Pathophysiology and classification:
Acute aortic dissection is an uncommon but potentially lethal disease. The basic pathophysiology of aortic dissection is disruption of the aortic intima and media (a “tear”) from aortic medial degeneration and mechanical forces that exert radial strain and shear stress on the luminal surface. Several congenital and acquired risk factors for aortic dissection are recognized. Aortic dissection is classically described by the extent and duration of dissection. Stanford type A dissection (proximal) originates in the ascending aorta, while type B (distal) originates distal to the ascending aorta. Acute dissection is characteristically defined as a dissection that presents within two weeks of symptom onset. The International Registry of Aortic Dissection (IRAD) details the distribution of symptoms and physical findings at presentation for aortic dissection. The diagnostic modality of choice is multi-slice, helical CT arteriography whereby intimal flap location, dissection extent, branch vessel involvement, false lumen patency, and aortic size may readily be determined. Magnetic resonance arteriography is an acceptable alternative modality. Management of type A aortic dissection requires urgent operative therapy. However, in uncomplicated type B aortic dissections (no mal-perfusion syndrome), medical therapy is preferred and is not inferior to operative management.

Medical Management:
The goal of medical management is manifold: to reduce the force of left ventricular contraction, to decrease the rate of rise of the aortic pulse wave (dP/dT), and to reduce systemic arterial pressure. These goals are achieved while ensuring adequate vital organ perfusion. Intensive care unit admission is requisite with appropriate invasive monitoring including continuous arterial pressure monitoring and central venous access when needed. The immediate goal of medical therapy is to relieve the pain of dissection and to rapidly reduce the systolic blood pressure and heart rate to a goal of 100-110 mmHg and less than or equal to 60 beats per minutes, respectively. The prompt institution of medical therapy attempts to stabilize the extent of dissection, reducing intimal flap mobility, relieving dynamic aortic branch obstruction, and decreasing risk of rupture. Even brief periods of poor control can result in extension of the dissection and may be organ or life threatening. Intravenous beta-blockers, which decrease aortic shear stress, are first-line drugs and should be administered before direct vasodilators to avoid undesirable reflex tachycardia. Preferred intravenous beta-blockers include labetalol and esmolol. Labetalol conveniently provides both α-receptor and β-receptor antagonism. If blood pressure control is suboptimal despite appropriate heart rate reduction, a direct arterial vasodilator is added such as nicardipine or nitroprusside. Centrally-acting, non-dihydropiridine calcium channel blockers (e.g. diltiazem) are acceptable agents in those who are intolerant to beta-blockers (e.g. severe COPD). A gradual transition from intravenous to oral antihypertensive agents is conducted while maintaining optimal hemodynamic parameters, a process that may extend up to one week. Preferred oral agents include an intermediate acting beta-blocker (e.g. metoprolol or labetaolol) in addition to agents with established benefit or efficacy in certain medical diseases (e.g. ACE inhibitors in diabetes). Frequently, two or more agents are required for optimal blood pressure management.
**Indications for surgical therapy:**
The cornerstone of surgical therapy is complication-specific intervention for states of mal-perfusion. Additional indications for operative intervention include rupture, false lumen expansion, and persistent dissection pain despite optimal medical treatment. A management algorithm is presented for reference (Figure). Most management guidelines are class I recommendations with level of evidence C (consensus). Long-term management includes institution of a stable blood pressure regimen, regular clinical evaluation, and serial non-invasive imaging.

**VUMC – SICU Management Protocol:**

- **Admit to SICU – enter aortic dissection order set**
- **Medical therapy goal endpoints:**
  - Blood pressure: SBP: 100-110mm of Hg at all times (SBP ≥ 120 – increase dosage of medical therapy)
  - Heart rate: ≤ 60 bpm (HR ≥ 70 – increase dosage of medical therapy)
  - Monitor measures of end-organ perfusion
    - Serial exam
    - Urine output
    - Serum lactate
  - Notify vascular service and faculty/fellow immediately in the following conditions:
    - Inability to rapidly (within 30 minutes) achieve blood pressure/heart rate goals
    - Evidence of inadequate end-organ perfusion
    - Persistent hemodynamic instability (SBP < 90, HR > 100)
    - Malperfusion states
      - Oliguria
      - Abdominal pain
      - Change in neurologic examination
      - Change in extremity vascular examination
      - Persistent back or chest pain despite goal blood pressure/heart rate control
  - Initiate beta-blockade therapy with esmolol (500 mcg/kg bolus and 50mcg/kg/min drip) or labetalol (bolus and drip dosages)
    - Titrate rapidly to goal HR/BP
    - Drip to remain hanging for 24 hrs after changed to oral regimen with BP control at targets throughout the period
  - *If SBP is inadequately controlled (SBP ≥ 120), initiate nicardipine drip – (bolus and drip dosing)*
    - Titrate rapidly to goal BP
    - Drip to remain hanging for 24 hrs after changed to oral regimen with BP control at targets throughout the period
  - Conversion to oral therapy:
    - Initiate oral therapy after adequate BP/HR control with IV agents
    - Oral therapy and IV therapy should overlap for 24 hours
    - Transition from IV to oral therapy should adhere to established hemodynamic parameters with 100% target achievement
### Box 1. Predisposing conditions for aortic dissection [9]

**Long-standing arterial hypertension**  
Smoking  
Dyslipidemia  
Use of cocaine/crack

**Connective tissue disorders**  
Hereditary fibrillinopathies  
- Marfan's syndrome  
- Ehlers-Danlos syndrome

**Hereditary vascular diseases**  
Bicuspid aortic valve  
Coarctation  
Vascular inflammation  
- Giant cell arteritis  
- Takayasu arteritis  
- Behcet's disease  
- Syphilis  
- Ormond's disease

**Deceleration trauma**  
Car accident  
Fall from height

**Iatrogenic factors**  
Catheter/instrument intervention  
Valvular/aortic surgery  
- Side- or cross-clamping/aortotomy  
- Graft anastomosis  
- Patch aortoplasty  
- Cannulation site  
- Aortic wall fragility

**Other**  
Pregnancy  
Turner's syndrome

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### Table:

<table>
<thead>
<tr>
<th>Category</th>
<th>Present/No. Reported (%)</th>
<th>No. Type A (%)</th>
<th>No. Type B (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Presenting symptoms</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any pain reported</td>
<td>443/464 (95.5)</td>
<td>271 (93.8)</td>
<td>172 (98.3)</td>
</tr>
<tr>
<td>Abrupt onset</td>
<td>379/447 (84.8)</td>
<td>234 (85.4)</td>
<td>145 (83.8)</td>
</tr>
<tr>
<td>Chest pain</td>
<td>331/455 (73.7)</td>
<td>221 (78.9)</td>
<td>110 (62.9)</td>
</tr>
<tr>
<td>Anterior chest pain</td>
<td>262/430 (60.9)</td>
<td>191 (73)</td>
<td>71 (44.1)</td>
</tr>
<tr>
<td>Posterior chest pain</td>
<td>149/415 (35.9)</td>
<td>85 (32.8)</td>
<td>64 (41)</td>
</tr>
<tr>
<td>Back pain</td>
<td>240/451 (53.2)</td>
<td>129 (46.6)</td>
<td>111 (63.8)</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>133/449 (29.6)</td>
<td>60 (21.6)</td>
<td>73 (42.7)</td>
</tr>
<tr>
<td>Severity of pain: severe or worst ever</td>
<td>346/382 (90.6)</td>
<td>211 (90.1)</td>
<td>135 (90)</td>
</tr>
<tr>
<td>Quality of pain: sharp</td>
<td>174/270 (64.4)</td>
<td>103 (62)</td>
<td>71 (68.3)</td>
</tr>
<tr>
<td>Quality of pain: tearing or ripping</td>
<td>155/267 (59.6)</td>
<td>78 (49.4)</td>
<td>57 (52.5)</td>
</tr>
<tr>
<td>Radiating</td>
<td>127/449 (28.3)</td>
<td>75 (27.2)</td>
<td>52 (30.1)</td>
</tr>
<tr>
<td>Migrating</td>
<td>74/446 (16.6)</td>
<td>41 (14.9)</td>
<td>33 (19.3)</td>
</tr>
<tr>
<td>Syncope</td>
<td>42/447 (9.4)</td>
<td>35 (12.7)</td>
<td>7 (4.1)</td>
</tr>
<tr>
<td><strong>Physical examination</strong></td>
<td></td>
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<tr>
<td>Hemodynamics (n = 451)</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Hypertensive (SBP ≥ 150 mm Hg)</td>
<td>221 (49)</td>
<td>99 (35.7)</td>
<td>122 (70.1)</td>
</tr>
<tr>
<td>Normotensive (SBP 100–149 mm Hg)</td>
<td>156 (34.6)</td>
<td>110 (39.7)</td>
<td>46 (26.4)</td>
</tr>
<tr>
<td>Hypotensive (SBP &lt; 100 mm Hg)</td>
<td>36 (8)</td>
<td>32 (11.6)</td>
<td>4 (2.3)</td>
</tr>
<tr>
<td>Shock or Tamponade (SBP ≤ 80 mm Hg)</td>
<td>38 (8.4)</td>
<td>36 (13)</td>
<td>2 (1.5)</td>
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<tr>
<td>Murmur of aortic insufficiency</td>
<td>69/457 (15.1)</td>
<td>117 (44)</td>
<td>20 (12)</td>
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<tr>
<td>Pulse deficit</td>
<td>69/457 (15.1)</td>
<td>53 (18.7)</td>
<td>16 (9.2)</td>
</tr>
<tr>
<td>Cerebrovascular accident</td>
<td>21/447 (4.7)</td>
<td>17 (6.1)</td>
<td>4 (2.3)</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>29/440 (6.6)</td>
<td>24 (8.8)</td>
<td>5 (3)</td>
</tr>
</tbody>
</table>
Clinical Suspicion of AAD

- Pain relief (IV morphine sulphate)
  - Begin IV beta-blocker to maintain systolic BP
  - ≈ 110 mm Hg. Add IV vasodilators (nitroprusside) if BP remains high after adequate beta-blockade

- Emergency Diagnostic Imaging
  - CT-scan or TEE or MRI

- Dissection confirmed
  - Type A Dissection
    - Emergency Surgery
  - Type B Dissection
    - Organ or limb ischemia
      - Endovascular or surgical treatment

- Hemodynamically Unstable
  - Intubation & Mechanical Ventilation

- Emergency bedside TEE
  - No Dissection
    - Work-up for alternative diagnosis
      - Discontinue IV beta-blocker

Medical treatment
- Beta-blocker ± vasodilator initially IV
- then PO for long term target
- BP<135/85 mm Hg in usual patients
- and <130/80 mm Hg for patients with Marfan’s syndrome
References:


