Since 1976, echocardiography (echo) has been used to evaluate cardiac structure and function. Echo uses sound in the high-frequency range (2 to 10 MHz). Frequency ranges between 2 and 5 MHz are typically used for imaging adults, while frequencies of 7.5 to 10 MHz are used for children and specialized adult applications. The transducer contains a piezoelectric crystal that converts electrical to sound energy, producing sound waves that are transmitted in the form of a beam. A complete transthoracic echocardiogram (TTE) consists of a group of interrelated applications including two-dimensional (2D) anatomic imaging, M-mode, and three Doppler techniques: pulsed-wave (PW), continuous-wave (CW), and color-flow (CF) imaging. In addition, the quantification of cardiac chamber dimensions, areas, and volumes is an important aspect of a complete examination. Using a combination of these ultrasound techniques, one can assess the anatomy and function of the cardiac valves, myocardium, and pericardium.

**TWO-DIMENSIONAL ECHOCARDIOGRAPHY**

Standard views are obtained along three orthogonal planes of the left ventricle (LV): long-axis, short-axis, and the 4-chamber plane (Fig. 49-1A to C). The long axis is parallel to the long axis of the LV. The short axis cuts the LV cross-sectionally, similar to slices of bread in a loaf, and is orthogonal to the long axis. Four standard transducer locations are used to obtain complete visualization of the entire heart: parasternal, apical, subcostal, and suprasternal.

**M-MODE ECHOCARDIOGRAPHY**

M mode is a one-dimensional “ice pick” view of the heart (Fig. 49-1D) and is often used for measuring LV systolic and diastolic chamber dimensions and wall thickness.

**DOPPLER ECHOCARDIOGRAPHY**

The Doppler effect is the phenomenon whereby the frequency of sound waves increases or decreases as the sound source moves toward or away from the observer. The resultant Doppler frequency shift can be detected and translated into blood-flow velocity. The velocity of blood can then be used to calculate valvular gradients and areas, intracardiac pressures, and volumetric flow.

**Pulsed-wave doppler**

Pulsed-wave (PW) Doppler is obtained when a pulse of ultrasound is transmitted intermittently, allowing for estimation of blood-flow velocity at a specific region of interest. It is thus “site-specific.” Its disadvantage is aliasing (or wraparound) of the signal at velocities that are one-half of the pulse repetition frequency (Nyquist limit). This property limits the maximal velocity that can be accurately measured with PW.

**Continuous-wave doppler**

Continuous-wave (CW) Doppler records all the velocities along the path of the ultrasound beam. Using CW, the magnitude of the blood-flow velocity and its direction can be accurately recorded. Its disadvantage is that it does not allow localization of the specific site of the velocity obtained along the beam path. CW is used to measure high-velocity jets associated with valvular dysfunction or intracardiac pressure gradients. The simplified Bernoulli equation can then be used to convert velocities into pressure gradients (\(\Delta P = 4V^2\)).

**Color-flow doppler**

Color-flow (CF) Doppler transforms Doppler velocity information into a color-coded scheme. Red denotes
flow toward the transducer, while blue represents flow away from the transducer. The relative velocity of blood flow is also depicted, with brighter shades of blue or red representing higher velocities. Turbulent flow is seen as multicolored jets (e.g., due to valvular disease or intracardiac shunts).

**TRANSESOPHAGEAL ECHOCARDIOGRAPHY**

TEE was first introduced clinically in the United States in the 1980s. By placing an echocardiographic probe in the esophagus, which is in close proximity to cardiac structures, TEE obtains significantly enhanced images...
with excellent resolution. The development of transducer crystals that rotate from 0 to 180 degrees also allows for examination of each cardiac structure from different planes and angles. Acquisition of images is from two basic locations: midesophageal (30 to 35 cm from the incisors) and midgastric (40 to 45 cm from the incisors).

**Indications**

TEE is useful for the evaluation of patients with limiting body habitus, such as obesity or emphysema, who are not optimally imaged by the transthoracic approach. In addition, certain structures that are not well visualized by transtracheal echo (TTE) [such as the left atrial (LA) appendage, thoracic aorta, and prosthetic valves] can be assessed by the transesophageal approach. A third common indication is to guide intraoperative management during cardiac surgery. Class I indications for perioperative TEE (conditions for which there is evidence and/or general agreement that TEE is useful and effective) are listed in Table 49-1.8 There are other situations in which TEE may also be useful but is not required (i.e., class II indications: conditions in which there is a divergence of opinion about the usefulness/efficacy of a procedure but in which the weight of opinion is in favor of usefulness/efficacy). These include ongoing surgical procedures in patients at increased risk of myocardial ischemia or hemodynamic instability, during minimally invasive surgery or the Cox-Maze procedure cardiac tumor resection or aneurysm repair, intracardiac thrombectomy or pulmonary embolectomy, for detection of intracardiac air or aortic atheromatous disease, or for selecting anastomotic sites during heart/lung transplantation.8 Antibiotic prophylaxis for infective endocarditis is usually unnecessary but is optional in the high-risk patient (e.g., complex congenital heart disease, prosthetic valve, poor dentition, or prior history of endocarditis).9

**Contraindications and complications**

Relative contraindications for TEE include significant esophageal pathology (e.g., strictures, varices), history of radiation therapy to the mediastinum, and recent esophageal or gastric surgery. Serious complications of TEE are uncommon (<1 percent) but may include aspiration and other problems related to oversedation, mucosal trauma, laryngospasm, esophageal or pharyngeal laceration or perforation, methemoglobinemia, and rarely death. TEE probe passage is “blind” and is done without direct visualization. The more severe complications of esophageal or pharyngeal trauma are signaled by patient complaints of severe pain and inability to swallow, which usually occur after the procedure has ended and sedation has worn off. Esophageal perforation during intraoperative TEE can go undetected for a period of time because of the anesthetized state of the patient and the lack of patient feedback regarding pain during probe passage.

**ASSESSMENT OF VENTRICULAR FUNCTION**

Routinely, a qualitative “eyeball” estimate of global LV systolic function is obtained visually by examining LV wall thickening and motion. In addition, quantitative measures of LV systolic function can also be obtained. LV segmental wall function can be analyzed using a semiquantitative grading scale (wall motion score).

**LEFT VENTRICULAR EJECTION FRACTION**

Ejection fraction (EF) is often reported qualitatively as increased (hyperdynamic), normal, mildly, moderately, or severely reduced.8 In addition, the reader often assigns an estimated value to the qualitative assessment. Normal LV EF is 61 ± 10 percent.10 Visual estimation of EF by experienced readers is generally reliable but is limited by reader variability and depends on optimal echocardiographic delineation of the endocardium.

EF can also be quantified from the equation below after determining end-diastolic volume (EDV) and end-systolic volume (ESV):

\[
EF = \frac{SV}{EDV} = \frac{(EDV - ESV)}{EDV}
\]

Where SV = stroke volume, EDV = end-diastolic volume, and ESV = end-systolic volume.

Volumes can be obtained by the modified Simpson’s method, which divides the LV cavity into a series of stacked cylinders of equal height that are summed to estimate the entire ventricular volume at end-diastole and end-systole. Further details of the technique can be found in several of the references.1,2,11

**LEFT VENTRICULAR FRACTIONAL SHORTENING (PERCENT FRACTIONAL SHORTENING)**

Percent FS is another method for estimating LV systolic function and reflects a percent change in LV systolic volume.
dimension with systolic contraction. It is calculated from LV end-diastolic and end-systolic dimensions measured by M mode. For ventricles that are roughly symmetrical without regional wall motion abnormalities, EF is approximately 2 (percent FS).

\[
\text{Percent FS} = \frac{(\text{EDD} - \text{ESD})}{\text{EDD}} \times 100
\]

Where EDD = end-diastolic dimension and ESD = end-systolic dimension.

### LEFT VENTRICULAR REGIONAL WALL MOTION

For the assessment of LV regional wall motion, the LV is divided into 16 segments (Fig. 49-2), each of which is given a score from 1 to 5 (1, normal or hyperkinetic; 2, hypokinetic; 3, akinetic; 4, dyskinetic; 5, aneurysmal or diastolically deformed). This grading system differs from the 1-to-5 grading system of TEE often used by cardiac anesthesiologists\(^{12}\) [1, normal (> 30 percent thickening); 2, mildly hypokinetic (10 to 30 percent thickening); 3, severely hypokinetic (< 10 percent thickening); 4, akinetic (no thickening); 5, dyskinetic (paradoxical systolic motion)]. The wall motion score index is calculated as the sum of segmental wall motion scores divided by the number of segments seen. A score of 1 is normal and higher wall motion scores indicate more extensive ventricular dysfunction. However, this grading system is more useful for research databases than for clinical use.

### LEFT VENTRICULAR FILLING

LV preload is the LV volume at end-diastole. Normal LV end-diastolic size is 3.5 to 5.7 cm in the parasternal long-axis view. The size of the ventricles and atria may be used for the qualitative evaluation of filling pressures and assessment of hyper- or hypovolemia, particularly intraoperatively with TEE.\(^{13}\) Small LA size and near cavity obliteration of the LV can indicate hypovolemia.\(^{14}\) Conversely, ventricular and atrial enlargement may indicate hypervolemia. Doming of the interatrial septum toward the right suggests elevated LA pressure and increased LV preload (the septum bulges toward the side with lower pressure). One limitation to using TEE for indirect measurement of volume status is that the LV may be foreshortened; it is therefore recommended that the LV be imaged from several different planes to get a more accurate estimate of LV volume.\(^{14}\)

### LEFT VENTRICULAR DIASTOLIC FUNCTION

Doppler echo is the most common diagnostic tool for assessing diastolic function. Indices of transmitral and pulmonary venous Doppler flows are commonly used to identify patterns of diastolic dysfunction.\(^{11}\)

#### Mitral valve inflow

When sinus rhythm is present, PW Doppler at the level of the mitral valve (MV) leaflets records two velocities separated by a period of diastasis (no flow), as shown in Fig. 49-3. After the MV opens, early rapid (E wave) diastolic filling of the LV occurs, followed by a period of diastasis, after which late filling occurs due to atrial contraction (A wave). Normally, in individuals less than 60 years old, the peak E:A wave velocity ratio is greater than 1. When there is impaired relaxation without elevated filling pressures, the peak E decreases, hence the E:A ratio becomes less than 1. In contrast, a “restrictive” filling pattern is seen when both impaired relaxation and elevated filling pressures are present, resulting in a smaller contribution of atrial contraction and an increased E:A ratio (> 1.5 to 2).
Pulmonary vein flow

Four velocities are seen in PW Doppler of the pulmonary veins (Fig. 49-4). Occurring in early systole, the first systolic forward flow velocity (PVS1) is due to the relaxation of the LA, which promotes pulmonary venous flow into the LA. In mid- to late systole, a second systolic forward flow (PVS2) occurs that is produced by the increase in pulmonary venous pressure occurring after right ventricular (RV) systole. In diastole, a third pulmonary vein velocity is seen (PVD). This is related to the decrease in LA pressure seen after MV opening. The fourth velocity is the atrial flow reversal velocity (PVa) that is related to LA contraction. A pulmonary vein systolic velocity peak less than the diastolic velocity peak is suggestive of elevated LA pressures. Restrictive filling indicative of very high filling pressures is seen when PVS2 is much less than PVD. When higher filling pressures are present, both the duration and peak velocity of PVa are increased. Another sign of elevated LA pressure is when the duration of PVa is longer than the duration of the mitral inflow A wave (by > 0.03 s).

RIGHT VENTRICULAR FUNCTION AND FILLING PRESSURES

Qualitative evaluation of RV wall thickening and motion is done visually, as in the evaluation of LV function, although it is more difficult to estimate RV function. Normal RV size is less than two-thirds of LV size (see Fig. 49-1). Septal wall flattening (resulting in a D-shaped LV in the short-axis view) or septal deviation into the LV can be seen in both pressure- and volume-overload conditions of the RV. When tricuspid regurgitation is present, the RV systolic pressure (RVSP) can be estimated (see also “Pulmonary Hypertension,” below), although TTE is preferred to TEE for measuring RVSP because of better alignment of the transducer beam with the direction of the regurgitant jet.

Examination of the size and respiratory changes of the venae cavae and hepatic veins can give helpful clues to the patient’s volume status and is also used in the evaluation of pericardial disease. Although TTE is preferred for examination of respirophasic collapse of the inferior vena...
cava, TEE provides better visualization of the superior vena cava. Dilatation of the superior vena cava (greater than one-half the aortic dimension in the short-axis view) is suggestive of hypervolemia.14

### VALVULAR DISEASE

#### MITRAL VALVE

**Mitral valve morphology**

Normal MV function depends on the normal function of all its component parts, including the mitral leaflets, annulus, chordae tendineae, papillary muscles, and LV. The rectangular posterior leaflet is composed of three scallops (P1, P2, P3), while the semicircular anterior leaflet is divided into thirds for descriptive purposes (Fig. 49-5). Normal MV area is 4 to 6 cm².

**Mitral stenosis**

Rheumatic heart disease is the most common cause of mitral stenotic lesions (Fig. 49-6), with leaflet thickening and fusion of the commissures (characteristic fish-mouth valve in the short-axis view and hockey-stick appearance in the long-axis view).

Two-dimensional (2D) echo is the “gold standard” for evaluating mitral stenosis (MS) and allows assessment of the structure and function of the mitral annulus, leaflets, chordae, papillary muscles, LV size and function, LA size, and pulmonary hypertension (RVSP). The Wilkins echo score15 for rheumatic MS is based on four variables (leaflet mobility, leaflet thickening, subvalvular thickening, and calcifications). Each variable receives a score of 1 to 4 and the individual scores are summed up. A total score equal to or greater than 8 is associated with better outcomes for mitral balloon valvuloplasty. For valves with less favorable scores (> 8), cardiac surgery with valve replacement may be the preferred treatment modality.

**Mitral valve area**

Calculation of MV area is usually obtained using the pressure half-time (PHT) method. PHT is the time (milliseconds) for the maximal pressure gradient to decrease by half and is usually equal to the deceleration time multiplied by 0.29.

\[
MV \text{ area (cm}^2\) = \frac{220}{PHT \text{ (ms)}}
\]

The PHT method has several important shortcomings. It is affected by concomitant aortic regurgitation or decreased LV compliance.4 The rapid increase in LV diastolic pressure associated with either of these conditions may shorten the PHT and underestimate the extent of stenosis. Other techniques to estimate valve area include planimetry and the continuity equation.1,4 The latter approach is also less reliable in the presence of significant aortic and mitral regurgitation. Details of this approach are outlined in the references 1 through 11.

**Mitral valve pressure gradients**

As the degree of obstruction to blood flow caused by a stenotic valve increases, the velocity of the blood flow also increases in order to maintain constant flow (conservation of mass or flow). Velocities can then be transformed into pressures with the simplified Bernoulli equation:

\[
\Delta P = 4V^2
\]

Where \(\Delta P\) = MV pressure gradient (mmHg) and \(V\) = velocity of blood flow across the MV (ms).

**Severity of mitral stenosis**

The mean MV pressure gradient and MV area are used to estimate the severity of mitral stenosis and the need for intervention (Table 49-2).

**Mitral regurgitation**

There are three basic mechanisms of mitral regurgitation (MR): primary abnormalities of the mitral leaflets, commissures, or annulus; malfunctioning of the subvalvular
structures (chordae tendineae and papillary muscles); and alterations in LV and LA dimensions and function.

The most common cause of isolated severe MR is myxomatous degeneration (Fig. 49-7). The valve itself can also be deformed from rheumatic fever, mitral annular calcification, infective endocarditis, and congenital lesions (cleft MV). Other less common causes of leaflet abnormalities include endomyocardial fibrosis, carcinoid disease, drugs (e.g., fenfluramine hydrochloride and phentermine, or Fen-Phen), radiation therapy, trauma, and collagen vascular disease.

Abnormalities in the subvalvular structures include dysfunctional and/or ruptured chordae tendineae and papillary muscles. Ruptured chordae tendineae account for a large proportion of MR lesions. Etiologies include idiopathic causes, MV prolapse, infective endocarditis, and thoracic trauma. Papillary muscle dysfunction (with or without frank rupture) is most often seen in the setting of myocardial ischemia or infarction. The posteromedial head of the papillary muscle is most vulnerable to ischemia because of its end-artery vascular supply. Other causes of dysfunction include dilated cardiomyopathy, myocarditis, hypertension, and chest trauma.

Global or regional LV enlargement may dilate the mitral annulus and change the position and axis of contraction of the papillary muscles, leading to dysfunction. Progressive LA and LV enlargement associated with chronic MR further exacerbate the extent of MR because of continued changes in chamber geometry.

Table 49-2  Severity of mitral stenosis

<table>
<thead>
<tr>
<th>Severity</th>
<th>Mitral valve area (cm²)</th>
<th>Mean gradient* (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>4–6</td>
<td>—</td>
</tr>
<tr>
<td>Mild</td>
<td>1.6–2.0</td>
<td>&lt; 5</td>
</tr>
<tr>
<td>Moderate</td>
<td>1.1–1.5</td>
<td>5–10</td>
</tr>
<tr>
<td>Severe</td>
<td>≤ 1</td>
<td>&gt;10</td>
</tr>
</tbody>
</table>

Two-dimensional echocardiography

Unlike the case in MS, where echo is the accepted gold standard for diagnosis, there is no gold standard for the assessment of MR; therefore multiple parameters are used in estimating the severity of MR.\(^1^6\) The purpose of echo in MR is to determine the etiology, mechanism, and severity of regurgitation; determine the need for surgery and the type of surgery that is necessary; and evaluate other valves, RV and LV function, and the presence of pulmonary hypertension. The most important aspect is to determine the hemodynamic significance of the regurgitation based on LV size (particularly end-systolic dimension), LV systolic function (EF), and the presence and degree of LA enlargement and pulmonary hypertension (RVSP). Regurgitant jet size and area may contribute to the assessment of MR but often correlate poorly with MR severity.\(^1^6\) The degree of MR intraoperatively may appear less or more severe compared to the degree preoperatively, particularly for ischemic MR, because of different hemodynamic and ischemic conditions (e.g., lower blood pressure or relief of ischemia will reduce the amount of regurgitation).\(^1^7\)

**Severity of mitral regurgitation**

Echo findings in severe MR\(^4^,^1^6\) include:

- Large regurgitant jet: > 40 percent of LA area.
- Wide vena contracta (the narrowest part of the regurgitant color jet at its origin): ≥ 7 mm.
- Eccentric wall-impinging regurgitant jet (Fig. 49-7B).
- Dilated LV: end-systolic dimension ≥ 45 mm or end-diastolic dimension ≥ 70 mm.
- LV EF < 55 to 60 percent.
- Dilated LA (≥ 5.5 cm), although the LA may not be dilated in acute MR.
- Pulmonary hypertension: resting RVSP ≥ 50 mmHg.
- Restrictive mitral filling pattern: high peak early transmitial velocity (E wave) > 1.5 ms.
- Pulmonary vein systolic flow reversal: A normal pulmonary vein Doppler pattern has a systolic (S) wave larger than the diastolic (D) wave. As the MR progresses, the S wave decreases until it may become reversed. While a reduced S wave is a nonspecific finding,\(^1^8\) the presence of a reversed S wave has high specificity for significant MR. The absence of a reversed S wave does not exclude significant MR.
- Effective orifice area (ERO) ≥ 0.4 cm\(^2\).

Table 49.3 summarizes the important echo features of MV stenosis and regurgitation.

**Mitral valve prolapse**

Mitral valve prolapse (MVP) is the most common cause of MR in patients undergoing cardiac surgery in the United States.\(^1^9\) MVP is the systolic billowing or displacement of at least 2 mm of either mitral leaflet into

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**Table 49-3**

<table>
<thead>
<tr>
<th>Echocardiographic assessment of the mitral valve</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Morphology</strong></td>
</tr>
<tr>
<td>Mitral annulus, leaflets, chordae, papillary muscles, LV size and function, LA size, pulmonary hypertension (RVSP)</td>
</tr>
<tr>
<td><strong>Stenosis</strong></td>
</tr>
<tr>
<td>Often rheumatic</td>
</tr>
<tr>
<td>Echo score: leaflet mobility, thickening, subvalvular thickening, calcifications, &lt; 8 favors valvuloplasty</td>
</tr>
<tr>
<td>Severe MS: area ≥ 1 cm(^2) or mean gradient &gt; 10 mmHg</td>
</tr>
<tr>
<td><strong>Regurgitation</strong></td>
</tr>
<tr>
<td>Regurgitant jet size does not correlate well with severity</td>
</tr>
<tr>
<td>Severe MR: dilated LV (ESD &gt; 45 mm), ↓ LV systolic function (EF &lt; 55–60%), dilated LA, pulmonary hypertension (RVSP &gt; 50)</td>
</tr>
</tbody>
</table>

**LV** = left ventricle; **RVSP** = right ventricular systolic pressure; **MS** = mitral stenosis; **MR** = mitral regurgitation; **ESD** = end-systolic dimension; **LA** = left atrium; **EF** = ejection fraction.
the LA beyond the plane of the mitral annulus in the parasternal or apical long-axis views (see Fig. 49-7). Recent criteria for echo diagnosis of MVP\(^9\) have become more stringent, leading to increased specificity of the criteria for MVP while at the same time preserving sensitivity for the detection of MVP complications. Classic MVP is defined as equal to or greater than 5 mm of leaflet thickening (myxomatous changes) of the prolapsing leaflet, while nonclassic prolapse is leaflet thickening less than 5 mm. Compared to nonclassic MVP, classic MVP has a worse prognosis, with most complications of MVP (significant MR and congestive heart failure, MV surgery, infectious endocarditis) arising in patients with classic MVP.\(^{19}\) Asymmetrical prolapse of the leaflets is associated with progression to more significant disease (flail leaflet and severe MR).

Determining which segment of the mitral leaflet tissue is prolapsing is essential for proper MV repair. MR associated with MVP often has an eccentric jet directed opposite to the prolapsing leaflet (e.g., anteriorly directed jet if posterior leaflet prolapse is present). In contrast, in MR associated with rheumatic mitral stenosis, the regurgitant jet is directed toward the affected leaflet due to restricted leaflet motion (e.g., anteriorly directed jet if the anterior leaflet is calcified). MR associated with MVP may be due to flail leaflet or chordal rupture resulting from myxomatous involvement of the chordae or leaflet. Chordal rupture is diagnosed when the chords are seen as mobile echodensities attached to a flail or partially flail leaflet (Fig. 49-8).\(^{21}\) These may be confused with or may be difficult to distinguish from vegetations of infective endocarditis. Mitral annulus calcification (MAC) may also be seen in patients who have MVP.

**Figure 49-8** Ruptured chord attached to a flail segment of the posterior mitral leaflet due to myxomatous disease. Intraoperative transesophageal echocardiogram showing the ruptured chord as an echodense linear filament attached to the flail leaflet, prolapsing into the left atrium during systole.

**Figure 49-9** Flail mitral leaflet with severe mitral regurgitation. Intraoperative transesophageal echocardiogram with color-flow Doppler consistent with severe mitral regurgitation. This patient underwent successful mitral valve repair with posterior leaflet quadrangular resection and an annuloplasty band.

**Flail leaflet**

Flail leaflet encompasses a spectrum of disease severity from partially to completely flail leaflets, resulting in excessive motion of the mitral leaflets and various degrees of MR (Fig. 49-9). Flail mitral leaflet is diagnosed when the leaflet tip is “upturned” toward the LA during MV closure (due to loss of coaptation). Most commonly, it is caused by MVP or endocarditis resulting in chordal rupture, but it may also be caused by ischemia or infarction of the papillary muscle (usually affecting the posteromedial papillary muscle). A partially flail leaflet (due to chordal rupture) is usually associated with moderate to severe MR, while a completely flail mitral leaflet is almost always associated with severe MR necessitating surgery.\(^{21}\)

**Ruptured papillary muscle**

Dysfunction of the papillary muscle results in severe MR and is most often due to acute myocardial infarction. Papillary muscle dysfunction should be differentiated from acute chordal rupture. Papillary muscle rupture is diagnosed in the appropriate clinical context when a triangular mass (the head of the papillary muscle), seen attached to the flail leaflet, prolapses into the LA during systole, accompanied by severe MR.\(^{17}\)

**Leaflet perforation**

This is most commonly caused by valvular endocarditis, while less common etiologies include congenital (cleft MV) or iatrogenic causes. With leaflet perforation, the regurgitant jet is often eccentric and originates at the site of perforation.

**Mitral valve repair**

TEE is essential in determining the suitability of a valve for repair. It can assess the mechanism and severity of
MR, identify the affected leaflets, determine the presence of coexisting valvular lesions, and estimate RV and LV function. The prognosis of MV repair differs based on the mechanism of MR (organic/primary vs. functional/secondary) and is strongly influenced by the preoperative EF. The most common indication for MV repair is myxomatous disease. Determining which leaflet or segment is involved is important in deciding on the suitability of the repair, the likelihood of successful repair (better with posterior leaflet prolapse), and the method of repair. This is done by analyzing the motion of the leaflets and the direction of the regurgitant jet.

TEE is also helpful intraoperatively to assess the success of the repair. Immediately postrepair (Fig. 49-10), the competency of the valve can be assessed for residual MR (1+, mild; 2+, moderate; 3+, moderate to severe; 4+, severe). Phenylephrine and volume may be administered in order to assess the effect of increased afterload and preload on the degree of residual MR. If there is MR postrepair equal to or greater than 2+, further surgery is indicated. Repeat imaging is also done after the patient is off cardiopulmonary bypass. Other findings that may be detected on TEE include residual mitral stenosis, global or regional LV systolic dysfunction, suture dehiscence or leaflet perforation, and MV systolic anterior motion (SAM) with outflow obstruction. Limitations to intraoperative TEE include the effect of changing hemodynamics, which may significantly affect the appearance and severity of valvular lesions, since the evaluation depends on preload and afterload conditions as well as ventricular function. Hence, it is important that loading conditions be similar in comparing the severity of MR.

**AORTIC VALVE**

Aortic valve morphology

Normally, the aortic valve (AV) is composed of three semilunar leaflets (cusps) that open and close passively due to pressure differences between the LV and the aorta. Small strands may be seen on the cusps (Lambl’s excrescences), particularly on TEE, and represent a normal variant. Bicuspid and rarely unicuspid or quadricuspid valves may be seen on echo (Fig. 49-11). The normal valve area is 3 to 4 cm² with a 2-cm leaflet separation during systole.

A bicuspid AV is the most common congenital heart defect and occurs in about 1 to 2 percent of the U.S. population. The morphologic features of a bicuspid AV are somewhat variable. In some patients, there are two equal-sized cusps with a single central commissure. In many others, the cusps may be unequal in size with an eccentric commissure, with the larger of the two cusps containing a raphe. In the parasternal long-axis views, bicuspid valves are characterized by systolic doming. In the short-axis views, the hallmark is an elliptical “football”-shaped systolic orifice. The valve leaflets themselves may be thickened and fibrotic, particularly with increasing patient age. A bicuspid valve may be functionally normal with no significant stenosis or regurgitation, particularly in adolescents and young adults, among whom up to one-third have no significant valvular dysfunction. However, over time, progressive “wear and tear” with resulting fibrosis and valve calcification leads to functional abnormalities. By the age of 60 years, over 50 percent of bicuspid valves are significantly stenotic. Valve regurgitation is frequently present.
as well and may be the predominant functional abnormality in younger patients. Etiology of the regurgitation may be retraction and fibrosis of the commissures or leaflets, cusp prolapse, dilatation of the aortic root or valve annulus, or damage from infective endocarditis.

It is important to recognize that bicuspid AVs are often associated with abnormalities of the aorta. Concomitant aortic coarctation occurs in a minority. Aortic dissection is another known association, with 5 to 9 percent of patients with dissecting aortic aneurysms having bicuspid valves. Aortic root dilatation may be due to a common developmental defect that affects both the aorta and the valve. Poststenotic dilatation of the ascending aorta can also occur.

**Aortic stenosis**

Rheumatic aortic stenosis (AS) (Fig. 49-12A) is usually associated with MV disease. Calcium deposits are present on both sides of the aortic cusps, resulting in commissural fusion and aortic regurgitation. Degenerative calcific disease is the most common cause of AS in the United States. It is often associated with MAC and coronary artery disease. Nodular calcification is often present on the aortic aspect of the valve along the bases of the cusps and may protrude into the sinuses of Valsalva (Fig. 49-12B and C). In contrast to rheumatic AS, there is no commissural fusion in degenerative AS, hence aortic regurgitation is rare.

**Two-dimensional echocardiography**

A thorough echo evaluation includes assessing the thickness and calcification of the leaflets, their mobility, looking for the etiology of stenosis (e.g., bicuspid, rheumatic, degenerative calcific) and its extent, the presence of other valvular lesions, and assessing LV size and function. TTE is often superior to TEE in assessing the severity of AS because obtaining maximal velocity jets (i.e., absolutely parallel to flow) is often difficult with TEE.

**Aortic valve area**

In aortic stenotic lesions, the AV area decreases by approximately 0.1 cm² per year, although large variations from patient to patient exist. The AV area is usually determined using the continuity equation. Direct planimetry (visualization of the orifice area) is less reliable for patients with heavily calcified valves (due to shadowing of the valve) and for critical (“pinhole”) stenoses. The continuity equation is based on the principle of conservation of flow (or mass), whereby flow before the valve must equal flow across the valve. Since the area of the LV outflow tract (LVOT) can be directly measured, as can velocities in the LVOT and across the valve, the AV area can then be calculated:

\[
A_1V_1 = A_2V_2
\]

Where \( A = \text{area or } \pi r^2; V = \text{velocity}; A_1V_1 = \text{flow proximal to the valve (LVOT)}; A_2V_2 = \text{flow across the valve} \).

The major limitation to the continuity equation is that small errors in the LVOT diameter become magnified in estimating the AV area (since the radius is squared in the equation). As a general rule, one may assume that the LVOT diameter is 2 cm \((r = 1 \text{ cm})\) with generally good estimates of the AV area. Another limitation is in obtaining the maximal aortic jet velocity, which requires that the echocardiographic Doppler beam be exactly parallel to the direction of blood flow.

**Aortic valve pressure gradients**

Like pressure gradients obtained for mitral stenosis, the simplified Bernoulli equation is used for estimating AV pressure gradients:

\[
\Delta P = 4V^2
\]

Where \( \Delta P = \text{AV pressure gradient (mmHg)} \) and \( V = \text{velocity of blood flow across the AV (ms)} \).
AV pressure gradients may be significantly underestimated because of inadequate envelopes (because the Doppler beam is not parallel to the blood flow) or an inadequate number of measurements from different locations. Maximal velocities may be present or obtainable from only certain locations in an individual patient. Hence complete assessment from all locations is needed. In addition, the velocity of flow is highly dependent on overall LV function. Pressure gradients may significantly underestimate the severity of AS in the presence of severe LV dysfunction (“low-gradient AS”). The opposite is also true, whereby increased flow velocity across the valve is seen in situations of increased cardiac output (i.e., anemia, aortic regurgitation, hyperthyroidism) and may not reflect true aortic stenosis. In such situations, valve area calculations may be more accurate, and correlation with valve morphology and visual assessment of leaflet mobility is important.

**Severity of aortic stenosis**

The mean AV pressure gradient and the AV area are used to estimate severity of AS and the need for surgical intervention (Table 49-4). If there is a significant discrepancy between the pre- and intraoperative grading of the severity of AS, the following should be considered:

1. Change in hemodynamic conditions: changes in heart rate or rhythm, contractility, and hemodynamics influence the gradients across the AV. The AV area is usually less affected by loading conditions.
2. Measurement variability in data recording: measurement errors in the LVOT diameter greatly influence the AV area, since the LVOT radius is squared in the continuity equation.

<table>
<thead>
<tr>
<th>Severity</th>
<th>Velocity (ms)</th>
<th>Area (cm²)</th>
<th>Mean gradient (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>1</td>
<td>3–4</td>
<td>—</td>
</tr>
<tr>
<td>Mild</td>
<td>2.5–2.9</td>
<td>&gt;1.5</td>
<td>&lt;25</td>
</tr>
<tr>
<td>Moderate</td>
<td>3–4</td>
<td>1–1.5</td>
<td>25–50</td>
</tr>
<tr>
<td>Severe</td>
<td>&gt; 4</td>
<td>&lt;1</td>
<td>&gt;50</td>
</tr>
</tbody>
</table>

*Assumes normal cardiac output.

Source: ACC/AHA Guidelines for the Management of Patients with Valvular Heart Disease.9 With permission.
Aortic regurgitation

Echo assessment of aortic regurgitation (AR) includes a comprehensive examination of AV morphology, aortic root dilatation, and LV size and function. TTE is generally the initial diagnostic test used for evaluating the severity of AR. However, TEE is also helpful particularly for patients with poor transthoracic windows, if prosthetic valves are present, and intraoperatively to guide surgical repair. TEE is particularly useful for determining the mechanism of regurgitation by distinguishing valvular from nonvalvular causes of AR. Common etiologies for AR include congenital malformations (bicuspid valves), degenerative calcific, rheumatic, and infective endocarditis; aortic aneurysm (Fig. 49-13) or dissection, Marfan’s syndrome, drug-induced (e.g., Fen-Phen) AR, and prosthetic valve dysfunction. Acute and chronic AR differ in both pathophysiology and echo findings. Many of the echo findings in chronic AR (e.g., LV dilatation and systolic dysfunction) may not be present in acute AR. Chronic AR is a state of both pressure and volume overload of the LV, resulting in LV hypertrophy and dilatation.

Two-dimensional echocardiography

This is helpful for evaluating LV size and function, AV structure and leaflet mobility, aortic root dilatation, aortic dissection, associated vegetations, diastolic fluttering motion of MV leaflets, and premature MV closure (indicative of significant AR).

Severity of aortic regurgitation

In assessing the severity of chronic AR, it is essential to combine data on LV size and function with Doppler data and not to rely solely on color Doppler, since it is often misleading. No study has yet demonstrated that quantification of the severity of AR by Doppler criteria alone is predictive of outcome. Instead, LV size and function are used for risk stratification of asymptomatic patients with chronic AR. Echo findings in severe AR include:

- Dilated LV: minor-axis dimension > 50 to 55 mm in systole or > 70 to 75 mm in diastole
- LV ejection fraction < 55 percent
- Pressure half-time < 200 ms
- Proximal regurgitant color jet width/LVOT diameter ≥ 65 percent
- Vena contracta > 6 mm
- Holodiastolic flow reversal in the descending thoracic aorta
- Restrictive filling pattern to the MV inflow

In comparison to men, women with AR should be considered for surgery before severe symptoms have developed.

![Figure 49-13](image-url) Aortic aneurysm of the sinuses of Valsalva and secondary aortic regurgitation. The aortic wall is thin at the sinuses of Valsalva and aneurysmal dilatation results in aortic regurgitation. Note the wide base of the regurgitant jet (vena contracta) consistent with severe aortic regurgitation (arrow). SoV = sinuses of Valsalva; LV = left ventricle; Ao = aorta; LA = left atrium; AR = aortic regurgitation.
and for smaller LV dimensions. In a recent study, intraoperative mortality was similar for men and women, but 10-year survival was significantly worse for women than for men (39 vs. 72 percent, respectively).26

In acute AR, many of the features of chronic volume overload will not be present. The severity will be need to be assessed by evaluation of color-flow Doppler jet width, presence of significant pulmonary hypertension, and evidence by Doppler of rapid equilibration of aortic and LV diastolic pressure (short diastolic half-time <200 ms, short mitral deceleration time <150 ms, or premature closure of the MV). Echo assessment of the AV is summarized in Table 49-5.

### TRICUSPID VALVE

#### Tricuspid valve morphology

Normal function of the TV depends on the normal function of its components: annulus, leaflets, chordae, papillary muscles, right atrium, and ventricle.

#### Tricuspid stenosis

The most common cause of tricuspid stenosis (TS) is rheumatic, which results in both stenosis and regurgitation of the TV and is often associated with concomitant mitral or AV disease. Echo assessment of TS is similar to that of MS. The mean gradient across the TV is normally < 2 mmHg. Tricuspid stenosis is considered severe when the mean gradient is ≥ 7 mmHg and the pressure halftime (PHT) is ≥ 190 ms.

#### Tricuspid regurgitation

As in the case of the AV and MV, TTE or TEE evaluation of tricuspid regurgitation (TR) focuses on the identification of the etiology or mechanism of TR as well as its hemodynamic severity. TR is the most common abnormality of the TV. Mild TR is a normal finding in 70 percent of individuals.16 Pathologic TR is often secondary to RV dysfunction, RV dilatation, or significant systolic pulmonary hypertension (systolic pulmonary artery pressure > 55 mmHg). Primary causes of TR are less common (Ebstein’s anomaly, endocarditis, trauma, anorectic drugs, carcinoid, myxomatous or rheumatic valvular disease, radiation). Patients with severe TR of any cause have poor long-term outcomes because of RV dysfunction and/or systemic venous congestion. TV reconstruction, annuloplasty, or valve replacement is indicated in some cases of severe TR.

TEE is useful intraoperatively to assess the need for TV surgery when TR is secondary to annular dilatation and/or elevated pulmonary artery pressures, particularly during MV surgery. After correction of MV disease, TV surgery may be required if persistent severe TR or annular dilatation (> 30 mm) is still present (Fig. 49-14).

### Severity of tricuspid regurgitation

Echo findings in severe TR include:

- A large regurgitant (color) jet (> 10 cm²), vena contracta > 7 mm, or eccentric jet
- Annular dilatation (≥ 30 mm), leaflet coaptation, anatomic clues (e.g., vegetations, myxomatous disease)
- Dilated right atrial (RA)
- Dilated RV and paradoxical interventricular septal wall motion
- Pulmonary hypertension
- Dilated venae cavae and hepatic veins with minimal respiratory flow variation and systolic flow reversal

### Pulmonary hypertension

In the absence of pulmonary stenosis, pulmonary artery systolic pressure is equal to RV systolic pressure (RVSP). RVSP is estimated using the simplified Bernoulli equation (∆P = 4V²) from the peak TR velocity (V). ∆P is then the pressure gradient across the TV, or the pressure difference between the RA and RV (∆P = P_{RV} - P_{RA}). In the absence of elevated RA pressures, the right atrial pressure (P_{RA}) is estimated at 10 mmHg. Therefore,

\[
RVSP = P_{RA} + ∆P = 10 + 4V^2
\]

Where RVSP = RV systolic pressure and V = peak TR velocity (ms).

### Severity of pulmonary hypertension

Normal pulmonary artery systolic values are 18 to 25 mmHg with a mean of 12 to 16 mmHg.2 Pulmonary hypertension is defined as systolic pulmonary artery pressure greater than 30 mmHg or mean pulmonary artery pressure less than 20 mmHg at rest.2 Commonly used values using RVSP to estimate the severity of pulmonary hypertension are shown in Table 49-6.
Any evaluation of prosthetic valve function should include an assessment of transvalvular pressure gradients and valve area, similar to the evaluation of native valve function. However, its sensitivity is impaired by difficulty in visualizing structures around and behind the prosthesis, particularly for mechanical valves. Prosthetic material attenuates the ultrasound beam and causes multiple reverberations, hampering interpretation. TEE is often required for the complete evaluation of prosthetic valve structure and function. Cinefluoroscopy may also be a relatively quick and useful method for determining leaflet mobility in mechanical valves and is indicated when mechanical valve thrombosis is suspected. When valve dysfunction is suspected, 2D echo with Doppler and color flow in addition to TEE may be necessary for a comprehensive evaluation of valve function. Such an evaluation of valve function is summarized in Table 49-7. TEE findings in common complications of prosthetic valves are shown in Table 49-8.

### Prosthetic Valve Pressure Gradients

Like native valve gradients, prosthetic valve pressure gradients can be obtained using the simplified Bernoulli equation:

$$\Delta P = 4V^2$$

Where:
- \(\Delta P\) = pressure gradient
- \(V\) = velocity

**Table 49-7** Evaluation of prosthetic valves

<table>
<thead>
<tr>
<th>Pressure gradients</th>
<th>(\Delta P = 4V^2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Valve area</td>
<td>MV prostheses: area = 220/PHT</td>
</tr>
<tr>
<td>Regurgitation/leaks</td>
<td>AV or MV prostheses: (A_1V_1 = A_2V_2)</td>
</tr>
<tr>
<td>Leaflet mobility/ restriction</td>
<td>Size, symmetry, velocity, eccentricity Degree of leaflet excursion</td>
</tr>
<tr>
<td>LV size/function</td>
<td>LV dimensions, EF</td>
</tr>
<tr>
<td>Pulmonary hypertension</td>
<td>RVSP = 4V^2 + 10</td>
</tr>
<tr>
<td>Compare with prior echo</td>
<td>Change in gradients, area, leaks</td>
</tr>
</tbody>
</table>

\(\Delta P = \) pressure gradient; \(V = \) velocity; MV = mitral valve; PHT = pressure half-time; AV = aortic valve; \(A_1V_1 = \) flow (area, \(\times\) velocity) proximal to the valve prosthesis; \(A_2V_2 = \) flow (area, \(\times\) velocity) across the valve prosthesis; LV = left ventricle; EF = ejection fraction; RVSP = right ventricular systolic pressure.

Source: From Zabalgoitia. With permission.
equation \( \Delta P = 4V^2 \). Compared to normal native valves, homografts and the newer nonstented bioprostheses have similar velocities and pressure gradients, while mechanical valves have higher flow velocities. Prosthetic valves except ball-cage valves normally have pressure gradients since they are by design obstructive, with gradients increasing as valve size decreases. High gradients are often seen with 19-mm AV prostheses. High gradients in prosthetic valves may be seen for other reasons as well, such as high cardiac output, valve obstruction, or significant valvular regurgitation (due to increased flow).

**Prosthetic valve area**

As in the case of native valves, prosthetic valve area can be calculated using the continuity equation or pressure half-time method. For AV prostheses, the continuity equation \( A_1V_1 = A_2V_2 \) is usually used. The LV outflow tract (LVOT) diameter or outer diameter of the sewing ring (not its internal diameter) should be measured for accurate estimation of \( A_1 \). For MV prostheses, use of the continuity equation is preferred, since the pressure half-time equation may overestimate the true prosthetic MV area. The continuity equation should not be used if there is significant aortic regurgitation or MR. The same equations may be used for the calculation of prosthetic TV areas.

**Paravalvular leaks**

Normal amounts of regurgitation are expected with prosthetic valves owing to the built-in transvalvular regurgitation (“closing volume”). The amount of regurgitation increases with valve size, the size of the gap between the occluder and the rim, and lower heart rates. Echo findings\(^4\) of normal prosthetic valve regurgitation include:

1. AV: regurgitant area < 1 cm\(^2\) and length of jet < 1.5 cm
2. MV: regurgitant area < 2 cm\(^2\) and length of jet < 2.5 cm
3. Characteristic flow patterns (Medtronic-Hall, one central jet; Star-Edwards, two curved side jets; Bjork-Shiley, two unequal side jets; St. Jude Medical, two side jets and one central jet)

Larger leaks in other locations (Fig. 49-15) are abnormal and may be associated with significant hemodynamic compromise, hemolysis, or valve dehiscence.\(^2\)\(^8\) TEE is often required to fully assess prosthetic valve regurgitation because of the limited sensitivity of TTE. Echo characteristics that differentiate physiologic from nonphysiologic regurgitation are summarized in Table 49-9.\(^2\)\(^8\)

**INFECTIVE ENDOCARDITIS**

TEE is the procedure of choice for the detection of vegetations in infective endocarditis, with better sensitivity (50 percent for TTE vs. 90 percent for TEE) and specificity (95 percent for TTE vs. >95 percent for TEE) for native valve endocarditis compared to TTE.\(^2\)\(^\text{9,30}\) Characteristics of valvar vegetations are listed in Table 49-10.\(^2\)\(^9\) However, early in the course of infective endocarditis, vegetations may not have these typical characteristics and TEE should be repeated if the clinical suspicion is high. Compared with native valve endocarditis, it is more difficult to identify vegetations on prosthetic valves because of artifact from the prosthetic materials. Often both TTE and TEE are useful to detect vegetations, although TEE has lower sensitivity compared with TEE for prosthetic valve endocarditis.\(^8\) TEE is particularly sensitive for identifying ring abscesses. Complications of infective endocarditis (Fig. 49-16) include:

1. Paravalvular abscesses
2. Valve destruction or perforation, leaflet rupture, or dehiscence of prosthetic valves
3. Fistulas
4. Pseudoaneurysms
5. Emboli
EVALUATION OF SPECIFIC DISORDERS

CORONARY ARTERY DISEASE

Myocardial ischemia/infarction

The echo manifestation of myocardial ischemia is a decrease in contractility or systolic wall thickening of the ischemic territory that is manifest within seconds of the onset of ischemia, prior to evidence of electrocardiographic ischemia. Abnormal wall thickening is a better indicator of ischemia than wall motion, since infarcted myocardium may be passively pulled or tethered by adjacent normal myocardium, resulting in apparent wall motion without active contraction. Ancillary signs of ischemia include an increase in end-systolic LV volume and a decrease in global contractility or EF. Hypokinesis is decreased contractility (<30 percent wall thickening); akinesis is the absence of contractility (<10 percent wall thickening); and dyskinesis is outward motion during systole. Echo is accurate at localizing the site of coronary obstruction (Fig. 49-17). However, it usually overestimates infarct size due to myocardial stunning, which has resulted in a lack of correlation between wall motion abnormalities detected on echo in the setting of an acute myocardial infarction and infarct extent.

Ischemic, infarcted, stunned, or hibernating myocardium?

Myocardial segments may be dysfunctional secondary to ischemia, infarction/scar, or stunned or hibernating myocardium. Stunned myocardium is postischemic ventricular dysfunction that occurs when reperfusion of the occluded artery has been achieved but the wall motion and thickening of the corresponding myocardial segment remain abnormal—a condition that may last for days to weeks. Hibernating myocardium results from chronic ischemic dysfunction when the myocardial tissue is chronically hypoperfused owing to inadequate blood flow, resulting in abnormal wall motion and thickening, but it usually recovers after successful revascularization. Resting echo may help differentiate viable (stunned or hibernating) myocardium from nonviable (infarcted or scarred) myocardium based on wall thickness. Thicker myocardium is more likely to be viable, while thinned and fibrotic myocardium most likely represents scar. The specificity of these criteria is quite low, however. Viability assessment

<table>
<thead>
<tr>
<th>Table 49-9</th>
<th>Physiologic and nonphysiologic valvular regurgitation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regurgitant Jet</td>
<td>Physiologic</td>
</tr>
<tr>
<td>Size</td>
<td>Small, narrow</td>
</tr>
<tr>
<td>Symmetrical</td>
<td>Yes</td>
</tr>
<tr>
<td>Velocity</td>
<td>Low</td>
</tr>
<tr>
<td>Eccentric</td>
<td>No</td>
</tr>
</tbody>
</table>

Source: Modified from Zabalgoitia. With permission.
can be significantly improved with dobutamine or stress echocardiography.\(^8\) A biphasic response on dobutamine echo is the most sensitive parameter for viable myocardium and is associated with improved survival after revascularization. This is evidenced by an improvement in wall motion and thickening or recruitment at low-dose dobutamine (10 to 20 \( \mu \)g/kg/min) followed by worsening of wall motion and thickening at higher doses (30 to 40 \( \mu \)g/kg/min) when the ischemic threshold is reached.

**Left ventricular aneurysm and mural thrombus**

A true ventricular aneurysm consists of a thin wall (< 7 mm) that is echogenic (and sometimes calcified) and has

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Vegetation</th>
<th>Nonvegetation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Echogenicity</td>
<td>Similar to myocardium</td>
<td>Similar to pericardium</td>
</tr>
<tr>
<td>Low echogenicity/gray</td>
<td>High echogenicity/white</td>
<td></td>
</tr>
<tr>
<td>Location</td>
<td>Upstream surface of valve near the regurgitant jet</td>
<td>Downstream surface of valve</td>
</tr>
<tr>
<td>Motion</td>
<td>Mobile, prolapses</td>
<td>Less mobile</td>
</tr>
<tr>
<td>Shape</td>
<td>Amorphous, lobulated</td>
<td>Filamentous, strand-like narrow base of attachment</td>
</tr>
<tr>
<td>Regurgitation</td>
<td>Severe regurgitant jet</td>
<td>Mild or no regurgitant jet</td>
</tr>
<tr>
<td>Other</td>
<td>Paravalvular abscess/leak</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td>Fistula</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Valve dehiscence</td>
<td></td>
</tr>
</tbody>
</table>

Source: Modified from Schiller.\(^{29}\) With permission.
outward motion in both systole and diastole. Aneurysms occur apically, with inferobasal aneurysms being the second most common. Aneurysms are complications of adverse remodeling following transmural myocardial infarcts. Spontaneous echo contrast (SEC) or mural thrombus may be present within an LV aneurysm and is associated with an increased risk of embolic events. The appearance of an acute mural thrombus (same echogenicity as myocardium) generally differs from that of a chronic thrombus, which tends to be layered with areas of calcification. Compared to TTE, TEE may be limited in detecting apical thrombi because of the often suboptimal visualization of the true LV apex with TEE.

Pseudoaneurysms can also be complications of acute myocardial infarction and are characterized by the lack of a true myocardial wall. They result from contained free wall myocardial rupture in which a portion of the pericardial space limits frank rupture. Pseudoaneurysms can generally be differentiated from true aneurysms by the presence of a narrow neck (less than half of the maximum diameter) compared to the wider base of a true aneurysm.

Postinfarct ventricular septal defect

Life-threatening mechanical complications after acute myocardial infarction include free wall rupture, papillary muscle dysfunction/rupture, and ventricular septal defect (VSD) (Fig. 49-18). TTE is usually sufficient for the diagnosis of mechanical complications, although TEE may be used as an adjunct. Postinfarct VSD is uncommon (less than 1 percent of total infarcts), although it is associated with the worst outcome of mechanical complications in patients with cardiogenic shock. Unlike postinfarct papillary muscle rupture, VSD occurs with approximately equal frequency after anterior and inferior infarcts. The posteroapical septum is the most common site of postinfarct...
VSD. Echo is the gold standard for diagnosing ventricular septal rupture complicating myocardial infarction. TTE using color-flow Doppler has a sensitivity of 85 to 95 percent, while TEE has a sensitivity and specificity of 100 percent.\(^1\) Echo characteristics of postinfarct VSD, in addition to the septal defect, include the presence of a small pericardial effusion with possible intrapericardial thrombus (echogenic mobile mass in the pericardial space) and echo evidence of tamponade.\(^1\)

**Table 49-11** Diagnostic tests for acute aortic dissection

<table>
<thead>
<tr>
<th>Test</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>Pluses and minuses</th>
</tr>
</thead>
<tbody>
<tr>
<td>TEE</td>
<td>99–100</td>
<td>&gt; 89</td>
<td><strong>Pluses</strong>: Quick, semi-invasive, assesses AR, coronaries, pericardial effusion, may assess IH&lt;br&gt;<strong>Minuses</strong>: Limited assessment of IH, “blind spot” of distal ascending aorta and anterior aortic arch, reverberation artifact</td>
</tr>
<tr>
<td>CT</td>
<td>&gt; 90</td>
<td>&gt; 85</td>
<td><strong>Pluses</strong>: Quick, noninvasive&lt;br&gt;<strong>Minuses</strong>: Cannot assess branch vessels or IH, dye load/allergy</td>
</tr>
<tr>
<td>MRI</td>
<td>98–100</td>
<td>100</td>
<td><strong>Pluses</strong>: Noninvasive, assesses branch vessels, IH&lt;br&gt;<strong>Minuses</strong>: Slow, may not be available, pacemakers/device, breath-hold necessary</td>
</tr>
<tr>
<td>Angiography</td>
<td>88–91</td>
<td>&gt; 95</td>
<td><strong>Pluses</strong>: Assesses coronaries, AR, branch vessels&lt;br&gt;<strong>Minuses</strong>: Slow, invasive, dye load, may miss dissection if lumen is completely thrombosed, does not detect IH</td>
</tr>
</tbody>
</table>

IH = intramural hematoma; AR = aortic regurgitation.
Sources: From Erbel et al.,\(^3\) Sabatine,\(^3\) and Nienaber et al.\(^4\) With permission.

**THORACIC AORTIC ANEURYSM AND DISSECTION**

TTE may diagnose aortic dissection by detecting an intimal flap in the aorta (specificity 95 percent), but it has low sensitivity (80 percent) for ascending aortic dissection and even lower sensitivity for distal thoracic aortic dissection (70 percent).\(^2\) TEE is one of three imaging modalities used for the diagnosis of acute aortic dissection—TEE, computed tomography (CT), and magnetic resonance imaging (MRI)\(^3\),\(^4\)—and for the diagnosis of perioperative aortic dissections (Table 49-11). The choice of imaging modality depends primarily on the availability of the imaging procedure and patient characteristics (e.g., hemodynamic instability, presence of a pacemaker, or contrast allergy), since the overall diagnostic accuracy for TEE, CT, and MRI is comparable.\(^2\) TEE is the imaging procedure of choice for patients who are hemodynamically unstable. Compared to CT or MRI, one limitation of TEE is that it cannot image the aortic segment located between the distal ascending aorta and the proximal arch, which may decrease its sensitivity for detection of aortic dissection, hematoma, or atheroma in this region.

The main criterion for TEE diagnosis of suspected acute aortic dissection is the presence of two lumina (false and true) separated by an intimal flap (Table 49-12 and Fig. 49-19). Other findings for diagnosing aortic dissection by TEE\(^2\) include:

1. Tear or disruption of the flap continuity or jets seen with color Doppler across the flap
2. Complete obstruction of the false lumen; presence of thrombus
3. Central displacement of intimal calcification or separation of intimal layers from thrombus
4. Periaortic hematoma (echo-free spaces around the aorta)
5. Intramural hematoma (crescent-shaped echodensity with vacuolization within it on the aortic short-axis view)
6. Pericardial or pleural effusion
7. AR

It is important to define the anatomic site and extension of the dissection, the degree of AR, involvement of the coronary arteries, LV dysfunction, and the presence of pericardial effusion or tamponade.

Intraoperatively, TEE is used during reconstructive surgery for hemodynamic status, entry/exit sites, evaluation of decompression of the false lumen, and assessment of concomitant valve surgery.\(^1\) Postoperatively, TEE is used for the detection of residual regurgitation and LV dysfunction. TEE is also indicated for defining the anatomic site and size of aortic aneurysms.

**Table 49-12** True versus false lumen in aortic dissection

<table>
<thead>
<tr>
<th>True lumen</th>
<th>False lumen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systole</td>
<td>Expansion</td>
</tr>
<tr>
<td>Diastole</td>
<td>Collapse</td>
</tr>
<tr>
<td>SEC/thrombus</td>
<td>Absent or minimal</td>
</tr>
<tr>
<td>Blood flow</td>
<td>Systolic forward flow</td>
</tr>
</tbody>
</table>

SEC = spontaneous echo contrast.
Source: Erbel et al.\(^2\) With permission.
PERICARDIAL DISEASE

Pericardial effusion and tamponade

Echo is the diagnostic test of choice for detection of pericardial effusion (PE) and assessing its hemodynamic significance. TEE is usually superior to TTE for evaluating pericardial thickness and adjacent structures, although CT and/or MRI are preferred for evaluating the pericardium. Normally, the pericardial space contains 10 to 50 mL of fluid and the pericardium measures 1 to 3 mm in thickness. An increase in the volume of the pericardial fluid results in elevated pericardial pressures, leading to reduced RV filling, followed by reduced LV filling (Fig. 49-20). PE usually appears as an echo-free space surrounding the myocardium, although as protein or cellular debris increases in the fluid, it becomes increasingly echogenic.35 PE is differentiated from pleural effusion by the anterior location of PE relative to the proximal descending thoracic aorta, while pleural effusion is located posteriorly to the aorta. Epicardial fat may be confused with PE, since it is also echolucent, although epicardial fat is usually more echogenic than pericardial fluid and is usually located anteriorly.35 TEE is useful in the postoperative patient with tamponade and a small loculated PE that may be difficult to visualize on TTE. Loculated PE in postoperative cardiac surgery patients may cause tamponade in the absence of typical

Figure 49-19 Dissection of the descending thoracic aorta. Transesophageal echocardiogram showing a large aortic dissection with the diagnostic flap separating the true (TL) and false lumen (FL). A. Short-axis view. B. Long-axis view. C. Long-axis with color-flow Doppler shows flow in both the true and false lumens.

Figure 49-20 Large pericardial effusion and cardiac tamponade. Transthoracic echocardiogram of the apical four-chamber view reveals an atrial myxoma attached to the interatrial septal with a large echolucent circumferential pericardial effusion (PE) and evidence of elevated intrapericardial pressure. Classic features of tamponade are shown with right ventricular (RV) diastolic collapse and abnormal interventricular septal motion (shifted toward the left during inspiration). RA = right atrium; LA = left atrium; LV = left ventricle.
Constrictive pericardial disease

In constrictive pericardial disease, the pericardium is thickened (>3 mm) and often calcified, which reduces ventricular filling in diastole and causes diastolic heart failure. However, the absence of pericardial calcification does not rule out the diagnosis of constriction. Although echo signs of constriction (Table 49-14) are not very sensitive or specific, a completely normal echo study usually rules out constriction.28 Other imaging modalities, such as CT or MRI, may be necessary to further evaluate the pericardium and distinguish constriction from restriction.

### CARDIAC SOURCES OF EMBOLI

One of the most common indications for TEE is for the evaluation of cardiac sources of emboli, since TTE does not visualize well the potential sources of emboli (LA appendage thrombus, aortic atheroma, patent foramen ovale or atrial septal defect, LV thrombus, valvular lesions, intracardiac tumors). Small thrombi in the LA or LA appendage can be detected using TEE. In addition, factors that may contribute to or accompany atrial thrombi are often seen in the absence of an obvious thrombus: LA or LA appendage enlargement, SEC consistent with blood stasis, and decreased LA appendage velocities (< 20 mm/s). Aortic atheromas are evaluated for mobile components, plaque rupture, and ulceration and are graded as mild (< 1 mm), moderate (1 to 3.9 mm), and severe (≥ 4 mm).

### ATRIAL SEPTAL DEFECTS

TEE is superior to TTE for visualizing atrial septal defects (ASDs). The anatomic defect is visualized using two-dimensional echo and confirmed with Doppler and contrast (bubble study) using maneuvers that increase RA pressure, such as Valsalva or cough. The hemodynamic significance of the shunt is assessed using Doppler by quantifying the shunt size and determining the presence of pulmonary hypertension. Shunt quantification is obtained as the ratio of pulmonary to systemic flow (Qp:Qs) using Doppler cardiac outputs across the pulmonary valve and AV.4 All four pulmonary veins should be visualized and the presence of associated anomalies excluded. TEE plays an important role in determining the suitability of ASDs for device closure versus cardiac surgery based on the size of the ASD and the rim of tissue surrounding it as well as the degree of septal tissue redundancy.28 TEE has become essential for guiding placement of catheter-deployed closure devices and in assessing residual shunts (Fig. 49-21).
CARDIAC TUMORS

TEE, because of its high sensitivity, is the imaging modality of choice and is superior to TTE, CT, MRI, and angiography for detecting cardiac tumors. Although MRI may not detect small cardiac tumors, it is usually performed after TEE for further differentiation of thrombus (presence of methemoglobin or hemosiderin) from neoplasm, since it is superior to TEE for tissue characterization. MRI examinations are multiplanar and typically include fast T1- and T2-weighted techniques with administration of gadolinium (a paramagnetic contrast agent) and a technique for imaging moving structures with single-slice breath-hold, such as fast gradient-echo sequences (e.g., FLASH). Primary tumors are more likely to affect the myocardium, while secondary tumors usually involve the pericardium with secondary intramyocardial infiltration. Atrial myxomas are the most common (up to 25 percent) primary cardiac tumors. Atrial myxomas as seen on TEE or TTE show several typical features:

- Ninety percent originate in the interatrial septum, near the fossa ovalis.
- A spherical mass with a speckled appearance is often seen in RA myxomas, while a villous amorphous mass may be seen in LA myxomas.

Metastatic tumors to the heart most commonly arise from the breast or lung but may include leiomyosarcoma (Fig. 49-22B). Metastatic tumors usually affect the pericardium and result in pericardial effusion. In addition, some tumors metastasize through the inferior vena cava (renal cell, hepatoma), affecting the right heart more than the left; TEE allows for visualization of the route of extension. Tumors involving the cardiac valves are rare (often fibroelastomas) and may affect valve competence and global LV function. MRI and ultrafast CT may be a useful adjunct in delineating tumors of the cardiac valves.

LEFT VENTRICULAR OUTFLOW TRACT OBSTRUCTION

TEE is used intraoperatively for septal myectomy for treatment of obstruction of the LV outflow tract (LVOT) due to...
to hypertrophic cardiomyopathy. Systolic anterior motion (SAM) of the MV may cause LVOT obstruction; TEE is used to define the structures involved in the SAM (e.g., chordae, anterior leaflet). As in aortic stenosis, LVOT gradients may differ intraoperatively versus preoperatively owing to different hemodynamics. Color-flow Doppler typically demonstrates turbulent blood flow (mosaic pattern) at the site of LVOT obstruction. An eccentric jet of MR may also be seen if there is abnormal coaptation of the mitral leaflets (usually a posteriorly directed jet owing to abnormal coaptation of the anterior mitral leaflet).

TEE is also helpful during resection of lesions causing subaortic stenosis. It can determine the location and severity of obstruction. It is also useful for evaluating the success of the surgery in relieving the obstruction and in detecting MR that may result from the surgery.22

References


