Spinal cord complications after thoracic aortic surgery: Long-term survival and functional status varies with deficit severity

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Objective: **Paraplegia after thoracoabdominal aneurysm (TAA) repair has been associated with poor survival. Little information exists concerning the spectrum of severity that characterizes spinal cord ischemic (SCI) complications. This study stratified SCI by deficit severity to determine its impact on late survival and functional outcomes.**

Methods: **A review of our prospectively maintained thoracic aortic database was performed from May 1987 through December 2005 to identify patients who experienced SCI of any extent after TAA repair. During this period, 576 patients underwent descending thoracic aortic repair (93 open, 105 endovascular [TEVAR]) or open TAA repair (279 extent I to III; 99 extent IV). To stratify severity of SCI, we created a spinal cord ischemia deficit (SCID) scale, which is defined as: I, flaccid paralysis; II, average neurologic muscle grade indicating <50% function; and III, average neurologic muscle grade indicating >50% function. Long-term outcomes were evaluated in relation to these groups by actuarial methods.**

Results: **During the study period, 64 (11.1%) patients developed SCI of any severity (7 of 105 [6.6%] TEVAR, 57 of 471 [12%] open). These were stratified by SCID level: I, 24 (37.5%); II, 31 (48.4%); and III, 9 (14.1%). SCI was immediate in 33 (54.1%) and delayed in 28 (45.9%). Most SCI (6 of 7) associated with TEVAR was delayed. The 30-day mortality was significantly higher in the SCI group than the overall patient cohort (15 of 64 [23.4%] vs 41 of 512 [8%], $P < .001$) and varied by SCID level: I, 11 of 24 (45.8%); II, 4 of 31 (12.9%); and III, 0 of 9 (0%; $P = .001$). The 5-year actuarial survival for all SCI was lower than for non-SCI patients (25% ± 6% vs 51% ± 3%, $P < .001$) and varied linearly with SCID level but was similar between SCID II/III and the non-SCI patients (41% ± 10% vs 51% ± 3%, $P = .281$). No SCID I patients were alive at 5 years. No patients with SCID I recovered the ability to walk, but eight of 11 (73%) with SCID II and the nine (100%) with SCID III could ambulate with or without assistance at last follow-up.**

Conclusion: **Survival and functional outcomes correlate with SCI severity. Patients with SCID I have a poor long-term outlook. Survival of SCID II/III patients is similar to non-SCI patients; most recover the ability to ambulate. (J Vasc Surg 2008;48:47-53.)**

Spinal cord ischemia (SCI) remains a potentially devastating complication of thoracic aneurysm (TA) and thoracoabdominal aneurysm (TAA) repair, with an overall incidence of 16% in the Crawford benchmark series.[1] Various surgical and medical adjuncts, including epidural cooling, cerebrospinal fluid drainage, systemic hypothermia, endorphin receptor blockade, and a variety of pharmacologic adjuncts have been used with varying degrees of success, leading to a significant reduction in the incidence of SCI.[2-5] Despite these advances, however, SCI continues to complicate central aortic surgery, affecting a minimum of 5% to 10% of patients; even in the hands of experts.[5-8]

A variety of reports have documented the major negative impact on short- and long-term survival in patients who sustain paraplegia after central aortic repair.[7,9,10] Yet, it is also recognized that the spectrum of SCI includes incomplete deficits, including transient, totally reversible, and sometimes unilateral leg weakness.[11,12] Indeed, little information exists concerning the spectrum of SCI complications and their respective late outcomes. In addition, the increased application of endovascular aortic repair to the thoracic aorta (TEVAR) has the apparent potential to diminish the overall risk of SCI.[13] The goal of the present study is to stratify SCI deficit severity after TA/TAA repair in an effort to determine differences in late survival and functional outcome.

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METHODS

We prospectively collected relative clinical and demographic data throughout the study interval of May 15, 1987 to December 30, 2005. A review of this database identified 576 patients who underwent descending TA or TAA repair during this interval and included those who underwent elective and emergency repair. The distribution of procedures was 93 (16%) open descending thoracic, 105 (18%) endovascular descending thoracic, 279 (49%) open type I to III thoracoabdominal, and 99 (17%) open type IV thoracoabdominal.

The medical records of all patients who were identified to have a neurologic deficit, regardless of the cause, were

then reviewed. Excluded were patients with neurologic deficits that resulted from verified strokes and patients who developed a neurologic deficit related either to prior surgery or resulting spontaneously from thoracic pathology before undergoing surgery. The 64 patients (11%) who were identified that developed a lower extremity neurologic deficit attributable to SCI after TA/TAA repair comprise the study cohort.

It has been our practice to awaken patients in the immediate postoperative period in the operating room to obtain a gross motor neurologic examination and establish a neurologic baseline. Thus, the onset of neurologic deficit was considered immediate if it was present at this initial examination and delayed if the deficit occurred after a period of normal function. The Institutional Review Board of the Massachusetts General Hospital approved this protocol, and individual consent was waived.

Preoperative and intraoperative variables as well as perioperative outcomes were prospectively compiled during the study period. Chronic obstructive pulmonary disease (COPD) was determined by preoperative pulmonary function tests, which were obtained on most patients. Urgent operation was defined as either rupture or presentation with acute aortic syndrome necessitating invasive monitoring in the intensive care unit (ICU) and operative repair ≤48 hours of admission. Renal insufficiency was defined as a serum creatinine level >1.5 mg/dL.

Spinal cord ischemia deficit. All patients who were suspected by the surgical team to have a neurologic deficit postoperatively underwent immediate clinical evaluation by a neurologist. In an effort to stratify the severity of SCI after descending TA and TAA repair, a spinal cord ischemia deficit (SCID) scoring system was established. This is based upon a modification of the American Spinal Injury Association (ASIA) impairment classification that uses standard neurologic muscle scores (range, 0-5) for three levels of motion, including the hip (extensor, flexor, abductor and adductor), knee (extensor and flexor), and foot (dorsiflexor and plantar flexor muscles).[14,15]

The documented physical findings of the neurologist and functional impairment/ability documented by physical therapists were used to assign a score to each patient that was averaged across the three levels to establish an overall functional score of 0 to 5. Patients who averaged 0 to 1 were assigned to SCID category I, which corresponds with an ASIA A classification and represents flaccid paralysis. Patients with an average score of 1 to 3 were assigned to SCID category II, which represents an average neurologic muscle grade that indicates <50% function (ASIA B and C). Patients with an average score >3 were assigned to SCID category III, indicating >50% muscle function (ASIA D). ASIA E patients have normal function and were not included in the SCID classification. Documented neurologic muscle grades were not available for four patients, all of whom had flaccid paralysis immediately postoperatively and subsequently died before a meaningful neurologic examination could be performed. These patients were assigned to SCID category I based on the immediate postoperative examination of the operating surgeon.

Operative conduct. The clamp and sew technique, with or without neuroprotective adjuncts, was used in 92% of open cases. Distal aortic perfusion was reserved for patients with an anticipated technically complex proximal anastomosis or significant renal insufficiency.

Epidural cooling has been used for spinal cord protection at our institution since July 1993 (types I to III TAA) and, when implemented, was supervised in all cases by a dedicated vascular anesthesia team. Epidural cooling was used in all elective type I to III TAA repairs after 1993 but was not routinely used in patients with type IV TAA. In addition, patients who were considered to be too hemodynamically unstable to have a drain placed did not receive epidural cooling.

Details of the clamp and sew technique and the epidural infusion system have been previously reported.[2,16,17] In brief, the epidural cooling system uses an iced saline epidural infusion, which provides for moderate (25° to 27° C) hypothermia to the spinal cord during the critical period when the aorta is cross-clamped. Patent intercostals vessels in the T9 to L1 region were reimplanted by means of a separate inclusion button or were preserved with a beveled anastomosis, when technically feasible. After reperfusion of the lower extremities, epidural cooling was discontinued, and continuous passive cerebrospinal fluid drainage to keep cerebrospinal fluid pressure at 10 mm Hg was initiated and continued for 48 hours.

Clinical end points. The primary end points of this study included perioperative mortality, long-term survival, and overall functional outcome measured by ambulatory status. Perioperative mortality was defined as any death ≤30 days of the procedure or any death occurring during the initial hospitalization. Early and long-term functional status and survival was determined by a review of office charts and the hospital's computerized medical record. Ambulatory status was classified into three categories for ease of data collection that included: (1) wheelchair bound, (2) ambulatory with assistance (walker/cane), and (3) ambulatory without assistance. Patients who were last known to be alive but had not been seen in more than a year were queried in the Social Security Death Index database to ensure an accurate account of current survival status.

A secondary end point included perioperative morbidity, defined as:

- Major pulmonary complications: mechanical ventilation >72 hours postoperatively, reintubation, respiratory failure requiring ICU monitoring, pneumonia, or need for tracheostomy.
- Significant renal failure: postoperative serum creatinine levels >3.0 mg/dL in patients with normal baseline levels or need for dialysis.
- Cardiac complications: myocardial infarction (MI) as confirmed by electrocardiogram, and appropriate lab-

Table I. Demographic and clinical data for 576 patients undergoing descending thoracic and thoracoabdominal repair

Variable	*SCI*	*No SCI*	P
Patients, No.	64	512	
Age, mean ± SD years	74 ± 7.9	71 ± 11.3	.09
Male sex, No. (%)	27 (42)	265 (52)	.13
Diabetes mellitus, No. (%)	5 (8)	41 (8)	.97
Hypertension, No. (%)	59 (92)	446 (87)	.29
Smoker (former or current), No. (%)	49 (77)	400 (78)	.74
Serum creatinine (>1.5 mg/dL), No. (%)	15 (23)	106 (21)	.54
CAD by history or ECG, No. (%)	54 (84)	276 (54)	<.001
Family history, No. (%)	4 (6)	41 (8)	.62
Max aneurysm diameter, mean ± SD, cm	7.2 ± 1.7	6.7 ± 1.5	.01
Dissection, No. (%)	15 (23)	57 (15)	.09

CAD, Coronary artery disease; *ECG,* electrocardiography; *SCI*, spinal cord ischemia.

Table II. Incidence of spinal cord ischemia stratified by aneurysm extent

Aneurysm extent	*SCI, no (%)*
Descending thoracic	
Open, No (%)	7/93 (7)
TEVAR, No (%)	7/105 (7)
Thoracoabdominal	
Type I, No (%)	14/58 (24)
Type II, No (%)	13/65 (20)
Type III, No (%)	21/156 (13)
Type IV, No (%)	2/99 (2)

SCI, Spinal cord ischemia; *TEVAR,* thoracic endovascular aneurysm repair.

oratory values or, any cardiac event that required intervention.

- Gastrointestinal complications, including bleeding episodes.
- Strokes, which were confirmed using appropriate imaging techniques.

Statistical analysis. Data were analyzed using χ^2 or Fisher exact tests, as appropriate, for nominal variables. The Student *t* and Wilcoxon tests were used to assess continuous variables. A multivariate logistic regression model was created to analyze differences between patients who experienced SCI and those who did not. Long-term survival was determined using Kaplan-Meier life-table methods. Values of $P < .05$ were considered significant.

RESULTS

The study cohort consisted of 64 patients who experienced SCI of any severity after TA/TAA repair. The clinical and demographic features of patients with SCI are compared with those of patients without SCI in Table I. A multivariate regression model was created, and only aneurysm size remained significant (coefficient = 1.255, 95% confidence interval, 1.033-1.524, $P = .02$). The incidence of SCI varied by aneurysm extent, as detailed in Table II. In the SCI cohort, 57 patients (89%) underwent open aneurysm repair and the remaining 7 (11%) were treated with TEVAR. Neuroprotective operative adjuncts of any nature were used in 41 patients (64%), and the combination of cerebrospinal fluid drainage and epidural cooling was applied to 27 (42%). The average total aortic cross-clamp time was 85.9 ± 31.2 minutes, and intraoperative hypotension was documented in 16 patients (25%).

Table III. Additional postoperative complications seen in 64 patients with spinal cord ischemia

Complication	*No. (%)*
Pulmonary	38 (59)
Renal failure	19 (30)
Cardiac	13 (20)
Gastrointestinal	9 (14)
Stroke	8 (13)
Epidural hematoma	3 (5)

The average ICU length of stay in patients with SCI was 9 days (range, 0-52 days), and the average hospital length of stay was 25 days (range, 0-120 days). All patients experienced postoperative complications in addition to SCI, which are summarized in Table III. Patients were stratified by severity of neurologic deficit and assigned to the following groups: SCID I, 24 (38%); SCID II, 31 (48%); and SCID III, 9 (14%). Predictors of SCID score are detailed in Table IV.

SCI deficit was immediate in 27 patients (42%) and had a delayed presentation in the remaining 37 (58%). There was no difference in timing of presentation with respect to SCID score (SCID I: immediate 52% vs delayed 36%; SCID II/III: immediate 48% vs delayed 64%; $P = .11$) or use of neuroprotective adjunct (immediate, 41% vs delayed, 59%; $P = .14$). Six of seven (85%) TEVAR patients with SCI had a delayed presentation, whereas 25 of 57 (44%) who had open surgery presented in the same manner ($P = .03$). Timing of presentation had no impact on perioperative mortality (immediate, 18% vs delayed, 30%; $P = .10$).

Perioperative mortality was 23% (15 of 64) in the SCI group, which was significantly higher than the 8% (41 of 512) seen in those without SCI ($P < .001$). When the perioperative mortality was stratified by SCID classification, there was a significant difference across the groups. Mortality was highest in those with SCID I deficits, at 46% (11 of 24), was 13% (4 of 31) in those with SCID II, and 0% in the remaining patients with SCID III lesions ($P = .001$).

A small number of patients did show improvement in their functional outcome while hospitalized, and seven (11%) had fully recovered neurologic function at the time of discharge. The average follow-up was 27 months. Excluding patients who died during the perioperative period,

Table IV. Analysis of variables predicting immediate functional status in patients with spinal cord ischemia

	Functional outcome, %		
Variable	*SCID I (n = 24)*	*SCID II & III (n = 40)*	P
Age <75 years	42	65	.07
Male sex	63	30	.01
Diabetes	4	10	.40
History of smoking	77	80	.84
Hypertension	92	93	.90
COPD	38	30	.54
History of DVT	8	5	.59
PVD	21	20	.94
History of MI	8	15	.43
History of CVA/TIA	25	10	.11
Family history	4	8	.59
Hyperlipidemia	25	30	.67
CAD	88	83	.59
Renal insufficiency	26	8	.04
Open descending	13	10	.76
TEVAR descending	13	10	.76
Type I TAA	8	30	.03
Type II TAA	29	15	.18
Type III TAA	29	35	.63
Type IV TAA	8	0	.06
Spinal drain + EC	26	53	.04
Any spinal adjunct	60	78	.14
Aneurysm <7 cm	61	68	.64

CAD, Coronary artery disease; *COPD,* chronic obstructive pulmonary disease; *CVA,* cerebrovascular accident; *DVT,* deep vein thrombosis; *EC,* epidural cooling; *PVD,* peripheral vascular disease; *SCID,* spinal cord ischemia deficit; *TIA,* transient ischemic attack; *TAA,* thoracoabdominal aneurysm; *TEVAR,* thoracic endovascular aneurysm repair.

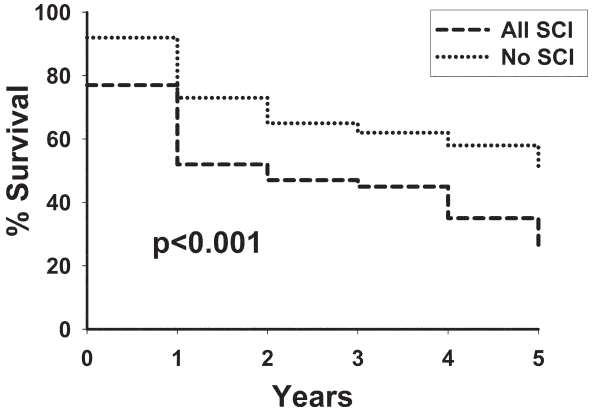

Fig 1. Kaplan-Meier estimate for survival of patients after thoracic aneurysm and thoracoabdominal aneurysm repair stratified by all patients who presented spinal cord ischemia (*SCI, dashed line*) and those who did not (*dotted line*). *P* value determined by Mantel-Cox long-rank univariate analysis.

	0-1 Year	*2-3 Years*	*4-5 Years*
All SCI			
At risk	64	23	24
% Survival (SE)	52 (6)	45 (5)	25 (6)
No SCI			
At risk	509	271	193
% Survival (SE)	73 (2)	62 (2)	51 (3)

the last known ambulatory status was determined for each patient (either current or before death), with 61% confined to a wheelchair, 9% ambulating with assistance, and the remaining 30% ambulating independently. When ambulatory status was stratified by SCID, 0% of SCID I were ambulatory, whereas 73% of SCID II and 100% of SCID III had recovered ambulatory status, with or without assistance, during follow-up.

Long-term all-cause survival was calculated using Kaplan-Meier life-table methods. The 5-year survival for all patients with SCI was 25% ± 6%, which was significantly lower than those without SCI (51% ± 3%, $P < .001$; Fig 1). When those without SCI were compared with patients with SCID II and III deficits, the curves were essentially the same (5-year survival of SCID II and III was 41% ± 9%, $P = .281$). Finally, as expected, the survival of patients with SCID I deficits was significantly lower than patients in the SCID II and III groups, at 0% vs 41% at 5 years ($P < .001$; Fig 2).

DISCUSSION

The benchmark report by Crawford et al[18] of their experience with 1500 patients treated during the interval 1960 to 1991 established the incidence of SCI after TA/TAA repair to be 16%. The addition of a variety of adjunctive strategies designed to minimize SCI have led to rather consistent results of 5% to 10% in contemporary series.[7,8,10] SCI remains a formidable obstacle despite these improved results, and few would suggest that SCI is a solved problem. Indeed, in the current study, the overall incidence of SCI of any severity was 11% (includes emergency cases). There is consensus with respect to the clinical variables that influence the risk of SCI during TA/TAA repair, but because this is not the focus of this report, these are reviewed elsewhere.[2,7,8,10,19,20]

The predominant cause of SCI is thought to be ischemic cell injury to the spinal cord. The neurologic deficits caused by SCI after aortic surgery can be conceptualized as varying on three major axes: location, severity, and time to onset/resolution. With rare exceptions,[21] the location (termed proximity) of the deficit is limited to the lower extremity or lower trunk musculature. In addition, the deficit can be left-side predominant, right-side predominant, or symmetric. Vertical location can also differ when the deficit does not cover the entire lower extremity. The severity of deficit ranges from minor weakness to total paralysis. In addition to motor deficits, sensory deficits, neuropathic pain, and bowel/bladder dysfunction have also been observed.[11,12]

Crawford classified the severity of the SCI deficit as paraplegia (defined as minimal function) and paraparesis (patient had motion against resistance or gravity across all joints).[1] Since this description, several measures of severity

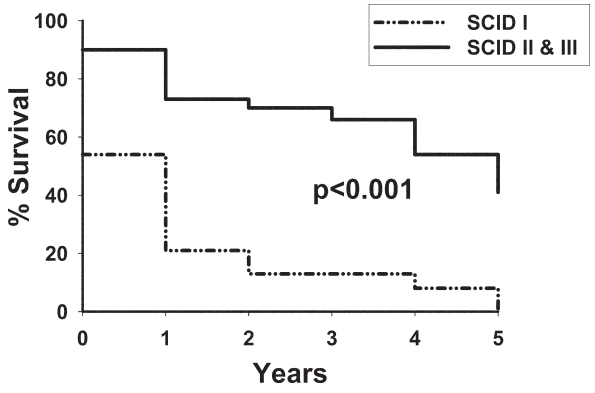

Fig 2. Kaplan-Meier estimate for survival of patients after thoracic aneurysm and thoracoabdominal aneurysm repair stratified by spinal cord ischemia deficit I (*SCID I, dot-dash line*) and SCID II and III *(solid line)*. *P* value determined by Mantel-Cox long-rank univariate analysis.

	0-1 Year	*2-3 Years*	*4-5 Years*
SCID I			
At risk, No.	24	3	1
% Survival (SE)	21 (8)	13 (8)	0 (0)
SCID II/III			
At risk, No.	40	20	13
% Survival (SE)	73 (7)	66 (8)	41 (10)

have been developed and applied to both human and animal models of spinal cord damage,[22,23] but the dichotic classification of paraplegia and paraparesis continues to be used in the surgical literature. The international standards for neurologic classification of spinal cord injury were established by the ASIA to provide a uniform method of comparison of neurologic deficit after SCI.[24] This involves evaluating patients with a 5-point scale and categorizing neurologic deficit according to nine different variables that are often confusing because motor deficits differ between upper and lower extremities.[24] The SCID scale was created to stratify patients into clinically useful categories without being too complex to reproduce. Patients with partial deficits after SCI often have varied motor function across different muscle groups, and the SCID classification takes this into account because the grade from each muscle group is averaged to create the total score.

One issue that confounds the stratification of SCI after TAA repair is the timing of the initial neurologic examination. A subset of patients initially presented with flaccid paralysis (SCID I) but experienced symptomatic improvement with the prompt institution of neuroprotective therapy. This includes transfer to an ICU setting, pharmacologic elevation of the blood pressure, and the institution of spinal drainage to increase the spinal cord perfusion pressure. Although our neurologists are involved early when SCI is expected, the logistics of implementing the above therapy can delay the initial extended neurologic examination, at which time patients may have already experienced a degree of neurologic recovery. This may also explain why no patients with SCID I deficits were ambulatory at the time of follow-up, because patients who are going to recover will usually show some early sign of improvement with the initiation of therapy.

Much has been written about the use of neuroprotective adjuncts in the prevention of SCI after TA/TAA repair.[5-7] Indeed, the adoption of this posture has significantly reduced the rate of SCI in our patients, especially those with type I to III TAA.[7] The role played by neuroprotective adjuncts in the determination of deficit severity has been previously reported.[25] Our finding that a significantly higher percentage of patients with SCID II and III deficits had received spinal drainage and epidural cooling compared with those with flaccid paralysis (SCID I) supports the continued use of neuroprotective adjuncts in anatomically high-risk patients.

Renal insufficiency has been described as a risk factor for early neurologic deficit[10] but was not predictive of SCI in our recent series.[7] Despite this, the finding that renal insufficiency is significantly higher in patients with flaccid paralysis is not surprising. This may be the result of added difficulty in managing volume status in patients who are not producing urine or could reflect patients who were hemodynamically unstable. Previous series have noted a protective effect of female sex against neurologic deficit,[10] but again, this has not been true in our recent experience.[7] In the current cohort, however, male sex was significantly associated with flaccid paralysis. A univariate analysis of the available variables stratified for sex revealed no confounding relationships, so the reason for this association is unclear.

Crawford noted a 30-day mortality of 23% in patients who developed paraplegia vs 9% in those with paraparesis.[1] Our mortality rate was twice as high in patients with flaccid paralysis (46%). This may be related to quality of life issues, because families of patients who developed a SCID I deficit that did not improve were more likely to withdraw care. Despite this phenomenon, there is no doubt that patients with SCID I paralysis experience a greater number of additional complications that increase their risk of early death. Comparatively, the 30-day mortality of SCID III patients was 0%, reflecting a more vital cohort of patients. This is further reflected in the long-term survival outcomes (Fig 2), where patients with SCID II and III lesions have a survival similar to those with no SCI, whereas all patients with SCID I deficits were dead at 5 years.

One metric used to determine the validity of any neurologic grading scale is its correlation with functional outcomes.[24] Admittedly, it would have been impossible to compare outcomes in these patients with standard quality of life measures such as questionnaires because all patients in SCID I group died. Instead, we used survival and ambulatory status as our desired outcomes. Both metrics appear to validate the SCID scale, because mortality rates and ambulatory status improved as neurologic deficit, as defined by the SCID scale, became less severe.

Endovascular stent grafting is likely the single most important advancement in the repair of TAs to date, and emerging technologies will allow this procedure to eventually be applied to patients with TAA.[26-28] Our experience with SCI after TEVAR is small, and no real conclusions can be made from a sample size of seven.[27] However, it is interesting that the distribution of SCI between SCID I and SCID II and III was very similar in this group, and most presented in a delayed fashion. This is similar to recent reports involving the TAG device (W. L. Gore and Assoc, Flagstaff, Ariz), where most incidents of SCI were delayed and resolved or improved with therapy.[13,29] These observations have led to our adoption of the practice of observing our TEVAR patients in an ICU setting with continuous arterial pressure monitoring for 48 hours to prevent sudden changes in blood pressure that can precipitate neurologic events.

CONCLUSION

Despite advances in operative technique and the application of various neuroprotective adjuncts, SCI remains a significant complication of the repair of aneurysms of the thoracic aorta. Patient survival and functional outcome after SCI are directly linked to deficit severity, and the application of a classification system similar to the SCID score can provide prognostic information for patients and their families during the initial postoperative period. These data underscore the importance of early and aggressive intervention when SCI is expected after TA/TAA repair, because early improvement to SCID II or III from flaccid paralysis has significant implications for survival and functional status.

AUTHOR CONTRIBUTIONS

Conception and design: MC, JD, RC
Analysis and interpretation: MC, TC, JD
Data collection: JY, TC
Writing the article: MC, JY, JD, RC
Critical revision of the article: MC, RC
Final approval of the article: MC, JY, JD, RC
Statistical analysis: JY, TC
Obtained funding: NA
Overall responsibility: MC

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