Management of the Patient with Aortic Stenosis undergoing Non-cardiac Surgery

Srinivasan Rajagopal M.D.
Assistant Professor
Division of Cardiothoracic Anesthesia

Objectives

• Describe the pathophysiology of aortic stenosis (AS)
• Review data from cardiac catheterization and echocardiogram
• Know anesthetic principles of management of patients with critical AS
Introduction

• Aortic stenosis is the most important valvular lesion
  – because of its potential for sudden death (15-20%)
  – inability to obtain systemic perfusion by external cardiac massage during a cardiac arrest

Case

• Case A
  – 75 year old male with critical aortic stenosis, mean gradient 25 mmHg, valve area 0.5cm², EF25%, presents with a hip fracture

• Case B
  – 65 year old female with an aortic valve area of 1 cm², mean gradient 45, EF 55 %, ventricular hypertrophy presents for lap cholecystectomy
Background

- AS is the most common valvular disease in elderly at 3%
- More common in men
- Usually congenital or degenerative in origin
- Long asymptomatic latent period followed by angina, syncope or dyspnea

Mechanism of Stenosis

- Degenerative calcific AS
  - Mechanical stress leads to progressive fibrosis and calcification of previous trileaflet
- Initial sclerosis that progresses to stenosis
  - Associated with risk factors for coronary artery disease, diabetes, hypercholesterolemia
Etiology

• Congenital Bicuspid Valve
  – most common congenital cardiac malformation
  – two leaflets leads to turbulent flow—produce fibrosis, calcification and orifice narrowing secondary to trauma
  – accounts for 50% of patients <70 yr requiring surgery for stenosis

Etiology

• Rheumatic heart disease
  – results from adhesions and fusions of the commissures and cusps
  – vascularization of the leaflets of the valve ring
    • leads to retraction and stiffening of the free borders of the cusps
  – calcific nodules develop on both surfaces
  – orifice reduced to a small round or triangular opening
  – rheumatic valve is often regurgitant and stenotic
**Classification of Severity**

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Valve Area</td>
<td>1.2 – 1.8 cm²</td>
<td>0.8 – 1.2 cm²</td>
<td>&lt;0.8 cm²</td>
</tr>
<tr>
<td>Mean Gradient</td>
<td>12-25 mmHg</td>
<td>40-50 mmHg</td>
<td>&gt;50 mmHg</td>
</tr>
</tbody>
</table>

**Pathophysiology**

- Normal aortic valve area (AVA) 2.6–3.5 cm² in adults
- Hemodynamically significant obstruction occurs as the AVA approaches 1.0 cm²
- Increasing obstruction → LV hypertrophy, – allows LV to maintain pressure gradient across the valve without reducing cardiac output
Pathophysiology

• Hypertrophied ventricle eventually becomes stiff
  – Diastolic dysfunction with a reduced compliance

• Reduced coronary flow reserve → ischemia in absence of CAD

• Heart Failure: Changes in left ventricle function may no longer be adequate to overcome outflow obstruction

Pathophysiology

• Cardiac output, stroke volume, and therefore, pressure gradient across the valve, fall

• Left ventricular dilatation occurs late in the disease process

• Primary contractile dysfunction cause for low ejection fraction
Pathophysiology

- Normal sinus rhythm beneficial as the left atrial kick accounts for 40% of LV filling
- LA hypertrophy secondary to this increased demand
  - increased chance of atrial fibrillation
- The ventricle becomes:
  - very sensitive to changes in preload
  - dependent on the maintenance of sinus rhythm
  - susceptible to ischemia, especially when arterial pressure is reduced
Echo

- Mean gradient - average gradient across the valve occurring during the entire systole
- Peak gradient
- Left ventricular size
- Ejection fraction

Echo Image
Echo

[Images and diagrams related to echocardiography showing different heart conditions and images]
2-D echo image measuring the diameter of the LVOT

Echocardiographic features of severe AS
Top left: upper esophageal short-axis view shows a heavily calcified tri-leaflet valve with an EOA of 0.55
Normal Aortic Valve

Critical Aortic Stenosis
Ventricular Hypertrophy

Dilated ventricle
Cardiac Catheterization

- Assesses coronary artery disease
- Gradient – difference in pressure between left ventricle and aorta
- Peak to peak gradient

Simultaneous pressure tracings from the left ventricle and aorta in aortic stenosis

