Mitigating Spinal Cord Ischemia in Endovascular Thoracoabdominal Repair

Adam W. Beck, M.D.
Associate Professor of Surgery
Division of Vascular Surgery and Endovascular Therapy
University of Alabama at Birmingham

April 28th, 2019
Disclosures

• Sponsored investigator for: Cook, Medtronic, W.L. Gore, Lombard

• Consultant for Medtronic, Inc, Terumo Aortic, W.L. Gore & Associates

• Physician Education: Cook Medical, Terumo

ALL Proceeds to UAB
Outline

• Spinal cord ischemia (SCI) and Aortic Repair
• Implications of Spinal Cord Ischemia (SCI)
• Spinal Cord Perfusion/Mechanisms of Injury
• SCI and endo-TAAA repair
• UF/UAB Experience
Thoracic Aneurysm

Etiology

- 80% Degenerative
- 15% Dissection
- 5% Trauma, Infection, Marfan’s Syndrome, Ehler’s-Danlos syndrome, Takayasu’s Arteritis
Open Thoracic Aneurysm Repair

- 1902: Obliterative endoaneurysmorrhaphy (Matas)
- 1951: First successful open repair (Lam and Aram)
- 1953: Cooley and Debakey
Background
Thoracoabdominal Aneurysm
Open Repair of TAA and TAAA

Mortality
10-25%

Morbidity
Pulmonary: 30-50%
Cardiac:  10-25%
Renal: 10-20%
Paralysis/Paraplegia: 10-40%
Stroke: 5-10%
Open Repair: Acute Spinal Cord Ischemia

- Aortic Cross-Clamp
- Hemodynamics (Hypotension)
- Intercostal, Subclavian, Hypogastric, Lumbar Arterial coverage/ligation
Open Repair of TAA and TAAA

Current Adjuncts

• CSF Drainage
• Distal Aortic Perfusion
• Intercostal Reimplantation
  • Expeditious Surgery
    • Hypothermia
    • Spinal Cooling
• Neuroprotective drugs

SCI: 5-20%
SCI and TEVAR
Background

TEVAR:

- 1994: First successful TEVAR
- 2005: Gore TAG approved for use in the U.S.
Risk factors, outcomes, and clinical manifestations of spinal cord ischemia following thoracic endovascular aortic repair

Brant W. Ullery, MD, a Albert T. Cheung, MD, a Ronald M. Fairman, MD, a Benjamin M. Jackson, MD, a Edward Y. Woo, MD, a Joseph Bavaria, MD, c Alberto Pochettino, MD, c and Grace J. Wang, MD, a Philadelphia, Pa

Objective: The purpose of this study was to assess the incidence, risk factors, and clinical manifestations of spinal cord ischemia (SCI) after thoracic endovascular aortic repair (TEVAR).

Methods: A retrospective review of a prospectively collected database was performed for all patients undergoing TEVAR at a single academic institution between July 2002 and June 2010. Preoperative demographics, procedure-related variables, and clinical details related to SCI were examined. Logistic regression analysis was performed to identify risk factors for the development of SCI.

Results: Of the 424 patients who underwent TEVAR during the study period, 12 patients (2.8%) developed SCI. Mean age of this cohort with SCI was 69.6 years (range, 44–84 years), and 7 were women. One-half of these patients had prior open or endovascular aortic repair. Indication for surgery was either degenerative aneurysm (n = 8) or dissection (n = 4). Six TEVARs were performed electively, with the remaining done either urgently or emergently due to contained rupture (n = 2), dissection with malperfusion (n = 2), or severe back pain (n = 2). All 12 patients underwent extent C endovascular coverage. Multivariate regression analysis demonstrated chronic renal insufficiency to be independently associated with SCI (odds ratio [OR], 4.39; 95% confidence interval [CI], 1.2–16.6; P = .029). Onset of SCI occurred at a median of 10.6 hours (range, 0–229 hours) postprocedure and was delayed in 83% (n = 10) of patients. Clinical manifestations of SCI included lower extremity paraparesis in 9 patients and paraplegia in 3 patients. At SCI onset, average mean arterial pressure (MAP) and lumbar cerebrospinal fluid (CSF) pressure was 77 mm Hg and 10 mm Hg, respectively. Therapeutic interventions increased blood pressure to a significantly higher average MAP of 99 mm Hg (P = .001) and decreased lumbar CSF pressure to a mean of 7 mm Hg (P = .30) at the time of neurologic recovery. Thirty-day mortality was 8% (1 of 12 patients). The single patient who expired, never recovered any lower extremity neurologic function. All patients surviving to discharge experienced either complete (n = 9) or incomplete (n = 2) neurologic recovery. At mean follow-up of 49 months, 7 of 9 patients currently alive continued to exhibit complete, sustained neurologic recovery.

