TEVAR: Preventing Spinal Ischemia

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Medstar Heart and Vascular Institute
Incidence of Spinal Cord Ischemia with Endovascular Repair

• 3-5% incidence (Hnath et al. JVS 48(4) - 2008)

• 13% with no or inconsistent use of adjuncts

• Approach rates in open surgery in some series

• Paraplegia associated with death by 1 yr
Mechanism of Spinal Cord Ischemia

• **Arterial Insufficiency**
  - Robust circulation in high thoracic region including anterior spinal artery
  - Lower thoracic cord watershed region with less robust circulatory pathways—main supply is Artery of Adamkiewicz (off of intercostals) with lumbars and pelvic circulation contributing

• **Hemodynamics**
• **Cord swelling**
• **Embolization**
Considerations - Procedural

• Procedure
  – Emergent
  – Sustained hypotension
  – Blood loss
  – Extent of coverage
Considerations - Anatomy

- Anatomy
  - Prior abdominal repair
  - Length of coverage needed
  - Prior SCI
  - Risk of embolization
  - Coverage of contributing vascular beds
    - Left Subclavian
    - Internal Iliacs (already occluded)
    - Intercostals
    - Lumbars
Incidence and Determinants of Spinal Cord Ischaemia in Stent-graft Repair of the Thoracic Aorta

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Spinal cord ischemia after elective endovascular stent-graft repair of the thoracic aorta

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Considerations - Adjuncts

- Neuro monitoring
- Blood pressure Augmentation
- Lumbar Drain
- Steroids
- Mannitol
- Moderate Hypothermia
- Naloxone
Strategies to Manage Paraplegia Risk After Endovascular Stent Repair of Descending Thoracic Aortic Aneurysms

Albert T. Cheung, MD, Alberto Pochettino, MD, Michael L. McGarvey, MD, Jehangir J. Appoo, MD, Ronald M. Fairman, MD, Jeffrey P. Carpenter, MD, William G. Moser, RN, Edward Y. Woo, MD, and Joseph E. Bavaria, MD

Departments of Anesthesia, Neurology, and Surgery, University of Pennsylvania, Philadelphia, Pennsylvania

**Background.** Paraplegia is a recognized complication after endovascular stent repair of descending thoracic aortic aneurysms. A management algorithm employing neurologic assessment, somatosensory evoked potential monitoring, arterial pressure augmentation, and cerebrospinal fluid drainage evolved to decrease the risk of postoperative paraplegia.

**Methods.** Patients in thoracic aortic aneurysm stent trials from 1990 to 2004 were analyzed for paraplegic complications. Lower extremity strength was assessed after anesthesia and in the intensive care unit. A loss of lower extremity somatosensory evoked potential or lower extremity strength was treated emergently to maintain a mean arterial pressure 90 mmHg or greater and a cerebrospinal fluid pressure 10 mm Hg or less.

**Results.** Seventy-five patients (male = 49, female = 26, age = 75 ± 7.4 years) had descending thoracic aortic aneurysms repaired with endovascular stenting. Lumbar cerebrospinal fluid drainage (n = 23) and somatosensory evoked potential monitoring (n = 15) were performed selectively in patients with significant aneurysm extent or with prior abdominal aortic aneurysm repair (n = 17).

Spinal cord ischemia occurred in 5 patients (6.6%); two had lower extremity somatosensory evoked potential loss after stent deployment and 4 developed delayed-onset paraplegia. Two had full recovery in response to arterial pressure augmentation alone. Two had full recovery and one had near-complete recovery in response to arterial pressure augmentation and cerebrospinal fluid drainage. Spinal cord ischemia was associated with retroperitoneal bleed (n = 1), prior abdominal aortic aneurysm repair (n = 2), iliac artery injury (n = 1), and atheroembolism (n = 1).

**Conclusions.** Early detection and intervention to augment spinal cord perfusion pressure was effective for decreasing the magnitude of injury or preventing permanent paraplegia from spinal cord ischemia after endovascular stent repair of descending thoracic aortic aneurysm. Routine somatosensory evoked potential monitoring, serial neurologic assessment, arterial pressure augmentation, and cerebrospinal fluid drainage may benefit patients at risk for paraplegia.

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Routine somatosensory evoked potential monitoring, serial neurologic assessment, arterial pressure augmentation, and cerebrospinal fluid drainage may benefit patients at risk for paraplegia.

