Perioperative Management of Acute Stanford Type-A Aortic Dissection

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I have no disclosures.
Basic Information

• Aortic dissection involving the ascending aorta is a lethal condition, with mortality approaching 60% if surgical intervention is not performed early.

• Identification and resection of the primary intimal tear with re-approximation of the intima and adventitia remains the surgical principle of repair.

• The surgical repair of an ascending aortic dissection presents multiple challenges to the Surgeon, Perfusionist, Anesthesiologist and the OR Team.

• Preoperative discussion focuses on assessment, hemodynamic management, and risk stratification.

• The intraoperative section includes an overview of anesthetic management, as well as surgical overview that may influence anesthesia and perfusion management.
Stanford Classification of Aortic Dissection

Type A involves the ascending aorta and may progress to involve the arch and thoracoabdominal aorta.

Type B involves the descending thoracic or thoracoabdominal aorta distal to the left subclavian artery without involvement of ascending aorta.
General Principles of Managements

• Identification and resection of the primary intimal tear with re-approximation of the intima and adventitia remains the surgical principle of repair.

• The aim of surgical intervention should also include the management of the potentially fatal complications associated with it. These are
  • intrapericardial rupture and tamponade,
  • malperfusion phenomena (coronary, neurological, and visceral),
  • acute aortic valve regurgitation.
Etiologic classification —

- Sporadic – Aortic dissection that has a sporadic (degenerative) etiology is not associated with any known genetically mediated syndromes.

- Genetically mediated – Patients with genetically mediated syndromic thoracic aneurysm/dissection (TAAD) include those associated with Marfan syndrome, vascular Ehlers-Danlos syndrome and Turner syndrome. Non syndromic genetically mediated disorders are most commonly familial TAAD.

- Traumatic – Traumatic aortic dissection can be related to a blunt injury mechanism or iatrogenic in nature related to instrumentation (eg, catheterization, dissection following aortic repair).
Clinical Features

- Acute onset of severe, sharp, or knife-like pain in the anterior chest, with radiation to the neck, back, or abdomen. Pain may be migratory.
- Palpate carotid, subclavian, and femoral pulses; note any significant differences between sides. Obtain blood pressure in both arms.
- Auscultate for diastolic cardiac murmur of aortic regurgitation; assess for tamponade (muffled heart sounds, jugular venous distention, pulsus paradoxus).
- Evaluate for signs of ischemic stroke, spinal cord ischemia, ischemic neuropathy, hypoxic encephalopathy.
Findings suggesting involvement of the dissection include:

• Back pain, anterior chest pain
• Myocardial infarction
• Hemodynamic instability
• Diastolic Cardiac murmur
• Aortic insufficiency or heart failure
• Tamponade
• Syncope or Stroke
• Absent Pulses
• Acute Paraplegia
• Acute Paraplegia
• Abdominal Pain
Mechanisms of aortic regurgitation in ascending aortic dissection may include:
(A) Dilation of the aortic root
(B) Asymmetric cusp coaptation
(C) Disruption of aortic annulus
Malperfusion in aortic dissection
Static and Dynamic

Figure 3. UpToDate
Intraoperative Care

- On arrival in the operating room, have the whole team ready.
- Be prepared for emergency cannulation and CPB.
- A right radial arterial pressure monitoring catheter is placed prior to induction of anesthesia.
- After induction of anesthesia, central venous lines and a left radial or lower extremity pressure monitoring catheters, are placed.
- A transesophageal echocardiogram is obtained to confirm the diagnosis, as well as to delineate the anatomy and size of the aortic root and sinotubular junction, aortic insufficiency, and if possible, location of the tear. That provides critical information regarding whether the aortic valve or root will need replacement.
Traditional surgical intervention

- The current best practice guidelines for type A dissection repair involve ascending aortic replacement.
- Open distal anastomosis (hemiarch) under circulatory arrest.
- Varying degrees of hypothermia and selection of cerebral protection techniques including antegrade or retrograde cerebral perfusion, or deep hypothermia alone.
- Prolonged periods of circulatory arrest leads to a high incidence of neurologic complications following this operation.
Figure 4. Annals of Cardiothoracic Surgery
Figure 5. Annals of Cardiothoracic Surgery
Deep Hypothermic Circulatory Arrest (DHCA)

• Hypothermia provides neuroprotection during periods of circulatory arrest.

  1) reducing the metabolic requirements (thus raising the ischemic threshold) for both neuronal activity and cellular integrity.

  2) suppressing many of the complex excitotoxic, inflammatory, immune, and genetic pathways that result in neuronal injury and death when ischemia does occur.

  3) attenuating the secondary insult which occurs during reperfusion following ischemia
<table>
<thead>
<tr>
<th>Temperature (°C)</th>
<th>Cerebral metabolic rate (% of baseline)</th>
<th>Calculated safe duration of HCA (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>37</td>
<td>100</td>
<td>5</td>
</tr>
<tr>
<td>30</td>
<td>56 [52–60]</td>
<td>9 [8–10]</td>
</tr>
</tbody>
</table>

Data are means with 95% confidence intervals (CI).
<table>
<thead>
<tr>
<th>Mode of Monitoring</th>
<th>Advantages</th>
<th>Limitations</th>
</tr>
</thead>
</table>
| EEG                | • Sensitive indicator of post-operative cognitive function  
                    • Can detect “silent EEG” as trigger to begin DHCA  
                    • Can detect epileptiform activity | • Poor specificity  
                    • Cannot detect activity in sub-cortical/deep regions  
                    • Requires dedicated technician  
                    • Patient specific responses to hypothermia – isoelectric EEG at different temperatures in different patients.  
                    • Affected by ambient electromagnetic (EM) radiation, anaesthetic drugs |
| SSEP               | • Easy to assess and interpret  
                    • Highly sensitive and specific in predicting early postoperative neurological events | • Not significantly affected by anaesthetic drugs  
                    • Not significantly affected by EM interference. |
| SjO₂               | • Infers changes in cerebral metabolism. A low SjO₂ reflects high O₂ uptake indicating the potential for neurological damage  
                    • Can be used to measure cerebral temperature directly | • Only reflects “global” cerebral blood flow (doesn’t reflect regional ischaemia)  
                    • Not directly correlated with EEG findings (Low SjO₂ may be seen in EEG silence) |
| NIRS               | • Easy to use  
                    • Decreasing rSO₂ is an early indicator of neurological compromise. | • Only monitors small area of cortex- not global function  
                    • Diathermy interferes with function  
                    • Cannot differentiate cause of low rSO₂ (embolus versus hypoperfusion) |
Temperature Control in DHCA

• The degree of hypothermia and the route of application have been evolving and currently tend to use moderate hypothermia (MH) (20.1–28 °C) associated with unilateral or bilateral selective cerebral perfusion methods.

• Following the recommendations of The Society of Thoracic Surgeons, The Society of Cardiovascular Anesthesiologists, and The American Society of Extracorporeal Technology,

• Cooling should be performed gradually by maintaining a temperature gradient between the arterial outlet and the oxygenator venous inlet <10 °C to avoid the generation of gaseous embolism. Also during the rewarming, a gradient of T <10 °C must be maintained until the out-flow temperature reaches 30 °C where it should be lowered to <4 °C.

• Bladder and Nasopharyngeal temperature are most commonly used sites.
<table>
<thead>
<tr>
<th>System</th>
<th>Adverse Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular</td>
<td>Arrhythmias secondary to potassium loss</td>
</tr>
<tr>
<td></td>
<td>Increased plasma viscosity</td>
</tr>
<tr>
<td></td>
<td>Vasoconstriction impairing microcirculation</td>
</tr>
<tr>
<td>Coagulation</td>
<td>Impaired coagulation</td>
</tr>
<tr>
<td></td>
<td>Reduced platelet count</td>
</tr>
<tr>
<td>Renal and metabolic</td>
<td>Reduced glomerular filtration rate</td>
</tr>
<tr>
<td></td>
<td>Metabolic acidosis</td>
</tr>
<tr>
<td></td>
<td>Hyperglycaemia secondary to impaired glucose metabolism</td>
</tr>
<tr>
<td></td>
<td>Effects on pharmacodynamics and pharmacokinetics</td>
</tr>
<tr>
<td>Cerebral</td>
<td>Vasoconstriction during cooling</td>
</tr>
<tr>
<td></td>
<td>Brain injury from hyperthermia during rewarming</td>
</tr>
</tbody>
</table>
Right Axillary Cannulation

Figure 6. The Journal of Thoracic and Cardiovascular Surgery
Selective Antegrade Cerebral Perfusion (SACP)

- With the advent of ACP and RCP, there has been a shift in aortic surgery from deep hypothermic circulatory arrest (14°C–20°C) to more modest degrees of systemic cooling, with equivalent or improved neurological outcomes.

- In ACP, blood is directed from the axillary or innominate artery in an antegrade direction up the right common carotid artery to provide perfusion to the brain after clamping of the innominate artery and discontinuation of systemic blood flow.

- Can result in embolization or vascular injury during manipulation of the arch vessels.

- Risks - no uniform perfusion of the brain if only unilateral ACP is used.
• Monitoring of perfusion pressure during ACP can be accomplished via arterial blood pressure monitoring in the right upper extremity.

• ACP is typically performed by targeting cerebral blood flow, with values near 10 mL/kg/min used most frequently.

• The optimal perfusion pressure for ACP is unclear, with equivalent neurological outcomes demonstrated in patients managed with high (≈80 mm Hg) versus low (≈50 mm Hg) pressures.
• Arterial cannulation at any peripheral sites for example, femoral artery or axillary artery, can, on institution of cardiopulmonary bypass (CPB), precipitate or worsen malperfusion.

• The retrograde arterial flow towards the heart, even when the cannula lies within the vessel’s true lumen, may lead to differential false luminal dominance via a primary or secondary tear, false luminal pressurization, and cerebral, cardiac, or other organ malperfusion.

• Historically, the femoral artery has often been used for arterial cannulation due to ease of access, size, and ability to achieve adequate flow rates.

• However, retrograde femoral perfusion carries the risk of pressurization of the dissection false lumen, leading to compromise of true luminal flow and malperfusion.
• Commonly, an 8 mm graft is anastomosed to the artery in an end-to-side fashion which then allows antegrade blood flow during CPB without further aortic manipulation.

• It may also reduce emboli from an atheromatous aorta or retrograde femoral arterial flow.

• Increasingly the right axillary or subclavian artery has been used for arterial cannulation with well-documented results.

• Direct aortic cannulation, carotid artery cannulation, and transapical left ventricular-aortic cannulation have all been utilised to try and ensure true luminal flow.

• Thus, the institution of CPB is a timepoint for extravigilant monitoring.

• Detection of malperfusion at this point should prompt the surgeon to change the arterial cannulation site immediately to ensure true luminal flow.
The Impact of Cerebral Malperfusion

• The incidence of neurological compromise evidenced by cerebral ischaemia and malperfusion at presentation is quoted to be between 5–14% and is associated with poor early outcomes.

• Neurological injury may be secondary to hypotension, malperfusion, or thromboembolic phenomenon.

• The surgery itself for treatment of Type A Dissection is associated with permanent neurological injury rates of as high as 26% depending on the age of the patient with temporary deficit rates of up to 32%.

• A shorter time between symptom onset and surgical reperfusion is associated with improved outcomes particularly if presentation is less than five hours and has a reasonable prospect of limiting stroke progression.
Cerebral Protection

- HCA only allows for limited periods of repair as the rate of adverse cerebral outcomes is closely related to time and the temperature at which arrest occurs.
- HCA enables visualization of the aortic arch to inspect for intimal tears.

**Key points:**
- Deep hypothermic circulatory arrest (DHCA) is a technique to obtain optimal operating conditions while providing cerebral protection.
- The majority of patients will tolerate 30 min of circulatory arrest at 18°C without significant neurological impairment.
- Hypothermia is the main method of cerebral protection. Other neuroprotective strategies include pharmacological methods, glucose control, haemodilution, and acid–base management.
- Surgical techniques such as selective antegrade cerebral perfusion may be used to prolong the safe duration of DHCA.
Pharmacological neuroprotection

• In a recent survey of members of the Association of Cardiothoracic Anaesthetists, 83% reported using some form of pharmacological cerebral protection during DHCA in adult thoracic aortic surgery.

• Thiopental and propofol have both been investigated at doses sufficient to cause burst suppression.

• Methyl Prednisone 1 Gram.

• Mannitol appears to have a certain antiapoptotic effect in addition to the osmotic and free radical scavenging effect.

• Lidocaine, Calcium Channel Blockers and Magnesium have been used.
Summary

- Operative mortality for Type A aortic dissection depends largely on the presence of malperfusion, circulatory compromise at the time of presentation.
- Operative repair requires determination of the extent of aortic root and arch involvement, the location of intimal tears and the presence of malperfusion.
- The availability of endovascular grafts and experience in hybrid techniques has increased options.
- Refinement of organ protection using DHCA and selective antegrade cerebral perfusion have decreased the risk neurological complications.
References