Conclusion: Spinal cord ischemia after TEVAR is an uncommon, but important complication. Preoperative renal insufficiency was identified as a risk factor for the development of SCI. Early detection and treatment of SCI with blood pressure augmentation alone or in combination with CSF drainage was effective in most patients, with the majority achieving complete, long-term neurologic recovery. (J Vasc Surg 2011;54:677–84.)
UPenn TEVAR Experience

N=424

SCI=2.8%

Fig. 1. Study design.
Risk factors, outcomes, and clinical manifestations of spinal cord ischemia following thoracic endovascular aortic repair

Brant W. Ullery, MD,^a^ Albert T. Cheung, MD,^b^ Ronald M. Fairman, MD,^a^ Benjamin M. Jackson, MD,^a^ Edward Y. Woo, MD,^a^ Joseph Bavaria, MD,^c^ Alberto Pochettino, MD,^c^ and Grace J. Wang, MD,^a^ Philadelphia, Pa

Table II. Preoperative predictors of spinal cord ischemia after TEVAR, multivariate analysis

<table>
<thead>
<tr>
<th>Variable</th>
<th>Odds ratio (95% CI)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>CRI^a^</td>
<td>4.39 (1.16-16.58)</td>
<td>.029</td>
</tr>
<tr>
<td>Extent C coverage^b^</td>
<td>0.00</td>
<td>.995</td>
</tr>
</tbody>
</table>

CI, confidence interval; CRI, chronic renal insufficiency; TEVAR, thoracic endovascular aneurysm repair.

^a^Creatinine ≥1.5 mg/dL.

^b^Stent coverage from origin of left subclavian artery to diaphragm.
Extent of Aortic Coverage and Incidence of Spinal Cord Ischemia After Thoracic Endovascular Aneurysm Repair

Robert J. Feezor, MD, Tomas D. Martin, MD, Philip J. Hess Jr, MD, Michael J. Daniels, ScD, Thomas M. Beaver, MD, Charles T. Klodell, MD, and W. Anthony Lee, MD

Divisions of Vascular Surgery and Endovascular Therapy, and Thoracic and Cardiovascular Surgery, Department of Surgery, and Division of Biostatistics, Department of Epidemiology and Biostatistics, University of Florida, Gainesville, Florida


### Table 1. The Incidence of Any and Permanent Spinal Cord Ischemia Based on Aortic Pathology

<table>
<thead>
<tr>
<th>Aortic Pathology</th>
<th>No.</th>
<th>No SCI, No. (%)</th>
<th>Any SCI, No. (%)</th>
<th>p Value&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Perm SCI, No. (%)</th>
<th>p Value&lt;sup&gt;a&lt;/sup&gt;</th>
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</thead>
<tbody>
<tr>
<td>TAA</td>
<td>164</td>
<td>145 (88)</td>
<td>19 (12)</td>
<td>0.46</td>
<td>5 (3)</td>
<td>0.29</td>
</tr>
<tr>
<td>Dissection</td>
<td>80</td>
<td>70 (88)</td>
<td>10 (13)</td>
<td>0.40</td>
<td>7 (9)</td>
<td>0.05</td>
</tr>
<tr>
<td>Ulcer</td>
<td>43</td>
<td>39 (91)</td>
<td>4 (9)</td>
<td>1.00</td>
<td>2 (5)</td>
<td>1.00</td>
</tr>
<tr>
<td>Transection</td>
<td>21</td>
<td>21 (100)</td>
<td>0 (0)</td>
<td>0.15</td>
<td>0 (0)</td>
<td>0.61</td>
</tr>
<tr>
<td>Other</td>
<td>18</td>
<td>18 (100)</td>
<td>0 (0)</td>
<td>0.23</td>
<td>0 (0)</td>
<td>1.00</td>
</tr>
</tbody>
</table>

<sup>a</sup> Values for p are calculated for the incidence of SCI for each pathology relative to the aggregate of other patients.
Table 5. Multivariate Logistic Regression Analysis of Variables That Influenced the Development of any Spinal Cord Ischemia

<table>
<thead>
<tr>
<th>Variable</th>
<th>OR (95% CI)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (every 10 years)</td>
<td>1.6 (1.1–2.2)</td>
<td>0.006</td>
</tr>
<tr>
<td>Male</td>
<td>2.3 (0.90–5.7)</td>
<td>0.08</td>
</tr>
<tr>
<td>ASA class IV</td>
<td>2.3 (0.95–5.4)</td>
<td>0.06</td>
</tr>
<tr>
<td>Emergency</td>
<td>1.7 (0.73–4.0)</td>
<td>0.22</td>
</tr>
<tr>
<td>Procedure time (every 60 min)</td>
<td>1.3 (0.98–1.6)</td>
<td>0.07</td>
</tr>
<tr>
<td>Fluoroscopy time (every 10 min)</td>
<td>1.2 (0.98–1.5)</td>
<td>0.07</td>
</tr>
<tr>
<td>Contrast (every 10 mL)</td>
<td>1.0 (0.96–1.1)</td>
<td>0.38</td>
</tr>
<tr>
<td>Prophylactic spinal drainage</td>
<td>1.2 (0.50–2.7)</td>
<td>0.74</td>
</tr>
<tr>
<td>TAG</td>
<td>1.1 (0.46–2.6)</td>
<td>0.83</td>
</tr>
<tr>
<td>General endotracheal anesthesia</td>
<td>2.5 (1.0–6.3)</td>
<td>0.05</td>
</tr>
<tr>
<td>Estimated blood loss (every 50 mL)</td>
<td>1.0 (0.96–1.1)</td>
<td>0.86</td>
</tr>
<tr>
<td>Iliac conduit</td>
<td>1.2 (0.50–3.0)</td>
<td>0.66</td>
</tr>
<tr>
<td>AAA repair</td>
<td>1.6 (0.61–4.4)</td>
<td>0.33</td>
</tr>
<tr>
<td>Aortic length (every 20 mm)(^a)</td>
<td>1.2 (0.93–1.4)</td>
<td>0.17</td>
</tr>
<tr>
<td>Aortic coverage (every 20 mm)</td>
<td>1.3 (1.1–1.5)</td>
<td>0.0004</td>
</tr>
<tr>
<td>Uncovered distance</td>
<td></td>
<td></td>
</tr>
<tr>
<td>From L CCA (every 25 mm)</td>
<td>0.99 (0.87–1.1)</td>
<td>0.81</td>
</tr>
<tr>
<td>From celiac (every 20 mm)</td>
<td>0.61 (0.42–0.87)</td>
<td>0.006</td>
</tr>
<tr>
<td>L SCA perfusion(^b)</td>
<td>1.2 (0.39–3.7)</td>
<td>0.75</td>
</tr>
<tr>
<td>Patent hypogastric artery</td>
<td>0.84 (0.28–2.5)</td>
<td>0.75</td>
</tr>
</tbody>
</table>

