Ο ρόλος της απεικόνισης στις παθήσεις αορτικής βαλβίδος

Δυσαρμονία συμπτωμάτων και βαρύτητας αορτικής στένωσης

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Ιπποκράτειο Νοσοκομείο
Valvular Heart Disease

The Need for Concordance Between Examination and Echocardiogram

Essential questions in the evaluation for valvular intervention

Guidelines on the management of valvular heart disease

<table>
<thead>
<tr>
<th>Question</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Is valvular heart disease severe?</td>
<td></td>
</tr>
<tr>
<td>Does the patient have symptoms?</td>
<td></td>
</tr>
<tr>
<td>Are symptoms related to valvular disease?</td>
<td></td>
</tr>
<tr>
<td>What are patient life expectancy(^a) and expected quality of life?</td>
<td></td>
</tr>
<tr>
<td>Do the expected benefits of intervention (vs. spontaneous outcome)</td>
<td></td>
</tr>
<tr>
<td>outweigh its risks?</td>
<td></td>
</tr>
<tr>
<td>What are the patient's wishes?</td>
<td></td>
</tr>
<tr>
<td>Are local resources optimal for planned intervention?</td>
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</tbody>
</table>
Discordance between symptoms and aortic severity

Role of imaging

- **Natural History of AS**
- **Pathophysiology of AS**
- **Classification of AS**
- **Role of Imaging**
  - Echocardiographic assessment
    - Caveats to Be Considered
    - Specific Groups
  - Cardiac CT
  - CMR
  - Cardiac Catheterization
- **Conclusions**
Natural History of Medically Treated AS

Once symptoms develop in a patient with aortic stenosis the average survival with medical treatment is 2-5 years

Discordance between symptoms and aortic severity

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Patterns of Ventricular Remodeling in Valve Disease

Look at the ventricle!

Ventricular Remodeling is the alteration in ventricular architecture, with associated increased volume and altered chamber configuration, driven on a histological level by a combination of pathologic myocyte hypertrophy, myocyte apoptosis, myofibroblast proliferation, and interstitial fibrosis.

Pathophysiology of AS

Dweck et al, J Am Coll Cardiol 2012;60:1854–63
Severe AS

Flow, Mean Gradient and LV geometry

Pibarot et al, J Am Coll Cardiol 2012;60:1845–53
Discordance between symptoms and aortic severity

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## Stages of Valvular AS

<table>
<thead>
<tr>
<th>Stage</th>
<th>Definition</th>
<th>Valve Anatomy</th>
<th>Valve Hemodynamics</th>
<th>Hemodynamic Consequences</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>At risk of AS&lt;br&gt;• Bicuspid aortic valve (or other congenital valve anomaly)&lt;br&gt;• Aortic valve sclerosis</td>
<td>• Bicuspid aortic valve&lt;br&gt;• Aortic valve sclerosis</td>
<td>• Aortic (V_{\text{max}}) &lt;2 m/s</td>
<td>• None</td>
<td>• None</td>
</tr>
<tr>
<td>B</td>
<td>Progressive AS&lt;br&gt;• Mild-to-moderate leaflet calcification of a bicuspid or trileaflet valve with some reduction in systolic motion or&lt;br&gt;• Rheumatic valve changes with commissural fusion</td>
<td>• Mild AS: Aortic (V_{\text{max}}) 2.0–2.9 m/s or mean (\Delta P) &lt;20 mm Hg&lt;br&gt;• Moderate AS: Aortic (V_{\text{max}}) 3.0–3.9 m/s or mean (\Delta P) 20–39 mm Hg</td>
<td>• Early LV diastolic dysfunction may be present&lt;br&gt;• Normal LVEF</td>
<td>• None</td>
<td>• None</td>
</tr>
<tr>
<td>C</td>
<td>Asymptomatic severe AS&lt;br&gt;C1 Asymptomatic severe AS&lt;br&gt;C2 Asymptomatic severe AS with LV dysfunction</td>
<td>• Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening</td>
<td>• Aortic (V_{\text{max}}) ≥4 m/s or mean (\Delta P) ≥40 mm Hg&lt;br&gt;• AWA typically ≤1.0 cm² (or AWAi ≤0.6 cm²/m²)&lt;br&gt;• Very severe AS is an aortic (V_{\text{max}}) ≥5 m/s or mean (\Delta P) ≥60 mm Hg&lt;br&gt;• AWA typically ≤1.0 cm² (or AWAi ≤0.6 cm²/m²)</td>
<td>• LV diastolic dysfunction&lt;br&gt;• Mild LV hypertrophy&lt;br&gt;• Normal LVEF</td>
<td>• None: Exercise testing is reasonable to confirm symptom status&lt;br&gt;• LVEF &lt;50%</td>
</tr>
<tr>
<td>D</td>
<td>Symptomatic severe AS&lt;br&gt;D1 Symptomatic severe high-gradient AS&lt;br&gt;D2 Symptomatic severe low-flow/low-gradient AS with reduced LVEF&lt;br&gt;D3 Symptomatic severe low-gradient AS with normal LVEF or paradoxical low-flow severe AS</td>
<td>• Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening</td>
<td>• Aortic (V_{\text{max}}) ≥4 m/s or mean (\Delta P) ≥40 mm Hg&lt;br&gt;• AWA typically ≤1.0 cm² (or AWAi ≤0.6 cm²/m²) but may be larger with mixed AS/AR&lt;br&gt;• Aortic (V_{\text{max}}) ≥4 m/s at any flow rate</td>
<td>• LV diastolic dysfunction&lt;br&gt;• LV hypertrophy&lt;br&gt;• Pulmonary hypertension may be present</td>
<td>• Exertional dyspnea or decreased exercise tolerance&lt;br&gt;• Exertional angina&lt;br&gt;• Exertional syncope or presyncope&lt;br&gt;• HF&lt;br&gt;• Angina&lt;br&gt;• Syncope or presyncope&lt;br&gt;• LVEF &lt;50%</td>
</tr>
</tbody>
</table>
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Discordance between symptoms and aortic severity

