Echocardiographic evaluation of the aortic valve is an important aspect of the perioperative evaluation of surgical patients. The high resolution images of the aortic valve provided by TEE results from the close proximity of the valve to the esophagus. The application of Doppler echocardiography (pulsed wave, continuous wave, and color) with two- and three-dimensional imaging allows for the complete evaluation of regurgitant aortic valve pathology. At the conclusion of this lecture, the participant should be able to: 1) identify the 2-D and 3-D structural anatomy of the normal aortic valve 2) recognize anatomical pathology of aortic valve regurgitation 3) recite the mechanisms of aortic regurgitation 4) recognize how to quantify the degree of aortic regurgitation, and 5) diagnose the pathology of unknown examples of aortic regurgitation and their quantitative assessment.

Aortic Valve Anatomy

The aortic valve apparatus is comprised of the left ventricular outflow tract, valve cusps, sinuses of Valsalva, and proximal ascending aorta. The three aortic valve cusps are associated with three bulges or pouch-like dilations in the aortic wall called sinuses of Valsalva. Proximal to the aortic valve, the left ventricular outflow tract consists of the inferior surface of the anterior mitral leaflet, the interventricular septum, and the posterior left ventricular free wall. Abnormalities involving any of these structures can lead to aortic valve dysfunction.

A normally functioning aortic valve apparatus allows unrestricted flow of blood from the left ventricle to the ascending aorta during systole and prevents retrograde blood flow from the aorta to the left ventricle during diastole. The pressure change across the aortic valve during diastole generates considerable stress within the leaflets. An intact apparatus allows distribution of this stress from the leaflets to the surrounding fibrous structure to which the leaflets are attached. The leaflets are also supported by one another along the region of coaptation on the leaflet called the lunula. The stress is then distributed along the leaflet edges to corners of the commissures. Further stress reduction occurs through distribution of stress to the sinuses of Valsalva. The sinuses provide a reservoir of blood for developing vortices that move toward the ventricular arterial junction as the velocity of blood flow declines during late systole. These vortices within the sinuses of Valsalva prime the leaflets for closure, so that as soon as the pressure between the ventricle and aorta equalize, the leaflets rapidly close. All components of the aortic valve apparatus are important for the proper function and durability of the valve. A complete understanding of these mechanisms is a requirement for the echocardiographer.

Etiology of Aortic Regurgitation

Aortic regurgitation (AR) is caused by either intrinsic disease of the aortic cusps or secondarily from diseases affecting the ascending aorta. Intrinsic valvular problems include rheumatic, calcific, myxomatous disease, endocarditis, traumatic injury, and congenital abnormalities. Conditions affecting the ascending aorta that lead to aortic regurgitation include annular dilation and aortic dissection (secondary to blunt trauma or hypertension), mycotic aneurysm, cystic medial necrosis, connective tissue
disorders (Marfan’s syndrome), and chronic hypertension. The most common cause of pure aortic regurgitation is no longer post-inflammatory with the decreasing prevalence of rheumatic heart disease among cardiac surgical patients. Aortic root dilation is now the most common etiologic factor due to the increased prevalence of degenerative disease, followed by post-inflammatory and bicuspid valve disease (1).

Chronic left ventricular volume overload causes progressive left ventricular dilation over many years, while systolic function is preserved. Ejection fraction is initially normal while end-diastolic dimensions are increased. In contrast to AS, the ventricle remains relatively compliant until systolic dysfunction ensues late in the course of disease progression. Another contrasting feature is that systolic dysfunction is not reversible. Acute aortic regurgitation is not associated with left ventricular dilation because the adaptive left ventricular dilation has not yet occurred (2). This lack of adaptation is associated with decreased left ventricular compliance and a rapid onset of symptoms. Other echocardiographic findings of chronic AR include premature mitral valve closure and fluttering of the mitral valve leaflets, most effectively demonstrated using M-mode echocardiography. Depending on the etiology of the AR, aortic root abnormalities may also be present, including aortic dissection or aneurysm.

**Echocardiographic Evaluation of Aortic Regurgitation**

The aortic valve, ascending aorta, and LVOT are inspected using the ME AV LAX view. Normal leaflets are often not visible during diastole, because they are parallel to the Doppler beam when closed. Stenotic leaflets that dome during systole, often do not completely coapt during diastole leading to aortic regurgitation. The diagnosis of leaflet prolapse is made when aortic leaflet tissue is imaged in the LVOT below the annular plane during diastole. An aortic dissection in the aortic root causes disruption of leaflets from the aortic annulus and may also cause leaflet prolapse. Two-dimensional echocardiography is used to determine the etiology of the aortic regurgitation by identifying structural abnormalities of the leaflets or aortic root.