Intraoperative Monitoring

- Somatosensory Evoked Potentials
- Motor Evoked Potentials
- SSEP’s
  - Easier to use—not affected by neuromuscular blockade
  - Longer delay between detection and incidence of SCI
  - Increased false negatives
in 99% of cases (102/103). Loss of somatosensory evoked potential was encountered in 26% of cases (27/102), and return of somatosensory evoked potentials occurred in all cases after intraoperative maneuvers. Motor evoked potentials achieved adequate readings in 96% of cases (99/103). Loss of motor evoked potential was encountered in 50% of cases (50/99), and return of motor evoked potentials occurred in all but 1 case (95%). This patient awoke with an immediate spinal neurologic deficit.

<table>
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<td>Increasing the mean central pressure to &gt; 80 mm Hg</td>
</tr>
<tr>
<td>Increasing distal aortic pressure to &gt; 60 mm Hg</td>
</tr>
<tr>
<td>Decreasing cerebrospinal fluid pressure by free gravity drainage</td>
</tr>
<tr>
<td>Increasing hemoglobin levels by transfusion</td>
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<tr>
<td><strong>Surgical maneuvers:</strong></td>
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<td>Immediate reimplantation of T8 to T12</td>
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<td>Reimplant other patent ICA, T4–T7, L1, L2</td>
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*DAP, Distal aortic perfusion; ICA, internal carotid artery.*
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### Conclusion: Neuromonitoring useful

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| Surgical maneuvers:                                         |
| Immediate reimplantation of T8 to T12                      |
| Establish DAP                                              |
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Perioperative Management to Improve Neurologic Outcome in Thoracic or Thoracoabdominal Aortic Stent-Grafting

Ernst Weigang, MD, Marc Hartert, Michael P. Siegenthaler, MD, Nicholas A. Beckmann, Ronen Sircar, MD, Gábor Szabò, MD, Christian D. Etz, MD, Maximilian Luehr, Patrick von Samson, MD, and Friedhelm Beyersdorf, MD

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**Background.** Thoracic or thoracoabdominal aortic stent-graft repair has shown a reduction in morbidity and mortality rates due to the procedure's advantages (no aortic cross-clamping, continuous distal aortic perfusion, no reperfusion injury). However, 3% to 12% of the patients are at risk of spinal cord ischemia. We investigated spinal cord protective measures with evoked potentials, cerebrospinal fluid drainage, and prevention of hypotension to minimize postoperative neurologic deficit.

**Methods.** Between November 2000 and July 2005, vital parameters and spinal cord function were monitored, including cerebrospinal fluid pressure and transcranial motor-evoked and somatosensory-evoked potentials in 36 stent-graft procedures (31 patients) on the thoracic or thoracoabdominal aorta.

**Results.** Stent-graft placement was technically successful in all patients. We achieved a survival rate of 100% without neurologic deficit after fast-track extubation. Eleven of 31 patients exhibited changes in evoked potentials during stent-graft deployment. In 12 of 31 patients (including the 11 with evoked potential alterations), cerebrospinal fluid pressure exceeded 15 mm Hg. Cerebrospinal fluid drainage and vital parameter adjustment were executed in those instances. We observed intraoperative evoked potential total recovery in 10 of 11 patients after these interventions.

**Conclusions.** Interventions to improve spinal cord perfusion led to total recovery of spinal function in most patients (10/11). Therefore, spinal cord protective measures with motor- and somatosensory-evoked potential monitoring, cerebrospinal fluid drainage, and prevention of hypotension can reduce the incidence of spinal cord ischemia and improve the neurologic outcome of patients undergoing endovascular thoracic or thoracoabdominal aortic repair.

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(Ann Thorac Surg 2006;82:1679–87)
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Intraoperative Monitoring

- All cases should be monitored
- Changes in waveforms should lead to intervention
- No flattening or improvement in waveforms does not always equate with spinal cord preservation or recovery
- Flattening of waveforms almost always sign of SCI
Cerebrospinal Fluid Drainage

• Lumbar drain
  – Needs to be in correct area
  – “Bloody tap”
    • Case cancellation
    • ?bleed
  – Postpull leak—may need blood patch
  – Complications of placement or removal uncommon
CSF Drain

- Drain to 10mm Hg or for symptoms
- Protocol
  - Symptomatic-continue drainage
  - Asymptomatic
    - Cap after 24hrs or after “critical period”
    - Cap for 12hrs-if no sx, remove
Cerebrospinal fluid drainage reduces paraplegia after thoracoabdominal aortic aneurysm repair: Results of a randomized clinical trial

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Objective: Despite the use of various strategies for the prevention of spinal cord ischemia, paraplegia and paraparesis continue to occur after thoracoabdominal aortic aneurysm (TAAA) repair. Although cerebrospinal fluid drainage (CSFD) is often used as an adjunct for spinal cord protection, its benefit remains unproven. The purpose of this randomized clinical trial was to evaluate the impact of CSFD on the incidence of spinal cord injury after extensive TAAA repair.

