The Role of Echocardiography in Assessment of Aortic Stenosis

Ramzi El Accaoui, MD
February 12, 2016
Pathophysiology

Rheumatic  Calcific  Bicuspid

Pathophysiology

Stenotic valves from 932 patients (age 26-91 years):
  – 504 (54%) had congenitally malformed valves
  – 417 (45%) had tricuspid valves

<table>
<thead>
<tr>
<th>Aortic Valve Structure</th>
<th>Cases, n (%)</th>
<th>Ages (y) of Patients by Decades at Time of Aortic Valve Replacement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unicuspid</td>
<td>34 (6)</td>
<td>3</td>
</tr>
<tr>
<td>Bicuspid</td>
<td>309 (53)</td>
<td>1</td>
</tr>
<tr>
<td>Tricuspid</td>
<td>234 (40)</td>
<td>0</td>
</tr>
<tr>
<td>Uncertain</td>
<td>7 (1)</td>
<td>0</td>
</tr>
<tr>
<td>Subtotals, n (%)</td>
<td>584 (100)</td>
<td>4 (&lt;1)</td>
</tr>
<tr>
<td>Women</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unicuspid</td>
<td>12 (3)</td>
<td>1</td>
</tr>
<tr>
<td>Bicuspid</td>
<td>149 (43)</td>
<td>1</td>
</tr>
<tr>
<td>Tricuspid</td>
<td>183 (53)</td>
<td>0</td>
</tr>
<tr>
<td>Uncertain</td>
<td>4 (1)</td>
<td>0</td>
</tr>
<tr>
<td>Subtotals, n (%)</td>
<td>348 (100)</td>
<td>2 (&lt;1)</td>
</tr>
</tbody>
</table>

### Epidemiology

Population-based data from Olmsted County (11,911 adults)

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Participants</th>
<th>Moderate to severe Aortic stenosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>18-44</td>
<td>4351</td>
<td>1 (0.02%)</td>
</tr>
<tr>
<td>45-54</td>
<td>696</td>
<td>1 (0.1%)</td>
</tr>
<tr>
<td>55-64</td>
<td>1240</td>
<td>2 (0.2%)</td>
</tr>
<tr>
<td>65-74</td>
<td>3879</td>
<td>50 (1.3%)</td>
</tr>
<tr>
<td>≥75</td>
<td>1745</td>
<td>48 (2.8%)</td>
</tr>
</tbody>
</table>

Nkomo VT et al. Lancet. 2006 Sep 16;368(9540):1005-11
Prognosis

Valvular Aortic Stenosis in Adults
Average Course
(Post Mortem Data)

Percent Survival

Age, Years

Latent Period
(Increasing obstruction, Myocardial overload)
Onset Severe Symptoms

Angina
Syncope
Failure

Average age death (♂)

PARTNER Trial

2014 ACC/AHA Guidelines

Class I (Level of Evidence: B)

TTE is indicated in patients with signs or symptoms of AS or a bicuspid aortic valve for:

– determining the cause of AS
– assessing hemodynamic severity
– measuring LV size and systolic function
– determining prognosis, and
– determining timing of valve intervention

Nishimura RA et al. J Am Coll Cardiol. 2014 Jun 10;63(22):2438-8
Invasive Valve Assessment

• Gorlin Formula:

\[
\text{Valve Area (cm}^2) = \frac{\text{Cardiac Output (ml/min)}}{\text{Heart rate (beats/min) \cdot Systolic ejection period (s) \cdot 44.3 \cdot \sqrt{mean \ Gradient \ (mmHg)}}}
\]

• Hakki Formula:

\[
\text{Aortic Valve area (cm}^2) \approx \frac{\text{Cardiac Output (litre/min)}}{\sqrt{\text{Peak to Peak Gradient (mmHg)}}}
\]
Echo vs. Cath

Pressure Recovery

- Flow convergence at stenotic valve $\rightarrow$ conversion of potential energy to kinetic energy
- Divergence of distal to vena contracta $\rightarrow$ reconversion of some kinetic energy to potential energy $\rightarrow$ recovery of a proportion of the pressure lost
- Doppler detects peak flow velocity at the vena contracta $\rightarrow$ higher gradients than cath

Bach DS. JACC Cardiovasc Imaging. 2010 Mar;3(3):296-304
Pressure Recovery

1,563 patients in the SEAS trial

Pressure recovery = \(4v^2 \times \frac{2AVA}{Aa[1 - (AVA/Aa)]}\)
Pressure recovery

• Conversion of kinetic energy to heat dominates if there is turbulent flow (dilated ascending aorta)
• The effect of pressure recovery should be accounted for in patients with proximal ascending aorta diameter ≤3.0 cm

Bach DS. JACC Cardiovasc Imaging. 2010 Mar;3(3):296-304
# Prior Guidelines

## 2008 ACC/AHA Guidelines

### A. Left-sided valve disease

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jet velocity (m per s)</td>
<td>Less than 3.0</td>
<td>3.0–4.0</td>
<td>Greater than 4.0</td>
</tr>
<tr>
<td>Mean gradient (mm Hg)*</td>
<td>Less than 25</td>
<td>25–40</td>
<td>Greater than 40</td>
</tr>
<tr>
<td>Valve area (cm²)</td>
<td>Greater than 1.5</td>
<td>1.0–1.5</td>
<td>Less than 1.0</td>
</tr>
<tr>
<td>Valve area index (cm² per m²)</td>
<td></td>
<td></td>
<td>Less than 0.6</td>
</tr>
</tbody>
</table>

## 2009 ASE Guidelines

<table>
<thead>
<tr>
<th>Aortic sclerosis</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aortic jet velocity (m/s)</td>
<td>≤2.5 m/s</td>
<td>2.6–2.9</td>
<td>3.0–4.0</td>
</tr>
<tr>
<td>Mean gradient (mmHg)</td>
<td>=</td>
<td>&lt;20 (&lt;30°)</td>
<td>20–40</td>
</tr>
<tr>
<td>AVA (cm²)</td>
<td>=</td>
<td>&gt;1.5</td>
<td>1.0–1.5</td>
</tr>
<tr>
<td>Indexed AVA (cm²/m²)</td>
<td>&gt;0.85</td>
<td>0.60–0.85</td>
<td>&lt;0.6</td>
</tr>
<tr>
<td>Velocity ratio</td>
<td>&gt;0.50</td>
<td>0.25–0.50</td>
<td>&lt;0.25</td>
</tr>
</tbody>
</table>

Discrepancies

1. Valve leaflets are heavily calcified with restricted mobility but the echocardiographic parameters do not support severe AS

2. AVA <1.0 cm² but the transvalvular gradients fall in the mild or moderate range
2012 ESC Guidelines

• Valve area measurements are operator-dependent and are less robust than gradient estimates in clinical practice

• Valve area alone cannot be relied upon for clinical decision-making and should be considered in combination with flow rate, pressure gradients, ventricular function, size and wall thickness, degree of valve calcification and blood pressure, as well as functional status

### 2014 ACC/AHA Guidelines

<table>
<thead>
<tr>
<th></th>
<th>At risk of AS</th>
<th>Progressive AS</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>- Bicuspid aortic valve (or other congenital valve anomaly)</td>
<td>- Aortic $V_{\text{max}} &lt; 2 \text{ m/s}$</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Aortic valve sclerosis</td>
<td>- Mild AS:</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Aortic $V_{\text{max}} 2.0\text{–}2.9 \text{ m/s or mean } \Delta P &lt; 20 \text{ mm Hg}$</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Moderate AS:</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Aortic $V_{\text{max}} 3.0\text{–}3.9 \text{ m/s or mean } \Delta P 20\text{–}39 \text{ mm Hg}$</td>
<td></td>
</tr>
<tr>
<td>B</td>
<td></td>
<td>- Mild to-moderate leaflet calcification of a bicuspid or trileaflet valve with some reduction in</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>systolic motion or</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Rheumatic valve changes with commissural fusion</td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>Asymptomatic severe AS</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
# 2014 ACC/AHA Guidelines

### D: Symptomatic severe AS

<table>
<thead>
<tr>
<th>D1</th>
<th>Symptomatic severe high-gradient AS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>- Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening</td>
</tr>
<tr>
<td></td>
<td>- Aortic $V_{max} \geq 4$ m/s or mean $\Delta P \geq 40$ mm Hg</td>
</tr>
<tr>
<td></td>
<td>- AVA typically $\leq 1.0$ cm$^2$ (or AVA$I \leq 0.6$ cm$^2$/m$^2$) but may be larger with mixed AS/AR</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>D2</th>
<th>Symptomatic severe low-flow/low-gradient AS with reduced LVEF</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>- Severe leaflet calcification with severely reduced leaflet motion</td>
</tr>
<tr>
<td></td>
<td>- AVA $\leq 1.0$ cm$^2$ with resting aortic $V_{max} &lt; 4$ m/s or mean $\Delta P &lt; 40$ mm Hg</td>
</tr>
<tr>
<td></td>
<td>- Dobutamine stress echocardiography shows AVA $\leq 1.0$ cm$^2$ with $V_{max} \geq 4$ m/s at any flow rate</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>D3</th>
<th>Symptomatic severe low-gradient AS with normal LVEF or paradoxical low-flow severe AS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>- Severe leaflet calcification with severely reduced leaflet motion</td>
</tr>
<tr>
<td></td>
<td>- AVA $\leq 1.0$ cm$^2$ with aortic $V_{max} &lt; 4$ m/s or mean $\Delta P &lt; 40$ mm Hg</td>
</tr>
<tr>
<td></td>
<td>- Indexed AVA $\leq 0.6$ cm$^2$/m$^2$ and</td>
</tr>
<tr>
<td></td>
<td>- Stroke volume index $&lt; 35$ mL/m$^2$ and</td>
</tr>
<tr>
<td></td>
<td>- Measured when patient is normotensive (systolic BP $&lt; 140$ mm Hg)</td>
</tr>
</tbody>
</table>

Nishimura RA et al. J Am Coll Cardiol. 2014 Jun 10;63(22):2438-8
Doppler

- Any deviation from a parallel intercept angle results in velocity underestimation
- Degree of underestimation is $\leq 5\%$ if the intercept angle $\Theta$ is $<15^\circ$ of parallel

Doppler shift = $\Delta f = f_r - f_o$

$$= 2f_o \frac{v \cos \theta}{c}$$

$f_o =$ transmitted frequency
$f_r =$ reflected frequency
$v =$ velocity of red blood cells
$c =$ speed of ultrasound in blood

Doppler

- 100 Patients with severe AS
- $V_{\text{max}}$ was obtained in:
  - apical window in 39%
  - right parasternal window in 50%

ASE guidelines: “accurate data recording mandates multiple acoustic windows in order to determine the highest velocity”

Bernoulli Equation

\[ P_1 + \frac{1}{2} \rho v_1^2 + \rho gh_1 = P_2 + \frac{1}{2} \rho v_2^2 + \rho gh_2 \]

- Pressure Energy
- Kinetic Energy per unit volume
- Potential Energy per unit volume

Flow velocity \( v_1 \)
Flow velocity \( v_2 \)

The often cited example of the Bernoulli Equation or "Bernoulli Effect" is the reduction in pressure which occurs when the fluid speed increases.

\[ A_2 < A_1 \]
\[ v_2 > v_1 \]
\[ P_2 < P_1 \]

Increased fluid speed, decreased internal pressure.
Bernoulli

• Simplified Bernoulli:
  - \( \Delta P = 4V^2 \)
  - \( \Delta P_{\text{max}} = 4V_{\text{max}}^2 \)

• Proximal velocity should be included if it is >1.5 m/s (aortic insufficiency, fever, AV fistula...) or if aortic velocity is <3.0 m/s
  - \( \Delta P_{\text{max}} = 4(V_{\text{max}} - V_{\text{proximal}})^2 \)

AVA Calculation

\[ A_2 = \frac{A_1 \cdot v_1}{v_2} \]

LVOT Diameter

Assuming LVOT is circular

\[ A_1 = 3.14 \times \left( \frac{\text{Diameter}}{2} \right)^2 \]

- If \( D = 2 \text{ cm} \), then \( A_1 = 3.14 \text{ cm}^2 \)
- If \( D = 1.8 \text{ cm} \), then \( A_1 = 2.54 \text{ cm}^2 \)
- \( 2 \text{ mm} \rightarrow 20\% \) difference

LVOT Area

Tandon A et al. JACC Cardiovasc Imaging. 2013 Feb;6(2):184-95
LVOT Area

- 50 patients (25 with AS and 25 without AS)
- LVOT area and AVA were estimated using 2D TTE
- LVOT area and diameters were measured using 256-slice CCTA and 3D TTE
- LVOTs were oval in 96% of the patients without AS
- Eccentricity index \((D2/D1) = 1.26\pm0.09\) by CCTA
- This resulted in underestimation of LVOT area and AVA on 2D TTE by 17%

Dimensionless Index

\[ DI = \frac{LVOT \text{ VTI}}{AV \text{ VTI}} \]

Severe AS if DI $<$ 0.25

Dimensionless Index

488 patients with:
- at least mild AS
- LVEF ≥50%
- No or minimal subjective symptoms

Rusinaru D et al. JACC Cardiovasc Imaging. 2015 Jul;8(7):766-75
Planimetry

Planimetry

- Geometric orifice area (GOA) is difficult to trace, especially when the valve is heavily calcified
- As blood flows toward a stenotic valve, it converges beyond the GOA
- Vena contracta is the effective orifice area (EOA)

EOA vs. GOA

\[
C_c = \frac{\text{Effective orifice area}}{\text{Anatomic area}}
\]

= 0.71-0.90 \ (N = 35)

Low-Flow Low-Gradient Aortic Stenosis

• AVA <1 cm² but aortic jet velocity < 4.0 m/s
• Low flow = stroke volume index (SVI) ≤35 mL/m²
• PARTNER trial (971 patients):
  – 530 patients (55%) had low flow
  – 225 patients (23%) had low flow and low LVEF
  – 147 patients (15%) had low flow, low LVEF, and low mean gradient (<40 mmHg)
  – 2-year mortality was significantly higher in patients with low flow (47% vs. 34%; HR = 1.5 [1.25–1.89])

Hermann HC et al. Circulation 2013 Jun 11;127(23):2316-26
2014 ACC/AHA Guidelines

CLASS IIa (*Level of Evidence: B*)

- Low-dose dobutamine stress testing using echocardiographic or invasive hemodynamic measurements is reasonable in patients with stage D2 AS with all of the following:
  - calcified aortic valve with reduced systolic opening
  - LVEF <50%
  - calculated AVA <1.0 cm², and
  - aortic velocity <4.0 m/s

Nishimura RA *et al.* J Am Coll Cardiol. 2014 Jun 10;63(22):2438-8
Protocol

- Start dobutamine at 2.5-5 mg/kg/min
- Increased every 3-5 min (maximum 10-20 mg/kg/min)
- Stop infusion when:
  - positive result is obtained
  - heart rate begins to rise >10-20 bpm over baseline
  - heart rate exceeds 100 bpm, or
  - if patient develops arrhythmia
- Record LVOT velocity and optimal AS jet velocity
- LVOT diameter is measured at baseline and used to calculate AVA at each stage
- Measure biplane EF to assess for contractile reserve

Dobutamine Stress Echocardiogram

• Patients with **fixed** AS demonstrate increased cardiac output and transvalvular gradient with no change in AVA (<1 cm²)
• Patients with **relative** AS have an increased aortic valve area but no change in gradient
• Patients without contractile reserve have **indeterminate** AS because they are unable to increase their cardiac output with dobutamine

*deFilippi CR et al. Am J Cardiol. 1995 Jan 15;75(2):191-4*
True Stenosis vs. Pseudo-stenosis

Cardiac Output (L/min, assumes HR 76 bpm, SEP 300 ms)

Transvalvular Gradient (mmHg)

Transvalvular Flow (ml/sec)

AVA 0.7
AVA 1.0
AVA 1.5

Grayburn PA et al. Circulation. 2006 Feb 7;113(5):604-6
Aortic Valve Calcification by CT

179 patients with severe AS

Aortic Valvular Area, cm²

Aortic valve calcification, AU

R = -0.63

Adjusted Aortic Valvular Area, cm²/m²

Aortic valve calcification, AU

R = -0.67

Mean Pressure Gradient, mm Hg

Aortic valve calcification, AU

R = 0.78

Mean Peak Velocity, cm/sec

Aortic valve calcification, AU

R = 0.79

Cueff C et al. Heart. 2011 May;97(9):721-6
Aortic Valve Calcification

<table>
<thead>
<tr>
<th>LVEF ≤40% (N = 47)</th>
<th>Severe AS</th>
<th>Non-severe AS</th>
<th>PPV 97%</th>
<th>NPP 80%</th>
</tr>
</thead>
<tbody>
<tr>
<td>CS ≥1651</td>
<td>36</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CS &lt;1651</td>
<td>2</td>
<td>8</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Sensitivity 95% Specificity 89%

Cueff C et al. Heart. 2011 May;97(9):721-6
Low-Flow Low-Gradient Severe AS

Prognosis

157 patients with:
- AVA ≤ 0.75 cm²
- EF ≤ 35%
- Mean gradient ≤ 30 mmHg

Contractile Reserve

Increase in SV (VTI) of ≥20% following dobutamine

136 patients with AS:
- AVA 0.7 cm² [0.6-0.8]
- Mean gradient 29 mmHg [23-34]
- Group I: contractile reserve
- Group II: no contractile reserve

Contractile Reserve

- 81 patients with symptomatic AS:
  - AVA ≤1 cm²
  - EF ≤ 40%
  - Mean gradient ≤40 mmHg
  - No contractile reserve

- Operative mortality was 22%

Patterns of Severe Aortic Stenosis

Causes of Low Flow with Normal EF

- Pronounced Concentric Remodeling
- Impaired Longitudinal systolic function
- Atrial Fibrillation
- Mitral Regurgitation
- Mitral Stenosis
- Tricuspid Regurgitation

LV ejection time → Reduced Forward Stroke Volume → Reduced Transvalvular flow rate → Low-Flow, Low gradient AS with Preserved LVEF

Prevalence of Paradoxical Low-Flow Low-Gradient Severe Aortic Stenosis

## Prognosis

<table>
<thead>
<tr>
<th></th>
<th>Normal Flow (SVI &gt;35 mL/m²)</th>
<th>Low Flow (SVI ≤35 mL/m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>N</strong></td>
<td>331</td>
<td>181</td>
</tr>
<tr>
<td><strong>AVA (cm²)</strong></td>
<td>0.84±0.18</td>
<td>0.76±0.23</td>
</tr>
<tr>
<td><strong>Peak velocity (m/s)</strong></td>
<td>4.0±0.7</td>
<td>3.5±0.9</td>
</tr>
<tr>
<td><strong>Mean gradient (mmHg)</strong></td>
<td>40±15</td>
<td>32±17</td>
</tr>
<tr>
<td><strong>DI</strong></td>
<td>0.23±0.05</td>
<td>0.24±0.07</td>
</tr>
<tr>
<td><strong>LVEF (%)</strong></td>
<td>68±7</td>
<td>62±8</td>
</tr>
<tr>
<td><strong>CI (L/min/m²)</strong></td>
<td>2.80±0.54</td>
<td>2.15±0.42</td>
</tr>
</tbody>
</table>

Hachicha Z et al. Circulation. 2007 Jun 5;115(22):2856-64
Prognosis

Hachicha Z et al. Circulation. 2007 Jun 5;115(22):2856-64
Prognosis

A

Event-Free Survival, (%)

Follow-up Time, (years)

N patients at risk: 187 157 128 98 55 32 20 11 6

B

Aortic Valve Replacement, (%)

Follow-up Time, (years)