Although two-dimensional echocardiography is not useful for quantifying the severity of AR, there are several associated echocardiographic features. The left ventricle is dilated and more spherical in shape with chronic AR, but not necessarily with acute AR. The mitral valve exhibits premature closure and fluttering of the anterior mitral leaflet during diastole. An eccentric AR jet directed towards the anterior mitral valve leaflet may cause doming of the anterior leaflet with convexity towards the left atrial side of the mitral valve.

Doppler echocardiography is used to quantify the severity of AR by several techniques that involve color, pulsed wave, and continuous wave Doppler. These techniques are sensitive and reliable, but all have limitations. Color Doppler applied to ME AV SAX is useful for localizing the site of regurgitation. Despite the orthogonal relationship between the aortic valve flow and Doppler beam in this short axis view, the regurgitant orifice is identifiable because the AR jet is usually not completely orthogonal to the Doppler beam, particularly if the jet is eccentric. Utilizing the ME AV SAX the cross sectional area of the regurgitant jet can be measured by planimetry within 1cm of the valve. The cross sectional area of the jet and its ratio to the LVOT area can be used to quantify the severity of AR (3). This method defines severe AR as a ratio ≥ 0.6 as indicated in the table (2).

The ME AV LAX view is the most useful for quantifying the severity of AR. Color Doppler reveals a flow disturbance in the left ventricular outflow tract originating from the aortic valve and directed into the left ventricle. A central jet is usually caused by aortic root dilatation whereas an eccentric jet usually implies a leaflet problem. The width of the jet at the orifice compared to the width of the LVOT correlates with angiographic determinants of aortic regurgitation (3). A jet width/LVOT width ratio of less than 0.25 is mild AR, while a ratio ≥ 0.65 is indicative of severe
AR (2). The length of the AR jet into the receiving chamber does not correlate with the AR severity (3).

There are limitations to the use of color flow Doppler echocardiography to estimate severity of aortic regurgitation. One such limitation to this technique is that the regurgitant jet orifice and the true LVOT diameter (not foreshortened) may not be in the same imaging plane (4). This limitation is most apparent if “color M-mode” is used to determine the jet/LVOT ratio. Color M-mode refers to the application of M-mode imaging to a color flow Doppler image. M-mode evaluation of the LVOT in the patient with AR is more useful for determination of the duration of AR into the diastolic phase rather than the jet/LVOT ratio. Another limitation to the jet/LVOT ratio method of assessing AR is that the shape of the regurgitant orifice may not be circular or symmetric. An irregularly shaped regurgitant orifice may cause the jet to appear wider in one imaging plane than another (5); hence the importance of examining multiple imaging planes. The AR jet may also be eccentric or converge with the mitral valve inflow, rendering the jet particularly difficult to evaluate in patients with mitral stenosis (6). An eccentric AR jet directed towards the anterior mitral valve leaflet tends to underestimate severity with the jet width/LVOT width ratio method (7). If color Doppler cannot be applied to the LVOT from the ME AV LAX view because of annular calcification or shadowing of the LVOT from a prosthetic mitral or aortic valve, a deep TG LAX or TG LAX view is used. The AR jet in this view appears red or mosaic in color with the jet directed away from the aortic valve towards the left ventricular cavity. Multiple imaging planes should be utilized to appreciate the three-dimensional character of the jet.

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Angiographic Grade</td>
<td>1+</td>
<td>2+</td>
<td>3+</td>
</tr>
<tr>
<td>Jet width/LVOT width</td>
<td>&lt;0.25</td>
<td>0.25-0.46</td>
<td>0.47-0.64</td>
</tr>
<tr>
<td>Vena contracta width (cm)</td>
<td>&lt;0.3</td>
<td>0.3-0.6</td>
<td>&gt;0.6</td>
</tr>
<tr>
<td>Deceleration slope (m/sec²)</td>
<td></td>
<td></td>
<td>&gt;3</td>
</tr>
<tr>
<td>Pressure half-time (msec)</td>
<td>&gt;500</td>
<td>500-200</td>
<td>&lt;200</td>
</tr>
<tr>
<td>Regurgitant volume (ml/beat)</td>
<td>&lt;30</td>
<td>30-59</td>
<td>≥60</td>
</tr>
<tr>
<td>Regurgitant fraction, %</td>
<td>&lt;30</td>
<td>30-49</td>
<td>≥50</td>
</tr>
<tr>
<td>Regurgitant orifice area (cm²)</td>
<td>&lt;0.10</td>
<td>0.10-0.29</td>
<td>≥0.30</td>
</tr>
</tbody>
</table>

The ME AV LAX also allows for measurement of the vena contracta. The vena contracta is slightly different than the jet width in that it is the smallest diameter of regurgitant color flow at the level of the aortic valve and is usually smaller than the jet width in the LVOT. Some consider the vena contracta measurement to be a stronger measurement than the jet width/LVOT ratio (8). Despite the simplicity of the vena contracta measurement in estimating the severity of AR, there are few limitations, such as the presence of multiple or abnormally shaped regurgitant jets and the inability to accurately determine the annular plane of the aortic valve due to pathology.