\(^a\) Left common carotid artery to celiac artery. \(^b\) Includes those cases with either uncovered or revascularized left subclavian artery.
Preoperative prediction of spinal cord ischemia after thoracic endovascular aortic repair

Salvatore T. Scali, MD, a S. Keisin Wang, MD, a Robert J. Feezor, MD, a Thomas S. Huber, MD, PhD, a Tomas D. Martin, MD, b Charles T. Klodell, MD, b Thomas M. Beaver, MD, MPH, b and Adam W. Beck, MD, a Gainesville, Fla

Objective: Spinal cord ischemia (SCI) after thoracic endovascular aortic repair (TEVAR) is a potential morbid complication that requires prompt recognition and intervention. We sought to identify clinical and procedural variables that were predictive of SCI and determine if a logistic regression model based on these conditions can predict SCI in patients undergoing TEVAR.

Methods: All consecutive patients undergoing TEVAR at our institution were included in the analysis. Preoperative and procedural variables of interest were collected, and SCI was defined according to the International Spinal Cord Injury Classification Committee (2002). Logistic regression models were developed to predict SCI and its severity (permanent SCI vs. nonpermanent SCI).

Results: From 2006 to 2017, 741 patients underwent TEVAR (permanent: n = 43; 7.4%) were subsequently dichotomized into two groups: SCI (n = 23; 3.1%) and no SCI (n = 718; 96.9%) (P < .0001) and had significantly higher rates of multiple cardiovascular risk factors. The stepwise selection procedure identified five variables as the most important predictors of SCI: age (odds ratio [OR] multiplies by 1.3 per 10 years; 95% confidence interval [CI], 0.9-1.8, P = .06), aortic coverage length (OR multiplies by 1.3 per 5 cm; CI, 1.1-1.6; P = .002), chronic obstructive pulmonary disease (OR, 1.9; CI, 0.9-4.1; P = .1), chronic renal insufficiency (creatinine concentration ≥ 1.6 mg/dL; OR, 1.9; CI, 0.8-4.2; P = .1), and hypertension (defined as chart history or medication; OR, 6.4; CI, 2.6-18; P < .0001). A logistic regression model with just these five covariates had excellent discrimination (area under the receiver operating characteristic curve = .83) and calibration (χ² = 9.8, P = .28).

Conclusions: This analysis generated a simple model that reliably predicts SCI after TEVAR. This clinical tool can assist decision-making about when to proceed with TEVAR, guide discussions about intervention risk, and help determine when maneuvers to mitigate SCI risk should be implemented. (J Vasc Surg 2014;60:1481-90.)
Preoperative prediction of spinal cord ischemia after thoracic endovascular aortic repair

A  Age  

B  Aortic Coverage Length  

Observed Probability of SCI

Age in Years

<50  50-54  55-58  60-64  65-69  70-74  75-79  80-84  85+

Observed Probability of SCI

Aortic Coverage Length

<151  151-175  176-200  201-225  225-250  251-275  276-300  >300

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Division of Vascular Surgery and Endovascular Therapy
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Table IV. Outcomes after TEVAR in all patients with or without spinal cord ischemia (SCI)

<table>
<thead>
<tr>
<th>Feature</th>
<th>No SCI (n = 673)</th>
<th>SCI (n = 68)</th>
<th>P value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thirty-day mortality</td>
<td>25 (4)</td>
<td>4 (6)</td>
<td>.3</td>
</tr>
<tr>
<td>Length of stay</td>
<td>5 (3-9)</td>
<td>13 (8-22)</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Complications</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulmonary</td>
<td>51 (8)</td>
<td>15 (22)</td>
<td>.0004</td>
</tr>
<tr>
<td>Renal</td>
<td>35 (5)</td>
<td>10 (15)</td>
<td>.005</td>
</tr>
<tr>
<td>Bleeding</td>
<td>25 (4)</td>
<td>4 (6)</td>
<td>.3</td>
</tr>
<tr>
<td>Stroke</td>
<td>21 (3)</td>
<td>4 (6)</td>
<td>.3</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>20 (3)</td>
<td>3 (4)</td>
<td>.5</td>
</tr>
<tr>
<td>Cardiac</td>
<td>20 (3)</td>
<td>4 (6)</td>
<td>.3</td>
</tr>
</tbody>
</table>
What happens after SCI?
Fate of patients with spinal cord ischemia complicating thoracic endovascular aortic repair

Kenneth DeSart, MD,a Salvatore T. Scali, MD,a Robert J. Feezor, MD,a Michael Hong, MD,a Philip J. Hess Jr, MD,b Thomas M. Beaver, MD, MS,b Thomas S. Huber, MD, PhD, and Adam W. Beck, MD,a Gainesville, Fla

Objective: Spinal cord ischemia (SCI) is a potentially devastating complication of thoracic endovascular aortic repair (TEVAR) that can result in varying degrees of short-term and permanent disability. This study was undertaken to describe the clinical outcomes, long-term functional impact, and influence on survival of SCI after TEVAR.

