Aortic Stenosis: 
Spectrum of Disease, 
Low Flow/Low Gradient and Variants

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Basic root structure
Parasternal Long Axis View

- Fibrous Annulus
- Sinotubular junction
- Leaflets
- Sinuses of Valsalva
Normal AV M-Mode
cloaptation in center of aortic root
Normal AV orientation and opening

Diastole  Systole

Aortic Stenosis

Etiology

- Senile/Degenerative Calcific
  - Calcification resembles ectopic bone
  - Risk factors similar to those for atherosclerosis
  - Renal dysfunction may accelerate

- Premature Calcific Bicuspid / Congenital

- Rheumatic
  - Less common in the United States

- Less common
  - Type 2 Hyperlipidemia, SLE, Irradiation, Paget’s Dz
Calcific Aortic Stenosis: *Progressive reduction in leaflet motion*

Spectral Doppler of the AV
*Apical Five Chamber*
Bicuspid Aortic Valve

- Most common congenital anomaly (1-2%)
- Commissure may be horizontal or vertical
  - Horizontal: Anterior and Posterior leaflets
  - Vertical: Right and Left (coronary) leaflets
- Accel. Calcification → premature stenosis
- Proximal aortopathy (even in normals)
  - Associated abnormalities - coarctation

Bicuspid Aortic Valve
PLAX View – Doming

Diastole

Systole
Bicuspid Aortic Valve
*PSAX view morphology*

**Diastole**

**Systole**

Systolic ellipsoid orifice identifies as raphe

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Rheumatic Aortic Stenosis:
*Less calcification, More commissural fusion*
Aortic Valve: 
*Other Anomalies associated with AS*

Unicuspid AoV  Quadracuspid AoV

Aortic Stenosis: 
*Physiologic Sequelae*

- **Chronic LV pressure overload**
  - Myocardial Hypertrophy – Progressive, Concentric
  - LA dilatation

- **Progressive diastolic & systolic dysfunction**
  - END STAGE: Limited Cardiac Output

- **After long latency… SYMPTOMS:**
  - Early: Dyspnea and Fatigue (often subtle)
  - Late: “Cardinal Symptoms”
    - Chest Pressure, Syncope, Congestive Heart Failure
Evaluation of AS: *Echo Essentials*

- Valve Anatomy - establish etiology
  - Exclude other forms of LVOT obstruction
- Severity of stenosis
- Physiologic sequelae
  - LV hypertrophy, diastolic fxn, systolic fxn
  - LA dilatation, Pulmonary hypertension
- Evaluate concurrent disease
  - Proximal aorta and arch
  - Aortic Valve Regurgitation, Mitral Disease

Aortic Stenosis: *Assessing Severity*

- Peak AV Jet Velocity
- Mean AV Gradient
- Valve Area by continuity equation
- Velocity Ratio (“Dynamic Index”, “Dimensionless Index”…)
- Planimetry
Aortic Stenosis: Prognosis of Velocity

- Variable Rate of Progression
  - Avg ~0.3 m/sec/year

- High rate of events, even for "asymptomatic" AS

- Peak Jet Velocity, Rate of velocity change, Functional status: predict clinical outcome

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Aortic Stenosis: 
Peak Velocity – Continuous Wave (CW) Doppler

- Generally in A5C View, also Apical LAX (A3C)
- **Parallel to the ejection jet!**
- Confirm – Right Parasternal
  - Suprasternal also possible
- Use highest velocity
  - Avoid feathery signals at tip
  - Piedoff –
    - “non-imaging” probe
    - “Dual Crystal CWD Transducer”

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Aortic stenosis
Assessment by Peak Velocity

- Mild stenosis: 2.0 – 2.9 m/s
- Moderate stenosis: 3.0 – 3.9 m/s
- Severe stenosis: > 4.0 m/s
- “Very Severe” or “Critical” stenosis: > 5.0 m/s
Beware the Dynamic Gradient!!

