SPINAL CORD PROTECTION DURING THORACOABDOMINAL AORTIC ANEURYSM REPAIR

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1. Introduction

- Classification of TAAA
- Problem definition
- Types of repair
- Morbidity and mortality
Crawford Classification

Figure 1 The Crawford classification of thoracoabdominal aortic aneurysms classifies aneurysms involving the descending thoracic and abdominal aorta according to anatomic extent.

Incidence of spinal cord ischemia according to Crawford extent of aneurysm:

<table>
<thead>
<tr>
<th>Extent</th>
<th>Endovascular repair</th>
<th>Open surgical repair</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>10%</td>
<td>14%</td>
</tr>
<tr>
<td>II</td>
<td>19%</td>
<td>22%</td>
</tr>
<tr>
<td>III</td>
<td>5%</td>
<td>10%</td>
</tr>
<tr>
<td>IV</td>
<td>3%</td>
<td>2%</td>
</tr>
</tbody>
</table>

Aortic aneurysms isolated to the descending thoracic aorta between the left subclavian artery and the diaphragm are designated as descending thoracic aortic aneurysms (DTAs or TAAs). Aneurysm extent and type of surgical repair influence the risk of spinal cord ischemia.

Artery of Adamkiewicz = large segmental artery

→ origin between T5-L2 (75% between T9-T12)
Problem definition

- During aortic repair:
  - aortic cross-clamping or circulatory arrest
    - temporary or permanent interruption of arterial collaterals (blood supply)
  - oxygen debt
  - risk of ischemia/infarction
  - risk of paraparesis/paraplegia
Risk Factors for ischemia

- Longer extent of aneurysm (greatest risk in Crawford type II, least in type IV)
- Perioperative hypotension
- Emergency operation (16.7 vs 3.9%)
- Open operative repair
- Acute aortic rupture/dissection
- Longer duration of aortic cross-clamp
- Failure to re-implant segmental arteries
- Prior distal aortic surgery
- Severe peripheral vascular disease
- Anemia (impairing oxygen supply)
- Systemic vasodilatation with vascular steal (for control of hypertension associated with aortic clamping)
Goal = augmenting spinal cord perfusion pressure:
- Increasing mean arterial pressure
- Lumbar cerebrospinal fluid drainage
- Reattachment of segmental arteries

Early detection is very important to prevent permanent paraplegia:
- Intraoperative monitoring in the anesthetised patient
- Postoperative neurological examination in the awake patient
Types of repair

- Open: replace diseased segments with a prosthetic vascular interposition graft

  vs

- Endovascular (=TEVAR): exclude diseased segments with an endovascular stent graft (fenestrated or branched) -> beneficial effects on the spinal collateral arterial network

→ Risk of ischemia greatest in open repair: 8-28% vs 4-7%
Morbidity and mortality

- Incidence of paraplegia after TAAA-repair: 0.5-40%
  - significant medical, social and financial aspects
  - limited mobility, assistance in activities of daily living
2. Pathophysiology of spinal cord injury

- Anatomy of spinal cord blood supply
- Autoregulation
- Spinal blood flow after Thoracic Aortic Occlusion (Aortic cross clamping)
Anatomy of spinal cord blood supply
Anatomy of spinal cord blood supply

- The collateral network concept
FIGURE 2. Porcine vasculature demonstrated by radiograph following latex barium emulsion infusion. Top, Entire pig. Bottom left, Eviscerated trunk of a normal pig. Bottom right, Eviscerated trunk of a pig 5 days after ligation of all segmental vessels. Note neovascularization connecting distal segmental vessels with the sacral vessels and the subclavian vessels via the internal thoracic arteries, as well as the complete backfilling of the segmental vessels to the ligation sites adjacent to the aorta. Reprinted with permission. 

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Autoregulation

- Spinal cord blood flow is constant
  - Between 10 - 50 mmHg CO₂
  - Between 50 - 135 mmHg MABP

- Mechanism:
  - Sympathetic ganglia
  - Sensory control center caudal to the medulla
  - Further research needed
Spinal blood flow after thoracic aortic occlusion (aortic cross clamping)

- Spinal cord perfusion pressure (SCPP) = MABP – CSF pressure
  - > 50 – 60 mmHg to protect spinal cord from ischemia
  - Normal CSF pressure = 13 – 15 mmHg

- Temporary aortic cross-clamping decreases SCBF and distal organ perfusion
  - Distal hypotension
  - Proximal hypertension
  - Increase in left ventricle afterload

- AoX ➔ Proximal hypertension + intracranial pressure ↑
  ➔ Autoregulation: CSF pressure ↑ ➔ SCPP ↓

- Spinal cord injury ➔ hypotension
  - Interrupting sympathetic fibers
  - Direct myocardial dysfunction

- Release of aortic cross-clamping:
  - CSFP remains elevated for 5 minutes
  - CSFP returns to normal after 25 minutes
  - Hyperemia is observed
Anatomy of spinal cord blood supply

- The collateral network concept: implications
  - Peroperatively provide adequate blood flow
    - Arterial pressure in the high physiologic range
    - Reduction of resistance to flow
      - Drainage of cerebrospinal fluid (CSF) to maintain an intrathecal pressure of 10 cmH₂O or less
      - Low central venous pressure
    - Importance of hypogastric arteries
      - Distal aortic perfusion
  - Adequate spinal cord blood flow in the early postoperative period
    - Maximization and stability of hemodynamics
    - Continued CSF drainage
    - Monitoring of spinal cord functions
3. Prevention of spinal cord injury

- Minimize spinal cord ischemia time
- Increase tolerance to ischemia
- Augmentation of spinal cord perfusion
- Early detection of spinal cord ischemia
Minimize spinal cord ischemia time

- Decrease duration of surgery
- Preservation of subclavian artery flow
- Distal aortic perfusion
  - Passive shunt (Gott shunt)
  - Left heart bypass = Atrial-femoral bypass
- Thoracic endovascular aortic repair
- Staged repair
Staged repair

- Principle based on “dynamic collateral vascular network”
  - Endovascular repair in different stages
  - Dividing extensive aneurysm repair into multiple steps may mitigate the impact of diminished blood flow to the collateral network
  - Allowing new blood vessels to grow
  - Reduce chance of ischemia
  - Less/no neurological deficit postoperatively
Left heart bypass
Increase tolerance to ischemia

- Deliberate mild systemic hypothermia
- Deep hypothermic circulatory arrest
- Selective spinal cord hypothermia by epidural cooling
- Pharmacologic neuroprotection
- CSF drainage
Deliberate mild systemic hypothermia

= 32-34°C

* Hypothermia is the only intervention in humans that has been proven consistently to be effective for protecting the CNS
  → decreased metabolic demands
  → cell membrane stabilization
  → attenuating the inflammatory and excitotoxic responses to ischemia during reperfusion

CAVE: arrhythmias, coagulation abnormalities

* Allowing body core temperature to decrease after induction
  → Re-warming after reperfusion: gradually, avoid systemic hyperthermia
Deep hypothermic circulatory arrest

= 10-18°C → requires CPB

* For TAAA that extends into the aortic branch requiring temporary interruption of cerebral blood flow

* Electro cortical silence by electroencephalography

* CAVE: Risks associated with DHCA:
  → stroke caused by cerebral atheroembolism from retrograde blood flow during CPB
  → postoperative encephalopathy
  → cerebral hyperthermia during re-warming
Selective spinal cord hypothermia by epidural cooling

= 26°C

→ Infusion of 4°C saline into the epidural space through an epidural catheter placed in the Th11-Th12 vertebral interspace

+ Second catheter into the subarachnoidal space at the L3-L4 level to measure CSF temperature and allow CSF drainage to control CSF pressure

→ CAVE risk of excessive CSF pressure and inability to monitor spinal cord function!

→ Only limited clinical experience
Pharmacologic neuroprotection

Should increase neural tolerance to ischemia

- Methylprednisolone 1g IV (role is controversial)
- Mannitol 12.5-25g IV
- Magnesium 1-2g IV
- Lidocaine 100-20mg IV
- Thiopental 0.5-1.5g IV
- Naloxone 1mcg/kg/hr IV
- Papaverine 30mg IT

Promising roles for an array of agents (laboratory experiments):
- Allopurinol, adenosine, activated prot C, barbiturates, carbamazepine, prostaglandines, volatile anesthetics

Effectiveness has not been proven, ideal candidate has not yet been found!
Pharmacologic neuroprotection

Recent studies:

→ macrophages in the spinal cord (the microglia) are thought to play a central role in the development of neural death during spinal ischemia → R/ Macrolide AB

→ acetazolamide, drug used to counteract mountain sickness: reduces CSF production → lowers ICP
CAVE: responders vs non-responders, unwanted side effects
Augmentation of spinal cord perfusion

- Deliberate hypertension
  - MAP 80-100 mmHg
  - CVD < CSF pressure
  - For at least 24h-48h postoperatively

- Lumbar cerebrospinal fluid (CSF) drainage
  - CSF pressure < 10 -15mmHg
  - Complications!
    - CSF drainage < 25 ml/hr

- Re-implantation of intercostals and lumbar segmental arteries
Early detection of spinal cord ischemia

= monitoring function of the spinal cord

- Intraoperative MEP
- Intraoperative SSEP
- Serial postoperative neurologic examination
- Biochemic measurements
Intraoperative MEPs

= Anterior horn of the spinal cord

→ Elicited through transcortical electrical stimulation
→ Myogenic potentials produced in extremity muscle groups by electrical stimulation through the scalp overlaying the motor cortex

CAVE! NMB and other general anesthetic agents (such as volatile anesthetics) influence amplitude
→ TIVA and low dose or no NMB
Intraoperative SSEPs

= Posterior horn of the spinal cord

→ placing stimulating electrodes on the skin adjacent to peripheral nerves in the arms or legs
→ electrical stimulation of the peripheral nerves generates AP’s that can be measured from recording electrodes over the lumbar/brachial plexus, spine, brainstem, thalamus and cerebral cortex

LIMITATION: anterior spinal cord infarction causes a selective motor deficit with intact sensation

ADVANTAGE: easier to interpret compared to MEP’s
Serial postoperative neurologic examination

- Start early!
- CAVE local/epidural analgesia: difficult postop neurologic examination
- Strength in the proximal and distal muscle groups of the extremities and the presence/absence of sensation
- Diagnosis should trigger a protocol for spinal cord rescue, including permissive systemic hypertension with or without CSF drainage
Biochemic measurements

Some studies: monitoring MEP en SSEP alone is inaccurate to predict spinal cord ischemia

→ monitoring the biochemical parameters of the CSF:
  CSF lactate levels, S-100 protein and neuron-specific enolase (NSE) concentrations
  → specific for neuroglia and neuronal injury
  → study the reversible aerobic/anaerobic metabolic changes

→ Significant CSF lactate increase in patients with spinal cord injury + remained elevated throughout the whole operation
  → Significant reduction in CSF lactate concentrations when distal femoral bypass and/or hypothermia were used
Anesthetic plan for neuroprotection during thoracoabdominal aortic aneurysm repair

- **Induction**
  - Full ASA monitoring
  - Intravenous, arterial access (arterial blood gas sample, ACT)
  - Intravenous induction after adequate preoxygenation
  - MEPs → lower dose inhalational anesthetics, short-/intermediate acting muscle relaxants

- **Airway**
  - Left sided double-lumen tube (DLT)
  - Cave compromised airway (endobronchial blocker/right-sided DLT)
  - Normal ventilation (PaCO2 10-50 mmHg) and oxygenation

- **IV access**
  - Large bore peripheral
  - Central venous access
  - Pulmonary artery catheter (optional)

- **Spinal catheter for cerebrospinal fluid monitoring**
Anesthetic plan for neuroprotection during thoracoabdominal aortic aneurysm repair

- **Ancillary measures**
  - MEP / SSEP
  - TEE (optional)
  - Nasopharyngeal temperature
  - Prophylactic intravenous antibiotics
  - Cell-saver, rapid infusion device

- **Minimize spinal cord ischemia time**
  - Decrease duration of surgery
  - Preservation of subclavian artery flow
  - Distal aortic perfusion (passive shunt (Gott shunt), left heart bypass = atrial-femoral bypass)
  - Thoracic endovascular aortic repair if possible
  - Staged repair

- **Increase tolerance to ischemia**
  - Hypothermia: deliberate mild systemic hypothermia (= 32-34°C) or deep hypothermic circulatory arrest (= 10-18°C: CPB)
  - Selective spinal cord hypothermia by epidural cooling
  - Pharmacologic neuroprotection eg methylprednisolone, mannitol, ,,,
  - CSF drainage (cfr. Infra)
Anesthetic plan for neuroprotection during thoracoabdominal aortic aneurysm repair

- **Augmentation of spinal cord perfusion**
  - Deliberate hypertension
    - MAP 80-100 mmHg
    - CVD < CSF pressure
    - For at least 24h-48h postoperatively
  - Lumbar cerebrospinal fluid (CSF) drainage
    - CSF pressure < 10 - 15 mmHg
    - CSF drainage < 25 ml/hr to avoid complications (intracranial/subdural hematoma)
  - Re-implantation of intercostals and lumbar segmental arteries

- **Early detection of spinal cord ischemia**
  - Intraoperative MEP / SSEP
  - Serial postoperative neurologic examination
  - (Biochemic measurements in the CSF)