N patients at risk: 187 157 128 98 55 32 20 8 5 3

C

Overall Survival, (%)

Follow-up Time, (years)

N patients at risk: 187 178 163 141 101 75 56 35 22

D

Cardiovascular Survival, (%)

Follow-up Time, (years)

N patients at risk: 187 178 163 141 101 75 56 35 22

Prognosis - PARTNER Trial

A - high risk
B - inoperable

PARTNER (N=971)

Normal Flow (N=441, 45%)

Low Flow (N=530, 55%)

LF NEF (N=304, 31%)

LF LEF (N=225, 23%)

LF LEF NG (N=78, 8%)

LF LEF LG (N=147, 15%)

TAVR (N=170)
SAVR (N=180)
TAVR (N=85)
MM (N=95)
Prognosis - PARTNER Trial

Hermann HC et al. Circulation. 2013 Jun 11;127(23):2316-26
Prognosis – SEAS Trial

## Prognosis

<table>
<thead>
<tr>
<th></th>
<th>Moderate AS</th>
<th>LG/LF AS</th>
<th>LG/NF AS</th>
<th>HG AS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>N</strong></td>
<td>420</td>
<td>57</td>
<td>85</td>
<td>247</td>
</tr>
<tr>
<td><strong>AVA (cm²)</strong></td>
<td>1.3 [1.1-1.5]</td>
<td>0.8 [0.7-0.9]</td>
<td>0.9 [0.8-0.9]</td>
<td>0.7 [0.6-0.8]</td>
</tr>
<tr>
<td><strong>Peak velocity (m/s)</strong></td>
<td>2.9 [2.5-3.3]</td>
<td>3.3 [2.9-3.7]</td>
<td>3.7 [3.5-3.9]</td>
<td>4.6 [4.2-5.0]</td>
</tr>
<tr>
<td><strong>Mean gradient (mmHg)</strong></td>
<td>20.0 [15.0-27.0]</td>
<td>30.0 [20.5-34.5]</td>
<td>34.0 [30.0-37.0]</td>
<td>53.0 [45.0-65.0]</td>
</tr>
<tr>
<td><strong>LVEF (%)</strong></td>
<td>64.0 [59.0-68.0]</td>
<td>60.0 [55.0-67.0]</td>
<td>65.0 [60.0-69.0]</td>
<td>65.0 [59.0-70.0]</td>
</tr>
<tr>
<td><strong>SVI (mL/m²)</strong></td>
<td>43.7 [37.0-50.4]</td>
<td>30.1 [27.2-32.2]</td>
<td>42.4 [39.0-45.4]</td>
<td>41.0 [33.5-48.0]</td>
</tr>
</tbody>
</table>

Prognosis

A

Medical management

B

Medical and surgical management

2014 ACC/AHA Guidelines

Class IIa (Level of Evidence: C)

- AVR is reasonable in symptomatic patients with low-flow/low-gradient severe AS (stage D3) with an LVEF ≥50%, a calcified aortic valve with significantly reduced leaflet motion, and a valve area ≤1.0 cm² only if clinical, hemodynamic, and anatomic data support valve obstruction as the most likely cause of symptoms and data recorded when the patient is normotensive (systolic BP <140 mmHg) indicate:
  - aortic velocity <4 m/s or mean gradient <40 mmHg
  - stroke volume index <35 mL/m², and
  - indexed valve area ≤0.6 cm²/m²

Nishimura RA et al. J Am Coll Cardiol. 2014 Jun 10;63(22):2438-8
Treatment of Aortic Stenosis

Nishimura RA et al. J Am Coll Cardiol. 2014 Jun 10;63(22):2438-8
Conclusion

• Echocardiography remains the main diagnostic modality for the assessment of valvular heart disease
• However, it is prone to operator errors
• When assessing the severity of aortic stenosis, you should incorporate 2D-image, Doppler measurements, LVEF, SVI, and other available imaging modalities
• Dobutamine echocardiography is useful when LVEF is depressed
• AVR is indicated for symptomatic patients with paradoxical low-flow, low-gradient, severe aortic stenosis
THANK YOU