Methods: A retrospective review of all TEVAR patients at the University of Florida from 2000 to 2011 was performed to identify individuals experiencing SCI, defined by any new lower extremity neurologic deficit not attributable to another cause. SCI was dichotomized into immediate or delayed onset, with immediate onset defined as SCI noted upon awakening from anesthesia, and delayed characterized as a period of normal function, followed by development of neurologic injury. Ambulatory status was determined using database query, record review, and phone interviews with patients and/or family. Mortality was estimated using life-table analysis.

Results: A total of 607 TEVARs were performed for various indications, with 57 patients (9.4%) noted to have postoperative SCI (4.3% permanent). SCI patients were more likely to be older (63.9 ± 15.6 vs 70.5 ± 11.2 years; P = .002) and have a number of comorbidities, including chronic obstructive pulmonary disease, hypertension, dyslipidemia, and cerebrovascular disease (P < .0001). At some point in their care, a cerebrospinal fluid drain was placed in 54 patients (95%), with 54% placed postoperatively. In-hospital mortality was 8.8% for the entire cohort (SCI vs no SCI; P = .45). SCI developed immediately in 12 patients, delayed onset in 40, and indeterminate in five patients due to indiscriminate timing from postoperative sedation. Three patients (25%) with immediate SCI had measurable functional improvement (FI), whereas 28 (70%) of the delayed-onset patients experienced some degree of neurologic recovery (P = .04). Of the 34 patients with complete data available, 26 (76%) reported quantifiable FI, but only 13 (38%) experienced return to their preoperative baseline. Estimated mean (± standard error) survival for patients with and without SCI was 37.2 ± 4.5 and 71.6 ± 3.9 months (P < .0006), respectively. Patients with FI had a mean survival of 53.9 ± 5.9 months compared with 9.6 ± 3.6 months for those without improvement (P < .0001). Survival and return of neurologic function were not significantly different when patients with preoperative and postoperative cerebrospinal fluid drains were compared.

Conclusions: The minority of patients experience complete return to baseline function after SCI with TEVAR, and outcomes in patients without early functional recovery are particularly dismal. Patients experiencing delayed SCI are more likely to have FI and may anticipate similar life-expectancy with neurologic recovery compared with patients without SCI. Timing of drain placement does not appear to have an impact on postdischarge FI or long-term mortality. (J Vasc Surg 2013;58:635-42.)
609 TEVARs

57 patients (9.4%) developed Spinal Cord Ischemia

- In-hospital mortality with SCI was 7.0%
- 26 (76%) noted subjective functional improvement after discharge
- 12 (35%) reporting return to baseline
Median survival

- No SCI
  - 72 months

- SCI
  - 37 months
Fate of patients with spinal cord ischemia complicating thoracic endovascular aortic repair
Median survival

- Functional Improvement: 56 months
- No improvement: 3 months
Fate of patients with spinal cord ischemia complicating thoracic endovascular aortic repair

*P=0.03

Return to Baseline

Did not Return to Baseline

Percent Survival after TEVAR

Months after TEVAR

<table>
<thead>
<tr>
<th>RTB</th>
<th>At Risk</th>
<th>SE</th>
<th>At Risk</th>
<th>SE</th>
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<td></td>
<td>13</td>
<td>.00</td>
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<tr>
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<td>.00</td>
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<td>.13</td>
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<tr>
<td></td>
<td>3</td>
<td>.09</td>
<td>3</td>
<td>.12</td>
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</table>
Implications of SCI

Perioperative Mortality (Open Repair)

With SCI: 45.5%
Without SCI: 4.5%

12-Month survival (TEVAR)

With SCI: 25%
Without SCI: 85%
Endovascular Repair: Acute Spinal Cord Ischemia

- Aortic coverage/Intercostal Thrombosis
- Hemodynamics (Hypotension)/Blood loss/Anemia
- Intercostal, Subclavian, Hypogastric, Lumbar Arterial coverage/ligation
- Transient hypogastric/femoral malperfusion due to sheaths
- Secondary injury due to spinal cord edema
What about Endovascular Thoracoabdominal Repair?
Contemporary Analysis of Descending Thoracic and Thoracoabdominal Aneurysm Repair
A Comparison of Endovascular and Open Techniques

Roy K. Greenberg, MD; Qingsheng Lu, MD; Eric E. Roselli, MD; Lars G. Svensson, MD, PhD; Michael C. Moon, MD; Adrian V. Hernandez, MD, MSc, PhD; Joseph Dowdall, MD; Marcelo Cury, MD; Catherine Francis, BS; Kathryn Pfaff, BS; Daniel G. Clair, MD; Kenneth Ouriel, MD; Bruce W. Lytle, MD

Table 1. Definitions of Types of Repair

<table>
<thead>
<tr>
<th>Type</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Aortic repair distal to the left common carotid artery terminating above the diaphragm</td>
</tr>
<tr>
<td>I</td>
<td>A repair distal to the left common carotid artery to beyond the diaphragmatic border to above the renal arteries (with or without reimplantation of the mesenteric vessels)</td>
</tr>
<tr>
<td>II</td>
<td>Aortic repair distal to the left common carotid artery, proximal to the 6th intercostal space, to a region below the renal arteries (requiring reimplantation of the visceral segment)</td>
</tr>
<tr>
<td>III</td>
<td>An aortic repair originating distal to the 6th intercostal space but above the diaphragm terminating below the renal arteries</td>
</tr>
<tr>
<td>IV</td>
<td>A repair originating below the diaphragm and above the renal arteries terminating below the renal arteries</td>
</tr>
</tbody>
</table>
SCI
Endovascular vs. Open

![Graph showing comparison between Open Surgery and Endovascular Surgery](image)

Log-rank test = 0.08
# Contemporary Analysis of Descending Thoracic and Thoracoabdominal Aneurysm Repair

A Comparison of Endovascular and Open Techniques

<table>
<thead>
<tr>
<th>Extent</th>
<th>Repair Technique</th>
<th>n</th>
<th>Mortality at 30 d</th>
<th>Mortality at 1 y</th>
<th>SCI</th>
</tr>
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<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>n</td>
<td>%*</td>
<td>Rate†</td>
</tr>
<tr>
<td>None</td>
<td>ER</td>
<td>163</td>
<td>8</td>
<td>5</td>
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<tr>
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<td>SR</td>
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<td>12</td>
<td>1.68</td>
</tr>
<tr>
<td>IV</td>
<td>ER</td>
<td>69</td>
<td>3</td>
<td>4</td>
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</tr>
<tr>
<td></td>
<td>SR</td>
<td>64</td>
<td>4</td>
<td>6</td>
<td>0.80</td>
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<tr>
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<td>ER</td>
<td>352</td>
<td>20</td>
<td>6</td>
<td>0.72</td>
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<tr>
<td></td>
<td>SR</td>
<td>372</td>
<td>31</td>
<td>7</td>
<td>1.07</td>
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</table>

*Kaplan-Meier estimate.
†Incidence rates are defined as deaths per person-year.
‡Statistically significant difference between ER and SR with log-rank test.
Endovascular Repair of Type II and Type III Thoracoabdominal Aneurysms

Tara M. Mastracci, MD, FRCSC, MSc, and Matthew J. Eagleton, MD, FACS

Table 5. Incidence of Spinal Cord Ischemia (SCI) Based on Extent of Aortic Aneurysm Repaired Using an Endovascular Approach

<table>
<thead>
<tr>
<th>Extent of Repair</th>
<th>n</th>
<th>Incidence of SCI (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All</td>
<td>352</td>
<td>4% (15)</td>
</tr>
<tr>
<td>I</td>
<td>82</td>
<td>10% (8)</td>
</tr>
<tr>
<td>II</td>
<td>16</td>
<td>19% (3)</td>
</tr>
<tr>
<td>III</td>
<td>22</td>
<td>5% (1)</td>
</tr>
<tr>
<td>IV</td>
<td>69</td>
<td>3% (2)</td>
</tr>
</tbody>
</table>
UF Branched/Fenestrated Spinal Cord Ischemia

N=243

In-house mortality: N=10 (4.1%)

No SCI

Any SCI

Juxtarenal: 43
Suprarenal: 52
IV: 53
III: 54
II: 33
I: 8
UF Branched/Fenestrated Spinal Cord Ischemia Temporary vs. Permanent

- Recovered Function
- Permanent SCI

<table>
<thead>
<tr>
<th>Level</th>
<th>Recovered Function</th>
<th>Permanent SCI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Juxtarenal</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>Suprarenal</td>
<td>0%</td>
<td>10.71%</td>
</tr>
<tr>
<td>IV</td>
<td>0%</td>
<td>4.9%</td>
</tr>
<tr>
<td>III</td>
<td>0%</td>
<td>10.71%</td>
</tr>
<tr>
<td>II</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>I</td>
<td>0%</td>
<td>0%</td>
</tr>
</tbody>
</table>
What can we do?

Prevention is key

Rapid detection is equally important

- Physical Exam?
- Biomarkers of Spinal Cord Ischemia?
- SSEP/MEP

Treatment should be protocol-guided
A Modern Theory of Spinal Cord Ischemia/Injury in Thoracoabdominal Aortic Surgery and Its Implications for Prevention of Paralysis

Martha M. Wynn, MD,* and C.W. Acher, MD†

Pathophysiology of Spinal Cord Ischemia/Infarction

Blood flow

O₂ delivery

Cell injury

↓ Arterial flow
↑ CVP & SFP

↓ Cord perfusion

O₂ Supply < Demand

↓ Ischemia

ATP deficit, cells lose resting membrane potentials

Free radicals, excitatory amino acids, NO

Reperfusion hyperemia, edema, inflammation

Interrupt all intercostal arteries

Direct arterial input to spinal cord blood flow

Fig 3. Pathophysiology of spinal cord ischemia/infarction in TAAA surgery. (Color version of figure is available online.)
A Modern Theory of Spinal Cord Ischemia/Injury in Thoracoabdominal Aortic Surgery and Its Implications for Prevention of Paralysis

Martha M. Wynn, MD,* and C.W. Acher, MD†

![Diagram of spinal cord ischemia and intervention points](image)

