Aortic Stenosis

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Upon completion of this session, the participant should be able to:

- Describe the mechanisms and causes of aortic stenosis
- Recognize the 2D TEE findings of aortic stenosis
- Assess and quantify aortic stenosis

Introduction

Aortic valve sclerosis refers to the thickening and calcification of the aortic valve leaflets and affects about ¼ of the U.S. population over age 65. In about 2% of Americans, this results in a large enough reduction in the orifice to obstruct flow, leading to the diagnosis of aortic stenosis (AS).\(^1\) Over 56,000 aortic valves are replaced annually in the United States, making AS the second most common reason for heart surgery, behind only coronary artery disease.\(^2\)

The triad of angina, syncope, and congestive heart failure describe the clinical manifestations of aortic stenosis that appear as the disease slowly progresses, usually after several asymptomatic decades. Chest auscultation revealing a course, late-peaking systolic ejection murmur may be the first sign of AS in asymptomatic patients. Echocardiography, however, is the main method of diagnosis and the technique of choice to follow disease progression.

Causes of Aortic Stenosis

Calcific AS, often called “senile” AS, is the most common underlying etiology of aortic stenosis. Although the underlying mechanism was once thought to be degeneration of the leaflets due to “wear and tear,” there is evidence to suggest the disease process is similar to that of plaque formation by atherosclerosis.\(^3,4\)

Whether due to injury or plaque, calcium deposits eventually accumulate on the valve cusps, leading to leaflet stiffness, fusion, and reduced mobility.

While a normal aortic valve has three cusps, a bicuspid valve has only two and is the most common congenital heart anomaly. There is a failure of one
of the commissures to fully develop, resulting in 2 leaflets of unequal size. A raphe, or ridge, is often present in the larger leaflet, giving the appearance of 3 cusps during diastole on echocardiographic examination. Bicuspid aortic valves are associated with aortic coarctation and dilatation of the thoracic aorta. The development of aortic stenosis results from leaflet calcification, much like a trileaflet valve, but occurs much earlier in life, often in the patient’s 40’s.5

Rheumatic heart disease is uncommon in developed countries, but still remains a potential cause of AS seen in the operating room. Unlike calcific AS, rheumatic AS results from fusion and calcification at the valve commissures rather than the leaflets themselves. The mitral valve is almost always also involved, and isolated rheumatic AS is quite rare. The three main etiologies of AS are summarized in Table 1 below.

Table 1. Summary of most common underlying causes of adult aortic stenosis:

<table>
<thead>
<tr>
<th>Cause</th>
<th>Calcific AS</th>
<th>Bicuspid Aortic Valve</th>
<th>Rheumatic AS</th>
</tr>
</thead>
<tbody>
<tr>
<td>OR Cases6</td>
<td>46%</td>
<td>33%</td>
<td>18%</td>
</tr>
<tr>
<td><strong>Leaflets</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adapted from (7)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calcified nodules</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Increased stiffness</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Raphe in larger leaflet</td>
<td></td>
<td>Leaflet thickening</td>
<td></td>
</tr>
<tr>
<td>May appear tricuspid</td>
<td></td>
<td>Commissural fusion</td>
<td></td>
</tr>
<tr>
<td>Secondary calcification</td>
<td></td>
<td>Leaflet thickening</td>
<td></td>
</tr>
<tr>
<td>Chordal shortening</td>
<td></td>
<td>Symptomatic at 45-60 yrs</td>
<td></td>
</tr>
<tr>
<td>Note:</td>
<td>Occurs ages 70+ yrs</td>
<td>½ AS, ½ AI, ½ no Sx</td>
<td>10-30 years after RF</td>
</tr>
<tr>
<td>“Senile” AS</td>
<td></td>
<td>Mitral valve almost always involved too</td>
<td></td>
</tr>
<tr>
<td>Normal “wear &amp; tear”</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

2D TEE Findings in Aortic Stenosis

Leaflet Imaging

Regardless of the underlying etiology of AS, there is a reduction in the valve orifice area and a decrease of leaflet separation. Examination with 2-D echo in the mid esophageal (ME) long axis view of the aortic valve will usually show the leaflets to be thickened in appearance. They often curve toward the midline of the aorta rather than opening parallel to the aortic wall, a finding known as “leaflet doming.” Leaflet separation of less than 15 mm is suggestive of significant AS. Short axis views of the valve may reveal leaflet calcification or commissural fusion. It may be
difficult to discern individual leaflets due to the high amount of calcium present. In these situations, lowering the transmit gain can help reduce reverberation artifact.

**Associated TEE Findings**

Left ventricular hypertrophy is a common compensatory mechanism in AS patients. According to the Law of Laplace, wall stress is equal to transmural pressure multiplied by the sphere’s radius divided by 2 times the wall thickness ($\sigma = \frac{P_r}{2h}$). As the left ventricle (LV) must generate higher pressures to force blood past the stenotic aortic valve, concentric hypertrophy allows the increased stress to be distributed over a greater myocardial mass. Unfortunately, this also leads to a decrease in LV compliance and coronary perfusion.

Post-stenotic aortic dilatation is present in about ¼ of AS patients, affecting mainly the sinotubular junction, although the proximal ascending aorta is frequently involved as well. Hemodynamic factors likely contribute to the process and dilation may stop following valve replacement. Abnormal aortic tissue may also play a role, as patients with congenital bicuspid aortic valves are more likely to have significant dilatation and progression of disease. The management of this finding is controversial and ranges from simple observation, to reinforcing the dilated portion with a synthetic wrap, to full ascending aortic replacement.

