, reduced blood loss, fewer pulmonary and cardiac complications, and shorter hospital recovery. These advantages are particularly relevant in older patients and in individuals with significant comorbidities, in whom the physiological burden of open thoracoabdominal surgery may be considerable. However, the greater anatomical reach of FEVAR and BEVAR is achieved at the cost of increased technical complexity. Unlike standard EVAR, these procedures require the precise incorporation of one or more target arteries through fenestrations or branches. This creates additional potential failure points, including bridging stent-graft stenosis or occlusion, target vessel instability, endoleaks, component separation, and the need for secondary interventions. Long-term follow-up is therefore particularly important. Contemporary series demonstrate high technical success and satisfactory mid-term outcomes, but secondary interventions remain relatively frequent, largely because of target-vessel-related complications and endoleaks. In a recent five-year analysis of 436 patients treated with F/BEVAR, greater aneurysm extent was associated with higher early mortality, spinal cord ischemia, and need for secondary intervention, emphasizing the importance of both anatomical complexity and long-term surveillance. Thus, FEVAR and BEVAR should not simply be regarded as “better EVAR.” Rather, they provide an expanded therapeutic option for anatomies that cannot be adequately treated with standard EVAR, while introducing additional technical and device-related challenges. Their principal advantage is the ability to preserve perfusion of otherwise covered visceral vessels while obtaining a secure proximal seal; their principal limitation is the greater procedural complexity and the increased number of potential complications associated with target-vessel incorporation.

The same principle applies to neurological complications. Although endovascular treatment generally avoids many of the physiological insults associated with open thoracoabdominal surgery, extensive endovascular coverage can still compromise spinal cord perfusion. In

particular, the exclusion of large portions of the thoracic and abdominal aorta may interrupt multiple intercostal and lumbar arteries that contribute to the spinal cord collateral network. Consequently, the reduced invasiveness of endovascular repair does not eliminate the risk of spinal cord ischemia; rather, it changes the mechanisms through which spinal cord perfusion may be compromised.

From complex endovascular repair to spinal cord ischemia

The progressive development from standard EVAR to FEVAR and BEVAR has therefore allowed endovascular treatment to extend from the infrarenal aorta to increasingly proximal and extensive segments of the thoracoabdominal aorta.

This evolution, however, introduces a fundamental physiological consequence: the greater the length of aorta covered by the endograft, the greater the number of segmental arteries that may be excluded from direct antegrade perfusion.⁽¹⁰⁾ In extensive thoracoabdominal repair, these vessels include intercostal and lumbar arteries that contribute to the collateral network supporting spinal cord perfusion. The anatomical extent of the repair can be described according to the Ishimaru classification of proximal and distal landing zones, which identifies the different aortic segments used for proximal and distal endograft anchoring and allows for standardized characterization of the extent of aortic coverage. Progressive extension of the proximal landing zone toward more proximal thoracic aortic segments and extension of the distal landing zone into the thoracoabdominal aorta or iliac territories progressively result in the exclusion of a greater number of segmental arterial branches.⁽¹¹⁾ The aorta is divided into 11 landing zones (0–11) according to major anatomical landmarks. Zone 0 extends from the ascending aorta to the distal margin of the origin of the innominate artery; Zone 1 lies between the innominate artery and the left common carotid artery; Zone 2 extends between the left common carotid and left subclavian arteries. Zone 3 comprises the proximal descending thoracic aorta, extending approximately from the left subclavian artery to the level of the T4 vertebral body, whereas Zone 4 extends from the distal limit of Zone 3 to the mid-descending thoracic aorta, approximately at the level of T6. Zone 5 encompasses the distal descending thoracic aorta between Zone 4 and the celiac artery. Zone 6 includes the segment from the celiac artery to the superior mesenteric artery, Zone 7 extends from the superior mesenteric artery to the renal arteries, and Zone 8 encompasses the renal artery level and extends to the infrarenal abdominal aorta. Zone 9 comprises the infrarenal aorta to the level of the aortic

bifurcation, while Zone 10 corresponds to the common iliac arteries and Zone 11 to the origins of the external iliac arteries. (8)

The spinal cord does not depend on a single arterial source but on a complex network of longitudinal and segmental vessels. When several of these arterial inflows are simultaneously interrupted, spinal cord perfusion becomes increasingly dependent on the remaining collateral pathways and on adequate systemic haemodynamics. The risk becomes particularly relevant when extensive thoracic coverage is combined with previous aortic interventions, occlusion or coverage of the left subclavian or hypogastric arteries, hypotension, anemia, or other conditions capable of reducing oxygen delivery. This relationship explains an important paradox of modern endovascular aortic surgery: the procedures have become progressively less invasive from a surgical perspective while potentially becoming more demanding from a spinal cord perfusion perspective. The neurological risk is therefore not determined solely by the endovascular nature of the procedure, but by the anatomical extent of aortic exclusion and the consequent alteration of the vascular network supplying the spinal cord.

Understanding this concept is essential to interpret the pathophysiology of spinal cord ischemia following complex endovascular aortic repair. The next chapter will therefore examine in detail the arterial anatomy of the spinal cord, including the longitudinal spinal arteries, segmental arteries, the artery of Adamkiewicz, and, above all, the extensive collateral network that allows the spinal cord to tolerate progressive interruption of individual arterial inflows.

Spinal Cord Blood Supply

Surgical anatomy of the spinal cord blood supply

A detailed understanding of spinal cord vascular anatomy is fundamental to interpreting the mechanisms of spinal cord ischemia (SCI) during complex aortic repair. Unlike organs supplied predominantly by one or a few major arterial territories, the spinal cord depends on an intricate and highly variable vascular network in which longitudinal arteries, segmental vessels, and collateral pathways interact continuously to maintain tissue perfusion.

This organization provides substantial physiological redundancy and allows the spinal cord to tolerate the interruption of individual arterial branches. At the same time, it creates a particular vulnerability during extensive thoracoabdominal aortic interventions, when multiple segmental arteries may be simultaneously excluded.

The arterial supply of the spinal cord can be conceptually divided into three interconnected components:

1. a longitudinal arterial system, consisting primarily of the anterior and posterior spinal arteries;
2. a segmental arterial system, consisting of radicular and radiculomedullary arteries arising from segmental vessels;
3. an extensive collateral network, linking the spinal circulation with the subclavian, intercostal, lumbar, hypogastric, and paraspinal arterial territories.

The interaction between these systems, rather than the integrity of any individual vessel, is the principal determinant of the spinal cord's ability to withstand interruption of arterial inflow.

Longitudinal spinal arteries

The longitudinal spinal circulation consists principally of a single anterior spinal artery (ASA) and two posterior spinal arteries (PSAs). The ASA originates intracranially from branches of the vertebral arteries and descends along the anterior median fissure of the spinal cord. It provides the predominant arterial supply to approximately the anterior two-thirds of the spinal cord, including the anterior horns, corticospinal tracts, spinothalamic pathways, and much of the central grey matter. The anatomical predominance of the ASA is clinically relevant because the motor pathways are largely contained within its vascular territory. Consequently, ischemia affecting the anterior spinal circulation may produce severe bilateral motor deficits and autonomic dysfunction, while the dorsal column modalities of vibration and proprioception may be relatively preserved. The paired PSAs course along the dorsolateral surface of the spinal cord and primarily supply the posterior one-third, including the dorsal columns and portions of the posterior horns. Their territory is interconnected with the anterior circulation through a network of pial vessels known as the vasocorona. The vasocorona forms a circumferential arterial plexus around the spinal cord and provides additional communication between the anterior and posterior vascular territories. This organization

contributes to the intrinsic redundancy of the spinal circulation and provides alternative pathways for redistribution of blood flow.

Despite their longitudinal extension from the craniovertebral junction toward the conus medullaris, the spinal arteries cannot independently provide adequate perfusion to the entire length of the spinal cord. Their caliber and contribution vary along the spinal axis, and the thoracic spinal cord in particular depends substantially on reinforcement from segmental arterial branches.(12)

Segmental arterial supply

The segmental arterial system provides the principal reinforcement of the longitudinal spinal circulation. Segmental vessels arise from the subclavian and thoracoabdominal arterial territories and include intercostal, lumbar, and sacral arteries.

After entering the spinal canal through the intervertebral foramina, these vessels give rise to radicular arteries. Most radicular arteries terminate within the spinal canal or supply the nerve roots and surrounding structures. Only a limited number continue medially as radiculomedullary arteries, reaching the spinal cord itself and reinforcing the longitudinal spinal arteries. This distinction is clinically important. The spinal cord therefore does not receive a direct and equivalent arterial contribution from every intercostal or lumbar artery. Rather, a variable number of segmental arteries provide meaningful reinforcement to the longitudinal spinal circulation. The number, caliber, origin, and distribution of radiculomedullary arteries show substantial interindividual variability. Consequently, two patients undergoing apparently similar aortic repairs may have markedly different residual spinal cord perfusion following exclusion of the same anatomical segment.

This anatomical variability is one of the principal reasons why spinal cord ischemia cannot be predicted solely on the basis of the loss of a particular named artery.(2)

Artery of Adamkiewicz

Among the radiculomedullary arteries, the great anterior radiculomedullary artery, commonly known as the artery of Adamkiewicz, represents the most prominent contributor to the anterior spinal circulation in the thoracolumbar region.

The artery of Adamkiewicz most commonly originates between the lower thoracic and upper lumbar levels, frequently between T8 and L2, and more often arises from the left side. However, its exact level of origin is highly variable. After entering the spinal canal, the vessel typically follows a characteristic hairpin-shaped course before joining the ASA. The artery contributes importantly to perfusion of the lower thoracic spinal cord, lumbar enlargement, and conus medullaris.(13) Its interruption may therefore have substantial neurological consequences, particularly when other components of the collateral network are simultaneously compromised.

Historically, spinal cord protection during thoracoabdominal aortic repair focused heavily on identifying and preserving the artery of Adamkiewicz. The development of more sophisticated vascular imaging and the progressive understanding of spinal cord collateral circulation have, however, demonstrated the limitations of considering this vessel in isolation. Preservation of the artery of Adamkiewicz may be desirable when technically feasible, but its identification does not guarantee preservation of spinal cord perfusion. Conversely, interruption of the artery does not necessarily result in SCI when sufficient collateral pathways remain available.

The contemporary concept of spinal cord protection has therefore shifted from the preservation of a single dominant vessel toward preservation of the overall collateral network.(14)

The collateral network concept

The collateral network represents the central anatomical concept underlying contemporary spinal cord protection.

Spinal cord perfusion is maintained through extensive interconnections between the spinal arterial circulation and multiple extracord vascular territories. These include the subclavian and vertebral arteries, internal thoracic arteries, intercostal arteries, lumbar arteries, hypogastric arteries, and paraspinal muscular branches. Through these connections, blood flow can be redistributed when individual segmental arteries are interrupted. The spinal cord therefore possesses a substantial degree of vascular redundancy, allowing it to tolerate the loss of individual arterial contributors under appropriate haemodynamic conditions.(15) This concept is particularly important during extensive aortic repair. Endograft deployment may intentionally exclude numerous intercostal and lumbar arteries. The resulting reduction in direct segmental inflow does not necessarily produce SCI because blood may continue to reach the spinal circulation through patent collateral pathways. The neurological outcome therefore depends on the balance between arterial inflow lost and collateral capacity preserved. When several collateral territories are simultaneously compromised, however, the physiological reserve of the network may become insufficient. Extensive thoracic coverage, previous infrarenal aortic reconstruction, previous TEVAR, occlusion or intentional coverage of the left subclavian artery, loss of hypogastric perfusion, and perioperative hypotension may act synergistically to reduce spinal cord blood flow.

Thus, the relevant question is not simply whether a particular intercostal artery or the artery of Adamkiewicz remains patent, but whether the remaining network can provide sufficient perfusion to meet the metabolic requirements of the spinal cord.

Collateral network and previous aortic interventions

The concept of cumulative collateral loss is particularly relevant to patients undergoing repeated aortic interventions.

A patient presenting for extensive FEVAR or BEVAR may have already undergone previous EVAR, TEVAR, open aortic reconstruction, or hybrid procedures. Each intervention may have altered one or more components of the spinal cord collateral circulation. The neurological risk associated with the current procedure must therefore be considered within the context of the patient's entire aortic history. Previous exclusion of infrarenal or thoracic segmental arteries may reduce the available collateral reserve before the new endograft is implanted. Additional coverage may then represent the final insult in a progressively compromised vascular network. The same principle applies to the major collateral territories supplied by the subclavian and hypogastric arteries. Loss of one of these territories may be tolerated in isolation but become clinically relevant when combined with extensive thoracic coverage or previous aortic interventions.

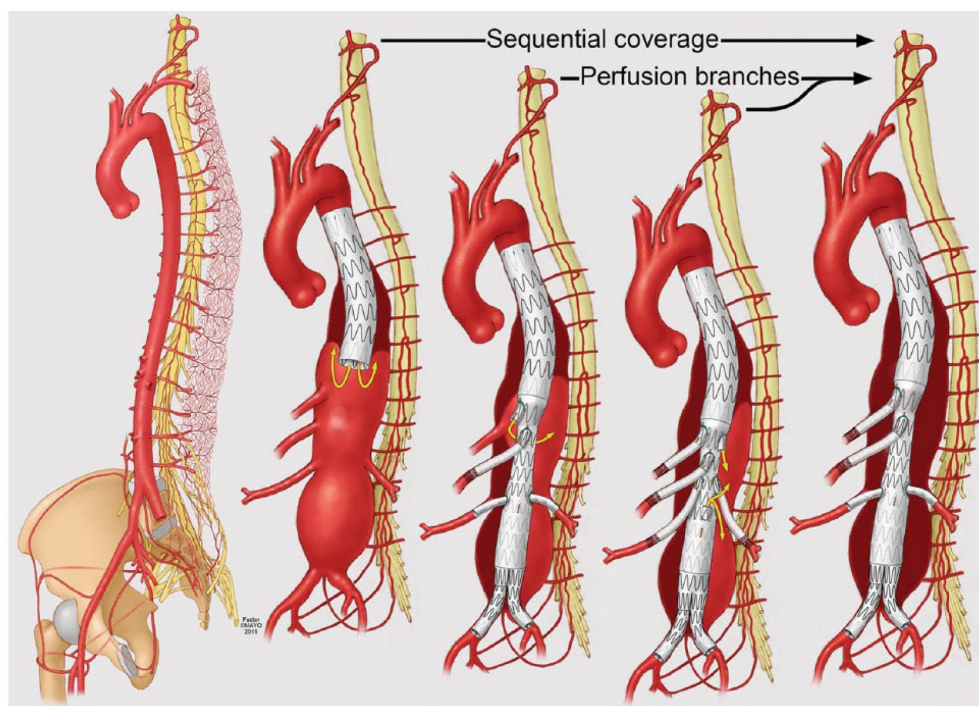
This cumulative model provides an anatomical explanation for why apparently similar endovascular procedures may result in substantially different neurological outcomes between patients.