**Interventions to Reduce Spinal Cord Ischemia/Infarction**

- **Clamp Aorta**
  - ↓MAP (CNP) & CI
  - ↑CVP, ↑SFP
  - ↓SFP, ↑CNP, ↑CI
  - ↑Cord perfusion
  - **Hypothermia**
  - Neuroprotective drugs
  - O₂ Supply = O₂ Demand
  - ↓Ischemia
  - ATP deficit, cells lose resting membrane potential
  - **Hypothermia**
  - Free radicals, excitatory amino acids, NO

- **Replace Aorta**
  - Interrupt all intercostal arteries (ICA)
  - Reimplant ICA ↑ direct arterial input
  - ↓Ischemia
  - Reperfusion edema, hyperemia, inflammation
  - Neuroprotective drugs
  - Hypothermia

- **Unclamp Aorta**
  - Neuroprotective drugs

---

Fig 7. Interventions to reduce spinal cord ischemia/infarction in TAAA surgery. (Color version of figure is available online.)
**Preoperative:**
1. No perioperative blood pressure medications (except beta-blockers) two days prior to the procedure for permissive hypertension.
2. All patients should be on a statin started in the preoperative setting unless there is an appropriate contraindication.
3. Preoperative placement of a cerebrospinal fluid drain
4. After induction of anesthesia, 30mg/Kg of methylprednisolone is given to reduce the swelling that might occur with spinal cord ischemic injury.
5. Staged TEVAR procedures will be performed when appropriate, and will generally be done while we are awaiting the arrival of the custom device. This will include proximal TEVAR and any adjunctive procedures required such as femoral bypass/carotid-subclavian bypass.

**Intraoperative:**
1. Permissive hypertension with a goal Mean Arterial Pressure (MAP) of 90.
2. Optimization of cardiac index with inotropic agents, as necessary. Tissue Doppler echo will be used to help guide therapy in patients with an abnormal ejection fraction at baseline.
3. Mannitol is given to patients who have any issues with the potency of their spinal drain at a dose of 12.5gms prior to graft deployment and 12.5gms afterward.
4. If the CSF drain is working well, we drain the patient at 10mm of Hg and avoid mannitol.
5. Norepinephrine drip that is started at the beginning of the procedure and continued for 48 hours.
6. Goal hemoglobin of 10mg/dL.

**Post-operative:**
1. Permissive hypertension with a mean goal MAP of 90 for 48 hours or until the spinal drain is out.
   a. Patients will have continuous CVP monitoring in the SICU. If the patient is on the ventilator, the goal CVP will be ≥10mm Hg. If extubated, the goal CVP will be 7.5mm Hg. These numbers are a guideline only and will not be absolute. They may change depending on the patient’s cardiac history.
   b. Patients should receive 100mL of 25% albumin as their first bolus, unless contraindicated for some reason. If blood is necessary to meet the goal hemoglobin (mentioned below), PRBCs should be the first bolus given.
   c. Cardiac status will be monitored by trans-thoracic echocardiogram (bedside) by qualified ICU team members. The bedside echo will preferentially be used to direct volume management/diuresis when images are adequate. If necessary due to poor imaging, other methods may be used including PA Catheter or Echo, as deemed appropriate by the ICU team.
2. Goal hemoglobin of ≥9g/dl for the first 3 days post-op, with red blood cell transfusion if necessary. If the patient develops SCI, a goal hemoglobin of ≥10g/dl may be instituted as directed by the ICU and Vascular Surgery team.
3. CSF drainage for 24 hours at 10mm of Hg. If no symptoms, the drain is clamped at 24 hours after the operation and left clamped for 18-24 hours with q1 hour physical exams. If there are no symptoms of SCI during that time period, the drain is removed. If symptoms occur, the patient’s drain is left in for at least 72 hours after the onset of symptoms at 10mm Hg. The pressure will be lowered if necessary to alleviate symptoms.
4. Trans-thoracic echocardiogram will be used to optimize intravascular volume, cardiac index and oxygen delivery with intravenous inotropes as necessary. Additionally, we will keep a goal ScVO2 >70 for the first 24 hours post-operatively and lactate levels will be followed q4 hours to help guide therapy. If necessary, patients may be transfused to a hemoglobin 10 to achieve this goal.
5. If the patient’s lactate is >10 in the first 24 hours, a hemoglobin goal of 10 may be utilized, as deemed appropriate by the ICU and Vascular Surgery team. This goal of 10 will be in the first 24-48 post-operative hours and will be relaxed to a goal hemoglobin of ≥9mg/dL out to five days. After five days, if the patient is stable and without symptoms of spinal cord ischemia, transfusion goals will be relaxed to the usual ICU protocol of ≥7, unless the clinical scenario warrants blood transfusion (see existing transfusion protocol).
6. Continuation of the norepinephrine drip mentioned above.
Implementation of a bundled protocol significantly reduces risk of spinal cord ischemia after branched or fenestrated endovascular aortic repair

Salvatore T. Scali, MD, Moses Kim, MD, Paul Kubilis, MS, Robert J. Feezor, MD, Kristina A. Giles, MD, Brittney Miller, BS, Javairah Fatima, MD, Thomas S. Huber, MD, PhD, Scott A. Berceli, MD, PhD, Martin Back, MD, MS, and Adam W. Beck, MD, Gainesville, Fla; and Birmingham, Ala

ABSTRACT

Objective: Spinal cord ischemia (SCI) is a devastating complication after branched or fenestrated endovascular aortic repair (B/FEVAR) for thoracoabdominal aortic disease. The purpose of this analysis was to describe the impact of a bundled clinical care protocol designed to reduce the risk of SCI in this population of patients.