Possible causes

- Measurement errors
- Small body size
- Inconsistency in guidelines criteria
- Low flow state
Measurement Pitfalls: Vmax and PG

Underestimation of Vmax

Overestimation of Vmax

Underestimation


Overestimation

**Measurement Pitfalls: LVOT Diameter**

- **Underestimation of LVOT$_D$:**
  - Poor acoustic window
  - Image truncated
  - LVOT elliptical

- **Overestimation of LVOT$_D$:**
  - Too far in the LV
  - Oblique measure
Measurement Pitfalls: LVOT Velocity

- **Underestimation of $V_{\text{LVOT}}$:**
  
  Too far from the valve
  
  Doppler beam not well aligned

- **Overestimation of $V_{\text{LVOT}} (>1.5\text{m/s})$:**
  
  Subvalvular flow acceleration
  
  Too far in the valve/aorta
Energy Loss Index
Impact of pressure Recovery

- Becomes relevant in patients with moderate to severe AS and small aortas and leads to overestimation of severity.
- Pressure recovery can be calculated with ‘Energy Loss Index’ measurement:
  - $\text{ELI} = [(\text{AVA} \times \text{AA}) / (\text{AA} - \text{AVA})] / \text{BSA}$,
  * AA: CSA aorta at SNJ

Symptomatic AS

Does Systemic Hypertension Play an Additional Role?

- In hypertensive symptomatic AS patients, symptoms develop at a relative earlier stage of the disease, with larger valve areas and lower stroke loss, probably because of the additional overload due to the hypertension itself.

- In patients with coexisting hypertension and AS, hypertension should be treated more aggressively to delay the occurrence of symptoms, and these patients should be followed-up more closely.

**Antonini-Canterin et al, Hypertension. 2003;41:1268-1272**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Hypertensives</th>
<th>Normotensives</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>n (%)</td>
<td>62 (32)</td>
<td>131 (68)</td>
<td></td>
</tr>
<tr>
<td>Age, y</td>
<td>67 ± 9</td>
<td>67 ± 8</td>
<td>NS</td>
</tr>
<tr>
<td>Male/female</td>
<td>36/26</td>
<td>77/54</td>
<td>NS</td>
</tr>
<tr>
<td>Systolic BP, mm Hg</td>
<td>142 ± 26</td>
<td>134 ± 23</td>
<td>0.032</td>
</tr>
<tr>
<td>Diastolic BP, mm Hg</td>
<td>70 ± 13</td>
<td>67 ± 13</td>
<td>NS</td>
</tr>
<tr>
<td>Aortic valve area, cm²</td>
<td>0.82 ± 0.2</td>
<td>0.74 ± 0.2</td>
<td>0.021</td>
</tr>
<tr>
<td>Stroke work loss, %</td>
<td>25.5 ± 7</td>
<td>28.0 ± 7</td>
<td>0.022</td>
</tr>
<tr>
<td>Mean aortic gradient, mm Hg</td>
<td>49 ± 17</td>
<td>52 ± 16</td>
<td>NS</td>
</tr>
<tr>
<td>NYHA class</td>
<td>2.09 ± 1.0</td>
<td>2.01 ± 1.1</td>
<td>NS</td>
</tr>
<tr>
<td>Angina/heart failure/syncope</td>
<td>24/41/5</td>
<td>47/75/14</td>
<td>NS</td>
</tr>
</tbody>
</table>
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Discordance in aortic severity

Specific Groups

• Asymptomatic severe AS
• Low Flow- low gradient severe AS
Is the patient truly asymptomatic?

Treatment Algorithm for Asymptomatic Severe Aortic Stenosis:

Basis of Current American and European Guidelines

---

<table>
<thead>
<tr>
<th>Patient with Asymptomatic Severe Aortic Stenosis (AS)</th>
<th>ESC/EACTS Aortic valve replacement (AVR) recommendation</th>
<th>AHA/ACC AVR recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean gradient (MG): ≥40 mm Hg</td>
<td>Class I</td>
<td>Class I</td>
</tr>
<tr>
<td>Aortic valve area: ≤1.0 cm²</td>
<td>Class Ia</td>
<td>Class Ia</td>
</tr>
<tr>
<td>Ejection fraction: &gt;50%</td>
<td>Class Iib</td>
<td>Class Iib</td>
</tr>
<tr>
<td>Limiting symptoms: Angina</td>
<td>≥5.5 m/s = Class Ila</td>
<td>≥5 m/s = Class Ila</td>
</tr>
<tr>
<td>Syncope</td>
<td>Class Ia</td>
<td>Class Iib</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>Decreased exercise tolerance</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>Abnormal systolic blood pressure response (drop or</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>&lt;20 mm Hg rise)</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>Stress imaging shows increase in MG with exercise by</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>&gt;20 mm Hg</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>Severe left ventricular hypertrophy</td>
<td>≥5 m/s = Class Ila</td>
<td>Class Iib</td>
</tr>
<tr>
<td>Sustained B-type Natriuretic Peptide (BNP) elevation</td>
<td>Class Ia</td>
<td>Class Ia</td>
</tr>
<tr>
<td>Peak velocity</td>
<td>Class Ia</td>
<td>Class Ia</td>
</tr>
<tr>
<td>Peak velocity progression ≥0.3 m/s/year</td>
<td>Class Ia</td>
<td>Class Ia</td>
</tr>
</tbody>
</table>

If no AVR performed, recommend clinical and echocardiographic follow-up each 6-12 months (Class I)

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Criteria of an abnormal exercise test

Asymptomatic severe AS

Patients with apparently asymptomatic AS with symptoms detected during exercise testing have lower peak myocardial oxygen consumption and lower peak stroke index than those patients who remained asymptomatic.

Amato et al, Heart 2001;86:381–386
Exercise Doppler echo versus resting echo and exercise ECG parameters

Incremental prognostic value

Prognosis in Asymptomatic severe AS

Treatment Algorithm for Asymptomatic Severe Aortic Stenosis:
Basis of Current American and European Guidelines

Prognosis

- Valve
- Left Ventricle
- Aorta
Evaluation of the aortic valve

The degree of AVC is a strong predictor of cardiac events

Jet velocity

Patients with a moderate/heavily calcified valve and an increase in jet velocity of .0.3 m/s/year had a particularly poor prognosis.

Although severe AS is defined as Vmax >4m/s, patients with a Vmax >5.0 or >5.5 cm/s have a higher risk of adverse events.

Otto et al, Circulation 1997;95:2262–2270

Rosenhek et al, Circulation 2010;121:151–156
The presence of an inappropriate LV mass (>110% of that expected for body size, gender, and wall stress) heralded a 4.5 increased risk of mortality independent of other known risk factors.

_Cioffi et al, Heart 2011;97:301–307_

Left Ventricular Diastolic Dysfunction

Symptomatic status as a function of diastolic grade

Symptomatic status in severe AS was independently associated with the severity of LV remodeling, diastolic dysfunction, and LA volume increase.

There was significantly higher 1-year survival rate in patients with E/E’ < 15 compared with patients with E/E > 15 among both asymptomatic (n = 39) and symptomatic (n = 79) patients.

Biner et al, J Am Coll Cardiol Img 2010;3:899–907
Left atrial size

Diastolic function, represented as left atrial diameter, is related to mortality in asymptomatic AS.
LA mechanics and stiffness for risk stratification in severe AS

Control

Asymptomatic severe AS

Symptomatic severe AS

Left Ventricular Mechanics
2D myocardial strain imaging

- Longitudinal strain was uniformly low in patients with severe AS and lower in symptomatic patients.
- Circumferential mechanics may be viewed as the “myocardial compensatory domain,” especially in increased afterload situations.

- Longitudinal strain progressively declined as patients became symptomatic, and the decline continued further with the development of systolic LV dysfunction.

*Carasso et al, J Am Soc Echocardiogr 2015;28:218-25*
• Apical rotation, showing supernormal values in asymptomatic patients and low values in symptomatic patients.
• Detection of lower than normal rotation at any point during follow-up may suggest loss of compensation, which may herald clinical and prognostic deterioration in patients with severe AS.

All-cause mortality according to GLS and treatment

Myocardial Strain in Low LVEF, Low-Gradient AS

Rest GLS

Stress GLS

Dahou et al, Circ Cardiovasc Imaging. 2015;8:e002117
Valvuloarterial Impedance

Total LV load is the sum of valvular load and arterial load and can be estimated with Valvuloarterial Impedance.

\[ Z_{va} = \frac{SAP + MG}{SVI} \]

where SAP is the systolic arterial pressure and MG is the mean transvalvular pressure gradient. Hence \( Z_{va} \) represents the valvular and arterial factors that oppose ventricular ejection.

Philippe Pibarot J Am Coll Cardiol 2012;60:169–80)
Global LV Afterload

Prognostic value of Zva

Increased Zva may be observed in hypertension and moderate AS or severe AS

## Risk factors corresponding threshold values

### Asymptomatic severe AS

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>High-risk values</th>
<th>Guideline recommendation</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Valve assessment</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak jet velocity</td>
<td>$&gt;5 \text{ m/s}$</td>
<td>AHA (IIb)</td>
<td>Bonow et al. (^{44}); Rosenhek et al. (^{8}); Bonow et al. (^{44}); Rosenhek et al. (^{7})</td>
</tr>
<tr>
<td>Aortic valve area</td>
<td>$&lt;0.6 \text{ cm}^2$</td>
<td>AHA (IIb)</td>
<td></td>
</tr>
<tr>
<td>Rate of progression of jet velocity</td>
<td>$\geq 0.3 \text{ m/s/year}$</td>
<td>ESC (IIa) if valve calcification moderate to severe</td>
<td></td>
</tr>
<tr>
<td>Degree of valve calcification</td>
<td>Heavily calcified (extensive calcification of all cusps)</td>
<td></td>
<td>Rosenhek et al. (^{7})</td>
</tr>
<tr>
<td>Valvulo-arterial impedance</td>
<td>$&gt;4.5–4.9 \text{ mmHg/mm/m}^2$</td>
<td></td>
<td>Hachicha et al. (^{34}); Lancellotti et al. (^{10})</td>
</tr>
<tr>
<td><strong>LV assessment</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left ventricular systolic dysfunction</td>
<td>Ejection fraction $&lt;50%$</td>
<td>ESC (I) and AHA (I)</td>
<td>Bonow et al. (^{45}); Vahanian et al. (^{46}); Vahanian et al. (^{46})</td>
</tr>
<tr>
<td>Left ventricular hypertrophy</td>
<td>$\geq 15 \text{ mm}$ unless this is due to hypertension</td>
<td>ESC (IIb)</td>
<td></td>
</tr>
<tr>
<td>Left ventricular mass</td>
<td>$&gt;110%$ of that expected for body size, gender and</td>
<td></td>
<td>Cioffi et al. (^{6})</td>
</tr>
<tr>
<td></td>
<td>wall stress</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Strain imaging</td>
<td>Global Longitudinal strain $&lt;15.9%$</td>
<td></td>
<td>Lancellotti et al. (^{10}); Lancellotti et al. (^{10})</td>
</tr>
<tr>
<td>Indexed left atrial area</td>
<td>$&gt;12.2 \text{ cm}^2/m^2$</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Stress testing</strong></td>
<td></td>
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<tr>
<td>Treadmill exercise stress test</td>
<td>Symptoms on exercise. Fall in blood pressure. Complex</td>
<td>ESC (I) and AHA (IIb); ESC (IIa) and AHA (IIb)</td>
<td>Amato et al. (^{11}); Das et al. (^{28})</td>
</tr>
<tr>
<td></td>
<td>ventricular arrhythmias on exercise</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exercise echocardiography</td>
<td>Mean pressure increase of $\geq 18–20 \text{ mmHg}$</td>
<td></td>
<td>Lancellotti et al. (^{9}); Maréchaux et al. (^{29})</td>
</tr>
<tr>
<td><strong>Biomarkers</strong></td>
<td></td>
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</tr>
<tr>
<td>Natriuretic peptides</td>
<td>BNP $\geq 130 \text{ pg/mL}$</td>
<td></td>
<td>Bergler-Klein et al. (^{33})</td>
</tr>
</tbody>
</table>
Discordance in aortic severity

Specific Groups

• Asymptomatic severe AS

• Low Flow - low gradient severe AS
LFLG severe AS

Evaluation with TTE

Low-flow, low-gradient severe AS
AVA <1.0 cm² (<0.6 cm²/m²)
LV stroke volume index < 35 mL/m²
Mean gradient < 40 mmHg

LVEF

< 50%

Classical low-flow low-gradient AS

AVR > 1.0 cm²
Mean gradient < 40 mmHg
Pseudo-severe AS

AVA < 1.0 cm²
Mean gradient > 40 mmHg
Low-dose dobutamine stress echocardiography
True severe AS

≥ 50%

Paradoxical low-flow low-gradient AS

No flow reserve
(Increase in stroke volume < 20%)
AVA unchanged
Mean gradient unchanged

MDCT
Aortic valve calcium score
Men: > 2065 AU
Women: > 1275 AU

Projected aortic valve area

TOPAS multicenter Group

AVA ≤ 1.2 cm² / LVEF ≤ 40% / MPG < 40 mm Hg

• Σε 101 ασθενείς που αντιμετωπίσθηκαν συντηρητικά το AVAproj ήταν ανεξάρτητος προγνωστικός δείκτης της θνησιμότητας.

Blais et al. Circulation. 2006;113:711-721
Clavel et al. Circulation. 2008; 118; S234-S242
Clavel et al, J Am Soc Echocardiogr 2010;23:380-6
Flow rate and Stress echo
Patients with LFLG AS

Flow rate (Q) = \frac{SV}{Ejection Time (ms)}

- In patients with flow rate >200ml/s
- AVA at rest is the true AVA

<table>
<thead>
<tr>
<th>Change in AVA During Stress, Stratified by Resting LVEF, SVi, and Flow</th>
<th>n</th>
<th>Rest AVA, cm²</th>
<th>Stress AVA, cm²</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEF &lt;50%</td>
<td>37</td>
<td>0.75 ± 0.14</td>
<td>0.87 ± 0.21</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LVEF ≥50%</td>
<td>30</td>
<td>0.79 ± 0.10</td>
<td>0.93 ± 0.23</td>
<td>0.007</td>
</tr>
<tr>
<td>SVi &lt;35 ml/m²</td>
<td>47</td>
<td>0.74 ± 0.12</td>
<td>0.86 ± 0.23</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>SVi ≥35 ml/m²</td>
<td>20</td>
<td>0.83 ± 0.10</td>
<td>0.98 ± 0.21</td>
<td>0.016</td>
</tr>
<tr>
<td>Q &lt;200 ml/s</td>
<td>48</td>
<td>0.74 ± 0.12</td>
<td>0.89 ± 0.25</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Q ≥200 ml/s</td>
<td>19</td>
<td>0.85 ± 0.09</td>
<td>0.89 ± 0.12</td>
<td>0.19</td>
</tr>
</tbody>
</table>

Adjusted Logistic Regression Analysis of Rest Function
Covariates Associated With TSAS