Aortic Stenosis:

**Peak Gradient**

- Peak Gradient = $4 \left( V_{AV} \right)^2$
  - Peak Instantaneous Gradient
**Instantaneous vs. Peak-to-Peak**

- Echo a more “physiologic” measurement
- Doppler peak gradient always higher
- Mean gradient and AVA should correlate
- Gradients are flow dependent

**Aortic Stenosis: Mean Gradient**

- **Mean Gradient**
  - Integration of velocity over time
  - Estimate – 0.7 * Peak Grad.
  - Correlates with cath Peak-to-Peak gradient
Aortic stenosis
Assessment by Mean Gradient

- Mild stenosis: < 20 mmHg
- Moderate stenosis: 20 – 39 mmHg
- Severe stenosis: \( \geq 40 \text{ mmHg} \)

Velocity and Gradient pitfall:
Influence of Cardiac Output

- **High CO = High gradient**
  - Aortic regurgitation
  - Hyperdynamic function

- **Low CO = Low gradient**
  - Reduced ejection fraction
  - Small ventricular cavity/LVH
  - High systemic vascular resistance/impedance
  - Significant mitral regurgitation
Aortic stenosis
Assessment of Valve Area

- Normal valve area: = 3 - 4 cm²
- Mild stenosis: > 1.5 cm²
- Moderate stenosis: 1.0 – 1.5 cm²
- Severe stenosis: < 1.0 cm²
- “Critical” stenosis: < 0.7 cm²

Calculation of AV Area: Continuity Equation

- Based on conservation of mass

\[ \text{Flow within LVOT} = \text{Flow across AV} \]

- LVOT area * VTI_{LVOT} = AVA * VTI_{AV}
- \[ \pi \times (\text{LVOT radius})^2 \times VTI_{LVOT} = AVA \times VTI_{AV} \]
- \[ \frac{\pi \times (\text{LVOT radius})^2 \times VTI_{LVOT}}{\text{VTI}_{AV}} = AVA \]
LVOT diameter
2.1 cm

The LVOT is never easy...

??? Go slightly off-axis

Flow through LVOT
*Pulse Wave Doppler*

- Spectral Envelope
  - With sample volume in LVOT

- **Velocity Time Integral (VTI)**
  - flow through a single point

\[ \text{VTI} = 19 \text{ cm} \]

Flow Across the Aortic Valve:
*Continuous Wave Doppler*

\[ \text{VTI} = 85 \text{ cm} \]
Calculating Aortic Valve Area

\[ \text{AVA} = \frac{(\text{Diameter}_{LVOT}/2)^2 \times \pi \times \text{VTI}_{LVOT}}{\text{VTI}_{AV}} \]

\[ \text{AVA} = \frac{(2.1 \text{ cm}/2)^2 \times 3.14 \times 19 \text{ cm}}{85 \text{ cm}} \]

\[ \text{AVA} = 0.7 \text{ cm}^2 \]

Continuity Equation:

**Pitfalls**

- **LVOT measurement**
  - Diameter\(^2\) - Can propagate large error
  - LVOT often elliptical – CSA best from 3D TEE or CT

- **LVOT velocity**

- **AV velocity**
  - Missing the Peak:
    - Use multiple sites / Piedoff / highest velocity
  - Beware MR!
Doppler Velocity Index

- Eliminates errors of LVOT measurement
  - \( DVI = \frac{VTI_{LVOT}}{VTI_{AV}} \)

- Criteria for Severe AS:
  - \( DVI < 0.25 \)

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Planimetry of the Aortic Valve

- \( AVA = 1.1 \text{ cm}^2 \)
**Planimetry**

- Correlates with invasively obtained areas
- Flow dependent
  - Difficult to distinguish decreased opening due to LV failure
- TEE superior - use of color flow area
- Dense calcification reduces accuracy

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**Summary**

**Table 3** Recommendations for grading of AS severity

<table>
<thead>
<tr>
<th></th>
<th>Aortic sclerosis</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak velocity (m/s)</td>
<td>1.0–1.5</td>
<td>2.6–2.9</td>
<td>3.0–4.0</td>
<td>≥4.0</td>
</tr>
<tr>
<td>Mean gradient (mmHg)</td>
<td>0.85</td>
<td>&lt;20</td>
<td>20–40</td>
<td>≥40</td>
</tr>
<tr>
<td>AVA (cm²)</td>
<td>1.5</td>
<td>&gt;1.5</td>
<td>1.0–1.5</td>
<td>&lt;1.0</td>
</tr>
<tr>
<td>Indexed AVA (cm²/m²)</td>
<td>0.60–0.85</td>
<td>&gt;0.85</td>
<td>0.60–0.85</td>
<td>&lt;0.6</td>
</tr>
<tr>
<td>Velocity ratio</td>
<td>0.1</td>
<td>&gt;0.50</td>
<td>0.25–0.50</td>
<td>&lt;0.25</td>
</tr>
</tbody>
</table>

So...
My patient has severe aortic stenosis! What do I do? Does echo help me?

WHEN SHOULD I OPERATE?

The Good Old Days:
The Symptomatic “Cliff”

“Stages” of Disease

- **Stage A:** At risk for disease
- **Stage B:** Progressive disease
- **Stage C:** Severe disease (asymptomatic)
- **Stage D:** Severe disease (symptomatic)

“Stage C” can be subdivided:

- **Stage A:** At risk for disease
- **Stage B:** Progressive disease
- **Stage C1:** Severe (asymptomatic) – Compensated LV
- **Stage C2:** Severe (asymptomatic) – Decompensated LV
- **Stage D:** Severe disease (symptomatic)
Guidelines Assist in Decision-Making

Calcified/Thickened leaflets
Reduced Systolic Opening

“Asymptomatic”

Vmax ≥5 m/s + low AVR risk
Vmax ≥4 m/s
EF <50%
EF ≥50%

Undergoing other CV Surgery
ETT ↓BP / ↓ex capacity
Rapid progression + low AVR risk

AVR (IIa) AVR (I) AVR (IIa) AVR (I) AVR (IIb)

“Low Gradient” Aortic Stenosis

Peak Velocity 2.74 m/sec
Mean Gradient 15 mmHg
Calculated AVA 0.5 cm²
Low Output – Low Gradient AS

Low Ejection Fraction

- AVA of 0.5 cm\(^2\), but MG of 15mmHg? WHY?
  - Because low SV (low flow) leads to low gradients

- “Real AS”
  - 1\(^{\circ}\) Prob: Severe obstruction to flow
  - 2\(^{\circ}\) Prob: Depressed LVEF

- “Pseudo-AS”
  - 1\(^{\circ}\) Prob: Depressed LVEF
  - 2\(^{\circ}\) Prob: Mild/Mod obstruction is made to look severe by ↓SV

Dobutamine Stress Testing

- Increase LV contractility -> Increase Stroke Volume

- Increase Stroke Volume by 20% ->
  - Real AS: Peak vel/mean gradient significantly ↑↑
    AVA stays unchanged or ↓slightly
  - Pseudo AS: Peak vel/mean gradient minimal ↑
    AVA↑

- What if LV contractility / SV don’t increase?

Improves with AVR
Low Gradient - Normal EF

- EF ≥50%, AVA <1 cm², mean grad <40mmHg
  - Whah???

- Still a stroke volume problem!!
  - SV\textsubscript{index} ≤35 ml/m² despite EF

- “Typical” patient:
  - Older, h/o hypertension, women
  - Concentric LVH, small cavity, impaired filling
  - Markedly increased vascular impedance

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Guidelines Assist in Decision-Making

Suspected "Low Flow" AS

Symptoms

$V_{\text{max}} \geq 4 \text{ m/s}$

- $V_{\text{max}} < 4 \text{ m/s}$
  - $\text{EF} < 50\%$
  - $\text{EF} \geq 50\%$

  - DSE
    - $\text{MG} \geq 40 \text{ mmHg}$ or $\text{Pk Vel} \geq 4 \text{ m/s}$
  - AVR (IIa)

- AVR (I)

No Symptoms

$V_{\text{max}} < 4 \text{ m/s}$

- $\text{AVR NOT Indicated}$

AVAindex $\leq 0.6 \text{ cm}^2/\text{m}^2$

and

SVindex $< 35 \text{ ml/m}^2$

Rule Out other causes for Sx!!

Thank You!