Although the jet width/LVOT width and vena contracta methods are easy to use and provide useful information for making clinical decisions, there are other methods to evaluate patients with AR. Continuous wave Doppler is used to determine the severity of AR by measuring the deceleration slope of the regurgitant jet and by calculating pressure half-time (PHT). Pressure half-time is the time required for the peak regurgitant pressure to decrease to half of its maximum value and is measured in milliseconds. A deep TG LAX or TG LAX view aligns the regurgitant jet with the Doppler beam. Color Doppler is used to identify the location and direction of the AR jet, while the continuous wave Doppler cursor is placed within the jet to obtain the continuous
wave spectral velocity profile. The velocity of the regurgitant jet declines more rapidly in patients with severe AR because the larger regurgitant orifice allows a more rapid equilibration of the aortic and left ventricular pressures. In other words, if the pressure difference between the aorta and left ventricle approaches zero rapidly, the regurgitant jet velocity also approaches zero rapidly, creating a steeper slope. A regurgitant velocity slope greater than 3 m/sec² is indicative of advanced (3 or 4 +) AR (9).

A more rapid decline in the regurgitant jet velocity also generates a shorter pressure half-time, which is the time it takes for the peak pressure difference between the aorta and LV to be halved. A pressure half-time shorter than 200 ms is indicative of severe aortic regurgitation. Factors other than regurgitant orifice size may influence the deceleration slope. Systemic vascular resistance and left ventricular compliance affect the rate of deceleration, irrespective of the regurgitant orifice size (10). Decreased systemic vascular resistance (sepsis) and reduced left ventricular compliance (ischemia, cardiomyopathy, acute AR) cause a steeper deceleration slope because aortic and left ventricular pressures equalize more rapidly in these conditions. Another limitation to this technique is that measurement of regurgitant jet velocity is difficult and unreliable in patients with eccentric jets, because it is difficult to align the Doppler beam with the regurgitant jet.

Pulsed wave Doppler is used to detect retrograde flow in the aorta during diastole. Holodiastolic flow reversal in the abdominal aorta is both sensitive and specific for severe AR. Detection of holodiastolic retrograde flow in the proximal descending thoracic aorta and aortic arch is a sensitive indicator of AR, but is not specific for severe AR. The short-axis TEE view of the descending thoracic aorta is used for placement of the pulsed wave sample volume distal in the aorta, near the diaphragm. Despite the orthogonal relationship between the aortic flow and Doppler beam in this short-axis view, the flow in the aorta is identifiable because the blood in the aorta tends to swirl as it travels down the aorta. The spectral Doppler display is examined for the duration of diastolic flow. Retrograde flow throughout diastole (holodiastolic) in the distal descending (11) or abdominal aorta (12) indicates severe AR.

Regurgitant volume and regurgitant fraction can also be used to evaluate the severity of AR. Regurgitant volume is the difference between the systolic flow across the aortic valve and “net forward” cardiac output. In the absence of intracardiac shunts and mitral regurgitation, flow through the pulmonary artery or mitral valve is equivalent to (net) cardiac output. Pulmonary artery blood flow is
reliably measured with TEE by measuring the pulmonary artery diameter (d), calculating its area \([\pi (d/2)^2]\), and multiplying the area by the pulmonary artery VTI and heart rate (13). Aortic valve systolic flow is the product of aortic valve area and VTI. The aortic regurgitant volume is the difference between aortic valve systolic flow and pulmonary blood flow (cardiac output). Regurgitant fraction is expressed as the proportion of aortic valve systolic flow that is regurgitant volume and indicates the severity of AR listed in the table above (14).

\[
\text{Regurgitant Volume} = \text{Aortic Valve Systolic Flow} - \text{Cardiac Output}
\]

\[
\text{Regurgitant Fraction} = \frac{\text{Regurgitant Volume}}{\text{Aortic Valve Systolic Flow}}
\]

The continuity equation could be theoretically used to determine regurgitant orifice size. Diastolic velocities just above (aortic root-VTI) and through the aortic valve (aortic valve-VTI) measured with Doppler echocardiography and the cross-sectional area of the aortic root measured with two-dimensional echocardiography are used in this calculation (15,16). This theoretical technique has not been widely accepted or validated.

\[
\text{Aortic Valve Regurgitant Orifice} = \frac{\text{Aortic Root Area} \times \text{Aortic Root Diastolic VTI}}{\text{Aortic Valve Regurgitant Jet VTI}}
\]

As the utility of three-dimensional echocardiography of the aortic valve is becoming more apparent for evaluation of structural anatomy among patients with AS, its use is also becoming more apparent among patients with AR. Online or offline software systems that allow planimetry measurements of area, may also provide accurate measurements of vena contracta and vena contracta area. Three-dimensional echocardiography can be used to identify and measure multiple jets, which can then be summated to accurately determine a true three-dimensional vena contracta (17).

References: