Neuromonitoring during Endovascular Thoracoabdominal Aortic Aneurysm Repair: A Systematic Review

Myat Soe Thet,[1] Mario D'Oria,[2] Davorin Sef,[3] Tomislav Klokocovnik,[4] Aung Ye Oo,[5] and Sandro Lepidi,[2] Southampton and London, United Kingdom; Trieste, TS, Italy; and Celje, Slovenia

Background: Spinal cord ischemia (SCI) is a potentially devastating complication of thoracic endovascular aortic repair (TEVAR) and fenestrated-branched endovascular aortic repair (F-BEVAR). The aim of this systematic review was to evaluate the efficacy of neuromonitoring modalities to mitigate the risk of SCI during TEVAR and F-BEVAR procedures.

Methods: Following the PRISMA guidelines, we conducted a detailed literature search of databases including PubMed, MEDLINE via Ovid, Embase, Scopus, and Cochrane CENTRAL, from 1998 to the present. Inclusion criteria were original research articles examining neuromonitoring during TEVAR and F-BEVAR. The primary outcome was the incidence of SCI, while the secondary outcome included early mortality. The quality of studies was assessed using the Newcastle–Ottawa Scale.

Results: From 1,450 identified articles, 11 met inclusion criteria, encompassing data from 1,069 patients. Neuromonitoring modalities included motor-evoked potentials (MEPs), somatosensory evoked potentials (SSEPs), and near-infrared spectroscopy. The combination of MEPs and SSEPs was most commonly used, with 93% sensitivity and 96% specificity for detecting SCI risks. SCI incidence ranged from 3.8 to 17.3%, with permanent deficits occurring in 2.7–5.8% of cases. In-hospital mortality ranged from 0.4 to 8%. Risk factors for SCI were identified, including operation duration and extent of aortic coverage.

Conclusions: Neuromonitoring with MEPs and SSEPs appears to be effective in detecting perioperative SCI risk during TEVAR and F-BEVAR. However, discrepancies between neuromonitoring changes and actual SCI outcomes suggest the need for cautious interpretation. While the incidence of SCI remains variable, identified risk factors may guide clinical decisions, particularly in high-risk procedures. Future research should focus on prospective studies and randomized controlled trials to validate these findings and improve SCI prevention strategies in TEVAR and F-BEVAR.

Myat Soe Thet and Mario D'Oria contributed equally to this work and should co-share first authorship.

[1]Department of Surgery and Cancer, Imperial College London, UK.

[2]Division of Vascular and Endovascular Surgery, Department of Clinical Surgical and Health Sciences, University of Trieste, Italy.

[3]Wessex Cardiothoracic Centre, University Hospital Southampton, Southampton, UK.

[4]Department of Cardiac Surgery, General Hospital Celje, Celje, Slovenia.

[5]Department of Cardiothoracic Surgery, St Bartholomew's Hospital, London, UK.

Correspondence to: Mario D'Oria, MD, Division of Vascular and Endovascular Surgery, Department of Medical Surgical and Health Sciences, University of Trieste, Strada di Fiume 447, 34149, Trieste, Trieste, Italy; E-mail: mario.doria88@outlook.com

Ann Vasc Surg 2024; 109: 206–215
https://doi.org/10.1016/j.avsg.2024.06.012

Manuscript received: April 23, 2024; manuscript accepted: June 10, 2024; published online: 14 July 2024

INTRODUCTION

Spinal cord ischemia (SCI) can be a devastating complication following thoracic endovascular aortic repair (TEVAR) and fenestrated-branched endovascular aortic repair (F-BEVAR) despite recent improvements in endovascular technology.[1] Neuromonitoring-guided repair of thoracic (TAA) and thoracoabdominal aneurysms (TAAA) can reduce the incidence of SCI and consists of different techniques including motor-evoked potentials (MEPs), somatosensory evoked potentials (SSEPs) and near-infrared spectroscopy (NIRS).[2] Perinterventional neuromonitoring is considered depending on the type and extent of TAA and TAAA, as well as based on the presence of additional risk factors for SCI. Anatomical factors such as a shaggy aorta and complexity of the endovascular procedure, due to technical reasons such as the performance of long aortic coverage with prolonged operative times are all considered substantial risk factors for developing SCI.[3,4]

Prevention of SCI requires appropriate risk assessment before the intervention, as well as employing a combination of various protective strategies peri- and postinterventional to ensure adequate perfusion of the spinal cord. However, there is still a lack of evidence demonstrating the benefit of additional spinal cord neuromonitoring among patients undergoing TEVAR and F-BEVAR as compared to the use of cerebrospinal fluid drainage.[5] Therefore, our aim was to conduct a systematic review that will analyze all available evidence on the effects of different neuromonitoring methods during TEVAR and F-BEVAR procedures.

MATERIALS AND METHODS

This systematic review follows standards set out in Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA).[6] The study protocol was registered on the International Prospective Register of Systematic Reviews with registration number CRD42023457508.

Literature Search

A comprehensive literature search was performed by a medical librarian across the following databases: PubMed, MEDLINE via Ovid, Embase, Scopus, and Cochrane CENTRAL. The literature search was restricted to publications from the year 1998 to reflect the current contemporary data. A combination of Medical Subject Headings terms and keywords related to the neuromonitoring methods in patients undergoing endovascular aortic repair were used in conjunction with Boolean operators to capture the maximum number of relevant studies. The specific search keywords include ''aneurysm, aortic,'' ''thoracic,'' ''thoracoabdominal,'' ''neuromonitoring,'' ''electrophysiology,'' ''spectroscopy,'' ''NIRS,'' ''evoked potential,'' ''cerebrospinal fluid drainage,'' ''spinal cord ischemia,'' ''paraplegia,'' ''TEVAR,'' ''FEVAR,'' ''BEVAR,'' and ''Endovascular.''

Study Selection

Following the initial literature search, duplicate studies were identified and removed. Two independent reviewers screened titles and abstracts against predefined inclusion and exclusion criteria. Studies were included if they were original articles focused on neuromonitoring methods in patients undergoing endovascular aortic intervention. Studies were excluded if they were not in English and if they were conference abstracts, review articles, letters, or editorials without original data. Case reports, case series, (less than 20 patients) and studies with hybrid procedures were also excluded. Any discrepancies between reviewers were resolved through consensus amongst the 3 authors.

Data Extraction and Outcome Measures

For each included study, data were extracted by 2 independent reviewers using a standardized extraction form. The extracted data included study characteristics, patient demographics, type of neuromonitoring methods, and outcome measures. The primary outcome was the incidence of SCI, including both transient and permanent paraplegia. The secondary outcomes included early postprocedural mortality and stroke. A qualitative synthesis was used to summarize and analyze the results from all included studies.

Quality Assessment

The quality of the included studies was independently assessed by 2 reviewers using the Newcastle−Ottawa Scale for observational studies. The studies were assessed in 3 categories consisting of 8 items: representation, selection, ascertainment, demonstration, comparability, assessment of outcome, length of follow-up, and adequacy of follow-up. Studies were categorized as high (7−9 points), moderate (4−6 points), or low quality (0−3 points) based on their scores.

RESULTS

Out of a total of 1,450 articles identified in the initial search, 756 were excluded as duplicates, and 597 were removed based on title and abstract screening. Consequently, 97 full-text articles were assessed for eligibility. Of these, 86 were excluded for various reasons, as described in the PRISMA flow chart (Fig. 1). Thus, 11 studies met the inclusion criteria and were included in this systematic review. These studies encompassed a total of 1,069 patients. A summary of the characteristics of the included studies is presented in Table I. Further details of patient demographics, indications and types of endovascular interventions are summarized in Table II.

Neuromonitoring

The primary modalities utilized for neuromonitoring during endovascular aortic repair include MEP, SSEP and NIRS. Six studies investigated both MEPs and SSEPs,[7−12] whereas 2 studies focused on the selective application of SSEPs.[13,14] One study examined MEPs,[15] and another study explored NIRS only.[16] There is one study that encompassed an examination of all the modalities: MEPs, SSEPs, and NIRS.[17] The sensitivity and specificity of combined MEP and SSEP for detecting neurological deficits in both open and endovascular interventions are 93% and 96%, respectively.[11] The neuromonitoring modalities reported a false positive rate of 1.8% and a striking false negative rate of 0%.[11] It was noted that MEP tends to be more sensitive than SSEP because SSEP displayed delayed alterations in amplitude, latency, and recovery following changes observed in MEP.[12]

Between 55% and 63% of patients demonstrated a decrease in both MEP and SSEP readings, with 75% of these decreases associated with the use of large bore sheaths.[7,10] Bilateral MEP alterations were typically observed 45−77 min subsequent to the introduction of a vascular sheath.[7,10] Banga et al. demonstrated that the introduction of large sheaths can lead to drops in transcutaneous oxygen saturation and MEP changes in some of the patients, which recovered with subsequent withdrawal.[7] Tenorio et al observed that changes in MEP manifested in 2 distinct patterns: simultaneous changes or a progressive proximal-to-distal change with a delay averaging 36 min. They observed that the delay was extended a further 23 min with the use of iliofemoral conduits.[10] Amplitudes typically improved and returned to baseline within 5 min once blood flow to the lower limb was restored. Notably, most of the patients displaying these MEP changes did not progress to develop SCI.[7]

Changes in SSEP were commonly noticed around 47−62 min postvascular sheath introduction.[7,10] The most frequently observed pattern of change in SSEP was an initial signal delay followed by a decrease in amplitude. It is of clinical significance to note that almost half of the patients who eventually developed SCI initially showed SSEP loss poststent deployment.[13] Transient unilateral loss of SSEP due to vascular insufficiency could result in 40% of patients, which was completely resolved upon restoration of the lower limb blood flow.[13]

When neuromonitoring signals were compromised, specific protective maneuvers could be applied to improve them, which include elevating the mean arterial pressure (MAP), reducing the cerebrospinal fluid (CSF) pressure to approximately 10 mm Hg, retracting large bore sheaths where possible, and lowering the central venous pressure (CVP).[7−10,12,13] MEP and SSEP returned to baseline in 65% of patients with these interventions.[10]

A significant drop in NIRS readings could occur with the complete coverage of the left subclavian artery, even in the presence of patent vertebral arteries, a basilar artery, and an intact circle of Willis.[14] Recovery of NIRS, in this case, could be achieved with a carotid-subclavian bypass. MEP, SSEP, and NIRS have shown utility in routinely guiding temporary aneurysm sac perfusion. A significant drop or disappearance of MEP, SSEP or NIRS poststent graft application requires a specific approach to terminate the procedure and scheduling a second-stage procedure between 2 weeks and 4 months after the index operation to allow recruitment of collateral blood supply to the spinal cord.[8,10,15,16]

Spinal Cord Ischemia (SCI)

Overall SCI rates ranged from 3.8 to 17.3%.[7,9,10,13−17] Permanent paraplegia or paraparesis resulted in 2.7%−5.8%,[7,13,15,16] whereas temporary neurologic deficit resulted in 2−7.2%.[7,13,15] Delayed SCI usually occurred on postoperative day 3 on average.[10] SCI typically occurred in 1% of patients with normal neuromonitoring and about 10% of patients with loss of monitoring signal.[10] Partial or complete resolution of SCI could be achieved with continued or re-institution of CSF drainage, intravenous hydration, increasing arterial pressure and cessation of antihypertensives.[7,10,13,14,16] Maier et al. showed that SCI incidence was much higher (4.7% vs 0.8%) in patients without CSF drainage.[8]

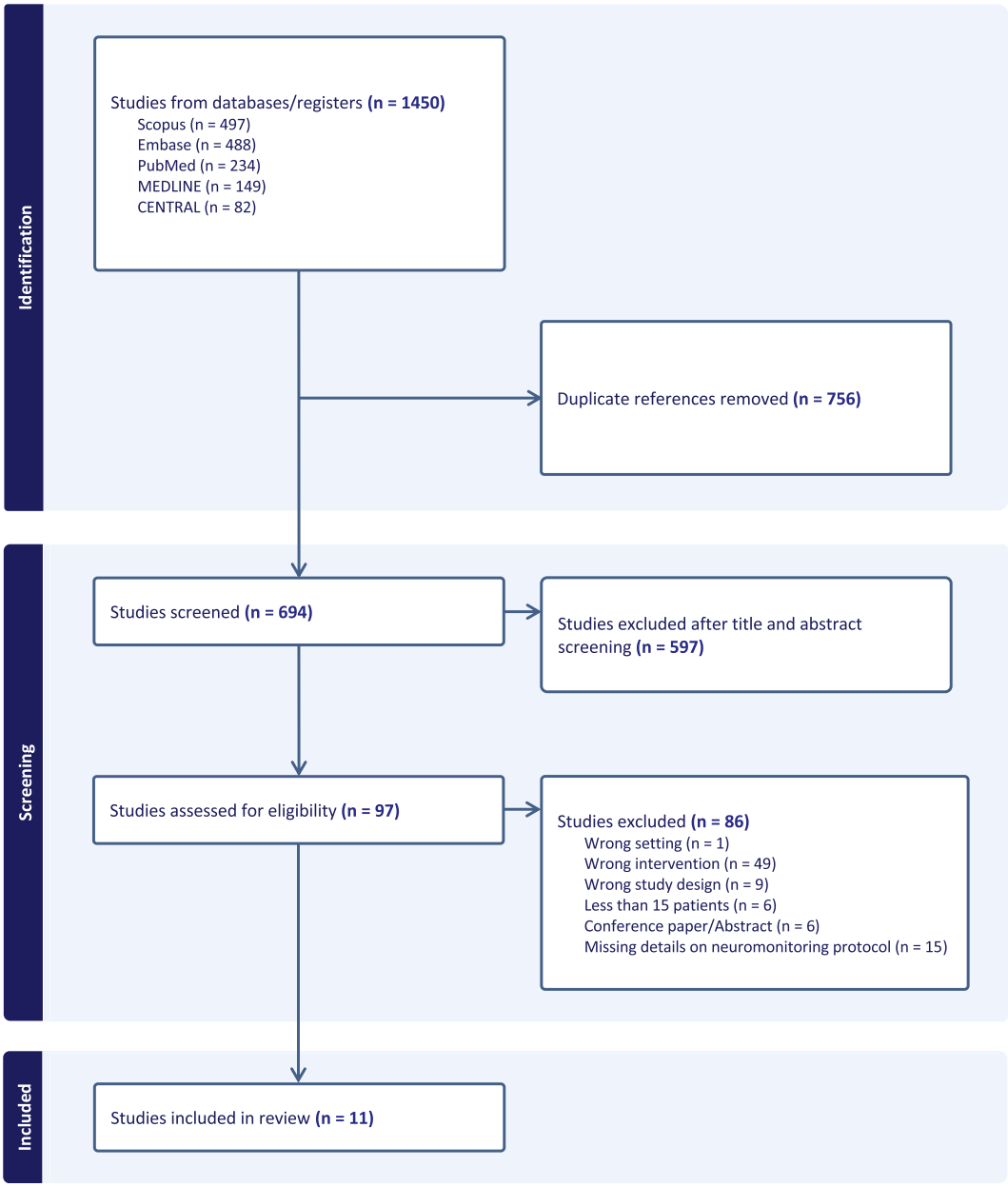

Fig. 1. PRISMA flowchart of study selection for the systematic review.

The possible causes of SCI included hemodynamic compromise, embolism and spinal hematoma.[10] The identified risk factors by multivariate analyses in different studies included operating time [OR 1.5, 95% CI: 1.1–2.2], persistent changes in neuromonitoring [OR 15.7, 95% CI: 2.9–86.2], percentage of thoracic coverage [OR 1.06, 95% CI: 1.00–1.11] and intraoperative blood loss [OR 1.0, 95% CI: 1.00–1.002].[10,16] While aorta coverage was identified as a risk factor,[16] some studies found no association of it with SCI.[15,17] Staged repair of the aorta at least 2 months apart could reduce the incidence of SCI by 80% [OR 0.19, 95% CI: 0.04–0.084].[17]

Mortality

In-hospital or 30-day mortality after endovascular thoracic and thoracoabdominal aortic repair ranged from 0.4 to 8%.[7–10,13,14] The 30-day mortality is the

Table I. Characteristics of the included studies

Study	Year	Study design	Study duration	Patient no.	Neuroprotective measures	Main outcomes
Banga et al.	2016	Prospective cohort study	2011–2014	49	MEP, SSEP	• SCI 6% (permanent 4%, temporary 2%) • 30-day mortality 4%
Cheung et al.	2005	Retrospective cohort study	1999–2004	75	Selective SSEP	• SCI - 6.6% (delayed - 2.7%, permanent SCI - 2.7%) • 30-day mortality - 3%
Haldenwang et al.	2023	Retrospective cohort study	2017–2021	52	Selective SSEP	• SCI - 2% • 30-day mortality - 8% • 1-year mortality - 8%
Kitpanit et al.	2020	Prospective cohort study	2014–2019	106	NIRS	• SCI 3.8% (paraplegia 1.9%, paraparesis 1.9%) • CSFD-related complications 7.6%
Maier et al.	2019	Retrospective cohort study	1998–2014	195	MEP, SSEP	• SCI - 2.7% • 30-day mortality - 2.2%
Rossi et al.	2015	Retrospective cohort study	2008–2014	69	MEP	• SCI 17% (12/69); 6% (4) had permanent paraplegia)
Scott et al.	2021	Retrospective cohort study	Not available	145	NIRS, MEP, Selective SSEP	• SCI 5.5% (permanent paraplegia 1.4%)
Sulzinski et al.	2022	Retrospective cohort study	2017–2018	130	MEP, SSEP	• SCI (delayed - 1.5%, permanent paraparesis - 0.8%) • In-hospital mortality - 7.7%
Tenorio et al.	2022	Prospective cohort study	2013–2018	170	MEP, SSEP	• SCI 4% (permanent 1%) • Mortality 0.4%
ter Wolbeek et al.	2010	Retrospective cohort study	2000–2007	57	MEP, SSEP	• SCI 0%
Weigang et al.	2006	Retrospective cohort study	2000–2005	21	MEP, SSEP	• Delayed SCI - 3.2% • 30-day mortality - 0%

MEP, Motor-Evoked Potential; SSEP, Somatosensory Evoked Potential; NIRS, Near Infra-Red Spectroscopy; SCI, Spinal Cord Ischemia.

Table II. Patient demographics, indications and type of endovascular intervention

Study	Aortic pathology	TAAA Crawford type	Age (year; mean or median)	Gender, male	Prior aortic repair	COPD	CKD	Carotid-subclavian bypass, debranching	CSF drainage	Type of intervention
Banga et al.	DTA, TAAA	I: 2 (4) II: 8 (16) III: 11 (22) IV: 23 (47)	75 ± 8	38 (78)	11 (22)	21 (43)	18 (37)	Debranching: 8 (16)	Routine	FEVAR, BEVAR
Cheung et al.	DTA	N/A	75 ± 7	47 (63)	17 (23)	46 (61)	19 (25)	N/A	Selective	TEVAR
Haldenwang et al.	Complicated TBAD, PAU, IMH	N/A	71 ± 11	N/A	N/A	N/A	N/A	Debranching: 4 (22)	Selective	TEVAR
Kitpanit et al.	TAAA	I: 6 (6) II: 8 (8) III: 25 (25) IV: 63 (62)	75 (70–80)	N/A	54 (52.5)	21 (20.6)	35 (34.5)	N/A	Selective	FEVAR, BEVAR
Maier et al.	TBAD, DTA, Traumatic rupture	N/A	69 (61–79)	155 (69)	28 (13)	46 (20)	61 (27)	Carotid-subclavian bypass: 31 (14) Debranching: 9 (4)	Selective	TEVAR
Rossi et al.	TAAA	I: 4 (6) II: 11 (16) III: 33 (48) IV: 14 (20) V: 7 (10)	72 (58–84)	51 (74)	25 (36)	17 (25)	N/A	N/A	Routine	FEVAR, BEVAR
Scott et al.	TAAA	I: 4 (3) II: 16 (11) III: 18 (12) IV: 39 (27)	70 (53–62)	104 (71)	N/A	66 (45)	41 (28)	N/A	Selective	FEVAR, BEVAR
Sulzinski et al.	TAAA, TBAD	I: 2 (2) II: 5 (5) III: 10 (8) IV: 14 (11) V: 8 (6)	72 (32–90)	62 (48)	45 (35)	23 (18)	7 (5)	Carotid-subclavian bypass: 27 (21) Debranching: 11 (8)	Routine	TEVAR
Tenorio et al.	TAAA	I: 9 (5) II: 53 (31) III: 24 (14) IV: 63 (37)	74 ± 8	122 (72)	90 (53)	62 (36)	74 (44)	N/A	Routine	FEVAR, BEVAR

(*Continued*)

Table II. Continued

Study	Aortic pathology	TAAA Crawford type	Age (year; mean or median)	Gender, male	Prior aortic repair	COPD	CKD	Carotid-subclavian bypass, debranching	CSF drainage	Type of intervention
ter Wolbeek et al.	TAAA, TBAD, Aortic Rupture	I: 16 (28) II: 7 (12) III: 1 (2) IV: 0 V: 0	65	37 (65)	6 (11)	N/A	N/A	N/A	N/A	TEVAR
Weigang et al.	TAAA	I: 7 (33) II: 9 (43) III: 5 (24) IV: 0	65 (53–81)	15 (71)	N/A	6 (29)	9 (43)	N/A	Routine	TEVAR

Data presented as *n* (%) unless stated otherwise.
DTA, Descending Thoracic Aortic Aneurysm; TAAA, Thoracoabdominal Aortic Aneurysm; TBAD, Type B Aortic Dissection; PAU, Penetrating Aortic Ulcer; IMH, Intramural Hematoma; COPD, Chronic Obstructive Pulmonary Disease; CSF, Cerebrospinal Fluid; TEVAR, Thoracic Endovascular Aneurysm Repair; FEVAR, Fenestrated Endovascular Aortic Repair; BEVAR, Branched Endovascular Aortic Repair; N/A, Not Available or Applicable.

highest with 8% in patients presenting with acute aortic syndromes and operated on within 24 hr.[14] Notably, there was no change in mortality at 6-month and 1-year follow-up for the patients with acute aortic syndrome.[14]

DISCUSSION

Continuous improvements in techniques and devices now allow for the treatment of the majority thoracic and thoracoabdominal aortic pathology by endovascular means, with low overall mortality and morbidity. However, the incidence of SCI and subsequent paraplegia after thoraco-abdominal and thoracic endovascular aneurysm repair is estimated to be between 2.5% and 10%.[18,19] In fact, SCI and subsequent paraplegia are among the most dreaded complications of TEVAR and F-BEVAR, as they portend high mortality rates, poor quality of life, and severe healthcare costs. Several risk factors for SCI have been identified, amongst which the length of aortic disease and subsequent need for endograft coverage remain the most consistent ones.[20] Lower SCI incidences are achieved in high volume and experienced centers, and paraplegia rates seem to be declining in recent years; this decline may be largely attributed to the use of rigorous multimodality SCI detection and prevention strategies.[21–23] However, our study highlights the fact that there is no clear strategy to prevent SCI during TEVAR in the current literature.

Our paper aimed to provide a contemporary overview of the current evidence on the implementation and effectiveness of peri-interventional strategies to detect SCI during endovascular thoracic and thoracoabdominal aortic repair and possibly recommend optimal neuromonitoring strategies based on the available data. Although the endovascular repair gained traction in the late 2000s, we have chosen the year 1998 to encompass all studies published in the contemporary period, where recruitment began. Overall, the use of SSEP, usually coupled with MEPs, seems to be the most employed method, achieving good sensitivity for the identification of critical reduction in SCI perfusion parameters that should trigger appropriate corrective responses. In a recent systematic review on the use of neuromonitoring techniques after open TAAA repair, evidence showed that rates of postoperative SCI can be kept at low levels with adequate precautions and perioperative maneuvers.[2] Furthermore, simultaneous monitoring of MEP and SSEP seemed to be the most reliable method that allows rapid detection of

important findings and can guide adequate protective maneuvers during open TAAA repair.[2]

Current literature seems to suggest that rates of postoperative SCI can be kept at low levels after TEVAR and F-BEVAR with the implementation of adequate precautions, notwithstanding that the occurrence of this devastating complication may be related to a multitude of patient-related and procedure-related factors.[8,11,14,16] Amongst the various strategies that have been developed to reduce the ischemic insult to the spinal cord, temporal staging of most extensive repairs has been reported as the preferred method[24,25] (although this may be counterbalanced by an increase in the risk of aneurysm rupture during waiting time,[26] thereby making the time in between procedural steps a critical factor in this strategy). However, the risk of aneurysm rupture during the interval period can be around 3–7%.[27] Also, the use of CSF drainage remains a widely adopted technique although lately, concerns have arisen over the risk of drain-related complications. In contrast, to open TAA/TAAA repair, where neuromonitoring with MEP and SSEP may provide the surgeons with objective criteria to direct selective intercostal reconstruction, less can be done in the endovascular setting to directly counteract the effects of reduced spinal cord perfusion. Amongst the most used techniques to reverse the hemodynamic compromise of spinal cord circulation, there are protective anesthetic and surgical maneuvers that mainly act through indirect mechanisms (such are restoration of hypogastric blood flow, increase of MAP, and lowering of CSF pressure). Nonetheless, simultaneous monitoring of MEP and SSEP seems to be a reliable method that can rapidly detect important findings and thereby direct adequate strategies during endovascular TAA/TAAA repair in the highest-risk individuals, thereby lowering the occurrence and severity of SCI. However, it should also be noted that SCI after minimally invasive endovascular procedures can be a late event, owing to the different pathophysiology from open repair which may entail progressive thrombosis of the aneurysm sac after successful endovascular exclusion. In this particular subset of patients, intraoperative neuromonitoring may not necessarily detect any changes and close clinical observation is therefore mandatory after these operations, even in patients with smooth intraoperative course.

Regarding the preventive measures for SCI after TEVAR and endovascular thoraco-abdominal repair, most of these strategies have proven their effectiveness in preventing SCI during open repair. Obviously, not all the preventive measures used during open surgery are applicable, given the minimally invasive nature of these procedures.[28] Prior research has found that a multimodal approach, including the staging of multiple repairs, preservation of the collateral blood flow network from the subclavian and hypogastric arteries, augmented spinal cord perfusion strategies, and selective CSF drainage, all appear to be important in reducing the risk of SCI. Also, the use of local anesthesia for the final stages of multistep complex endovascular repair may allow for the immediate identification of clinically overt deficits that would trigger immediate actions. Notably, the results of a recent international survey and Delphi consensus have shown that there is broad consensus on the importance of protecting the spinal cord via monitoring, CSF drainage, preoperative segmental coil embolization or staged procedures to avoid SCI in patients undergoing endovascular TAAA repair that require extensive coverage of the thoracoabdominal aorta (i.e. type II repair) or major side branches forming the collateral network.[29] However, concerns also exist regarding the relative safety profile of some of these measures, such as CSF drainage (which has been related to occurrence of potentially severe compications)[30] or the risk for rupture of the aneurysm during the waiting time between stages.[26,31] Future studies focused on the areas of variability may lead to more consistent and improved care for this high-risk population.[32]

Nonetheless, from a practical standpoint, the use of neuromonitoring devices is also dependent on the planned extent of the TAAA repair. Indeed, the same consensus found that neuromonitoring was used most frequently in type II, followed by type I and III, followed by type V, and was least applied in type IV TAAA; around half of the centers in this study used MEP and to a smaller extent SSEP. This explains why only moderate consensus was achieved amongst experts that for endovascular TAAA repair the use of an additional method for monitoring the spinal cord function should be adopted. In fact, the use of evoked potentials in TAAA surgery, whether open or endovascular, certainly demands additional knowledge to accurately detect and interpret the signals, and it may add to the time and costs of the overall procedure. Whether a future randomized trial would prove the superiority of one technique versus the available alternatives remains an area for future research in the field of complex endovascular aortic repair.

Study Limitations

One of the limitations of this systematic review is the availability of high-quality studies and data. Most of

the data are derived from observational studies or case series, rather than from randomized controlled trials, introducing potential inherent biases. Additionally, there is considerable heterogeneity among the included studies in terms of patient populations, types of endovascular procedures performed, and the neuromonitoring techniques employed. Therefore, it was not possible to analyze patients with chronic dissection or aneurysmal disease separately since the studies did not report them individually. Furthermore, the assessment of amplitude thresholds and cut-off values for SCI remains challenging. To further assess and confirm the effectiveness of these neuromonitoring modalities and to analyze whether changes correlate or predict SCI, large-scale adequately powered prospective studies and randomized controlled trials are still required.

CONCLUSIONS

In this systematic review, we provide a contemporary overview of the current evidence on the implementation and effectiveness of peri-interventional strategies to detect SCI during endovascular thoracic and thoracoabdominal aortic repair and recommend optimal neuromonitoring strategies. Overall, the use of SSEP, usually coupled with MEP, seems to be the most commonly employed method, achieving satisfactory sensitivity for the identification of critical SCI parameters that should trigger appropriate corrective treatment responses which can be particularly useful in high-risk patients. However, further studies are still needed to ascertain the optimal neuromonitoring techniques and devise appropriate strategies in response to changes in neuromonitoring findings.

CREDIT AUTHORSHIP CONTRIBUTION STATEMENT

Myat Soe Thet: Writing – original draft, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Mario D'Oria:** Writing – original draft, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Davorin Sef:** Writing – original draft, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Tomislav Klokocovnik:** Writing – review & editing, Visualization, Validation, Supervision. **Aung Ye Oo:** Writing – review & editing, Visualization, Validation, Supervision. **Sandro Lepidi:** Writing – review & editing, Visualization, Validation, Supervision.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at https://doi.org/10.1016/j.avsg.2024.06.012.

REFERENCES

1. Hiratzka LF, Bakris GL, Beckman JA, et al. 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM guidelines for the diagnosis and Management of Patients With Thoracic Aortic Disease: Executive Summary: A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, American Association for Thoracic Surgery, American College of Radiology, American Stroke Association, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of Thoracic Surgeons, and Society for Vascular Medicine *Endorsed by the North American Society for Cardiovascular Imaging*. Catheter Cardiovasc Interv 2010;76:e266–369.
2. Sef D, Thet MS, Miskolczi S, et al. Perioperative neuromonitoring during thoracoabdominal aortic aneurysm open repair: a systematic review. Eur J Cardio Thorac Surg 2023;63:ezad221.
3. Westin GG, Rockman CB, Sadek M, et al. Increased ischemic complications in fenestrated and branched endovascular abdominal aortic repair compared with standard endovascular aortic repair. J Vasc Surg 2020;72:36–43.
4. Maeda K, Ohki T, Kanaoka Y, et al. A novel shaggy aorta scoring system to predict embolic complications following thoracic endovascular aneurysm repair. Eur J Vasc Endovasc Surg 2020;60:57–66.
5. Gaudino M, Khan FM, Rahouma M, et al. Spinal cord injury after open and endovascular repair of descending thoracic and thoracoabdominal aortic aneurysms: a meta-analysis. J Thorac Cardiovasc Surg 2022;163:552–64.
6. Page MJ, McKenzie JE, Bossuyt PM, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ 2021;372:n71.
7. Banga PV, Oderich GS, Reis de Souza L, et al. Neuromonitoring, cerebrospinal fluid drainage, and selective use of iliofemoral conduits to minimize risk of spinal cord injury during complex endovascular aortic repair. J Endovasc Ther 2016;23:139–49.
8. Maier S, Shcherbakova M, Beyersdorf F, et al. Benefits and risks of prophylactic cerebrospinal fluid catheter and evoked potential monitoring in symptomatic spinal cord ischemia low-risk thoracic endovascular aortic repair. Thorac Cardiovasc Surg 2019;67:379–84.
9. Sulzinski MC, Rossi MJ, Alfawaz AA, et al. Optimization of factors for the prevention of spinal cord ischemia in thoracic endovascular aortic repair. Vascular 2022;30:199–205.
10. Tenorio ER, Ribeiro MS, Banga PV, et al. Prospective assessment of a protocol using neuromonitoring, early limb reperfusion, and selective temporary aneurysm sac perfusion to prevent spinal cord injury during fenestrated-branched endovascular aortic repair. Ann Surg 2022;276:e1028–34.
11. ter Wolbeek C, Hartert M, Conzelmann LO, et al. Value and pitfalls of neurophysiological monitoring in thoracic and thoracoabdominal aortic replacement and endovascular repair. Thorac Cardiovasc Surg 2010;58:260–4.
12. Weigang E, Hartert M, Siegenthaler MP, et al. Neurophysiological monitoring during thoracoabdominal aortic

endovascular stent graft implantation. Eur J Cardio Thorac Surg 2006;29:392–6.
13. Cheung AT, Pochettino A, McGarvey ML, et al. Strategies to manage paraplegia risk after endovascular stent repair of descending thoracic aortic aneurysms. Ann Thorac Surg 2005;80:1280–9.
14. Haldenwang PL, Heute C, Schero KJ, et al. Urgent endovascular aortic repair requiring coverage of the left subclavian artery. Thorac Cardiovasc Surg 2023 ((Haldenwang, Schlomicher, Strauch) Cardiothoracic Surgery, Berufsgenossenschaftliches Universitatsklinikum Bergmannsheil, Bochum, Germany(Heute, Nicolas) Institute for Radiologic Diagnostics, Interventional Radiology an Nuclear Medicine, Berufsgenossensch).
15. Rossi SH, Patel A, Saha P, et al. Neuroprotective strategies can prevent permanent paraplegia in the majority of patients who develop spinal cord ischaemia after endovascular repair of thoracoabdominal aortic aneurysms. Eur J Vasc Endovasc Surg 2015;50:599–607.
16. Kitpanit N, Ellozy SH, Connolly PH, et al. Risk factors for spinal cord injury and complications of cerebrospinal fluid drainage in patients undergoing fenestrated and branched endovascular aneurysm repair. J Vasc Surg 2021;73:399–409.e1.
17. Scott CK, Timaran DE, Malekpour F, et al. Selective versus routine spinal drain use for fenestrated/branched endovascular aortic repair (F-BEVAR). Ann Vasc Surg 2021;76:168–73.
18. Rinaldi E, Melloni A, Gallitto E, et al. Spinal cord ischemia after thoracoabdominal aortic aneurysms endovascular repair: from the Italian multicenter fenestrated/branched endovascular aneurysm repair registry. J Endovasc Ther 2023;30:281–8.
19. Pini R, Faggioli G, Paraskevas KI, et al. A systematic review and meta-analysis of the occurrence of spinal cord ischemia after endovascular repair of thoracoabdominal aortic aneurysms. J Vasc Surg 2022;75:1466–1477.e8.
20. Aucoin VJ, Motyl CM, Novak Z, et al. Predictors and outcomes of spinal cord injury following complex branched/fenestrated endovascular aortic repair in the US Aortic Research Consortium. J Vasc Surg 2023;77:1578–87.
21. Dijkstra ML, Vainas T, Zeebregts CJ, et al. Editor's choice – spinal cord ischaemia in endovascular thoracic and thoracoabdominal aortic repair: review of preventive strategies. Eur J Vasc Endovasc Surg 2018;55:829–41.
22. Aucoin VJ, Eagleton MJ, Farber MA, et al. Spinal cord protection practices used during endovascular repair of complex aortic aneurysms by the U.S. Aortic Research Consortium. J Vasc Surg 2021;73:323–30.
23. Marturano F, Nisi F, Giustiniano E, et al. Prevention of spinal cord injury during thoracoabdominal aortic aneurysms repair: what the anaesthesiologist should know. J Pers Med 2022;12:1629.
24. Dias-Neto M, Tenorio ER, Huang Y, et al. Comparison of single- and multistage strategies during fenestrated-branched endovascular aortic repair of thoracoabdominal aortic aneurysms. J Vasc Surg 2023;77:1588–1597.e4.
25. Bertoglio L, Katsarou M, Loschi D, et al. Elective multistaged endovascular repair of thoraco-abdominal aneurysms with fenestrated and branched endografts to mitigate spinal cord ischaemia. Eur J Vasc Endovasc Surg 2020;59:565–76.
26. D'Oria M, Wanhainen A, Mani K, et al. Frequency and type of interval adverse events during the waiting period to complex aortic endovascular repair. J Vasc Surg 2022;75:1821–1828.e1.
27. Kasprzak PM, Gallis K, Cucuruz B, et al. Editor's choice–Temporary aneurysm sac perfusion as an adjunct for prevention of spinal cord ischemia after branched endovascular repair of thoracoabdominal aneurysms. Eur J Vasc Endovasc Surg 2014;48:258–65.
28. Lella SK, Waller HD, Pendleton A, et al. A systematic review of spinal cord ischemia prevention and management after open and endovascular aortic repair. J Vasc Surg 2022;75:1091–106.
29. Schachner T, Gottardi R, Schmidli J, et al. Practice of neuromonitoring in open and endovascular thoracoabdominal aortic repair—an international expert-based modified Delphi consensus study. Eur J Cardio Thorac Surg 2023;63:ezad198.
30. Leone N, D'Oria M, Mani K, et al. Systematic review and meta-analysis of cerebro-spinal fluid drain-related mortality and morbidity after fenestrated-branched endovascular aortic repair. J Vasc Surg 2024 (Epub ahead of print).
31. Cirillo Penn NC, Mendes BC, Tenorio ER, et al. Incidence and risk factors for interval aortic events during staged fenestrated-branched endovascular aortic repair. J Vasc Surg 2023;78:874–82.
32. Chung JC, Lodewyks CL, Forbes TL, et al. Prevention and management of spinal cord ischemia following aortic surgery: a survey of contemporary practice. J Thorac Cardiovasc Surg 2022;163:16–23.e7.