Methods: After randomization, 145 patients underwent extent I or II TAAA repairs with a consistent strategy of moderate heparinization, permissive mild hypothermia, left heart bypass, and reattachment of patent critical intercostal arteries. The repairs were performed with CSFD (n = 76) or without CSFD (n = 69). In the former group, CSFD was initiated during the operation and continued for 48 hours after surgery. The target CSF pressure was 10 mm Hg or less.

Results: The two groups had similar risk factors for paraplegia. Aortic clamp time, left heart bypass time, and number of reattached intercostal arteries were also similar in both groups. Thirty-day mortality rates were 5.3% (four patients) and 2.9% (two patients) for CSFD and control groups, respectively (P = .68). Nine patients (13.0%) in the control group had paraplegia or paraparesis develop. In contrast, only two patients in the CSFD group (2.6%) had deficits develop (P = .03). No patients with CSFD had immediate paraplegia. Overall, CSFD resulted in an 80% reduction in the relative risk of postoperative deficits.

Conclusion: Perioperative CSFD reduces the rate of paraplegia after repair of extent I and II TAAAs. (J Vasc Surg 2002;35:631-9.)
Fig 1. Diagram shows trial profile, including participant flow after randomization, patient availability for intention-to-treat analysis, and withdrawals for secondary efficacy analysis. TAAA, Thoracoabdominal aortic aneurysm; CSFD, cerebrospinal fluid drainage; LHB, left heart bypass; DTA, descending thoracic aortic aneurysm.
Table IV. Postoperative lower extremity neurologic deficits after repair of extensive thoracoabdominal aortic aneurysms

<table>
<thead>
<tr>
<th>Neurologic injury</th>
<th>CSFD (n = 76)</th>
<th>Control (n = 69)</th>
<th>P value</th>
<th>Risk reduction</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Absolute</td>
</tr>
<tr>
<td>All lower extremity</td>
<td>2 (2.6%)</td>
<td>9 (13.0%)</td>
<td>.03</td>
<td>10.4%</td>
</tr>
<tr>
<td>neurologic deficits</td>
<td>1 (1.3%)</td>
<td>7 (10.1%)</td>
<td>.03</td>
<td>8.8%</td>
</tr>
<tr>
<td>Paraplegia</td>
<td>0</td>
<td>6 (8.7%)</td>
<td>.01</td>
<td>8.7%</td>
</tr>
<tr>
<td>Paraparesis</td>
<td>1 (1.3%)</td>
<td>1 (1.4%)</td>
<td>1.0</td>
<td></td>
</tr>
<tr>
<td>Delayed neurologic</td>
<td>1 (1.3%)</td>
<td>2 (2.9%)</td>
<td>.60</td>
<td></td>
</tr>
<tr>
<td>deficits</td>
<td>1 (1.3%)</td>
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<td>1.0</td>
<td></td>
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<tr>
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<td>0</td>
<td>1 (1.4%)</td>
<td>.48</td>
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CSFD, Cerebrospinal fluid drainage.

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Pathophysiology of Delayed-Onset Paraplegia

Spinal Cord at Risk

Spinal Cord Infarction
T2-weighted MRI Thoracolumbar Spine.

- Hypoperfusion
  - Spinal Cord Ischemia (paraplegia/paraparesis)
    - Neurogenic Shock (autonomic dysfunction)
      - Hypotension
        - Spinal Cord Infarction (permanent paraplegia)
Hemodynamics

- Hypotension associated with SCI
  - Avoidance of significant blood pressure swings
- Augmentation of Mean Arterial Pressure
  - CPP = MAP - ISP
- Central access
  - Measure central filling pressures
  - Rapid infusion
  - Pressors
- Invasive arterial lines-upper and lower
Hemodynamics

• Augmentation of MAP
  – First line of intervention if symptoms or changes in neuromonitoring
  – Volume expansion
  – Vasopressors
  – Increase MAP to 90-110
Postoperative Management

• Immediate neuro exam as soon as patient awake
  – Nursing staff education on exam
• Protocol for MAP augmentation
• Ability to place or replace CSF drain
• Hemodynamic control
• Prophylactic moves
  – Antiarrhythmics
  – Respiratory care
Conclusions

• SCI devastating complication
• Largely avoidable with consistent use of preoperative planning and use of adjuncts
• When it occurs it is largely treatable
• Protocols must be in place and team educated to rapidly recognize and treat