Methods: A bundled SCI prevention protocol including cerebrospinal fluid drainage, blood pressure parameters, transfusion goals, and pharmacologic adjuncts (steroids, naloxone) was initiated in May 2015. Before that date, portions of the protocol (cerebrospinal fluid drainage in particular) were used in an informal fashion in patients perceived to be at high risk. B/FEVAR cases completed from January 2012 to May 2016 were reviewed, and outcomes before (n = 223) and after (n = 70) SCI bundle application were compared. The primary end point was the incidence of SCI events. Secondary end points included length of stay, complications, and survival. High-risk patients for SCI were defined as those undergoing B/FEVAR resulting in aortic coverage equivalent to open Crawford extent I to III thoracoabdominal aortic aneurysm (TAAA) repair. Survival was estimated using Kaplan-Meier life-table analysis.

Results: Postprotocol patients were more likely to be older (75 ± 7 vs 72 ± 8 years; P = .03), to have an American Society of Anesthesiologists class 4 designation (94% vs 81%; P = .04), and to be treated for TAAA (67% vs 56%; P = .004). Postprotocol pre-emptive spinal drain use was greater in high-risk patients (100% vs 87%; P = .04) but significantly decreased in lower risk patients (suprarenal aneurysm or extent IV TAAA: 5% after protocol implementation vs 21% before protocol implementation; P = .04). Rates of any SCI before and after implementation of the bundled protocol were 13% (n = 29 of 223) and 3% (n = 2 of 70; P = .007), respectively. In comparing high-risk patients, protocol use resulted in an even more significant reduction in SCI rate (19% [28 of 144] vs 4% [2 of 50]; P = .004). Postoperative morbidity (41% vs 33%; P = .2) and 30-day mortality (5% vs 1%; P = .3) were not different between groups. However, patients treated on protocol had significantly improved 1-year survival (99% ± 1% after protocol implementation vs 90% ± 2% before protocol implementation; log-rank, P = .05).

Conclusions: Implementation of a bundled multimodal protocol may significantly reduce risk of SCI after B/FEVAR, with the greatest risk reduction occurring in the most vulnerable patients. Interestingly, reduction in SCI risk was associated with improvement in 1-year survival. (J Vasc Surg 2017;1:1-15.)
Introduction

- Level 1 evidence lacking for all strategies to ↓ SCI
  - SCI sequelae likely ↓ evidence-based threshold

“Care bundle”
A set of interventions that when used together significantly improve patient outcomes.

- Care bundles highly successful in prevention of neonatal sepsis, VAP, CLABSI
Study Design

• All patients undergoing B/FEVAR between January 2012 to May 2016 were reviewed

• May 2015 a QI project was initiated to reduce the incidence of SCI
  – Protocol elements selected based on consensus review of relevant literature
  – Bundled Protocol was prospectively applied: May 1st, 2015
Study Definitions

• **SCI:** *any new* lower extremity motor and/or sensory deficit exclusive of other causes (e.g. epidural hematoma, intracranial pathology, peripheral neuropathy or neuropraxia)

• **High-risk:**
  - Crawford Extent I-III TAAA
  - Zone 5 coverage
  - Prior aortic repair
PERI-OPERATIVE MANAGEMENT

- **Naloxone**
  - Naloxone infusion started preoperatively 1μg/kg/hr.
  - Methylprednisolone 30mg/kg x 1 prior to induction

- **Steroid Bolus**

- **Hypothermia**

- **MAP ≥ 90 mm Hg**
  - **Hgb ≥ 9 gm/dL**
  - MAP ≥ 90mmHg post-deployment
  - Hgb ≥ 10gm/dL intraoperatively
  - Mannitol 12.5gm pre- and post-stent deployment
  - Mild hypothermia (No active warming devices or warmed IVF given)

- **Lumbar Drain**
  - Lumbar drain inserted in pre-op by anesthesia team
  - Continuous drainage at 10 mm Hg (14cm H2O), centered at tragus

POST-OPERATIVE MANAGEMENT

- **Naloxone stopped 48-hours postoperatively**

- **MAP and Hgb goals relaxed**
  - MAP goal is relaxed before lumbar drain removal
  - Hgb ≥ 9gm/dL
  - BP medications are generally held until first clinic visit
  - Chronic Beta-blockers continued throughout

- **Drain Removed**
  - Drain is clamped 24 hours post-procedure
  - Removal occurs 12-24 hours after clamping if no SCI occurs
  - Stopped after patient transferred to floor

- **Neuro-Checks**
  - Neuro exam performed prior to leaving OR
  - Q1h neurochecks continue in ICU
Fig 2. Different interventions performed when a patient develops evidence of spinal cord ischemia (SCI) after branched or fenestrated endovascular aortic repair (B/FEVAR). Several maneuvers are employed, including lowering the cerebrospinal fluid (CSF) drain to minimize the impact of cord edema and to enhance spinal cord perfusion as well as ensuring adequate preload and cardiac output with lower transfusion triggers. All of these efforts are made to maximize oxygen carrying and delivery capacity to mitigate SCI. CI, Cardiac index; HCT, hematocrit; Hgb, hemoglobin; MAP, mean arterial pressure.
Pay Attention to Details!

Stage operations when possible

Expeditious surgery is important
    Remove femoral sheaths early when possible

Avoid hypotension
    Slow leaks through the sheaths can be large volume
    Protamine
    Sheath removal
**Presentation & Procedure Details**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre-protocol (N = 223)</th>
<th>Post-protocol (N = 70)</th>
<th>P-value</th>
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<tbody>
<tr>
<td><strong>Indication</strong></td>
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<td></td>
<td></td>
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<tr>
<td>Thoracoabdominal aneurysm</td>
<td>56%</td>
<td>67%</td>
<td>.004</td>
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<td>Suprarenal aneurysm</td>
<td>34%</td>
<td>27%</td>
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<td>Dissection</td>
<td>2%</td>
<td>3%</td>
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<tr>
<td>Post-surgical</td>
<td>5%</td>
<td>2%</td>
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<td>Other</td>
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<td>Maximal aortic diameter, mm</td>
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<td>66±11</td>
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<td>Elective</td>
<td>78%</td>
<td>81%</td>
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<td>17%</td>
<td>17%</td>
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<td>.4</td>
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<td>Pre-emptive spinal drain, any indication</td>
<td>54%</td>
<td>50%</td>
<td>.6</td>
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<td>Pre-emptive spinal drain Extent I-II TAAA</td>
<td>87%</td>
<td>100%</td>
<td>.04</td>
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<td>Pre-emptive spinal drain, Suprarenal&gt;Type IV aneurysm</td>
<td>21%</td>
<td>4%</td>
<td>.03</td>
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<tr>
<td>Number of fenestrations</td>
<td>3.3</td>
<td>3.5</td>
<td>.08</td>
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<td>Adjunct procedure</td>
<td>25%</td>
<td>10%</td>
<td>.004</td>
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<td>Contrast exposure, mL</td>
<td>85±62</td>
<td>53±22</td>
<td>.0008</td>
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<td>Estimated blood loss, mL</td>
<td>300 [200, 400]</td>
<td>200 [150, 500]</td>
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<td>Intraoperative transfusion</td>
<td>0.6 +/- 1.2u</td>
<td>0.8 +/- 1.2</td>
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<td>Fluoroscopy time, min</td>
<td>77±40</td>
<td>64±35</td>
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<td>Procedure time, hours</td>
<td>4.6±2</td>
<td>4±2</td>
<td>.07</td>
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<td>Post-protocol (N = 70)</td>
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<td><strong>Outcomes</strong></td>
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<td>Length of stay, days</td>
<td>5[4, 9]</td>
<td>6.5[4, 10]</td>
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<tr>
<td>30-day mortality</td>
<td>5%</td>
<td>1%</td>
<td>.3</td>
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<tr>
<td>Any complication</td>
<td>41%</td>
<td>33%</td>
<td>.2</td>
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<tr>
<td>Intra-Operative Complication</td>
<td>8%</td>
<td>0</td>
<td>.01</td>
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<tr>
<td>Post-Operative Complications</td>
<td>41%</td>
<td>32%</td>
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<td>Neurological</td>
<td>17%</td>
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<td><strong>Any SCI</strong></td>
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<td>3%</td>
<td>.007</td>
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<tr>
<td>Cardiac</td>
<td>9%</td>
<td>6%</td>
<td>.4</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>11%</td>
<td>10%</td>
<td>.8</td>
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<tr>
<td>Renal</td>
<td>8%</td>
<td>4%</td>
<td>.4</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>5%</td>
<td>3%</td>
<td>.7</td>
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<tr>
<td>Wound</td>
<td>5%</td>
<td>6%</td>
<td>1</td>
</tr>
<tr>
<td><strong>Bleeding</strong></td>
<td>8%</td>
<td>6%</td>
<td>.6</td>
</tr>
<tr>
<td>Blood Tx: Total (units)</td>
<td>1.7±3.7</td>
<td>2+/−3.8</td>
<td>.5</td>
</tr>
<tr>
<td>Blood Tx: High risk (units)</td>
<td>1.5+/−3.3</td>
<td>0.7+/−1</td>
<td>.1</td>
</tr>
<tr>
<td>Ischemia</td>
<td>4%</td>
<td>0</td>
<td>.1</td>
</tr>
<tr>
<td>Other</td>
<td>9%</td>
<td>7%</td>
<td>.7</td>
</tr>
</tbody>
</table>
SCI Rate Before and After Protocol

All Patients

<table>
<thead>
<tr>
<th></th>
<th>Pre</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spinal Cord Ischemia Rate (%)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| All Patients  |     |      | * p = .007
| High Risk Patients |     |      |
One-Year Survival for B/FEVAR Patients Before and After Protocol

Log rank $P = 0.053$
Impact of SCI on One-Year Mortality

One-Year Mortality Rate (%)

- Persistent SCI: 50% (N = 10)
- Improved SCI: 9% (N = 11)
- No SCI: 9% (N = 194)
- Resolved SCI: 0% (N = 8)

PRE-PROTOCOL

POST-PROTOCOL

N = 68
Summary/Conclusions

• Spinal Cord Ischemia can be a devastating, life altering complication, and prevention, detection and treatment are key

• Protocol driven care can reduce spinal cord ischemia in high risk patients after endovascular aortic repair

• Integration of a Spinal Cord Ischemia prevention protocol should be central to any TAAA program
Thank You