Three Cardinal Symptoms

- **Angina**
  - occurs as oxygen demand from the hypertrophied LV outstrips the supply
  - initial symptom in 50-70%
- **Syncope**
  - uncompensated decrease in vascular tone with exertion
  - initial symptom in 15-30%
- **Dyspnea**
  - pulmonary congestion, CHF
  - late in AS

Surgical AVR

- Severe AS is a risk factor for perioperative morbidity and mortality
- AVR may be needed before planned noncardiac surgery
  - Postpone elective noncardiac surgery in patients with symptomatic severe AS

Bonow RO et al. Focused update Circulation 2008

Alternatives to Surgical AVR

- TAVR - Transcatheter Aortic Valve Replacement
  - Developed for severe symptomatic AS with unacceptably high risk for surgery

- Balloon valvuloplasty
  - High rate of restenosis with no decrease in mortality
  - Transient improvement
  - Palliative for elderly who are poor surgical candidates
Monitoring EKG

- Increased QRS duration and voltage
- T-wave inversion and ST depression as hypertrophy worsens
- AV, intraventricular blocks
Monitoring

- Arterial line
  - Slow rising pulse with narrow pulse pressure
- Place before patient goes to sleep

Central Venous and Pulmonary Artery Catheter

- Administration of vasoconstrictor therapy
- Can measure CO, derived hemodynamic parameters
- Mixed venous oxygen saturation
- Transvenous pacing
- Risk for arrhythmia
**TEE**

- Estimates mean gradient and aortic valve area
- Provides real-time assessment of ventricular dysfunction, hypertrophy
- Monitor ventricular filling
- Manipulation of hemodynamics
- Abnormalities of other valves
- well validated and compares with cardiac catheterization

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**Hemodynamic Goals**

- Avoid systemic hypotension
  - leads to myocardial ischemia, and then decreased contractility and a vicious cycle ensues
  - vasoconstrictors must be at hand – consider an infusion from the beginning treat hypotension aggressively

- Maintain sinus rhythm
  - sinus tachy decreases diastolic time for myocardial perfusion
  - sinus bradycardia limits CO in pts with fixed stroke volume
  - Treat arrhythmias promptly
Hemodynamic Goals

• Contractility
  – Stroke volume is maintained with a heightened contractile state
• Maintain adequate intravascular volume to ensure ventricular filling
• Non compliance Ventricle
  – LVEDP & LVEDV - preload augmentation is needed for a normal stroke volume

Systemic Vascular Resistance

• Afterload
• Already elevated, but relatively fixed
• Coronary perfusion pressure must be maintained
Anesthesia

• GA vs Regional:
  – successful use of spinal and epidural have been reported
  – can use combined lumbar plexus and sciatic PNB for hips
  – GA is safe, as long as care is taken to maintain blood pressure and sinus rhythm

Vasopressors

• Drugs to maintain systemic tone
  – Phenylephrine
  – Norepinephrine
  – Vasopressin

• Infusions rather than boluses facilitate stability

• Aim to maintain blood pressure at pre-anesthetic values
Arrhythmias

- Treat promptly
- Maintain sinus rhythm
- New onset atrial fibrillation may require cardioversion
- Sinus tachycardia detrimental (ischemia)
- Maintain normal electrolyte levels

Postoperative

- Monitored bed with invasive monitoring
- Adequate pain control avoids catecholamine induced tachycardia and hypertension
- Maintain appropriate intravascular filling, blood pressure and sinus rhythm
### CASE

<table>
<thead>
<tr>
<th>Case A</th>
<th>Case B</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial line</td>
<td>General anesthesia</td>
</tr>
<tr>
<td>Spinal</td>
<td>Arterial line</td>
</tr>
<tr>
<td>Norepinephrine drip</td>
<td>Echo</td>
</tr>
<tr>
<td>Defibrillator pads</td>
<td>Phenylephrine drip</td>
</tr>
<tr>
<td>Echo in room</td>
<td>Large bore IV</td>
</tr>
</tbody>
</table>

### At UIHC
- Patients with severe aortic stenosis for valve replacement
- Spinal: 0.75% Bupivacaine 5 ml and Duramorph 500 Mcg
- Minimal narcotic requirement
- Patients extubated at end of case
Summary

• Understand the pathophysiology of Aortic stenosis
• Understand the cardiac cath and echo reports
• Anesthetic management will tailor to the type of surgery and your skills
  – aggressive intraoperative and postoperative monitoring and therapy
  – Prompt recognition and therapy of intraoperative hypotension