The amount of aortic regurgitation should be evaluated in all patients with AS. Stiff, calcified leaflets that fail to properly open can also fail to properly close. The degree of regurgitation can influence cardioprotection and cannulation strategies.

**Assessment of Aortic Stenosis**

**Doppler Findings**

Although only qualitative, color flow Doppler (CFD) often provides the first indication of significant AS. Laminar flow is seen in the left ventricular outflow tract (LVOT), while severe aliasing is seen in the proximal aorta. This is caused by flow acceleration through the stenotic aortic valve.
Spectral Doppler is used to measure the peak velocity through the aortic valve and can quantify the severity of AS (see next section). The continuous wave Doppler (CWD) signal should be aligned as parallel as possible through the aortic valve using either the deep transgastric view or the transgastric long axis view. Peak velocities higher than 2.5 m/s indicate some degree of AS. The shape of the displayed pattern is rounded, with a mid-systolic peak. Often times a “double envelope” is created, which contains the lower velocity profile of the LVOT embedded in the higher velocity aortic jet.

Quantifying the Severity of Aortic Stenosis

In 2009, the American Society of Echocardiography released guidelines for the quantification of AS severity.11 While a number of parameters can be measured, three are considered appropriate for clinical use in all patients with AS. They are: 1) AS jet velocity, 2) mean gradient, and 3) valve area by continuity equation. The cut-off values for each indicator are given in Table 2, and described in more detail below.

Table 2. Classification of Aortic Stenosis Severity (ACC/AHA Practice Guidelines).12

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Normal</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jet Velocity</td>
<td>&lt;1.7 m/s</td>
<td>&lt;3 m/s</td>
<td>3 – 4 m/s</td>
<td>&gt;4 m/s</td>
</tr>
<tr>
<td>Mean Gradient</td>
<td>&lt;25 mm Hg</td>
<td>25 – 40 mm Hg</td>
<td>&gt;40 mm Hg</td>
<td></td>
</tr>
<tr>
<td>Valve Area</td>
<td>&gt;2.5 cm²</td>
<td>&gt;1.5 cm²</td>
<td>1.0 – 1.5 cm²</td>
<td>&lt;1.0 cm²</td>
</tr>
</tbody>
</table>

Jet Velocity and Gradients

The aortic jet velocity can be obtained by aligning the CWD cursor through the aortic valve, parallel to blood flow. In general this requires either the deep transgastric or transgastric long axis view. The peak velocity, which is used to grade AS severity, is the highest value obtained and represents a single instantaneous measurement. The entire velocity envelope can be traced to yield a velocity time integral (VTI) and mean velocity. Compared to transthoracic echo, TEE tends to underestimate aortic jet velocities. Therefore, several separate measurements should be made, utilizing the highest values obtained.

A peak gradient can be calculated from the peak velocity using the simplified Bernoulli equation (ΔP=4V²). This is a “peak instantaneous” gradient, and will be slightly higher than the “peak to peak” gradient obtained in most catheterization labs. Nearly all echo machines will calculate a mean gradient when the velocity envelope is traced, but the mean gradient can also be estimated from the formula ΔPmean=2.4(V_max)².

Regardless of whether velocity, peak gradient, or mean gradient is used to grade AS severity, LV function significantly influences the result. This is because all gradients are dependent upon flow. With a decrease in cardiac output, as occurs with poor ventricular function, the aortic jet velocity, as well as any gradient, will also decrease. Severe mitral regurgitation and any left to right intracardiac shunts will also limit transvalvular flow, thereby underestimating the severity of AS. In these situations it is helpful to obtain a valve area, which is considered more constant and less dependent upon flow.
Valve area

The anatomical area of a stenotic aortic valve can often be obtained directly using planimetry. In the midesophageal aortic valve short axis view, the probe should be manipulated to make the imaging plane at the level of the leaflet tips. Cutting the valve obliquely will overestimate the valve area. Ideally, all leaflets should appear equal in size and an equilateral triangle can be traced out. Unfortunately, heavy calcium deposits can create significant shadowing, making it difficult to view the leaflets and impossible to identify the valve orifice. Additional limitations of planimetry for AS are tending to overestimate valve areas of less than 0.75cm², and underestimating the functional severity of stenosis in bicuspid aortic valves.13,14

The functional area of the stenotic aortic valve can be obtained by using the continuity equation, which also avoids some of the limitations of planimetry. The basis of the continuity equation is the assumption that the stroke volume (SV) proximal to the stenotic aortic valve (i.e. in the LVOT) must be equal to the SV just distal to the stenotic aortic valve (i.e. in the ascending aorta).

The cross sectional area (CSA) of the LVOT can be calculated by assuming it is a circle and using the formula CSA=πr². In practice, the diameter of the LVOT is measured in the ME aortic valve long axis view and the formula used is “CSA=π(d/2)²,” which simplifies to “CSA=0.785(d²).” The measurement should be taken in mid systole.
and, since the term is squared, repeated several times to minimize errors. As discussed above, the VTI of the aortic valve can be obtained using CWD. If a good double envelope is present, the VTI of the LVOT can be acquired by tracing out the LVOT velocity profile. Alternatively, pulsed wave Doppler (PWD) can be placed in the LVOT using the deep transgastric or transgastric long axis views and then tracing the LVOT velocity profile. As a short-cut, peak velocities of the LVOT and aortic valve can be substituted for their respective VTIs (sometimes called the ‘simplified continuity equation’), as long as the velocity curves are not atypically shaped.

Measure LVOT diameter in mid-systole. This value is squared so small errors result in large differences.

VTI of the LVOT measured using PWD. Aliasing will occur if sample volume cursor is actually in the aorta.