Dynamic adaptation of the collateral circulation

The collateral network is not a static anatomical structure. Following progressive interruption of segmental arteries, the remaining vascular pathways may undergo functional and structural adaptation.

This response includes vasodilation, increased flow through pre-existing anastomoses, recruitment of previously underperfused vascular channels, and arteriogenic remodeling. These mechanisms may increase the capacity of the collateral circulation to compensate for the progressive loss of direct segmental arterial inflow. The dynamic nature of this process provides an important physiological rationale for staged aortic repair. When extensive aortic coverage is divided into separate procedures, the interval between stages may allow collateral adaptation to occur before additional segmental arteries are excluded. Conversely, abrupt interruption of a large number of segmental arteries during a single-stage procedure may provide insufficient time for effective collateral recruitment. The spinal cord may consequently become dependent on a severely reduced vascular reserve at a time when systemic haemodynamic fluctuations are common. Collateral adaptation therefore represents an important bridge between vascular anatomy and the clinical strategies used to prevent SCI.

FIGURE VIII. — *Schematic representation of a staged endovascular procedure aimed at preserving spinal cord perfusion and promoting collateral network adaptation.*



Pathophysiology of Spinal Cord Ischemia

Spinal Cord Perfusion Pressure

Spinal cord ischemia results from an imbalance between oxygen delivery and the metabolic demands of spinal cord tissue. Although anatomical disruption of the spinal cord blood supply plays a central role, neurological injury ultimately develops when spinal cord perfusion falls below the threshold required to maintain neuronal viability. Consequently, spinal cord perfusion is determined not only by vascular anatomy but also by systemic hemodynamics and cerebrospinal fluid dynamics.

The principal physiological determinant of spinal cord blood flow is the spinal cord perfusion pressure (SCPP), conventionally defined as the difference between mean arterial pressure (MAP) and cerebrospinal fluid pressure (CSFP):

$$\{SCPP = MAP - CSFP\}$$

This relationship highlights the dual mechanisms through which spinal cord perfusion may deteriorate. A reduction in MAP decreases arterial inflow to the spinal cord, whereas an increase in CSFP reduces the pressure gradient across the spinal cord microcirculation, impairing tissue perfusion despite preserved systemic blood pressure. During extensive endovascular aortic repair, both mechanisms frequently coexist. Temporary hypotension, blood loss, vasoplegia, prolonged operative time, and extensive aortic coverage may simultaneously reduce arterial inflow while spinal cord edema increases intrathecal pressure, resulting in a progressive decline in SCPP. Under physiological conditions, spinal cord autoregulation maintains relatively stable blood flow across a broad range of arterial pressures through adaptive vasoconstriction and vasodilation of the intramedullary circulation. However, this autoregulatory capacity becomes progressively impaired during prolonged ischemia or when collateral circulation is extensively compromised. Once autoregulation fails, spinal cord blood flow becomes directly dependent on systemic arterial pressure, rendering even brief episodes of hypotension potentially detrimental.(16)

For this reason, maintenance of adequate MAP during and after complex aortic repair has become one of the cornerstones of contemporary spinal cord protection protocols. Likewise, reduction of CSFP through cerebrospinal fluid drainage represents a complementary strategy aimed at restoring an adequate perfusion gradient despite partial interruption of arterial inflow.

Mechanisms of Ischemic Injury

The development of SCI is not solely the consequence of reduced blood flow but rather the result of a complex cascade of metabolic, inflammatory, and cellular events triggered by ischemia and further amplified during reperfusion.

Reduced spinal cord perfusion initially leads to depletion of intracellular oxygen and glucose, forcing neuronal metabolism to shift from aerobic oxidative phosphorylation to anaerobic glycolysis. This transition results in rapid depletion of adenosine triphosphate (ATP), intracellular acidosis, and failure of energy-dependent ion transport mechanisms. Dysfunction of sodium-potassium ATPase pumps promotes intracellular sodium and water accumulation, leading to neuronal and glial cell swelling. Simultaneously, excessive membrane depolarization induces uncontrolled release of excitatory neurotransmitters, particularly glutamate. Overactivation of N-methyl-D-aspartate (NMDA) receptors allows massive calcium influx into neurons, initiating a process of excitotoxicity characterized by activation of calcium-dependent proteases, phospholipases, and endonucleases. These enzymes contribute to degradation of cytoskeletal proteins, mitochondrial dysfunction, and irreversible cellular injury. Mitochondrial damage further impairs ATP production while promoting the generation of reactive oxygen species (ROS). Oxidative stress damages membrane lipids, structural proteins, and nucleic acids, ultimately amplifying neuronal apoptosis and necrosis.

Concurrently, ischemia activates endothelial cells and resident microglia, initiating a local inflammatory response characterized by cytokine release, leukocyte recruitment, and increased vascular permeability. Breakdown of the blood–spinal cord barrier facilitates extravasation of inflammatory cells and plasma proteins into the spinal cord parenchyma, exacerbating tissue edema and further compromising microvascular perfusion.⁽¹⁵⁾ Unlike acute arterial occlusion of other organs, spinal cord ischemia often develops progressively through the interaction of these multiple mechanisms rather than as an immediate consequence of complete vascular interruption. Consequently, neurological injury evolves dynamically over several hours, providing a potential therapeutic window during which restoration of spinal cord perfusion may still prevent irreversible neuronal loss.⁽¹⁷⁾

Ischemia–Reperfusion Injury

Paradoxically, restoration of blood flow after transient ischemia may itself contribute to secondary spinal cord injury. Reperfusion is essential for tissue survival; however, abrupt

reoxygenation initiates several molecular pathways capable of amplifying neuronal damage beyond that produced by ischemia alone.

Reintroduction of oxygen into previously ischemic tissue markedly increases mitochondrial production of reactive oxygen species. These highly reactive molecules promote lipid peroxidation, protein oxidation, DNA damage, and further mitochondrial dysfunction. At the same time, reperfusion activates complement pathways and enhances neutrophil adhesion to the vascular endothelium, intensifying local inflammation and microvascular dysfunction.

Endothelial injury contributes to impaired capillary autoregulation and increased permeability of the blood–spinal cord barrier. Progressive interstitial edema elevates intrathecal pressure, thereby reducing spinal cord perfusion pressure according to the MAP–CSFP relationship. This creates a vicious cycle in which edema further compromises perfusion, promoting additional ischemia despite restoration of macroscopic arterial blood flow.

Experimental studies have also demonstrated the occurrence of the no-reflow phenomenon, in which persistent obstruction of the spinal cord microcirculation limits effective tissue reperfusion despite successful restoration of proximal arterial inflow. Microvascular thrombosis, endothelial swelling, leukocyte aggregation, and capillary compression all contribute to this phenomenon, explaining why neurological recovery may remain incomplete even after correction of systemic hemodynamic abnormalities.

Recognition of ischemia–reperfusion injury has important therapeutic implications. Contemporary spinal cord protection protocols therefore aim not only to restore arterial perfusion but also to minimize secondary injury through maintenance of adequate systemic blood pressure, optimization of oxygen delivery, reduction of cerebrospinal fluid pressure, correction of anemia, and prompt treatment of neurological deterioration.

Delayed Spinal Cord Ischemia

One of the distinctive features of SCI following complex endovascular aortic repair is its frequently delayed clinical presentation. Although neurological deficits may develop immediately after the procedure, a substantial proportion of patients experience symptom onset several hours or even days after apparently uneventful surgery.

Delayed SCI reflects the dynamic nature of spinal cord perfusion. Following extensive exclusion of segmental arteries, the collateral vascular network undergoes progressive

remodeling to compensate for the reduction in direct arterial inflow. During this transitional period, spinal cord perfusion remains critically dependent on systemic hemodynamic stability. Even transient episodes of hypotension, hypovolemia, cardiac dysfunction, respiratory failure, or anemia may overwhelm the limited compensatory capacity of the collateral circulation and precipitate neurological deterioration. Furthermore, postoperative spinal cord edema may progressively increase cerebrospinal fluid pressure during the first 24–72 hours after surgery, further reducing spinal cord perfusion pressure. This phenomenon explains why neurological deficits may develop despite technically successful endovascular repair and initially normal postoperative neurological examination. The recognition of delayed SCI has profoundly influenced perioperative management in high-volume aortic centers. Current spinal cord protection protocols emphasize prolonged postoperative monitoring, maintenance of elevated mean arterial pressure, avoidance of hypoxia and anemia, frequent neurological assessment, and early implementation of rescue measures—including cerebrospinal fluid drainage—whenever neurological impairment is suspected.

Rather than representing an isolated intraoperative event, SCI should therefore be regarded as a dynamic pathophysiological process extending throughout the perioperative period. This concept underlies the modern multidisciplinary approach to spinal cord protection and provides the rationale for combining anatomical preservation of collateral circulation with meticulous postoperative hemodynamic management.

Risk of Spinal Cord Ischemia in the Endovascular Treatment of Complex Aortic Disease

The management of complex aortic aneurysms has undergone a profound evolution driven by the widespread adoption of advanced endovascular techniques. Fenestrated and branched endovascular aortic repair (f/bEVAR) has significantly expanded the indications for minimally invasive treatments over the last two decades: compared with open surgery, these procedures are associated with reduced perioperative morbidity and mortality, shorter recovery, and lower physiological stress. Nevertheless, SCI remains one of the most devastating complications following extensive endovascular aortic repair.

Although relatively uncommon, SCI may result in permanent neurological deficits ranging from lower limb weakness to irreversible paraplegia, profoundly impairing functional independence, quality of life, and long-term survival. The occurrence of SCI also represents

a major determinant of postoperative resource utilization and healthcare costs, making spinal cord protection a fundamental component of perioperative management in complex aortic interventions. The pathophysiology of SCI is multifactorial and reflects the vulnerability of the spinal cord blood supply to alterations in regional perfusion. Extensive aortic coverage during f/bEVAR may interrupt multiple segmental arteries, including intercostal and lumbar vessels, thereby reducing collateral perfusion to the anterior spinal circulation. This effect may be exacerbated by perioperative hypotension, previous aortic interventions, embolic phenomena, occlusion of collateral vascular networks, or prolonged procedural complexity. Consequently, spinal cord perfusion depends not only on the preservation of collateral circulation but also on the maintenance of an adequate spinal cord perfusion pressure throughout the perioperative period. For this reason, contemporary management relies on a multimodal spinal cord protection strategy rather than on a single preventive measure. Current protocols integrate meticulous preoperative risk stratification, staged repair in selected patients, optimization of systemic hemodynamics, maintenance of adequate hemoglobin and oxygen delivery, selective revascularization of critical collateral vessels, neuromonitoring when available, and intensive postoperative neurological surveillance. These measures aim to maximize spinal cord perfusion while minimizing the duration and extent of ischemic insult.

Among the available neuroprotective strategies, cerebrospinal fluid drainage (CSFD) remains the most extensively investigated. Its physiological rationale is based on increasing spinal cord perfusion pressure through reduction of cerebrospinal fluid pressure, according to the relationship between mean arterial pressure and cerebrospinal fluid pressure. By lowering intraspinal pressure, CSFD may improve spinal cord blood flow despite the reduction in arterial inflow associated with extensive aortic coverage. Despite its well-established role during open thoracoabdominal aortic repair, the use of prophylactic CSFD in endovascular procedures remains controversial. The lower incidence of SCI following f/bEVAR compared with open surgery has limited the availability of high-quality evidence supporting its routine use. Furthermore, CSFD is an invasive procedure associated with potentially severe complications, including spinal epidural hematoma, intracranial hemorrhage, cerebrospinal fluid leakage, infection, and catheter-related adverse events. Consequently, many high-volume aortic centers have adopted a selective approach, reserving prophylactic CSFD for patients considered at highest risk of spinal cord injury while employing rescue drainage in cases of postoperative neurological deterioration. Given the absence of universally accepted selection criteria and the heterogeneity of institutional protocols, real-world evidence from

experienced centers remains essential to refine patient selection and optimize perioperative spinal cord protection strategies.

The present study aims to evaluate the single-center experience of the Vascular Surgery Unit of IRCCS Ospedale Policlinico San Martino, Genoa, with the use of cerebrospinal fluid drainage in patients undergoing fenestrated and branched endovascular aortic repair. Specifically, we analyzed patient selection criteria, perioperative management, neurological outcomes, and drainage-related complications, with the objective of assessing the role of CSFD within a contemporary multimodal spinal cord protection protocol during complex endovascular aortic repair.

Spinal Cord Ischemia after Endovascular Aortic Repair

Epidemiology

The increasing adoption of complex endovascular techniques has substantially expanded the treatment options for patients with thoracoabdominal and complex abdominal aortic aneurysms. Fenestrated and branched endovascular aortic repair (f/bEVAR) has demonstrated excellent technical success rates and lower perioperative morbidity compared with conventional open repair, particularly in elderly patients and those with significant comorbidities. Nevertheless, SCI remains one of the most feared complications following extensive endovascular aortic reconstruction because of its potentially irreversible neurological consequences.(18)

The reported incidence of SCI after f/bEVAR varies considerably across published series, reflecting differences in patient selection, aneurysm extent, procedural complexity, spinal cord protection protocols, and definitions of neurological injury. Contemporary studies generally report overall SCI rates ranging between approximately 3% and 15%, while the incidence of permanent paraplegia is substantially lower, particularly in experienced high-volume centers employing standardized spinal cord protection strategies. This wide variability is largely explained by differences in the anatomical extent of aortic coverage. Procedures involving Crawford extent I–III thoracoabdominal aneurysms require exclusion of a greater number of intercostal and lumbar arteries, resulting in a significantly higher risk of spinal cord hypoperfusion than repairs limited to the juxtarenal or pararenal aorta. Likewise, extensive thoracic stent-graft coverage, previous aortic reconstruction, and multistage procedures influence the observed incidence of neurological complications. Importantly, SCI should not be considered solely a technical complication of endograft implantation. Instead, it represents the final manifestation of multiple interacting anatomical, physiological, and procedural factors that collectively impair spinal cord perfusion. Consequently, considerable attention has shifted from estimating the absolute incidence of SCI toward identifying patients at increased risk and implementing individualized preventive strategies.(19)

Although permanent neurological deficits remain relatively uncommon, even transient spinal cord dysfunction has been associated with prolonged intensive care unit stay, increased hospital length of stay, reduced functional recovery, and impaired long-term survival.

Prevention of SCI has therefore become a primary objective of perioperative management in patients undergoing complex endovascular aortic repair.(20)

Clinical Presentation

The clinical manifestations of SCI encompass a broad spectrum of neurological deficits, ranging from mild lower extremity weakness to complete and irreversible paraplegia. Symptoms may develop immediately following the procedure or, more commonly, several hours after apparently successful intervention, reflecting the dynamic evolution of spinal cord perfusion during the postoperative period.(13)

Motor impairment is the predominant clinical feature and typically involves bilateral lower limb weakness or paralysis. Sensory abnormalities may accompany motor deficits and include diminished pain and temperature sensation, whereas proprioception and vibration sense are frequently preserved due to the relative resistance of the posterior spinal circulation. This pattern reflects the predominant involvement of the anterior spinal artery territory, which supplies the anterior horns and corticospinal tracts. In addition to motor dysfunction, patients may develop autonomic impairment manifested by urinary retention, bowel dysfunction, and reduced anal sphincter tone. The severity of neurological deficits varies considerably and may fluctuate during the early postoperative period in response to changes in systemic blood pressure, cerebrospinal fluid pressure, or collateral spinal cord perfusion. A characteristic feature of postoperative SCI is the occurrence of delayed neurological deterioration. Patients who awaken neurologically intact after anesthesia may subsequently develop progressive lower limb weakness several hours later, often following transient hypotension, hypovolemia, cardiac dysfunction, or respiratory compromise. Recognition of this phenomenon has emphasized the importance of frequent neurological assessment during the first 48–72 postoperative hours. (17)

Standardized neurological examination should include assessment of lower limb motor strength, sensory function, deep tendon reflexes, and sphincter function. The American Spinal Injury Association (ASIA) Impairment Scale remains the most widely accepted tool for grading neurological deficits and monitoring recovery over time. Its standardized classification facilitates comparison between studies and provides an objective measure of neurological outcome in both clinical practice and research.(12)

Diagnosis

The diagnosis of postoperative SCI is primarily clinical and relies on prompt recognition of new neurological deficits following aortic repair. Early identification is essential because timely implementation of rescue measures, including hemodynamic optimization and cerebrospinal fluid drainage, may reverse neurological impairment before irreversible spinal cord injury occurs.

Whenever SCI is suspected, alternative causes of postoperative neurological dysfunction should be excluded. Differential diagnoses include intracranial stroke, peripheral nerve injury, epidural or spinal hematoma, residual effects of neuraxial or general anesthesia, metabolic disturbances, and critical illness neuropathy. Comprehensive neurological examination therefore remains the cornerstone of initial evaluation. Magnetic resonance imaging (MRI) represents the imaging modality of choice for assessing spinal cord injury. Diffusion-weighted imaging and T2-weighted sequences may demonstrate cord edema or ischemic changes, although MRI findings are frequently normal during the early stages of ischemia. Consequently, a normal MRI does not exclude the diagnosis in patients with compatible clinical findings. Computed tomography has limited sensitivity for detecting spinal cord ischemia but may be useful for excluding compressive lesions or procedural complications. In the immediate postoperative setting, imaging should never delay initiation of therapeutic measures when clinical suspicion of SCI is high.

Because delayed neurological deterioration is common, continuous postoperative neurological surveillance is considered an integral component of spinal cord protection protocols. Serial neurological examinations performed by experienced clinicians allow early detection of subtle motor deficits before progression to complete paraplegia, thereby maximizing the opportunity for successful intervention.

Risk Factors for Spinal Cord Ischemia

The development of spinal cord ischemia following complex endovascular aortic repair is multifactorial and cannot be attributed to a single mechanism. Rather than depending exclusively on the anatomical interruption of spinal cord blood supply, SCI results from the interaction between patient characteristics, vascular anatomy, procedural complexity, and perioperative hemodynamic conditions. Consequently, contemporary risk stratification relies on the comprehensive evaluation of multiple variables rather than on individual predictors.

For practical purposes, risk factors may be classified into four major categories: patient-related factors, anatomical factors, procedure-related factors, and perioperative hemodynamic factors.(2)(19)(7)(1)

Patient-Related Risk Factors

Several baseline patient characteristics have been associated with an increased susceptibility to SCI. Advanced age represents one of the most consistently reported predictors, likely reflecting reduced physiological reserve, impaired collateral vessel adaptability, and a higher prevalence of diffuse atherosclerotic disease. Although age itself does not directly compromise spinal cord perfusion, elderly patients appear less capable of tolerating transient reductions in blood flow and systemic oxygen delivery. Pre-existing cardiovascular comorbidities, including coronary artery disease, chronic heart failure, peripheral arterial disease, and chronic kidney disease, further increase the risk of postoperative neurological complications. These conditions may impair cardiac output, limit compensatory hemodynamic responses, or reflect generalized vascular disease affecting the collateral circulation supplying the spinal cord. Renal dysfunction deserves particular attention because it has repeatedly emerged as an independent predictor of adverse neurological outcomes following complex aortic repair. Beyond its association with systemic atherosclerosis, chronic kidney disease frequently coexists with endothelial dysfunction, chronic inflammation, anemia, and impaired autoregulatory capacity, all of which may contribute to reduced spinal cord tolerance to ischemia. Similarly, chronic obstructive pulmonary disease may compromise oxygen delivery through impaired respiratory reserve, while diabetes mellitus has been associated with microvascular dysfunction and reduced endothelial responsiveness. Although the independent contribution of individual comorbidities varies across published studies, the cumulative burden of systemic disease consistently correlates with increased neurological risk.

Anatomical Risk Factors

Among all predictors of SCI, anatomical characteristics of the aneurysm and the planned repair play a predominant role. The extent of aortic coverage remains the single most important anatomical determinant of spinal cord injury. Progressive exclusion of thoracic and abdominal segmental arteries reduces direct arterial inflow to the spinal cord and increases dependence on collateral circulation. Consequently, repairs involving Crawford extent I–III thoracoabdominal aneurysms carry a substantially higher neurological risk than procedures limited to the juxtarenal or pararenal aorta. Coverage exceeding approximately 20 cm of

thoracic aorta has been consistently associated with increased SCI incidence, reflecting interruption of a larger number of intercostal arteries contributing to spinal cord perfusion. Likewise, extensive coverage involving both thoracic and abdominal segments simultaneously compromises multiple collateral pathways. Previous aortic interventions represent another major anatomical risk factor. Prior infrarenal EVAR, open abdominal aortic reconstruction, previous TEVAR, or hybrid procedures may have already interrupted portions of the collateral network. Subsequent endovascular repair further reduces residual collateral perfusion, leaving the spinal cord increasingly dependent on a limited number of remaining vascular pathways. Patency of the left subclavian artery and hypogastric arteries also plays a critical role in maintaining collateral spinal cord blood supply. Occlusion or intentional coverage of these vessels without revascularization may significantly reduce collateral perfusion, particularly in patients undergoing extensive thoracoabdominal repair. Finally, anatomical variations of the spinal cord vascular network, including the origin and distribution of the artery of Adamkiewicz and the density of radiculomedullary arteries, contribute to the marked interindividual variability observed in susceptibility to SCI. Although routine preoperative identification of these vessels remains technically challenging, their anatomical variability likely explains why patients with apparently similar procedures may experience markedly different neurological outcomes.

Procedure-Related Risk Factors

Beyond anatomical considerations, several procedural characteristics directly influence the likelihood of postoperative SCI. Procedural duration has consistently been associated with neurological complications. Longer interventions generally reflect greater anatomical complexity and expose patients to prolonged periods of hemodynamic instability, increased blood loss, greater contrast administration, and repeated catheter manipulation within the thoracoabdominal aorta. Complex branched repairs involving multiple target vessels frequently require prolonged fluoroscopy time and repeated device exchanges, increasing the cumulative physiological stress imposed on the spinal cord. Likewise, technically demanding procedures may necessitate temporary interruption of visceral or iliac perfusion, further compromising collateral circulation. Single-stage repair of extensive thoracoabdominal aneurysms has also been associated with increased SCI risk compared with staged procedures. Progressive exclusion of segmental arteries over multiple interventions allows collateral remodeling to occur before complete interruption of arterial inflow, thereby reducing spinal cord vulnerability. Consequently, staged repair has become an increasingly adopted strategy in selected high-risk patients. Another important procedural factor is intentional coverage of

collateral vessels, particularly the left subclavian artery and internal iliac arteries. Whenever feasible, preservation or revascularization of these vessels should be considered as part of a comprehensive spinal cord protection strategy.

Perioperative Hemodynamic Risk Factors

While anatomical and procedural characteristics determine the baseline susceptibility to SCI, perioperative hemodynamic management frequently represents the decisive factor influencing whether neurological injury ultimately develops. Hypotension remains the most important modifiable risk factor. Because spinal cord perfusion depends directly on the relationship between mean arterial pressure and cerebrospinal fluid pressure, even relatively brief episodes of systemic hypotension may critically reduce spinal cord blood flow in patients with compromised collateral circulation. Current spinal cord protection protocols therefore emphasize strict avoidance of perioperative hypotension and maintenance of supranormal MAP values during the early postoperative period. Hypovolemia, excessive blood loss, and perioperative anemia further compromise oxygen delivery by reducing both spinal cord perfusion and arterial oxygen content. Similarly, postoperative cardiac dysfunction, arrhythmias, myocardial ischemia, or vasoplegia may impair systemic perfusion despite apparently acceptable blood pressure measurements. Respiratory insufficiency and hypoxemia reduce arterial oxygen delivery, while prolonged mechanical ventilation may adversely affect venous return and spinal cord venous drainage through elevated intrathoracic pressure. Metabolic disturbances, including acidosis and hyperglycemia, have also been implicated in exacerbating ischemic neuronal injury, although their independent contribution remains less clearly established. Finally, increased cerebrospinal fluid pressure secondary to postoperative spinal cord edema represents an additional determinant of reduced spinal cord perfusion pressure. This mechanism provides the physiological rationale for cerebrospinal fluid drainage, particularly in patients presenting with postoperative neurological deterioration.

Concluding Remarks

Risk stratification for spinal cord ischemia has progressively evolved from the identification of isolated predictors toward an integrated assessment of cumulative neurological risk. No single factor is sufficient to determine the occurrence of SCI; rather, neurological injury reflects the combined effect of anatomical vulnerability, procedural complexity, and perioperative physiological stress. Recognition of these interacting mechanisms has led to the

development of contemporary multimodal spinal cord protection protocols, which combine meticulous preoperative planning with aggressive perioperative hemodynamic optimization and selective use of adjunctive protective measures. Among these, cerebrospinal fluid drainage remains one of the most extensively investigated strategies and will be discussed in detail in the following chapter.

Risk Stratification for Spinal Cord Ischemia

Accurate preoperative risk stratification has become a cornerstone of contemporary spinal cord protection strategies in complex endovascular aortic repair. Because SCI results from the cumulative interaction of multiple anatomical, procedural, and physiological factors, no single variable accurately predicts neurological injury. Instead, current practice relies on comprehensive assessment of the patient's overall risk profile to guide the implementation of preventive measures.

Although no universally accepted risk prediction model currently exists, several institutions have developed structured protocols based on combinations of clinical, anatomical, and procedural variables. These institutional algorithms aim to identify patients most likely to benefit from intensive spinal cord protection measures, including prophylactic cerebrospinal fluid drainage, staged repair, aggressive postoperative hemodynamic support, and prolonged neurological monitoring. The cornerstone of risk stratification remains the anticipated extent of aortic coverage. Patients undergoing repair of Crawford extent I–III thoracoabdominal aneurysms or requiring extensive thoracic stent-graft implantation consistently represent the highest-risk population because of the large number of segmental arteries excluded during the procedure. However, anatomical extent alone is insufficient to estimate neurological risk accurately. Previous aortic interventions substantially modify the collateral circulation and should therefore be considered cumulative rather than independent risk factors. Patients with previous infrarenal EVAR, open abdominal aortic replacement, thoracic endovascular repair, or hybrid procedures frequently present with pre-existing interruption of collateral pathways, rendering subsequent repairs considerably more hazardous despite similar anatomical characteristics. Risk assessment should also incorporate preservation of major collateral vessels. Planned coverage of the left subclavian artery without revascularization, bilateral hypogastric artery occlusion, or extensive iliac reconstruction may significantly reduce collateral spinal cord perfusion and should therefore be integrated into preoperative decision-making.

Equally important are patient-specific physiological factors. Advanced age, chronic kidney disease, impaired left ventricular function, chronic pulmonary disease, and limited cardiopulmonary reserve may reduce the patient's capacity to tolerate transient reductions in spinal cord perfusion. These variables, although individually modest predictors, may substantially increase overall neurological risk when combined with extensive aortic coverage.

Procedural complexity further contributes to cumulative risk. Long operative duration, multiple target vessel incorporation, anticipated technical difficulty, prolonged fluoroscopy time, and the likelihood of significant blood loss or perioperative hypotension should all be considered during procedural planning.

Rather than relying on isolated predictors, many high-volume aortic centers currently adopt a risk-adapted approach, categorizing patients into low-, intermediate-, and high-risk groups according to the cumulative burden of adverse factors. Although specific criteria differ among institutions, patients generally considered at high risk share several common characteristics, including extensive thoracoabdominal aortic coverage, previous aortic surgery, compromised collateral circulation, and the anticipated need for prolonged or technically demanding procedures. This individualized approach has important therapeutic implications. Low-risk patients may undergo standard perioperative management with routine neurological surveillance, whereas intermediate-risk patients often require intensified postoperative hemodynamic monitoring. In contrast, high-risk patients are typically managed using comprehensive multimodal spinal cord protection protocols that may include prophylactic cerebrospinal fluid drainage, maintenance of supranormal mean arterial pressure, staged repair when anatomically feasible, preservation or revascularization of collateral vessels, and extended postoperative intensive care monitoring. It should be emphasized that risk stratification is not a static preoperative assessment but rather a dynamic process extending throughout the perioperative period. Intraoperative events such as prolonged hypotension, unexpected technical complexity, excessive blood loss, or unplanned coverage of collateral vessels may substantially modify the patient's neurological risk and should prompt immediate reassessment of the spinal cord protection strategy.

Ultimately, the objective of contemporary risk stratification is not merely to estimate the probability of SCI but to identify patients in whom preventive interventions are most likely to improve neurological outcomes. This philosophy underpins current multimodal spinal cord protection protocols and provides the rationale for the selective application of cerebrospinal

fluid drainage, which remains one of the most extensively studied adjunctive strategies in complex endovascular aortic repair.

Strategies for Spinal Cord Protection

Spinal cord protection during complex endovascular aortic repair is based on a multimodal strategy aimed at preserving spinal cord perfusion, maintaining adequate oxygen delivery, and promoting the development or preservation of collateral circulation. No single intervention is sufficient to prevent spinal cord ischemia in all patients. Rather, contemporary protocols integrate hemodynamic optimization, limitation or staging of aortic coverage, preservation of major collateral vessels, intraoperative neurological monitoring, and optimization of systemic oxygen delivery. The intensity of these measures should be adapted to the patient's cumulative neurological risk and to the anatomical extent of the planned repair. The rationale for this multimodal approach derives from the complex and dynamic nature of spinal cord perfusion. During extensive thoracoabdominal endovascular repair, multiple sources of spinal cord blood supply may be simultaneously compromised. However, the collateral network can partially compensate for the loss of individual arterial inputs, provided that sufficient perfusion pressure and oxygen delivery are maintained. Accordingly, spinal cord protection should not be regarded as the preservation of a single critical artery, but rather as the preservation of an adequate perfusion gradient and of the capacity of the collateral network to sustain spinal cord blood flow. (21)

Hemodynamic optimization

Maintenance of adequate spinal cord perfusion pressure represents one of the fundamental principles of perioperative spinal cord protection. As previously discussed, SCPP can be approximated by the difference between MAP and CSFP. Consequently, systemic hypotension or an increase in CSFP may substantially reduce the perfusion gradient across the spinal cord, particularly in patients in whom the collateral circulation has already been compromised by extensive aortic coverage.

Avoidance of hypotension is therefore particularly important during and after complex endovascular repair. Intraoperative reductions in arterial pressure may occur as a consequence of anaesthesia, blood loss, hypovolemia, vasodilation, cardiac dysfunction, or procedural events. These episodes may be poorly tolerated when multiple segmental arteries have been excluded, because the spinal cord may become increasingly dependent on the remaining collateral pathways. Postoperatively, hypotension is similarly important because delayed SCI may develop after an initially uncomplicated procedure, particularly when collateral perfusion

remains marginal. Hemodynamic management should therefore include adequate intravascular volume, preservation of cardiac output, prompt treatment of vasoplegia, and avoidance of prolonged hypotensive episodes. Blood pressure targets should be individualized according to the patient's baseline physiology, extent of aortic coverage, neurological status, and institutional spinal cord protection protocol rather than being considered as a single universal threshold. In patients who develop neurological deterioration, prompt augmentation of systemic arterial pressure is commonly incorporated into rescue protocols in order to increase SCPP. (22)

Hemodynamic optimization should also extend beyond arterial pressure. Adequate cardiac output and arterial oxygen content are necessary to ensure effective oxygen delivery to the spinal cord. Consequently, hypoxemia, significant blood loss, severe anemia, and impaired cardiac function should be promptly recognized and corrected whenever clinically appropriate. These measures are particularly relevant in the early postoperative period, when the spinal cord may remain vulnerable despite technically successful exclusion of the aneurysmal segment.

Staged repair

Staged endovascular repair is one of the principal strategies used to reduce the neurological burden associated with extensive aortic coverage. The underlying rationale is based on the capacity of the collateral network to adapt progressively following interruption of segmental arterial inflow. Rather than simultaneously excluding a large number of intercostal and lumbar arteries, staged procedures allow the spinal cord to adapt to a partial reduction in blood supply before definitive completion of the repair.

In patients undergoing extensive thoracoabdominal repair, the first stage may consist of partial exclusion of the diseased aorta, followed by completion of the repair after an interval that allows collateral recruitment and remodeling. The exact timing between stages varies according to the underlying pathology, anatomical configuration, clinical urgency, and procedural strategy. Staging is therefore not simply a technical modification, but represents a deliberate attempt to reduce the abruptness of the reduction in spinal cord perfusion. The clinical rationale for staged repair is supported by observational evidence and by contemporary guidelines. The 2024 EACTS/STS guidelines recommend considering staged TEVAR followed by branched or fenestrated repair in patients at increased risk of SCI

undergoing treatment of extensive Crawford I–III and V thoracoabdominal aneurysms. Similarly, the 2026 ESVS guidelines recommend considering staged repair in extensive thoracic or thoracoabdominal endovascular procedures.

The strategy is nevertheless not universally applicable. Staging may be unsuitable in patients requiring urgent exclusion because of rupture, impending rupture, symptomatic disease, infection, or other circumstances in which delaying definitive treatment would expose the patient to substantial risk. Moreover, the interval between procedures introduces a period during which the aneurysm or residual aortic pathology remains incompletely excluded. Consequently, the decision to stage should balance the potential neurological benefit against the risks associated with an incompletely treated aorta.(6,18)

Left subclavian artery revascularization

The left subclavian artery (LSA) represents an important component of the collateral circulation supplying the spinal cord. Its contribution is particularly relevant in patients undergoing extensive thoracic aortic coverage, in whom exclusion of the LSA may further reduce the number of available collateral pathways. Intentional LSA coverage may therefore increase the neurological risk associated with extensive endovascular aortic repair, particularly when other collateral territories are simultaneously compromised. The relevance of the LSA is further influenced by the vertebral circulation, the presence of a dominant left vertebral artery, previous cerebrovascular or upper-limb disease, and the status of other collateral vessels. For these reasons, revascularization of the LSA should be considered when its coverage is required, particularly in patients undergoing zone 2 thoracic endovascular repair or in those with additional factors suggesting substantial dependence on the subclavian–vertebral collateral pathway. The 2024 EACTS/STS guidelines recommend LSA revascularization in patients undergoing zone 2 TEVAR to reduce neurological complications. In the setting of fenestrated or branched thoracoabdominal repair, the decision should remain individualized according to the planned proximal landing zone, the anatomy of the arch and supra-aortic vessels, and the overall collateral network. LSA preservation or revascularization should therefore be regarded as one component of a broader spinal cord protection strategy rather than as an isolated intervention.(5)

Preservation of hypogastric arteries

The internal iliac, or hypogastric, arteries represent another important source of collateral blood flow to the spinal cord through the pelvic and paraspinal arterial networks. Their preservation becomes particularly relevant when extensive thoracoabdominal aortic coverage is combined with previous infrarenal aortic interventions or additional iliac reconstruction. Bilateral hypogastric artery occlusion may substantially reduce the available collateral pathways and may therefore increase vulnerability to SCI, especially when other major sources of spinal cord perfusion have already been compromised. For this reason, preservation of at least one patent hypogastric artery is generally incorporated into the planning of extensive aortic repair whenever anatomically and technically feasible. The importance of this principle is particularly evident in patients with previous aortic surgery, in whom part of the collateral network may already have been interrupted. In such cases, further exclusion of the hypogastric circulation may result in cumulative loss of collateral inflow. Preoperative imaging should therefore assess the patency and anatomy of the iliac arteries and identify situations in which preservation or revascularization may be particularly important. (6)

Temporary aneurysm sac perfusion

Temporary aneurysm sac perfusion (TASP) is an emerging strategy designed to combine the benefits of staged repair with the technical advantages of a single endovascular procedure. The underlying concept is to maintain temporary perfusion of the aneurysm sac and its associated segmental arteries after partial endovascular exclusion, thereby preserving blood flow through collateral pathways during the interval preceding definitive completion of the repair.(13)(15) In its typical application, a temporary endoleak is deliberately maintained through a dedicated branch or fenestration during the first stage of treatment. Definitive exclusion of the aneurysm is subsequently performed after an interval intended to allow collateral remodeling. In this way, TASP attempts to reproduce the physiological principle of staged repair while avoiding the need for a completely separate initial intervention. Early clinical studies have reported encouraging results. However, the available evidence remains predominantly observational and is based on relatively small cohorts. Potential disadvantages include persistent endoleak, aneurysm expansion or rupture during the interval between stages, and other complications associated with maintaining temporary perfusion. In addition, the technique has not been established as equivalent to conventional staged repair. Consequently, TASP should currently be regarded as a promising but still selective and

investigational adjunct to spinal cord protection rather than a universally adopted standard of care.

Neuromonitoring

Intraoperative neuromonitoring provides a functional assessment of spinal cord integrity during complex aortic procedures. The principal techniques include motor evoked potentials (MEPs) and somatosensory evoked potentials (SSEPs), while near-infrared spectroscopy (NIRS) has also been investigated as a non-invasive method of indirectly assessing regional tissue oxygenation. MEPs evaluate the integrity of motor pathways and are particularly sensitive to acute reductions in spinal cord perfusion. A sudden decrease or loss of MEP amplitude may indicate an impairment of spinal cord blood flow and can prompt immediate corrective interventions, including optimization of systemic blood pressure, restoration of perfusion to compromised vascular territories, or modification of the procedural strategy. SSEPs provide complementary information regarding sensory pathways, although their relationship with clinically relevant SCI appears less consistent than that of MEPs.

The principal advantage of neuromonitoring is the possibility of detecting neurological compromise before irreversible clinical deficits become apparent. However, interpretation is influenced by several factors, including anaesthetic agents, hypotension, temperature, technical issues, and neuromuscular blockade. Furthermore, neuromonitoring is primarily useful for detecting intraoperative or immediate neurological compromise and cannot reliably exclude delayed postoperative SCI. Consequently, the absence of intraoperative abnormalities does not eliminate the need for careful postoperative neurological and hemodynamic surveillance. Neuromonitoring should therefore be regarded as an adjunct to, rather than a replacement for, anatomical planning and physiological spinal cord protection. Its greatest potential value lies in allowing rapid identification of a potentially reversible perfusion deficit and facilitating immediate intervention.(23)

Temperature management

Temperature represents an additional determinant of spinal cord metabolic demand. Hypothermia reduces cellular metabolism and oxygen consumption and can therefore increase tissue tolerance to temporary reductions in blood flow. This principle has been extensively investigated in open thoracoabdominal aortic surgery, in which systemic or selective

hypothermia may be used during periods of circulatory interruption. Its role in complex endovascular repair is considerably less established. Unlike open repair, f/bEVAR does not generally require prolonged complete interruption of systemic circulation, and the potential benefits of deliberate systemic hypothermia must therefore be balanced against complications such as arrhythmias, coagulopathy, impaired drug metabolism, and infection risk. Consequently, routine deep hypothermia is not considered a standard component of endovascular spinal cord protection. In the endovascular setting, particular attention should instead be paid to avoiding inadvertent hypothermia and maintaining physiological temperature throughout the perioperative period. Temperature should be interpreted as one component of the overall metabolic and hemodynamic environment rather than as an isolated neuroprotective intervention. (22)

Hemoglobin optimization

Adequate oxygen delivery is essential for maintaining spinal cord viability, particularly when arterial inflow is reduced following extensive aortic coverage. Because oxygen delivery depends on both blood flow and arterial oxygen content, significant anemia may further compromise a spinal cord that is already dependent on a reduced collateral circulation. Perioperative blood loss is common during complex aortic interventions and may be associated with a progressive reduction in hemoglobin concentration. Severe or rapidly developing anemia should therefore be recognized and corrected according to the overall clinical context. Optimization of hemoglobin should be considered together with adequate cardiac output, oxygenation, and systemic perfusion rather than as an isolated numerical target. At the same time, indiscriminate transfusion solely to achieve a predefined hemoglobin concentration is not supported by sufficient evidence to establish a universal spinal cord-specific threshold. Transfusion decisions should therefore consider the degree and rate of blood loss, hemodynamic status, tissue oxygenation, comorbidities, and the patient's overall clinical condition. In patients at high risk of SCI, avoidance of significant untreated anemia is particularly important because reduced oxygen-carrying capacity may amplify the consequences of marginal spinal cord perfusion.(21)

Multimodal approach

The strategies described above are most effective when integrated into a coordinated perioperative protocol rather than applied independently. Extensive aortic coverage may simultaneously compromise segmental arterial inflow, the left subclavian circulation, and pelvic collateral pathways; therefore, preservation of any single vascular territory cannot completely eliminate neurological risk. Contemporary spinal cord protection consequently relies on a combination of anatomical planning and physiological optimization. Preoperative assessment should identify the extent of planned aortic coverage, previous aortic interventions, patency of the subclavian and hypogastric arteries, and other factors associated with increased neurological vulnerability. During the procedure, preservation of collateral circulation, avoidance of hypotension and hypoxemia, and, when appropriate, staged repair or adjunctive perfusion techniques may reduce the abrupt reduction in spinal cord blood flow. Postoperatively, maintenance of adequate systemic perfusion, avoidance of anemia and hypoxia, and close neurological surveillance remain essential because SCI may occur despite an initially uncomplicated procedure. Within this multimodal framework, cerebrospinal fluid drainage occupies a particularly important position because it directly modifies the physiological determinant of spinal cord perfusion represented by CSFP. Its potential benefits, indications, technical aspects, complications, and areas of controversy will therefore be examined separately in the following chapter.(21)(17)

Cerebrospinal Fluid Drainage

CSFD represents one of the most widely used adjunctive strategies for the prevention and treatment of SCI during extensive thoracic and thoracoabdominal aortic repair. Its physiological rationale derives directly from the relationship between SCPP, MAP, and CSFP. By reducing CSFP, CSFD increases the pressure gradient available for spinal cord perfusion and may therefore partially compensate for the loss of arterial inflow resulting from extensive aortic coverage. Although the effectiveness of CSFD is well established in open thoracoabdominal aortic aneurysm (TAAA) repair, its role in endovascular treatment remains more controversial. In particular, the balance between the potential neurological benefit of prophylactic drainage and the risk of drain-related complications has led to a progressively more selective, risk-adapted approach. Current guidelines generally support prophylactic CSFD in patients undergoing open TAAA repair and recommend considering it in patients undergoing endovascular thoracic or thoracoabdominal repair who are considered to be at increased risk of SCI. (24)(25)

Physiological rationale

The physiological basis of CSFD is closely related to the concept of spinal cord perfusion pressure previously described. SCPP is conventionally approximated as:

$$\text{SCPP} = \text{MAP} - \text{CSFP}$$

Consequently, spinal cord perfusion can be improved either by increasing systemic arterial pressure or by reducing CSFP. During extensive endovascular aortic repair, interruption of multiple segmental arteries may markedly reduce arterial inflow to the spinal cord. At the same time, ischemia-reperfusion injury and spinal cord edema may increase intrathecal pressure, further reducing the perfusion gradient. CSFD acts on the latter mechanism by lowering CSFP and thereby increasing SCPP despite a potentially compromised arterial supply. The importance of this mechanism is particularly evident in patients with an already compromised collateral network. When extensive aortic coverage, previous aortic interventions, occlusion of the left subclavian artery, or loss of hypogastric flow have reduced the available collateral pathways, spinal cord perfusion may become critically dependent on the remaining arterial inflow. In this setting, even relatively modest reductions in MAP or increases in CSFP may result in clinically significant neurological dysfunction. CSFD may therefore be used both prophylactically and therapeutically. Prophylactic drainage aims to

maintain an adequate SCPP throughout a high-risk procedure and the early postoperative period, whereas therapeutic drainage is initiated in response to neurological deterioration suggestive of SCI. In the latter setting, CSFD is generally combined with aggressive optimization of systemic blood pressure and correction of other potentially reversible causes of reduced spinal cord perfusion. The 2022 American College of Cardiology/American Heart Association guideline specifically recommends timely measures to optimize spinal cord perfusion and decrease intrathecal pressure in patients developing delayed spinal cord dysfunction after open or endovascular TAAA repair.

Importantly, reduction of CSFP does not restore arterial inflow to the spinal cord. Rather, it increases the perfusion gradient across the spinal cord and may allow the remaining collateral circulation to provide sufficient blood flow. CSFD should therefore be considered a complementary intervention within a multimodal spinal cord protection protocol rather than a substitute for adequate systemic perfusion or preservation of collateral circulation.

Indications

The indication for CSFD should be based on an individualized assessment of neurological risk. The risk of SCI increases with the extent of planned aortic coverage and with the cumulative disruption of collateral pathways. Patients undergoing extensive thoracoabdominal repair, particularly Crawford extent I–III aneurysms, those with previous aortic interventions, and those with compromised left subclavian or hypogastric circulation, are generally considered to be at increased risk.

In the endovascular setting, additional factors may further support prophylactic CSFD, including extensive thoracic aortic coverage, planned coverage of multiple segmental arteries, previous infrarenal or thoracic aortic repair, extensive branched or fenestrated reconstruction, and a history of neurological complications following previous aortic intervention. The decision should also take into account the possibility of postoperative hypotension, the expected duration and complexity of the procedure, and the availability of rapid neurological assessment and rescue treatment. The contemporary approach has consequently shifted from routine CSFD for all endovascular procedures toward selective prophylactic drainage in patients with a high cumulative risk of SCI. This approach attempts to maximize the potential neurological benefit while avoiding unnecessary exposure to the complications associated with neuraxial catheterization.

CSFD also has an important role as a rescue therapy. The development of new-onset paraplegia, paraparesis, or other neurological deficits following extensive endovascular aortic repair should prompt immediate evaluation for SCI and rapid implementation of a spinal cord rescue protocol. Such protocols generally combine elevation of systemic arterial pressure with reduction of CSFP through CSFD, together with correction of hypoxemia, anemia, hypovolemia, and other potentially reversible contributors to impaired spinal cord perfusion. The 2024 EACTS/STS guidelines specifically recommend immediate CSF drainage in patients with new-onset paraplegia after TEVAR or open TAAA repair. (13,16)(2)

Current guidelines

The evidence supporting CSFD differs substantially between open and endovascular aortic repair. In open TAAA repair, randomized clinical evidence has demonstrated a reduction in perioperative SCI with prophylactic CSFD, and the technique has consequently become an established component of spinal cord protection. The 2022 ACC/AHA guideline recommends CSFD in patients undergoing open TAAA repair who are at high risk for SCI (Class I, Level of Evidence A). The 2024 EACTS/STS guidelines similarly recommend CSF drainage for spinal cord protection during open TAAA replacement (Class I, Level B).(5) For patients at increased risk of SCI undergoing endovascular treatment of thoracic or thoracoabdominal aortic disease, prophylactic CSFD should be considered (Class IIa, Level C), reflecting the lower quality and predominantly observational nature of the available evidence in the endovascular setting. The recently published 2026 ESVS Guidelines on the Management of Descending Thoracic and Thoraco-Abdominal Aortic Diseases maintain this distinction. (6) Prophylactic CSFD is recommended for patients undergoing open thoracic or thoracoabdominal aortic repair (Class I, Level A), whereas the guidelines emphasize early postoperative neurological assessment after open or endovascular repair and incorporate other strategies, including staged repair and preservation of internal iliac artery flow, into the broader spinal cord protection pathway. Thus, contemporary recommendations do not support indiscriminate prophylactic CSFD for all patients undergoing endovascular repair. Rather, the current evidence favours a risk-adapted strategy, particularly for extensive thoracic and thoracoabdominal procedures in which the anticipated risk of SCI is sufficiently high to justify the potential complications of lumbar drainage.

This distinction is particularly relevant to fenestrated and branched endovascular aortic repair. Although f/bEVAR can avoid the physiological burden associated with open surgery,

extensive treatment may require exclusion of a large number of intercostal and lumbar arteries and may therefore produce a substantial risk of SCI. Consequently, prophylactic CSFD is frequently incorporated into institutional protocols for selected high-risk f/bEVAR patients, although the optimal patient-selection criteria remain incompletely standardized.

Drain Placement and Technical Considerations

Lumbar cerebrospinal fluid drainage is performed using a technique similar to that employed for epidural anesthesia, although it differs in that the procedure ultimately involves access to the subarachnoid space. Placement of the CSF drain is performed by the anesthesiology team during the perioperative period. The patient may be awake or may have previously undergone preanesthetic medication. Performing the procedure in an awake patient allows for monitoring of pain and the development of any neurological symptoms during needle insertion and catheter placement, thereby minimizing the potential risk of neurological injury. The patient is positioned either in the lateral decubitus or sitting position. The sitting position, with the head and back flexed forward, provides better visualization of the anatomical landmarks and facilitates midline access to the intervertebral space. This positioning also reduces the risk of puncturing the epidural venous plexus, which is predominantly located in the paramedian region. Conversely, the lateral decubitus position results in lower hydrostatic CSF pressure. Since CSF pressure physiologically increases in the craniocaudal direction, this positioning limits the amount of CSF that escapes at the time of puncture, thereby reducing the potential risk of cerebellar tonsillar herniation resulting from rapid and excessive CSF loss. CSF drainage is performed at the L3–L4 or L4–L5 intervertebral space, corresponding approximately to the line connecting the iliac crests. Selection of the intervertebral level must take into consideration the extent of the spinal cord, which generally terminates above the level of L2. The CSF drain is inserted under sterile conditions following skin disinfection and preparation of the sterile field. Local anesthesia is then administered. A 14-gauge Tuohy needle is advanced until the subarachnoid space is reached, as confirmed by the appearance of CSF flow (Figure IX). Alternatively, when a technique using a syringe filled with sterile saline is employed, entry into the epidural space is identified by the loss-of-resistance technique. The syringe is then removed, and the Tuohy needle is carefully advanced through the dura mater into the subarachnoid space.(9)

FIGURE IX. — *Insertion and advancement of the Tuohy needle at the L3–L4 intervertebral space at Policlinico San Martino, Genoa.*



Subsequently, a flexible, multi-hole catheter is inserted and advanced by 5–10 cm. If catheter placement is unsuccessful, the needle and catheter should be withdrawn together to avoid damaging the catheter itself. Advancement should be performed without encountering resistance. If paresthesia or pain occurs, the procedure should be immediately interrupted and the catheter retracted until the symptoms resolve. In some studies, the catheter has been inserted to a greater length, up to 20 cm, which may reduce the risk of catheter dislodgement associated with patient movement. However, advancing a longer portion of the catheter into the subarachnoid space may increase the risk of neurological injury. Catheter patency is then assessed by flushing the catheter with 10 mL of normal saline. The proximal portion of the catheter is secured to the patient's back with an adhesive dressing (Figure X); the free end is connected to the pressure transducer system for monitoring cerebrospinal fluid pressure and to the collection bag for drained CSF. If the patient remains intubated and sedated at the end of the procedure, a neurological wake-up test should be performed as soon as feasible to assess the patient's neurological status.

FIGURE X. — *Insertion and fixation of a prophylactic CSFD at Policlinico San Martino, Genoa.*



The timing of catheter placement varies among institutions. In elective high-risk procedures, placement may be performed before the aortic intervention, allowing the catheter to be tested and ensuring immediate availability for both prophylactic and therapeutic drainage. Other centres favour placement after induction of anaesthesia on the day of surgery. In urgent or emergent procedures, particularly in the presence of rupture or significant coagulopathy, prophylactic catheter placement may be avoided because the risks associated with neuraxial instrumentation may outweigh the potential preventive benefit. Correct pressure measurement is essential because CSFP and arterial pressure must be referenced at comparable anatomical levels and expressed in compatible units. The relationship between CSFP and MAP determines the effective SCPP, and inappropriate transducer positioning may therefore lead to erroneous estimation of the spinal cord perfusion gradient. There is no universally accepted target CSFP or drainage rate. Contemporary protocols generally aim to maintain CSFP within a low-normal range while avoiding excessive drainage. Values around 10–12 mmHg have historically been used in many protocols, although some centres employ targets below 10 mmHg in patients with neurological deficits. Drainage is generally performed in a controlled fashion, with commonly reported maximum rates in the range of approximately 10–20 mL/h, although institutional protocols differ substantially. The absence of a universally validated

pressure target is clinically important. Excessive CSF removal does not necessarily translate into further neurological benefit and may increase the risk of intracranial hypotension and haemorrhagic complications. The objective should therefore be to achieve adequate SCPP using the lowest effective CSFP reduction and the minimum necessary drainage volume. In patients without neurological deficits, CSFD is usually maintained during the period of greatest neurological vulnerability and subsequently weaned once the patient's haemodynamics and neurological examination are considered stable. Different protocols maintain the drain for approximately 24–72 hours, although the duration should be individualized according to the extent of aortic coverage and the patient's neurological risk. If new neurological deficits develop, CSFD becomes therapeutic. In this situation, the treatment objective is to rapidly increase SCPP through a combination of CSF pressure reduction and augmentation of MAP. Contemporary rescue protocols therefore generally involve immediate neurological assessment, elevation of arterial pressure, verification of drain patency, and controlled CSF drainage. (16)

Particular attention must also be paid to haemostasis. Because lumbar catheterization represents a neuraxial procedure, anticoagulant and antiplatelet therapy must be managed carefully according to the patient's thrombotic and bleeding risk and institutional protocols. Coagulopathy, thrombocytopenia, active bleeding, or difficult spinal anatomy may substantially increase the procedural risk and should therefore be considered before prophylactic catheter placement.

Complications

The principal limitation of CSFD is that the procedure itself may cause clinically significant complications. These range from relatively minor symptoms, such as post-dural puncture headache or CSF leakage, to potentially devastating neurological and intracranial haemorrhagic complications.

Intracranial hypotension and intracranial haemorrhage are among the most serious complications. Excessive or uncontrolled CSF drainage may produce a rapid reduction in intracranial pressure, leading to downward displacement of intracranial structures and stretching of intracranial vessels. Subdural or intracranial haemorrhage may consequently occur, particularly in patients with predisposing cerebral or vascular conditions.

Spinal or epidural haematoma represents another particularly relevant complication because it may itself cause neurological deterioration and may be difficult to distinguish clinically from SCI caused by aortic repair. The risk is increased in the presence of coagulation abnormalities, thrombocytopenia, or antithrombotic therapy.

The largest published series specifically evaluating CSFD complications during first-stage and completion fenestrated-branched endovascular aortic repair included 187 patients who underwent drainage during 240 procedures. CSFD-related complications occurred in approximately 9% of procedures, including intracranial hypotension, intracranial haemorrhage, spinal haematoma, and bleeding during attempted catheter placement. Notably, some neurological events were directly attributed to the drainage procedure itself, highlighting the importance of careful patient selection and meticulous catheter management. More recent data confirm that these complications are not negligible. In a cohort of patients undergoing complex endovascular aortic repair with prophylactic CSFD, approximately 12% developed a drain-related complication, with a smaller proportion experiencing disabling complications. A 2026 study including patients undergoing TEVAR and complex EVAR reported major CSFD-related complications in 4.4%, all of which were haemorrhagic, while minor complications occurred in 16.7%. (26)

Other complications include post-dural puncture headache, persistent CSF leakage, infection or meningitis, catheter obstruction or displacement, traumatic insertion, and technical failure. In patients with previous spinal surgery or difficult spinal anatomy, fluoroscopic guidance may facilitate catheter placement and reduce technical difficulties. The occurrence of these complications emphasizes an important principle: CSFD is not a risk-free intervention. Its use should therefore be based on a careful balance between the anticipated risk of SCI and the individual risk associated with lumbar catheterization and drainage.(16)

Current controversies

Despite its widespread use, several aspects of CSFD remain controversial, particularly in endovascular aortic repair. The first and most important question concerns the efficacy of routine prophylactic drainage. While randomized evidence supports CSFD in open TAAA repair, comparable evidence in TEVAR and f/bEVAR is lacking. A systematic review and meta-analysis including 4,793 patients undergoing TEVAR found no significant difference in overall SCI rates between patients receiving prophylactic CSFD and those managed without

drainage. The authors therefore emphasized the need for randomized trials capable of defining which patients derive a meaningful neurological benefit from CSFD. (25)

This uncertainty is particularly relevant because prophylactic drainage exposes patients who would never have developed SCI to a procedure carrying its own risk of haemorrhagic and neurological complications. Conversely, selective or therapeutic drainage requires the ability to recognize neurological deterioration rapidly and to establish CSF drainage without delay. This has led to two broad approaches: a prophylactic strategy, in which the catheter is placed before or during a high-risk procedure, and a selective or rescue strategy, in which drainage is reserved for patients who develop neurological deterioration or who are considered to have particularly high baseline risk. The optimal CSFP target and drainage rate also remain uncertain. Published protocols vary considerably, with target pressures ranging from approximately 7–15 mmHg and drainage rates commonly ranging from 10 to 20 mL/h. Higher drainage volumes have been associated with increased rates of drain-related complications, suggesting that more aggressive drainage does not necessarily provide greater neurological protection. The timing and duration of CSFD are similarly not standardized. Some centres place the catheter before the procedure, whereas others insert it after induction of anaesthesia or reserve placement for patients developing postoperative neurological deficits. Likewise, the optimal duration of drainage after an uncomplicated procedure remains uncertain and is generally determined according to institutional protocols and individual neurological risk. Another important unresolved issue concerns the management of delayed SCI. Neurological deficits may occur several hours after an apparently successful procedure, when the patient is no longer in the operating room and when the effects of anaesthesia may have disappeared. This emphasizes the importance of early postoperative neurological assessment and continuous haemodynamic surveillance. The 2026 ESVS guidelines specifically recommend early clinical neurological evaluation after open or endovascular thoracic or thoracoabdominal aortic repair to facilitate prompt recognition and treatment of SCI.

Finally, there is increasing interest in replacing a binary “drain versus no drain” approach with risk-adapted, protocol-based management. Such protocols integrate anatomical risk, previous aortic interventions, planned extent of coverage, preservation of collateral vessels, haemodynamic targets, neurological monitoring, and the availability of rapid rescue CSFD. This approach reflects the current understanding that SCI is a multifactorial complication and that the effectiveness of CSFD depends on its integration within a broader spinal cord protection strategy.

Concluding remarks

Cerebrospinal fluid drainage remains one of the most extensively investigated interventions for spinal cord protection during aortic repair. Its physiological rationale is compelling: by reducing CSFP, CSFD increases SCPP and may preserve spinal cord perfusion when arterial inflow has been compromised. Its role is firmly established in high-risk open TAAA repair, whereas its prophylactic use in endovascular repair remains more selective because the evidence of benefit is less robust and the procedure carries potentially serious complications.

For patients undergoing extensive f/bEVAR, the decision to use prophylactic CSFD should therefore be based on the cumulative neurological risk and integrated with other protective measures, including preservation of collateral circulation, staged repair when feasible, maintenance of adequate systemic perfusion, and intensive postoperative neurological surveillance.

The uncertainty surrounding the optimal use of CSFD, together with the heterogeneity of institutional protocols and the potential discrepancy between prophylactic benefit and procedure-related complications, provides the rationale for continued evaluation of real-world clinical experience. In this context, the present study investigates the experience of a high-volume vascular centre with CSFD during fenestrated and branched endovascular aortic repair, with particular focus on its association with neurological outcomes and drain-related complications.

Aim of the study

Spinal cord ischemia represents one of the major and most feared neurological complications associated with the endovascular treatment of thoracoabdominal and complex abdominal aortic diseases. Its occurrence is related to impaired spinal cord blood supply resulting from the endovascular exclusion of extensive aortic segments and their collateral branches, potentially leading to neurological deficits of varying severity, including permanent paraplegia.

Cerebrospinal fluid drainage represents one of the main strategies for spinal cord protection and may be employed either prophylactically, in patients considered at high risk of spinal cord ischemia based on anatomical, procedural, and clinical characteristics, or therapeutically in the event of neurological deficit during the perioperative period. However, the role of prophylactic CSFD in the endovascular treatment of aortic disease remains controversial, particularly in view of the potential adverse events associated with the procedure.

In light of these considerations, the aim of the present study is to evaluate the incidence of neurological complications in our cohort of patients undergoing endovascular treatment of thoracoabdominal aortic disease, analyzing their relationship with the use of CSFD. Specifically, the study aims to compare the incidence of SCI between patients undergoing prophylactic CSFD and those treated without prophylactic drainage, while also evaluating the use of therapeutic CSFD in patients who develop postoperative neurological deficits. Finally, the analysis aims to identify the main factors associated with the occurrence of SCI and to assess neurological outcomes and complications related to the CSFD procedure.

Materials and Methods

A retrospective, single-centre study was conducted in patients who underwent endovascular treatment of complex abdominal or thoracoabdominal aortic disease between 2019 and 2026. Patients were divided into two groups: prophylactic cerebrospinal fluid (CSF) drainage (PD) and no prophylactic CSF drainage (ND). Prophylactic CSF drainage was chosen in patients deemed at high risk for SCI based on anatomical, clinical, and procedural factors. Our institutional experience included an early period during which CSF drainage was used exclusively for prophylactic purposes in patients considered at high risk for SCI, followed by a second period, during which CSF drainage was reserved exclusively for therapeutic purposes in patients developing neurological symptoms. The primary outcomes were the occurrence of SCI and identification of predictors of SCI in the ND group; secondary outcomes included 30-day mortality, and CSF drainage-related adverse events and long-term survival.

Statistical analysis

The statistical analysis was performed using “SPSS v.25 statistical software” (IBM Corporation, Armonk, NY, USA). Statistical and data reporting followed guideline recommendations. Categorical variables were presented as frequencies and percentages. Variables were presented as mean $\pm$ standard deviation (SD), or as median and interquartile range (IQR). Quantitative variables were compared through One-way ANOVA, Mann Whitney U or Student’s *t*-test and qualitative variables were compared through Pearson χ^2 test. Univariate analysis was used to identify predictors of SCI occurrence in the ND group. Variables showing an association with SCI at a P value <0.20 were considered for inclusion in the binary logistic regression analysis, using a Wald’s backward stepwise model. The strength of the association between variables and outcome was estimated by calculating odds ratios (OR) and 95% Confidence Intervals (95% CI). The Kaplan Meier method was employed to assess follow up outcomes; estimates were compared with the Log Rank test. Statistical significance was considered for P values <0.05 .

Results

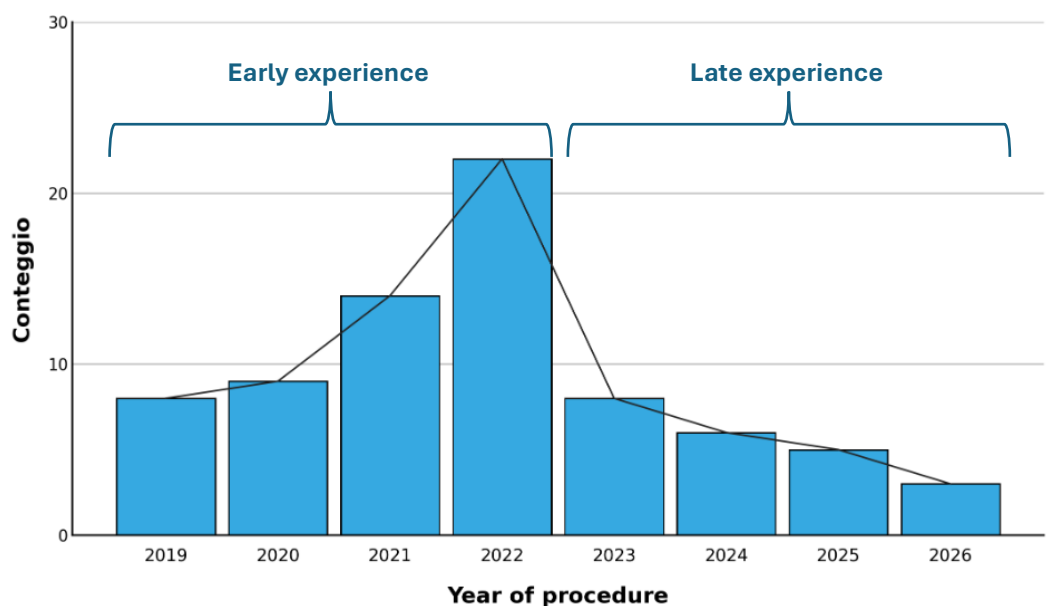
A total of 265 consecutive patients undergoing F/BEVAR for complex abdominal or thoracoabdominal aortic disease were included. 75 patients (28.3%) received prophylactic CSF drainage, whereas in 190 patients (71.7%) CSF drainage was not planned. Characteristics of the two groups are illustrated in Table I.

TABLE I. — *Characteristics of PD group vs ND group*

	Total cohort (N=265)	PD (N=75)	ND (N=190)	P value
Female (N. %)	60 (22.6)	24 (32)	36 (19.8)	0.022
Urgency (N. %)	87 (32.8)	9 (12)	78 (41.1)	<0.001
Crawford I-II-III (N.%)	93 (35.1)	47 (62.7)	46 (24.2)	<0.001
Hypertension (N. %)	217 (81.9)	62 (82.7)	155 (81.6)	0.690
Dyslipidemia (N.%)	137 (51.7)	36 (48)	101 (53.2)	0.500
Diabetes (N.%)	31 (11.7)	7 (9.3)	24 (12.6)	0.468
Ischemic Heart Disease (N. %)	117 (44.2)	29 (38.7)	88 (46.3)	0.288
COPD (N. %)	110 (41.5)	32 (42.7)	78 (9.9)	0.755
CAD (N. %)	36 (13.6)	8 (10.7)	28 (14.7)	0.366
PAD (N. %)	25 (9.4)	6 (8)	19 (10)	0.633
CKD (N. %)	225 (84.9)	59 (78.7)	166 (87.4)	0.075
Previous aortic surgery (N. %)	104 (54.7)	40 (53.3)	64 (33.7)	0.002
Aortic Dissection (N. %)	34 (12.8)	14 (18.7)	20 (10.5)	0.074

N.: number; COPD: chronic obstructive pulmonary disease; CAD: carotid artery disease; PAD: peripheral artery disease; IRC: chronic kidney disease.

FIGURE XI. — *Temporal distribution of patients in the Prophylactic Drainage group*



The overall incidence of SCI was 9.8% (26 patients), of whom 15 cases occurred in the no-drainage (ND) group (7.9%) and 11 in the prophylactic drainage (PD) group (14.7%). SCI

occurred more frequently in the PD group (14.7% vs. 7.9%), although the difference did not reach statistical significance ($p=0.095$).

Among the 15 cases of SCI in the ND group, 8 patients (4.2%) underwent therapeutic CSF drainage: complete neurological recovery was observed in 1 patient (12.5%), clinical improvement with a minor residual deficit in 3 patients (37.5%), and permanent SCI in 4 patients (50%). In the remaining 7 patients, despite the occurrence of SCI, CSFD was not performed, as the clinical conditions did not require or permit its placement. In the PD group, 11 patients (14.7%) experienced substantial neurological spine-related deficits, including 7 patients (63.6%) with minor permanent deficits and 4 (36.4%) with major permanent deficits.

Therapeutic drainage was initiated after a median of 2 days and maintained for a median of 4 days, while prophylactic drainage was maintained for a median of 3 days. SCI onset occurred in the PD group after a median of 1 day from the index procedure. During drainage, antithrombotic management mainly consisted of single antiplatelet therapy and prophylactic low-molecular-weight heparin, with intravenous heparin used in selected cases. At 30 days, 5 patients (6.7%) in the PD group developed mild CSF drainage-related symptoms (headache and local irritation), 2 (2.7%) developed a hematoma managed conservatively, and 2 (2.7%) developed a hematoma requiring decompressive laminectomy. Thirty-day mortality did not differ significantly between the two groups (6.7% PD vs. 10% ND; $p=0.482$).

In univariate analysis, female sex, urgent procedures, the need for additional procedures, perioperative reintervention, Crawford I–III extent, and perioperative hemoglobin <9 g/dL were associated with SCI at a significance threshold of $p < 0.20$ and were included in multivariate analysis (Table II).

TABLE II. —Details on perioperative characteristics in the ND group: univariate analysis.

	Total cohort (N=190)	SCI (N=15)	No SCI (N=175)	P value
Demographics				
Female (N. %)	36 (18.9)	7 (46.7)	29 (16.6)	0.008
Age >80 y.o. (N. %)	72 (37.9)	7 (46.7)	65 (37.1)	0.61
Early experience (N. %)	44 (23.2)	4 (26.7)	40 (22.9)	0.86
Urgency (N. %)	35 (18.4)	5 (33.3)	30 (17.1)	0.17
Crawford I-II-III (N. %)	46 (24.2)	7 (46.7)	39 (22.3)	0.16
Comorbidities				
Hypertension (N. %)	155 (81.6)	12 (80)	143 (81.7)	0.38
Dyslipidemia (N. %)	101 (53.2)	6 (40)	95 (54.3)	0.17
Diabetes (N. %)	24 (12.6)	2 (13.3)	22 (12.6)	0.97
Ischemic Heart Disease (N. %)	88 (46.3)	6 (40)	82 (46.9)	0.42
COPD (N. %)	78 (41.1)	7 (46.7)	71 (40.6)	0.86
CAD (N. %)	28 (14.7)	3 (20)	25 (14.3)	0.54
PAD (N. %)	19 (10)	1 (6.7)	18 (10.3)	0.59
CKD (N. %)	166 (87.4)	14 (93.3)	152 (86.9)	0.99
Risk Factors				
Previous aortic surgery (N. %)	64 (33.7)	5 (33.3)	59 (33.7)	0.95
Aortic Dissection (N. %)	20 (10.5)	3 (20)	17 (9.7)	0.26
Aortic coverage >400mm (N. %)	19 (10)	5 (33.3)	14 (8)	0.004
ICAs coverage >10 pairs (N. %)	44 (23.2)	6 (40)	38 (21.7)	0.31
No IIA patency (N. %)	3 (1.6)	1 (6.7)	2 (1.1)	0.12

Staged surgery (N. %)	39 (20.5)	4 (26.7)	35 (20)	0.67
Additional procedures (N. %)	34 (17.9)	8 (53.3)	26 (14.9)	<0.001
Perioperative reintervention (N. %)	15 (7.9)	3 (20)	12 (6.9)	0.09
Perioperative lab values				
Perioperative Hb <9 g/dl (N. %)	75 (39.5)	13 (86.7)	62 (35.4)	<0.001

N.: number; SCI: spinal cord ischemia; y.o.: years old; Early experience: f/bEVAR between year 2019 and year 2022; COPD: chronic obstructive pulmonary disease; CAD: carotid artery disease; PAD: peripheral artery disease; IRC: chronic kidney disease; ICAs: intercostal arteries; IIA: internal iliac arteries; Hb: hemoglobin.

Female sex (OR 3.89, 95% CI 1.08–13.97; $p=0.038$) and additional procedures (OR 5.57, 95% CI 1.55–20.06; $p=0.009$) were independently associated with SCI (Table III), while perioperative hemoglobin <9 g/dL showed a trend toward an independent association with SCI (OR 3.90, 95% CI 0.96–15.89; $p=0.058$). Intraoperative additional procedures mainly reflected the increased procedural complexity of the treatment, including both endovascular and open adjunctive interventions. These were performed to facilitate device delivery, optimize vascular access, maintain or restore vessel patency, or achieve adequate aneurysm exclusion, with the most commonly performed adjunctive procedures consisting of embolization (22 cases), stenting (14), angioplasty (12), graft interposition (7), bypass (4), and shockwave therapy (3). On the other hand, perioperative reinterventions were mainly required for the management of conditions arising after the main procedure, including vascular access-related complications, bleeding and retroperitoneal hematomas, ischemic events, endoleaks, and neurological complications such as subdural hematomas.

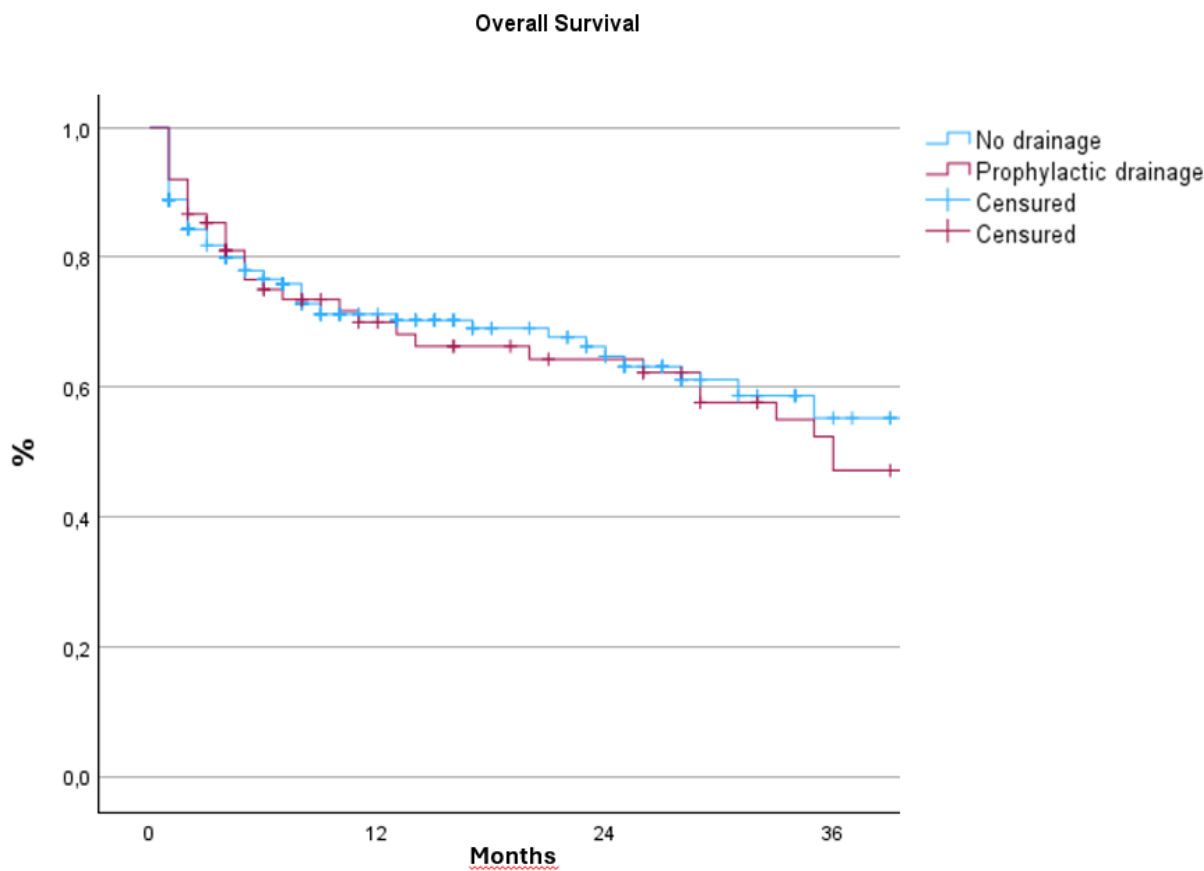
TABLE III. —Logistic regression for predictors of SCI in the ND group

Covariates	Predictors of SCI			
	Univariate	Multivariate		
	P value	OR	95%CI	P value
No IIA patency	0.12			
Perioperative Reintervention	0.09			
Crawford I-II-III	0.16			
Female	0.008	3.891	1.084-13.968	0.038
Urgency	0.17	3.464	0.856-14.026	0.082
Additional procedures	<0.001	5.570	1.547-20.056	0.009
Perioperative Hb <9 g/dl	<0.001	3.897	0.955-15.893	0.058

SCI: spinal cord ischemia; OR: odds ratio; 95%CI: 95% confidence interval; IIA: internal iliac artery; Hb: hemoglobin.

The mean follow-up was 15.7 ± 16.1 months. Long-term overall survival did not differ significantly between patients receiving prophylactic CSF drainage and those without prophylactic drainage (survival estimates at 36 months: no drainage group 55.2.2% vs prophylactic drainage group 52.4%, log-rank $p=0.982$, numbers at risk >10, standard error >10%, Figure XII).

FIGURE XII. —Kaplan-Meier curves of overall survival.



Discussion

Spinal cord ischemia represents one of the most feared complications of fenestrated and branched endovascular aortic repair (F/BEVAR) for thoracoabdominal aortic disease. Its pathogenesis is multifactorial and results from the interplay between the extent of aortic coverage, loss of segmental arteries, integrity of the collateral network, pelvic and subclavian perfusion, and perioperative hemodynamic conditions. In this context, CSFD represents one of the most extensively studied spinal cord protection strategies, owing to its ability to reduce cerebrospinal fluid pressure and, consequently, increase spinal cord perfusion pressure. However, its role in complex endovascular repair remains less clearly defined than in open repair of thoracoabdominal aortic aneurysms. The present study analyzed a single-center cohort of 265 consecutive patients who underwent F/BEVAR between 2019 and 2026, of whom 75 (28.3%) received prophylactic CSFD and 190 (71.7%) did not undergo prophylactic drainage. Importantly, the decision to perform prophylactic drainage was not randomized but was based on the presence of anatomical, clinical, and procedural characteristics considered to be associated with an increased risk of SCI. Furthermore, the cohort comprised two distinct phases of the institutional experience: an initial period characterized by the use of prophylactic CSFD in patients considered at high risk, followed by a subsequent period in which CSFD was predominantly reserved for therapeutic use in the event of neurological deficits. This feature of the study is essential for the correct interpretation of the findings. In particular, the direct comparison between patients with and without prophylactic CSFD cannot be considered equivalent to a comparison between two populations with an identical baseline risk of SCI. Rather, patients undergoing prophylactic drainage exhibited features indicative of greater anatomical and procedural complexity: Crawford I–III extent was present in 62.7% of patients in the prophylactic drainage (PD) group compared with 24.2% in the no-drainage (ND) group, while previous aortic surgery was reported in 53.3% and 33.7% of patients, respectively. Urgent procedures were also less frequent in the PD group, consistently with the greater feasibility of planning prophylactic drainage in patients undergoing elective high-risk procedures.

Prophylactic drainage and incidence of spinal cord ischemia

The overall incidence of SCI in the present cohort was 9.8%, corresponding to 26 events among 265 patients. The incidence was 14.7% in the prophylactic drainage group and 7.9%

in the no-drainage group, with the difference not reaching statistical significance ($p=0.095$). At first glance, the higher incidence of SCI in the PD group may appear counterintuitive given the physiological rationale underlying CSFD. However, this finding must be interpreted in light of the patient selection strategy. Prophylactic drainage was in fact reserved for patients considered to be at greater neurological risk. This resulted in an important confounding by indication, whereby patients receiving the intervention were precisely those with a higher baseline risk.

This represents one of the key interpretative aspects of the present study. The findings therefore do not support the conclusion that prophylactic CSFD increased the incidence of SCI. Conversely, the absence of a statistically significant difference does not allow the conclusion that prophylactic CSFD is ineffective. The observational study design and the unequal distribution of risk factors prevent a causal attribution of the observed SCI incidence to CSFD. Recent literature is consistent with this interpretation. A systematic review and meta-analysis including 4,793 patients undergoing TEVAR did not demonstrate a statistically significant reduction in SCI among patients receiving prophylactic CSFD compared with those undergoing repair without drainage. The authors also highlighted the presence of a, albeit limited, risk of major drainage-related complications. Even more directly relevant to the present cohort, a 2024 meta-analysis specifically addressing CSFD-related complications in patients undergoing F/B-EVAR demonstrated substantial heterogeneity across the available studies and a non-negligible overall incidence of drainage-related complications, underscoring the uncertainty surrounding the net benefit of prophylactic CSFD in the F/B-EVAR setting. These findings are part of a broader shift in the approach to spinal cord protection. A multicenter analysis from the United States Aortic Research Consortium, including 2,585 patients undergoing B/FEVAR between 2011 and 2024, documented a progressive reduction in the use of prophylactic CSFD over time without a concomitant deterioration in neurological outcomes. In the same study, SCI resolution did not differ significantly among patients receiving prophylactic drainage, therapeutic drainage, or no drainage. This finding is particularly relevant when considered alongside the experience reported in the present cohort, in which the transition from a predominantly prophylactic strategy toward a more selective and therapeutic approach is consistent with the trend observed in other contemporary series.

However, an overly simplistic interpretation should be avoided. The progressive decline in the use of prophylactic CSFD does not necessarily imply that CSFD has lost its clinical relevance. Rather, its role appears to be progressively evolving from that of a routine component of spinal

cord protection to that of a selective intervention integrated within a multimodal protocol and readily available when signs of neurological compromise develop.

The role of therapeutic drainage

A second finding of particular interest in the present experience concerns the therapeutic use of CSFD following the onset of SCI.

In the ND group, 15 patients developed spinal cord ischemia. Of these, 8 underwent therapeutic CSFD: one patient achieved complete neurological recovery, three experienced clinical improvement with a mild residual deficit, whereas four developed permanent SCI. Overall, therefore, 4 of the 8 patients undergoing therapeutic drainage experienced either complete recovery or clinically meaningful improvement. Therapeutic drainage was initiated after a median of 2 days and maintained for a median of 4 days. These findings should be interpreted with caution, particularly because no control group of patients with SCI managed without CSFD was available. It is therefore not possible to determine whether the neurological improvement observed was directly attributable to drainage or resulted from a combination of therapeutic interventions, the natural course of SCI, or other perioperative factors.

Despite this limitation, the finding remains clinically relevant. The rationale for rescue CSFD differs from that of prophylactic drainage. In a patient who has already developed a neurological deficit, the objective is not to prevent a hypothetical event but to intervene in an ongoing pathophysiological process. Reduction of cerebrospinal fluid pressure may increase spinal cord perfusion pressure and improve the perfusion gradient available to the spinal cord microcirculation, particularly when arterial inflow has been compromised by extensive aortic coverage.

Contemporary strategies for the management of SCI following F/BEVAR generally include a combination of CSFD, augmentation of arterial blood pressure, and optimization of hemoglobin levels, together with extremely rapid recognition of neurological deterioration.

A particularly relevant aspect of the present cohort is the timing of neurological events. In the PD group, SCI developed at a median of 1 day after the index procedure, whereas therapeutic drainage in the ND group was initiated at a median of 2 days. The occurrence of neurological deficits during the postoperative period therefore underscores the importance of structured neurological monitoring and the immediate availability of a rescue protocol. Timely

intervention may be particularly important in cases of potentially reversible SCI. Once prolonged ischemic injury has become established, the potential for neurological recovery may progressively decrease. Consequently, the effectiveness of a selective strategy based on rescue drainage depends not only on the technical availability of CSFD, but also on the ability of the treating center to recognize neurological deterioration at an early stage and intervene promptly.

Drainage-related complications

The potential benefit of CSFD must necessarily be balanced against the risks associated with the procedure. In the present cohort, among the 75 patients who underwent prophylactic drainage, 5 (6.7%) developed mild drainage-related symptoms, mainly headache and local irritation. Two patients (2.7%) developed a hematoma that was managed conservatively, whereas another two patients (2.7%) developed a hematoma requiring decompressive laminectomy. This finding is particularly relevant when considering whether CSFD should be routinely performed even in patients who may never develop SCI. Prophylactic drainage entails early procedural exposure: patients are subjected to the risks associated with catheter placement before the need to increase spinal cord perfusion pressure through reduction of CSF pressure has been established.

A 2024 meta-analysis specifically evaluating patients undergoing F/B-EVAR estimated CSFD-related mortality at 1.4% and overall morbidity at 25.6%, with major complications occurring in 6.1% of cases. The most severe complications reported included intracranial hemorrhage, spinal hematoma, subarachnoid hemorrhage, and drainage-related neurological deficits.

Although the findings of the present study are not directly comparable with the pooled estimates reported in the literature because of differences in event definitions and study populations, they support the fundamental principle that CSFD is not a risk-free procedure. This consideration helps explain why contemporary approaches to spinal cord protection increasingly favor individualized risk assessment rather than the uniform application of a single strategy.

Predictors of spinal cord ischemia

One of the most interesting aspects of the present study is the analysis of predictors of SCI in the SD group, which allows factors associated with the occurrence of SCI to be investigated in the absence of prophylactic intervention.

On univariate analysis, female sex, urgent procedures, Crawford I–III extent, the need for additional procedures, perioperative reintervention, and hemoglobin levels below 9 g/dL were associated with SCI according to the predefined criterion of $p < 0.20$. On multivariable analysis, female sex and the need for additional procedures were independently associated with the occurrence of SCI, whereas perioperative hemoglobin < 9 g/dL showed a trend toward an independent association.

Female sex

Female sex was associated with an odds ratio of 3.89 (95% CI, 1.08–13.97; $p = 0.038$). This finding is particularly interesting because it is not isolated from the available literature. In a previous analysis of 877 patients undergoing F/BEVAR, female sex was associated with SCI in the subgroup treated for complex abdominal aortic aneurysms, with an OR of 3.87.

More recent data also suggest differences in outcomes between men and women undergoing F/BEVAR. An analysis published in 2026 including 423 patients reported a higher frequency of major adverse events among women, including a higher incidence of SCI; however, the authors also highlighted differences in disease distribution and anatomical complexity between the sexes. The biological significance of this association cannot, however, be established on the basis of the present study. Female sex may represent a true independent risk factor, but it may also reflect differences in anatomy, vessel diameters, disease characteristics, or other incompletely measured determinants. Furthermore, the wide confidence interval observed in the present cohort reflects the relatively small number of events and warrants caution when interpreting the magnitude of the association.

Additional procedures

The second independent predictor identified was the performance of additional procedures, which was associated with an OR of 5.57 (95% CI, 1.55–20.06; $p = 0.009$). In the SD group, additional procedures were performed in 53.3% of patients with SCI compared with 14.9% of patients without SCI. This finding is pathophysiologically plausible. Additional procedures do not necessarily represent a direct cause of SCI but may instead be regarded as a marker of procedural complexity. In the present cohort, these procedures included embolization,

stenting, angioplasty, graft or prosthetic interposition, bypass procedures, and shockwave therapy.

More extensive endovascular manipulation may reflect more complex anatomy, longer procedures, a greater need for interventions involving access vessels, and greater involvement of collateral vascular territories. Furthermore, some additional procedures may directly alter pelvic or segmental perfusion. This finding is also supported by the literature. A systematic review of cases of SCI following EVAR identified difficult anatomy, longer procedural duration, additional procedures, and extensive intravascular manipulation among the recurrent factors associated with SCI.

Therefore, in the present study, the association between additional procedures and SCI may be interpreted not so much as an effect of any specific procedure, but rather as an indicator of a greater procedural burden. This interpretation is consistent with the multifactorial nature of SCI.

Perioperative hemoglobin

The finding regarding hemoglobin is particularly noteworthy. A perioperative hemoglobin level <9 g/dL was present in 86.7% of patients with SCI compared with 35.4% of patients without SCI and was strongly associated with the event on univariate analysis ($p<0.001$). On multivariable analysis, the association did not reach conventional statistical significance, but the OR remained 3.90 (95% CI, 0.96–15.89; $p=0.058$).

This finding is particularly consistent with the pathophysiology of SCI. When spinal cord perfusion is already compromised by the loss of segmental arteries, a reduction in hemoglobin concentration may further decrease tissue oxygen delivery. Anemia may therefore represent an additional vulnerability factor in a vascular system that already has limited perfusion reserve.

Recent studies have also demonstrated an association between perioperative hematological parameters and the severity of SCI following F/BEVAR. In a cohort of patients with SCI, an intraoperative hemoglobin level above 8.5 g/dL was associated with a lower likelihood of severe SCI, whereas the need for blood transfusion was significantly more frequent among patients who developed SCI. In the present study, however, the p value of 0.058 does not allow Hb <9 g/dL to be defined as a statistically significant independent predictor. It is therefore more appropriate to describe this finding as a clinically relevant signal of association that warrants further investigation in larger cohorts.

Urgency of the Procedure

Urgent procedures also showed a trend toward an independent association with the occurrence of SCI, with an odds ratio of 3.46 (95% CI 0.86–14.03; $p=0.082$). In the group without prophylactic drainage, urgent procedures were more frequent among patients who developed spinal cord ischemia than among those who did not (33.3% vs. 17.1%). Although this association did not reach conventional statistical significance, the direction and magnitude of the observed effect suggest that procedural urgency may represent a relevant factor in neurological risk stratification.

From a clinical perspective, urgent procedures may involve several factors potentially detrimental to spinal cord protection. Compared with elective interventions, the shorter time available for preoperative planning may limit the opportunity to optimize hemodynamic status, correct potential abnormalities in hemoglobin levels, and implement individualized protective strategies. Furthermore, the clinical circumstances necessitating urgent treatment may be associated with greater perioperative instability, potentially compromising spinal cord perfusion pressure. These mechanisms are physiologically plausible; however, they cannot be directly demonstrated by the data from the present cohort.

The interpretation of this finding therefore requires caution. The relatively wide confidence interval, which includes unity, reflects the uncertainty surrounding the estimate and may partly be attributable to the limited number of neurological events. Accordingly, urgency cannot be considered a statistically significant independent predictor of SCI in the present study. Nevertheless, the observed signal warrants attention and could be further investigated in larger cohorts. Overall, these findings highlight the importance of considering not only the anatomical extent and technical complexity of aortic repair, but also the clinical context in which the procedure is performed.

The role of multimodal spinal cord protection

Overall, the findings of the present study support a view of SCI as a multifactorial event in which CSFD represents only one component of the overall spinal cord protection strategy. Reduction of CSF pressure alone cannot fully compensate for a critical loss of arterial inflow to the spinal cord. The effectiveness of CSFD depends on the simultaneous maintenance of adequate arterial pressure, sufficient oxygen-carrying capacity, and an intact collateral network. This interpretation is consistent with contemporary SCI prevention protocols, in

which CSFD is integrated with hemodynamic targets, hemoglobin optimization, preservation of pelvic and subclavian perfusion, staged procedures, and neurological monitoring. In a recent study, a multimodal protocol incorporating selective CSFD, a MAP target >90 mmHg, and hemoglobin targets of ≥ 10 g/dL intraoperatively and ≥ 9 g/dL postoperatively was associated with a reduction in SCI after B/FEVAR.

This concept is particularly important because it suggests that successful spinal cord protection may not depend on the identification of a single “protective” intervention, but rather on the ability to preserve spinal cord perfusion reserve as much as possible throughout all phases of treatment. From this perspective, selection of patients for prophylactic CSFD should take into account the cumulative neurological risk, including the extent of aortic coverage, thoracoabdominal involvement, previous aortic procedures, the status of pelvic and subclavian circulation, procedural characteristics, and perioperative physiological conditions.

Mortality and long-term survival

Thirty-day mortality did not differ significantly between the groups, occurring in 6.7% of patients in the PD group and 10% in the SD group ($p=0.482$). Similarly, overall long-term survival did not differ significantly between the groups: at 36 months, estimated survival was 52.4% in the PD group and 55.2% in the SD group, with a log-rank test $p=0.982$. The mean follow-up duration for the entire cohort was 15.7 ± 16.1 months.

The absence of differences in survival should be interpreted in light of the observational nature of the study and the different composition of the two groups. In particular, the greater anatomical complexity and higher neurological risk among patients undergoing prophylactic drainage make it difficult to interpret this comparison as an assessment of the independent effect of CSFD on survival.

It is also important to distinguish the effect of CSFD from the effects of SCI itself. Previous studies have shown that the occurrence of SCI after F/BEVAR is associated with worse short- and long-term outcomes, including higher mortality among patients treated for TAAA.

Therefore, the primary clinical objective of CSFD remains plausibly the prevention or treatment of neurological injury rather than a direct effect on survival. The present cohort provides no evidence of an association between prophylactic drainage and survival; however,

the study design is not sufficiently powered or structured to exclude indirect effects mediated by the prevention or severity of SCI.

Clinical implications

In light of the findings of the present study and the available evidence, CSFD in F/BEVAR should be considered within a personalized spinal cord protection strategy, rather than as an inherently routine procedure.

A selective approach may be particularly appropriate in patients undergoing high-risk procedures, in whom the probability of SCI is determined by the combination of multiple risk factors. Conversely, systematic exposure of low-risk patients to CSFD may result in iatrogenic complications that are not necessarily offset by a neurological benefit.

At the same time, a selective strategy requires a highly structured clinical pathway. The decision not to place a prophylactic drain does not imply that spinal cord protection is being abandoned. Careful neurological monitoring, maintenance of adequate blood pressure, correction of anemia, preservation of collateral vascular territories whenever feasible, and immediate availability of CSFD in the event of neurological deterioration are essential components of this approach.

In other words, the clinical question may no longer simply be “to drain or not to drain?”, but rather “which combination of strategies provides the most appropriate balance between neurological protection and procedural risk for the individual patient?”.

This conceptual shift is consistent with the evolution of contemporary practice, in which the use of prophylactic CSFD has progressively declined alongside the development of multimodal SCI prevention protocols.

Study Limitations

The present study has several limitations that should be considered when interpreting its findings.

First, this was a retrospective, single-center study. Its observational nature precludes establishing a causal relationship between CSFD use and the occurrence of SCI and allows for the presence of residual confounding factors that could not be fully controlled. A

particularly important limitation is represented by confounding by indication. Prophylactic CSFD was used specifically in patients considered to be at higher risk; consequently, the PD group had a higher prevalence of Crawford I–III extent and previous aortic procedures, making the crude comparison between the two groups inherently influenced by their different baseline risk profiles.

Second, the absolute number of neurological events was relatively limited: SCI occurred in 26 patients overall and in only 15 patients in the SD group. This limits the statistical power of the predictor analysis and makes it particularly important to interpret the odds ratios and their corresponding confidence intervals with caution. Moreover, the transition over time from a predominantly prophylactic strategy toward a more therapeutic approach represents an additional potential confounding factor. Operator experience, advances in endovascular technologies, improvements in patient selection, and the progressive adoption of multimodal strategies may have influenced outcomes independently of CSFD use.

Another limitation is the heterogeneity of the treated population. The cohort included different forms of complex aortic disease and different anatomical extents, which may be associated with substantially different levels of neurological risk. A FEVAR procedure limited to the abdominal aorta does not, in fact, carry the same risk of SCI as an extensive BEVAR procedure performed for a type I–III TAAA.

Finally, the analysis does not allow a sufficiently detailed assessment of the potential effects of different protocols for the management of blood pressure, cerebrospinal fluid pressure, hemoglobin levels, and other components of spinal cord protection. SCI is the final result of numerous anatomical and physiological determinants, and the effect of a single component may be difficult to disentangle from that of the overall perioperative protocol.

These limitations highlight the need for prospective, multicenter studies, ideally incorporating risk-stratification strategies and standardized protocols, to more precisely determine which patients may benefit from prophylactic CSFD and which may be safely managed using a selective approach.

Future Perspectives

Future perspectives in spinal cord protection therefore appear to be moving toward an increasingly individualized and physiologically guided model.

Preoperative risk stratification should integrate the extent of aortic coverage, the presence of previous aortic procedures, patency of the hypogastric and subclavian arteries, the anatomy of the collateral network, and the characteristics of the planned procedure. During and after the intervention, maintenance of adequate perfusion pressure, optimization of hemoglobin levels, and neurological monitoring should be considered as components of a unified protection protocol.

Within this model, CSFD may assume different roles: prophylactic in patients at particularly high neurological risk, therapeutic in patients who develop SCI, and potentially as part of strategies adapted to the individual patient's physiological status.

The objective should therefore not simply be to increase the number of patients undergoing CSFD, but rather to better identify which patients, at what time, and with which combination of strategies are most likely to derive benefit while minimizing procedure-related complications.

Concluding Remarks

Overall, the findings of the present study did not demonstrate an association between prophylactic CSFD and a statistically significant reduction in the incidence of SCI after F/BEVAR. The observed incidence was, in fact, higher in the PD group than in the SD group; however, this difference must be interpreted in the context of the higher baseline risk of patients selected for prophylactic drainage and cannot be used to attribute a deleterious effect to CSFD.

Conversely, the experience with therapeutic drainage suggests a potential role for CSFD in the treatment of patients who develop neurological deficits, although the observational nature of the study does not allow a causal relationship between drainage and neurological recovery to be established.

The study also identified female sex and the need for additional procedures as factors independently associated with SCI in the group without prophylactic drainage, whereas a perioperative hemoglobin level below 9 g/dL showed a trend toward an independent association. These findings reinforce the concept that SCI should be regarded as the final expression of a complex interaction between anatomical characteristics, procedural factors, and perioperative physiological conditions.

In this context, CSFD should not be considered an isolated strategy, but rather as one component of a broader spinal cord protection protocol. Preservation of collateral circulation, consideration of staged procedures when appropriate, maintenance of adequate systemic perfusion, optimization of hemoglobin levels, and early neurological monitoring represent complementary and potentially synergistic components of this approach.

The main implication of the present experience is therefore that spinal cord protection in F/BEVAR should not be based on the uniform application of a single preventive measure, but rather on a multimodal and individualized strategy, in which the balance between the risk of SCI and the risks associated with CSFD is assessed for each patient. Within this paradigm, timely availability of therapeutic CSFD remains particularly important, even when prophylactic drainage is not employed.

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