<table>
<thead>
<tr>
<th>Coefficient</th>
<th>OR (95% CI)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resting LVEF, %</td>
<td>0.03</td>
<td>1.03 (0.98–1.10)</td>
</tr>
<tr>
<td>Resting SVi, ml/m²</td>
<td>0.001</td>
<td>1.00 (0.90–1.10)</td>
</tr>
<tr>
<td>Resting flow rate, ml/s</td>
<td>-0.05</td>
<td>1.05 (1.00–1.10)</td>
</tr>
</tbody>
</table>
Discordance between symptoms and aortic severity

Role of imaging

• Natural History of AS
• Pathophysiology of AS
• Classification of AS
• Role of Imaging
  – Echocardiographic assessment
    • Caveats to Be Considered
    • Specific Groups
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• Conclusions
Impact of AVC burden on survival in patients with AS

Measuring AVC load provides incremental prognostic value for survival beyond clinical and Doppler echocardiographic assessment. Severe AVC independently predicts excess mortality after AS diagnosis, which is greatly alleviated by AVI. Thus, measurement of AVC by MDCT should be considered for not only diagnostic but also risk-stratification purposes in patients with AS.

*Clavel et al, J Am Coll Cardiol. 2014 Sep 23;64(12):1202-13.*
AVC measured on non-contrast CT has shown correlation to severity of AS and mortality

Paulsen et al, Journal of Cardiovascular Computed Tomography (2016) 1e7
Discordance between symptoms and aortic severity

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- Role of Imaging
  - Echocardiographic assessment
    - Caveats to Be Considered
    - Specific Groups
  - Cardiac CT
  - CMR
  - Cardiac Catheterization
- Conclusions
Myocardial fibrosis is an important morphological substrate of postoperative clinical outcome in patients with severe aortic stenosis and was not reversible after AVR over the 9 months of follow-up examined in this study. Because markers of longitudinal systolic function appear to indicate sensitively both the severity of myocardial fibrosis and the clinical outcome, they may prove valuable for preoperative risk assessment in patients with aortic stenosis

Weidemann et al, Circulation. 2009;120:577-584
Low gradient is associated with a higher degree of fibrosis, decreased longitudinal function, and poorer clinical outcome.
Discordance between symptoms and aortic severity

Role of imaging

- Natural History of AS
- Pathophysiology of AS
- Classification of AS
- Role of Imaging
  - Echocardiographic assessment
    - Caveats to Be Considered
    - Specific Groups
  - Cardiac CT
  - CMR
  - Cardiac Catheterization
- Conclusions
The optimal method to measure the transaortic gradient in AS

Simultaneous LV pressure and central Ao pressure with side-hole catheters.

Central ascending Aorta  Femoral Aorta  Endhole catheter

Rick A. Nishimura, and Blase A. Carabello Circulation. 2012;125:2138-2150
Discordance between symptoms and aortic severity

Role of imaging

- Natural History of AS
- Pathophysiology of AS
- Classification of AS
- Role of Imaging
  - Echocardiographic assessment
    - Caveats to Be Considered
    - Specific Groups
  - Cardiac CT
  - CMR
  - Cardiac Catheterization

- Conclusions
Discordance between symptoms and AS stenosis

**Asymptomatic severe AS**
- Natural History of AS
- Measurement errors
  - Tracing of MR jet
  - Underestimation of LVOTd
  - LVOT velocity underestimation (too far from LVOT)
- Small BSA
- Small aortas

**Symptomatic moderate AS**
- Concomitant disease
- Measurement errors
  - Underestimation of Vmax
  - Overestimation of LVOTd
  - LVOT velocity overestimation (too far from LVOT)
- Diastolic dysfunction
- Arrhythmias
- Hypertension
- Increased ZvA
Conclusions

• Among patients evaluated in clinical practice for AS, a large percentage present with discordance between symptoms and severe of AS.

• Careful clinical examination and multi-imaging assessment may be performed.

• Risk stratification of the asymptomatic patient with severe AS has evolved from a simple assessment of clinical history into an assessment of multiple variables which interact to determine the risk profile of a patient.

• Future research should investigate the utility and prognostic value of newer imaging modalities and emerging prognostic markers.
Left Ventricular Torsion

Relative to ED, the LV at ES has shortened in both the longitudinal (L→L’) and circumferential (C→C’) directions (axial strains).

A characteristic wringing motion (arrows) gives rise to a torsional shear given by the angle $\theta_{CL} = 90^\circ - \alpha$. One significant effect of torsion is that the greatest shortening (P→P’) occurs obliquely to C and L, in the approximate direction of the sub-epicardial fibers.

Young et al, Journal of Cardiovascular Magnetic Resonance 2012, 14:49
Long axis excursion in AS

- Long axis excursion is reduced even in the presence of normal fractional shortening or ejection fraction

Linear regression for septal excursion of the mitral annulus (cm) and left ventricular mass index (LVMI) (g/m²).

Inverse relation between Fibrosis and MAPSE

- Mitral ring displacement (mean±SD) assessed by M-mode echocardiography within the 3 groups of histologically confirmed myocardial fibrosis before AVR.
- Gradual inverse relation between mitral ring displacement and the severity of fibrosis.
- *P<0.05, #P<0.001 vs no fibrosis (ANOVA).

Frank Weidemann et al. Circulation. 2009;120:577-584
In patients with severe AS and preserved LVEF, there is a significant relationship between LV untwisting and LV filling pressures, suggesting a role for impaired LV untwisting in the pathophysiology of diastolic dysfunction in this setting.

Significant negative correlation between LV peak systolic strain and peak aortic velocity by continuous Doppler in 50 patients with moderate AS.

Steine et al, Am J Cardiol 2008;102:897–901
Differential effects of afterload on LV long- and short-axis function

Longitudinal strain in 6 LV segments from the apical 4 chamber view. Low baseline strain increased after AVR.

Circumferential strain in 6 segments from the short-axis view. Abnormally high baseline strain decreased after AVR.

*Carasso et al, Am Heart J 2009;158:540-5*
Myocardial Mechanics in Severe AS to LV Ejection Fraction

• Compensatory mechanisms (high circumferential strain in patients with preserved LVEF and increased apical rotation in patients with mild to moderate LV dysfunction) were observed in patients with severe AS.
• Compensatory mechanics were lost in patients with severe LV dysfunction.

Carasso et al, Am J Cardiol 2011;107:1052–1057
There was a negative correlation between LGE and peak systolic longitudinal strain considering the total LV.

Hoffmann et al, Am J Cardiol 2014;114:1083e1088

There was a significant decrease of LGE from base to apex of the LV.
Different clinical outcomes in patients with asymptomatic severe aortic stenosis according to the stage classification: Does the aortic valve area matter?


Abstract

BACKGROUND: The ACC/AHA guidelines introduced a new classification of severe aortic stenosis (AS) mainly based on maximum jet velocity (Vmax) and mean pressure gradient (mP<sub>PG</sub>) but not on aortic valve area (AVA). However, prognostic value of this new classification has not yet been fully evaluated.

METHODS AND RESULTS: We studied 1512 patients with asymptomatic severe AS enrolled in the CURRENT AS registry in whom surgery was not initially planned. Patients were divided into 2 groups: Group 1 (N=1222) comprised patients who met the recommendation for surgery: high-gradient (HG)-AS (Vmax>4.0 m/s or mP<sub>PG</sub>&gt;40 mmHg) with ejection fraction (EF)&lt;50%, or very HG-AS (Vmax&ge;5.0 m/s or mP<sub>PG</sub>&ge;60 mmHg), and Group 2 (N=1390) comprised patients who did not meet this recommendation. Group 2 was further subdivided into HG-AS with preserved EF (HGpEF-AS; N=418) and low-gradient (LG)-AS, but AVA&lt;1.0 cm<sup>2</sup> (N=992). The excess risk of Group 1 relative to Group 2 for the primary outcome measure (a composite of aortic valve-related death or heart failure hospitalization) was significant (adjusted HR: 1.92, 95%CI: 1.37-2.68, P=0.001). The excess risk of HGpEF-AS relative to LG-AS for the primary outcome measure was also significant (adjusted HR: 1.45, 95%CI: 1.11-1.89, P=0.006). Among LG-AS patients, patients with reduced EF (<50%) (LGpEF-AS, N=103) had extremely high cumulative 5-year incidence of all-cause death (95%).

CONCLUSION: Trans-aortic valve gradient in combination with EF was a good prognostic marker in patients with asymptomatic AS. However, patients with LGpEF-AS had extremely poor prognosis when managed conservatively.

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