Echocardiographic Assessment of Valvular Heart Disease

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Key Points

General

- Echocardiography is the primary tool for the noninvasive detection, quantitation, and follow-up assessment of valvular heart disease.
- No single echocardiography method can be considered a robust "gold standard" for quantification of lesion severity in all situations.
- Valve lesion severity is often diagnosed by a synthesis of several anatomic, qualitative, and quantitative echocardiography severity indicators.
- For best practice, laboratories should routinely obtain as many severity parameters as possible, so that adequate scanning and interpretation skills are maintained for accurate diagnoses in difficult cases.
- Transesophageal echocardiography (TEE) may be needed in conjunction with the transthoracic exam (TTE) for a definitive diagnosis.

Color Doppler

- Imaging the full extent of an expanding jet within the receiving chamber using multiple views is necessary and helpful for determining lesion severity.
- The overall jet size within the receiving chamber viewed in isolation can be one of the least effective parameters for judging lesion severity, unless it is very small or very large.
- Applying several of the described quantitative methods requires an understanding of all of the component parts of a jet lesion as depicted by color Doppler.

Aortic Stenosis

- The degree of apparent cusp immobility does not always reflect the degree of hemodynamic severity, and quantitative measures are needed.
- The echocardiography imaging window that will yield the highest aortic valve velocity (most coaxial to the jet's net vector within the vena contracta) is not predictable from valve morphology or jet appearance by color Doppler.
- Flow velocities and gradients can vary tremendously beat-to-beat with irregular rhythms.
- Valve area by continuity equation: The left ventricular outflow tract (LVOT) diameter measurement is squared, so that any significant LVOT diameter (D) measurement error will also be squared, producing a potentially unacceptable error in the aortic valve area calculation.
- For a given valve area, the measured aortic valve gradient is strongly influenced by heart rate, stroke volume, ejection duration, left ventricle (LV) contractility and preload and afterload. Therefore, peak and mean velocity and pressure gradient measurements in isolation can be misleading.

Aortic Regurgitation

- Determining the severity of chronic aortic regurgitation can be one of the most challenging tasks for a cardiologist. The final diagnosis may be the result of an integrated analysis of several parameters.
- There are many potential pitfalls for using color Doppler jet size as an indicator for regurgitation severity. With
good imaging practices are employed, however, a very small aortic regurgitation (AR) jet by color Doppler likely represents mild AR, and a very large AR jet likely represents severe AR.

• In AR, pressure half-time (PHT) values are generally useful only in the extremes and when the continuous-wave (CW) Doppler signal’s outer edge is clearly definable and the interrogating beam is well aligned with the regurgitant jet.

• Acute severe AR usually represents a surgical emergency, in which case early TEE for confirmation of the diagnosis, its mechanism, and surgical planning is indicated.

Mitral Stenosis

• Severe mitral valve (MV) stenosis is most often rheumatic in origin.

• Mitral valve areas that cause clinical symptoms can vary markedly among patients depending on body size, cardiac output, and heart rate.

• The MV area should be confirmed using two or three methods, given the number of potential pitfalls of each.

• The mean MV gradient may be low in severe mitral stenosis (MS) when there is low cardiac output or bradycardia.

• In equivocal cases exercise stress Doppler may be useful.

Mitral Regurgitation

• Detection of mitral regurgitation (MR) by Doppler is relatively easy. However, accurate grading of MR severity can be technically challenging. No one method is sensitive and specific in all cases.

• When several findings are in agreement regarding lesion severity, an accurate “integrated diagnosis” is possible in most situations.

Tricuspid Valve Disease

• Tricuspid valve stenosis is an uncommon diagnosis. It should be considered in all symptomatic patients with rheumatic heart disease.

• Clinically significant tricuspid stenosis (TS) is poorly tolerated and is associated with modest resting tricuspid inflow gradients (mean gradient >5 mm Hg).

• Paradoxical septal motion by M-mode and two-dimensional (2D) examination and hepatic vein systolic flow reversal can be important signs of severe TR.

• Torrental “unobstructed” TR may be easily missed by color Doppler alone.

Pulmonary Valve Disease

• Low-velocity severe PR can be easily missed by color Doppler. Careful attention to the PV spectral Doppler signal and recognition of the signs of RV volume overload are important.

• PS is relatively uncommon in adult patients, although it is relatively easily to diagnose in most cases by routine PV Doppler examination.

Prosthetic Valve Assessment

• Distinctive imaging artifacts aid in recognition of mechanical prosthetic valves.

• Shadowing and reverberation artifacts, however, also hamper detection of either obstructive or insufficiency lesions and make their mechanisms (thrombus, pannus, vegetation, paravalvular leak, or leaflet malfunction) difficult to determine.

• Whenever prosthetic valve dysfunction is suspected, comprehensive TTE and TEE are frequently needed in order to address the situation.

Overview

Echocardiography is the primary tool for the noninvasive detection, quantitation, and follow-up assessment of valvular heart disease. In most cases, features of the two-dimensional (2D) [increasingly three-dimensional (3D)5] and M-mode and Doppler evaluation provide important measurable parameters and indirect clues regarding lesion severity and etiology.37 Because Doppler is used to evaluate a wide range of non–valve-related cardiovascular pathology, basic Doppler concepts are presented in Chapter 5. An echocardiogram can often quickly and accurately define valve lesion as clearly mild or clearly severe based upon certain highly specific features. Frequently, however, valve disease is neither clearly mild nor clearly severe. No single echocardiography method can be considered a robust gold standard for quantification of lesion severity in all situations. In challenging cases, lesion severity is often diagnosed by a synthesis of several anatomic, qualitative, and quantitative echocardiography severity indicators that are outlined herein. Valve disease can be simple (isolated obstructive or insufficiency lesions) or complex. Examples of complex valve disease include mixed stenosis and regurgitation of the same valve, multiple valve involvement, associated dynamic obstructive lesions, congenital malformations, and cardiomyopathies, or prosthetic valve dysfunction. Such complications and other variables imaging artifacts, tachycardia, irregular rhythm, low output states, and high or low afterload or preload influence the extent to which certain Doppler methods are applicable or valid. Accordingly, the most common pitfalls of each method will be addressed. For best practice, laboratories should routinely obtain as many lesion severity parameters as possible, so that adequate skills are maintained for accurate diagnoses in difficult cases. Transesophageal echocardiography (TEE) may be needed in conjunction with the transthoracic exam (TTE) for a definitive diagnosis.

Grading System for Valve Lesions

Valve lesions are reported as mild, moderate, or severe. The use of “mild-to-moderate” or “moderate-to-severe” is desirable, when appropriate, because lesions frequently lie within these gray zones. Left-sided valve regurgitant lesions [aortic and mitral] that are just detectable by color or continuous-wave (CW) Doppler and are of little or no hemodynamic significance may be called “trace” or “trivial.” Trace or trivial
and even mild right-sided [tricuspid and pulmonary] regurgitant lesions are common in individuals with normal anatomy, and reporting these often nonpathologic findings is not always necessary unless there has been a change in comparison with a prior examination. A numerical classification scheme [e.g., 1+, 2+, 3+, 4+] for regurgitation (or stenosis) is potentially confusing because the definitions may vary among laboratories [e.g., 3+ may mean “moderate” in one laboratory and “moderate-to-severe” in another].

Jet Lesion Anatomy by Color Doppler

Applying several of the quantitative methods described below requires an understanding of the component parts of a jet lesion as depicted by color Doppler. As illustrated in Figure 21.1, a jet lesion consists of a proximal flow convergence (PFC) zone, flow within the anatomic orifice, a physiologic orifice [vena contracta], and the expanding jet within the receiving chamber. Many of the following comments regarding jet anatomy by color Doppler apply more to regurgitant jets than to stenotic lesions.

Proximal Flow Convergence and Proximal Isovelocity Surface Area

Laminar blood flow exiting a pressurized chamber produces a localized, uniform PFC pattern by color Doppler just proximal to a narrowed orifice. The PFC size varies directly with the flow rate, which is related to the orifice size and driving pressure. Discriminable color bands are present within the PFC zone due to a velocity gradient and progressive aliasing of laminar blood flow proximal to the orifice. The geometric area of the color transition surface is known as a proximal isovelocity surface area [PISA]. The PISA and CW Doppler signal of a regurgitant jet can be used to calculate the effective regurgitant orifice area [EROA] and regurgitant volume [RV]. Under ideal circumstances [relatively flat anatomic orifice, relatively discrete opening, and appropriate color Doppler Nyquist limit setting], the outer edge PISA assumes a hemispheric shape with a measurable radius r. The area of a hemisphere [PISA] is 2πr². Blood flow velocity at the site of this hemisphere is the same as the aliasing velocity, Vₐ, which is obtained from the color map. Instantaneous flow is the cross-sectional area [PISA] multiplied by the instantaneous flow velocity [Vₐ]:

\[ \text{Flow} = 2\pi r^2 \times V_a \]

which is simplified as

\[ \text{Flow} = 6.28r^2 \times V_a. \]

The EROA can be calculated by a modification of the continuity equation because instantaneous peak PISA flow must equal instantaneous peak flow across the regurgitant orifice [Vₚk]. Using the PISA radius (r), Vₐ [from the color Doppler map], and peak regurgitation velocity (Vₚk) from the mitral or aortic regurgitation CW Doppler signal, EROA can be calculated:

\[ \text{EROA} = 6.28r^2 \times V_a / V_{pk}. \]

This method has been validated, when imaging conditions permit, for the mitral, aortic, and tricuspid valves.

Regurgitant volume [RV] is the cross-sectional area of flow [EROA] multiplied by the time-velocity integral [VTI] of flow at the EROA, as measured by CW Doppler (see examples below):

\[ \text{RV} = \text{EROA} \times \text{VTI}. \]

Analysis of the PFC for derivation of the PISA radius, combined with CW Doppler, can be used to calculate the area of a stenotic mitral valve (see example below). Although a PISA signal may not always be adequate for quantitative measurement, the presence of a prominent PFC is a potentially important finding that should raise suspicion for a hemodynamically important lesion.

Vena Contracta by Color Doppler

Immediately downstream from the anatomic orifice, is a physiologic orifice, where the highest velocity laminar flow occurs within an area slightly smaller than that of the upstream anatomic orifice. This region of compressed laminar flow is known as the vena contracta. The highest flow velocities measured by CW Doppler occur within the vena contracta and not within the anatomic orifice. The cross-sectional area of the vena contracta is known as the effective regurgitant orifice area (EROA), which too, is slightly smaller than the anatomic orifice area. With careful imaging [zooming the proximal jet region and applying color Doppler at a high frame rate [small color sector]], the vena contracta width can be measured (see examples below). The vena contracta width increases with increasing regurgitation severity, making this measurable parameter an important severity indicator.

FIGURE 21.1. Illustration of jet lesion “anatomy” by color flow Doppler. PFC, proximal flow convergence; VC, vena contracta.
Expanding Jet by Color Doppler

Imaging the full extent of an expanding jet within the receiving chamber using multiple views is necessary and helpful for determining lesion severity. However, the overall jet size within the receiving chamber viewed in isolation can be one of the least effective parameters for judging lesion severity, unless it is very small or very large. This is because color Doppler represents a velocity map display and not a volume map of blood flow. Due to convective forces, nonregurgitant “bystander” blood within the receiving chamber can be entrained by regurgitant blood contained in the jet and labeled by color Doppler. Accordingly, relatively small jets under high driving pressure (high velocity) can entrain significant surrounding blood in the receiving chamber, thereby producing a potentially misleadingly large expanding jet lesion by color Doppler. By the same token, severe regurgitation may produce a deceptively small color Doppler jet within a receiving chamber if the driving pressure is low [e.g., severe tricuspid regurgitation (TR) with normal RV systolic pressure, or severe mitral regurgitation (MR) with low systemic pressure and high left atrial (LA) pressure]. The extent of receiving chamber enlargement must be taken into account when thinking in terms of jet size and depth of the image sector, pulse repetition frequency (PRF), manufacturer. Operators should be familiar with these and other variables that may affect the color Doppler signal.

Technical Notes on Color Jet Area

Jet size is inversely related to the color Doppler Nyquist limit setting. As a standard, the color Doppler Nyquist limit should be set in the range of 50 to 60 cm/s. Color gain settings also influence jet size. The color gain should be increased until there are a few color pixels overlaying areas having no blood flow. Then the gain should be decreased gradually until these color “noise” speckles just disappear. Other factors may also affect the appearance of the jet size by color Doppler [size and depth of the image sector, pulse repetition frequency (PRF), manufacturer]. Operators should be familiar with these and other variables that may affect the color Doppler signal.

The sonographer should evaluate the possibility of separate [multiple] or geometrically complex proximal flow convergence zones. Using color Doppler in both short axis and multiple long axis and “off-axis” imaging planes along the entire extent of leaflet coaptation zones, the sonographer can often demonstrate all components of the jet lesion [PFC, anatomic orifice, vena contracta, expanding jet] within a single or a few “best” imaging planes. With eccentric jets, the proximal components of a lesion may be best viewed in one imaging plane and the expanding jet lesion in another. However, all elements should ultimately be included. Three-dimensional color Doppler imaging offers a potential means for the more expedient analysis of complex jet geometries and overall jet size.

Aortic Valve Disease

Aortic Stenosis

Two-Dimensional and M-Mode Exam and Differential Diagnosis

The normal, trileaflet aortic valve (AoV) is an almost perfectly symmetrical structure. Leaflet number and symmetry are best appreciated in the parasternal short axis view (Fig. 21.2). Normal aortic leaflets, also known as cusps, are thin and highly pliable and have a deeply semilunar or “cupped” conformation upon diastolic coaptation. During rapid systolic excursion, adjacent aortic cusps come into parallel alignment within the bloodstream (Fig. 21.3A) while simultaneously undergoing partial inversion so that the leaflets, in short axis, assume a circular [cylindrical] conformation corresponding to the circular aortic annulus (Fig. 21.2A). This overall “tubular” systolic valve shape minimizes blood flow resistance and shear forces within the leaflets. Some portions of thin and normal aortic valve leaflets may be difficult to visualize. On the other hand, certain portions of the normal semilunar-shaped aortic cusps behave as parabolic reflectors of the ultrasound beam, producing characteristic regional areas of increased echogenicity, which should not be mistaken for fibrosis or calcification. Disease or “degenerative” leaflets become increasingly thickened and well visualized throughout, except in advanced disease, in which case severe calcification can produce shadowing artifacts and leaflet dropout.

Calcific Aortic Stenosis

Calcific degeneration of a previously normal, trileaflet valve is the most common cause of acquired aortic stenosis (AS) in adults older than 65, and it is frequently encountered in almost all adult echocardiography laboratories. Aortic sclerosis is an even more common, possibly related, precursor lesion that is differentiated from AS because it produces no significant resting gradient [peak velocity <2.5 m/s]. Aortic sclerosis (without significant stenosis) is noteworthy in echocardiography reporting because it may be a marker for a more generalized atherosclerotic process and increased risk, including possible progression to AS. Aortic valve sclerosis is recognizable on the 2D exam as leaflet thickening and increased echogenicity with focal minimal or mildly reduced systolic cusp separation (Figs. 21.2B and 21.3B). Focal hyper-echoic regions indicating fibrosis and calcium deposits may occur throughout the leaflets, but they are typically more pronounced in the leaflet bases. Decreased cusp mobility also typically occurs first in the leaflet bases (Fig. 21.2B) and gradually works its way into the body of the leaflets (Fig. 21.3C-D). The degenerative process can be balanced among the three cusps or it may involve one or two cusps predominantly.

Insidiously, over decades, sclerotic aortic valve cusps can become increasingly thickened and immobilized, sometimes with bulky, protruding calcific lesions that can cause prominent shadowing and side lobe artifacts within the left ventricular outflow tract (LVOT) (Fig. 21.3D). Reduced aortic
cusp separation is easily detected by M-mode of the aortic cusps (Fig. 21.4). In severe AS, the aortic cusps can appear almost “frozen” in a near closed position throughout the cardiac cycle (Fig. 21.3D).

Calcific aortic stenosis is frequently accompanied by some lesser degree of aortic regurgitation due to inadequate closure of the dysmorphic leaflets. Mitral regurgitation also frequently accompanies calcific AS due to commonly associated degenerative mitral annular and mitral leaflet calcification (Fig. 21.3D).

**Congenitally Bicuspid Aortic Valves**

Congenitally bicuspid aortic valves are common (1% to 2% of all individuals, predominantly in males). Although not all are destined to develop important hemodynamic lesions, calcific degeneration of a congenitally bicuspid aortic valve is the most common cause of AS in adults younger than 65, and the second most common cause of severe AS overall. Bicuspid aortic valves can also exhibit predominant aortic regurgitation (AR) or mixed AS and AR. Congenital AS diagnosed in childhood, adolescence, or early adulthood is more commonly associated with a unicuspid or a commissural valve. The majority of bicuspid aortic valves appear to have fusion of any two of the three aortic cusps along a single “would be” coaptation line. This thickened fusion zone, between two aortic sinuses of Valsalva, is known as a “pseudo-raphe,” which can often exhibit prominent focal thickening or calcification. The pseudo-raphe’s presence can sometimes deceptively give the valve a normal trileaflet appearance in the parasternal short axis view upon diastolic closure, potentially resulting in a missed diagnosis. Upon
systolic opening, however, the two asymmetrical leaflets typically form an almond- or football-shaped opening [Fig. 21.5]. Truly bicuspid valves [two symmetrical sinuses of Valsalva with two symmetrical cusps] are less commonly encountered. Because bicuspid valves cannot undergo normal systolic leaflet straightening within the bloodstream, shear forces within the leaflet can be great, producing early degeneration. In the late stages of bicuspid valve AS, the valve can become heavily calcified and dysmorphic to the point of being indistinguishable on echocardiography from trileaflet degenerative calcific AS.

**Other Diagnostic Clues for Bicuspid Aortic Valves**

Abnormal dilatation of the aortic root, ascending aorta, or both are commonly associated with bicuspid aortic valves [28] [Fig. 21.5A]. When a dilated aortic root or dilated ascending aorta or both are encountered, without hypertension, particularly in young adults, a bicuspid aortic valve should be suspected. Efforts to clearly image the ascending aorta must always be carefully undertaken because the degree of aortic dilatation may influence surgical timing as much as, or more than, the presence of aortic valve stenosis (or regurgitation). Bicuspid aortic valves may exhibit relatively preserved mobility in their bases, resulting in systolic doming in the parasternal long axis view [Fig. 21.5B]. M-mode evaluation may show an eccentric closure line, owing to asymmetrical leaflet size and orientation in the parasternal long axis view [Fig. 21.5D]. Bicuspid valves may be severely stenotic due to small slit-like orifices, even though, by planar imaging, the overall cusp mobility and separation may appear deceptively adequate.

**Rheumatic Aortic Stenosis**

Rheumatic aortic stenosis is the third most common cause of adult valvular AS in Europe and North America.
Rheumatic AS rarely occurs in the absence of associated rheumatic mitral valve disease. In rheumatic AS, leaflet thickening due to inflammation occurs primarily in the leaflet leading edges and not the bases as with calcific AS (Fig. 21.6). Commissural fusion eventually produces a small, immobile “fish-mouth” orifice that may also produce significant AR. Systolic doming of the leaflets (Fig. 21.7B) may be present because of relatively preserved mobility of the leaflet bases. In advanced disease, severe cusp calcification, thickening, and immobility can render rheumatic AS indistinguishable from the other forms of advanced valvular AS.

**OTHER MORPHOLOGIC FEATURES OF AORTIC STENOSIS**

Concentric left ventricular hypertrophy in the presence of a morphologically abnormal aortic valve that has reduced systolic opening should always raise suspicion for clinically important AS or severe LVOT obstruction of any type. However, because hypertension is a frequent comorbid condition, this is a nonspecific finding. In the late stages of valvular AS, more often in men, the ventricle can become dilated.

![FIGURE 21.4. M-mode tracing of aortic valve (arrow), showing moderately reduced systolic cusp separation due to calcific AS. RVOT, right ventricular outflow tract; LA, left atrium.](image)

![FIGURE 21.5. Congenitally bicuspid aortic valve by surface echo. (A) Dilated aortic root (3.8 cm, red line). (B) Systolic doming of the cusps (arrow). (C) Short axis view shows ovoid or almond-shaped systolic opening. Note: This view likely represents exaggerated orifice area due to cusp systolic doming (see image B). (D) M-mode, eccentric cusp closure line (arrow), typical of a bicuspid aortic valve.](image)
Nonvalvular Left Ventricular Outflow Tract Obstruction

Some patients referred to the lab for possible valvular AS are found to have nonvalvular LVOT obstruction. Nonvalvular LVOT obstructive lesions identifiable by echocardiography include dynamic subaortic stenosis, caused by systolic anterior motion (SAM) of the mitral leaflets; fixed subaortic stenosis; and supravalvular aortic stenosis. These entities are discussed in other chapters.

Severity Assessment by Two-Dimensional Imaging

Cusp Mobility

When the aortic valve is abnormal, the echocardiogram report should generally include a statement regarding the degree of leaflet thickening and the extent to which cusp mobility or separation is reduced (mildly, moderately, or severely). However, the degree of apparent cusp immobility does not always reflect the degree of hemodynamic severity, and quantitative measures are needed.

Valve Area by Planimetry

Although aortic valve planimetry by surface imaging may be possible using state-of-the-art surface echocardiography, the method has been validated with TEE\textsuperscript{29,30} [Fig. 21.2D]. Aortic valve area by planimetry is most reliable in calcific AS because the relatively flat stenotic orifice is more likely to lie within a 2D imaging plane. Aortic valve area by TEE is most often employed when Doppler data (below) are indeterminate (e.g., irregular rhythm) or felt to be unreliable, when mixed AS and aortic insufficiency [AI] are present, or when Doppler and catheterization discrepancies are unexplained.

**Technical Considerations and Pitfalls**

Planimetry by TEE is frequently not possible when severe calcification shadows the leaflet’s leading edges. When the cusp margins are clearly defined, a dedicated effort should be made to obtain the most symmetrical short axis view possible, ideally with acquisition of three relatively reproducible area measurements. If gain settings are too low, leaflet “dropout” can lead to area overestimation of the aortic valve. If 2D gain settings are too high, “blooming” of the signal into the orifice can cause an underestimation of valve area.
The method is not reliable in congenital AS (bicuspid valves) because the stenotic orifice may be curvilinear and not lying within a single imaging plane. In this situation, attempted imaging planes that cut across a relatively funnel-shaped valve will overestimate valve area. The area of the systolic color Doppler flow map in short axis should not be measured by planimetry due to the low spatial resolution of color Doppler.

**Doppler Evaluation for Aortic Stenosis**

**Continuous-Wave Doppler**

The LVOT obstructive gradients are detected and measured using CW Doppler. Basic Doppler concepts, the pressure-velocity relationship, and sites of data acquisition are reviewed in Chapter 5. Spectral Doppler is accurate for clinical purposes when the vectors of the interrogating ultrasound beam and the jet lesion are approximately coaxial (within 20 degrees). Continuous-wave Doppler is needed for measuring pathologic velocities (2.5 to 7 m/s) that exceed the Nyquist limit of pulsed Doppler. Doppler-derived instantaneous velocity measurements are converted to a pressure drop or pressure gradient across the valve using the modified Bernoulli equation:

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

Using Doppler, the calculated peak pressure gradient occurs simultaneously with the maximum transvalvular flow velocity. The mean pressure gradient is derived by averaging instantaneously sampled gradients throughout the ejection period:

$$\Delta P_{\text{mean}} = 4V_1^2 + 4V_2^2 + 4V_3^2 + \ldots + 4V_n^2/n$$

The peak and mean gradients are automatically calculated by the ultrasound system or off-line analysis program when the user electronically “traces” the outer edges of the CW spectral Doppler envelope (Figs. 21.7C and 21.8). Both peak and mean gradients should be reported.
COMPARISON WITH CATHETERIZATION

Hemodynamic “loading conditions” and heart rate may be vastly different at the time of catheterization versus echocardiography, leading to potentially conflicting results. It is important to bear in mind that the peak instantaneous pressure gradient obtained by Doppler is not comparable to a reported catheter-derived peak-to-peak gradient. A catheter-derived peak-to-peak gradient is simply the difference between the peak pressure recorded in the left ventricle (LV) and in the aorta using either simultaneous fluid-filled transducer recordings or the “pull-back” method. Because peak left ventricular and aortic pressures do not occur simultaneously (left ventricular peak pressure occurs sooner than the aortic peak pressure), the peak-to-peak gradient is not, in fact, a gradient, but rather a useful clinical parameter that is not comparable to the physiologic Doppler-derived peak instantaneous pressure gradient. A better correlation is observed between Doppler and “cath” data, when the catheter-derived maximum instantaneous pressure gradient (occurring just before the peak ventricular pressure recording) and the mean systolic pressure gradient are compared with the Doppler-derived peak and mean instantaneous pressure gradients. Additionally, the phenomenon of pressure recovery can lead to overestimated gradients by Doppler as compared with catheterization. With good technique, AS velocities and gradients by CW Doppler have been shown to correlate well with simultaneous catheterization data over a broad range of pressures.

KEY TECHNICAL CONSIDERATIONS

The echocardiography imaging window that will yield the highest aortic valve velocity (most coaxial to the jet’s net vector within the anatomic orifice) is not predictable from valve morphology or jet appearance by color Doppler. Therefore, when AS is suspected, the sonographer must interrogate the aortic valve with CW Doppler from several acoustic windows, including apical, right parasternal, subcostal, and suprasternal notch. In many patients (hypertensive and older patients), the aortic valve plane can be abnormally rotated anteriorly (Fig. 21.9), which makes Doppler interrogation from the LV apical region inaccurate because of a poorly aligned interrogation angle (Ø). In addition, severe aortic calcification can strongly shadow a jet lesion’s vena contracta (zone of highest velocity, located on the aortic side of the valve) from apical views, making the right parasternal and sometimes the sternal notch windows more successful.
atrioventricular valve regurgitation (MR and TR) can be con-

From apical windows, CW spectral Doppler signals from

poor alignment, possibly “missing” severe AS.

Signal that is relatively low in velocity with reduced

signal density, because the interrogating beam may be in

A S signal that is relatively low in velocity with reduced

Signal density and a clearly defined envelope (Fig. 21.7C). An

early-peaking signal usually occurs with mild AS, and a mid-

peaking signal is a sign of severe AS [Fig. 21.8]. Continuous-

wave Doppler signals appearing much denser in the base may

indicate that the interrogating beam is cutting across the jet

lesion rather than being coaxial [Fig. 21.7D]. Therefore, one

should be highly suspicious of a late-peaking, possibly severe

AS signal that is relatively low in velocity with reduced

signal density, because the interrogating beam may be in

poor alignment, possibly “missing” severe AS.

Other Potential Pitfalls

From apical windows, CW spectral Doppler signals from

atrioventricular valve regurgitation (MR and TR) can be con-

fused with the aortic valve Doppler signal, because each is a

systolic signal, moving in roughly the same direction [below

the baseline] and occasionally at similar peak velocities. If

an MR CW Doppler signal [high velocity] is mistaken for an

aortic valve CW Doppler signal, severe AS could be errone-

ously diagnosed. Key observation: The aortic valve spectral

Doppler envelope is distinguished from atriocentric valve

regurgitation lesions (MR or TR) because of its shorter
duration. Both MR and TR occur throughout the isovolumic
contraction and isovolumic relaxation phases, in addition to
the systolic ejection phase. Therefore, MR and TR start
earlier and last longer than aortic outflow. These differences
in signal onset and duration can be easily determined using
the simultaneous ECG and time calibration display.

Irregular Rhythms

Flow velocities and gradients can vary tremendously beat to

beat with irregular rhythms as demonstrated in Figure 21.8.

With irregular rhythms, velocity and gradient determina-
tions should be based on an average of five to 10 different
samples. In sinus rhythm, measurement of ectopic beats or
postectopic beats should be excluded by recording three or
more regularly spaced sequential beats.

Pulsed Doppler

Techniques for determining flow and stroke volume within
the LVOT using pulsed Doppler are discussed in Chapter 5.
In a “best” apical view, the pulsed Doppler sample volume
is placed within the LVOT, very close to the aortic valve,
without placing it actually within the jet lesion. If the sample
volume is placed too far away from the aortic valve, the
pulsed Doppler signal will be erroneously small. A LVOT
pulsed Doppler velocity measurement (Fig. 21.7B) is neces-
sary for calculating the aortic valve area by the continuity
equation.

Valve Area by Continuity Equation

As discussed in Chapter 5, stroke volume (SV) within a
conduit having laminar flow is the product of the cross-
sectional area (CSA) and the VTI of flow measured at a
defined site. The SV just proximal to the aortic valve (LVOT)
and the stroke volume within the AoV must be equal. Flow
within a normal [unobstructed] LVOT and flow within the
aortic stenosis vena contracta (immediately distal to the ana-
tomic orifice) is laminar. Therefore, the following set of
equations can be applied to derive the simplified continuity
equation:

\[
SV = CSA \times VTI
\]

\[
SV_{LVOT} = SV_{AoV}
\]

\[
CSA_{LVOT} \times VTI_{LVOT} = CSA_{AoV} \times VTI_{AoV}
\]

\[
CSA_{AoV} = CSA_{LVOT} \times (VTI_{LVOT}/VTI_{AoV})
\]

The LVOT is assumed to be circular. The area of a circle =
\( \pi r^2 \). And \( \pi = 3.14 \). Because radius (r) is one half of the
diameter (D), the more conveniently measured LVOT diam-
eter can be inserted directly into the formula with the fol-
lowing conversion:
LVOT area = πr² = π(D/2)² = D² × 0.785

For aortic valve area, either of the following equations may be used:

\[ \text{AoV Area} = \pi(D/2)^2 \times \left( \frac{\text{VTI}_{\text{LVOT}}}{\text{VTI}_{\text{AoV}}} \right) \]

simplified:

\[ \text{AoV Area} = D^2 \times 0.785 \times \left( \frac{\text{VTI}_{\text{LVOT}}}{\text{VTI}_{\text{AoV}}} \right) \]

The ratio of LVOT and AoV velocity-time integrals (VTIs) and the ratio of LVOT and AoV peak velocities should be equal since the peak velocity at each site occurs simultaneously, and the instantaneous velocity ratios should be the same at any point during the ejection phase.

\[ \frac{\text{VTI}_{\text{LVOT}}}{\text{VTI}_{\text{AoV}}} = \frac{V_{\text{LVOT}}}{V_{\text{AoV}}} \]

Therefore, for clinical purposes, the continuity equation can be further simplified by substituting the VTIs with the peak velocity measurements:

\[ \text{AoV Area} = D^2 \times 0.785 \times \left( \frac{V_{\text{LVOT}}}{V_{\text{AoV}}} \right) \]

See Figure 21.8 for an illustration of aortic valve area calculation using the continuity equation.

**Potential Pitfall**

In all cases, the LVOT diameter measurement is squared, so that any significant LVOT diameter (D) measurement error will also be squared, producing a potentially unacceptable error in the aortic valve area calculation. Therefore, accurate LVOT imaging and diameter measurements are critical. The LVOT diameter can be difficult to measure when excessive aortic root and annular calcification is present or if parasternal images are poor. The LVOT diameter and the aortic annulus diameter are not always interchangeable (Figs. 21.7A and 21.10). The aortic annulus diameter is measured at the base or “hinge point” of the aortic valve leaflets (Fig. 21.3A). This annular measurement may be useful for prosthetic valve selection. Frequently, however, the LVOT, immediately proximal to the aortic annulus (site of pulsed Doppler sample volume placement) is impinged by an angulated basal septum, making the LVOT diameter smaller than the aortic valve annular diameter (Fig. 21.10). When the LVOT is very small or when there is turbulent or high-velocity LVOT flow proximal to the aortic annulus due to dynamic LVOT obstruction [systolic anterior motion of the mitral valve], the aortic valve area calculation by simplified continuity equation is not reliable, and the aortic valve area by continuity equation will likely be overestimated (Fig. 21.10).

**Dimensionless Velocity Index** \( \left( V_{\text{LVOT}} : V_{\text{AoV}} \right) \)

Velocity is proportional to flow across a fixed orifice. In the case of a completely normal aortic valve orifice, there should be no significant difference between the peak LVOT velocity \( V_{\text{LVOT}} \) by pulsed Doppler and the peak AoV velocity by CW Doppler \( V_{\text{AoV}} \). Under normal conditions, therefore, the peak velocities, \( V_{\text{LVOT}} : V_{\text{AoV}} \) should be close to 1.0 regardless of flow rate. On the other hand, \( V_{\text{LVOT}} : V_{\text{AoV}} \) will be <1.0 in the presence of aortic stenosis, because of increased flow velocity across the aortic valve relative to that within the LVOT. Severe obstruction is likely if \( V_{\text{LVOT}} : V_{\text{AoV}} \) ≤0.25, indicating an aortic valve area less than one-fourth the reference LVOT area. The dimensionless velocity index can be a quick, useful method for identifying potentially significant obstruction in either high- or low-output states when the area calculation by continuity equation is not possible or is in doubt (i.e., the LVOT diameter cannot be reliably measured). This index can be helpful in the assessment of possible prosthetic aortic valve obstruction (Figs. 21.11 and 21.12).

Calculations of aortic valve resistance \[^{35}\] and left ventricular stroke work loss \[^{36}\] are other indices of AS severity that may be useful but have not yet emerged as being clinically superior to the aortic valve area calculation.

**Dobutamine Stress Echocardiography**

A patient with dilated cardiomyopathy, heart failure, and severe AS based on valve area calculation by the continuity equation but with a low-velocity aortic valve gradient (peak velocity <4 m/s) can present a difficult clinical management situation. In some such individuals, the cardiomyopathy may be secondary to severe AS, with possible benefit to be had from aortic valve replacement surgery. Others, however, may have a primary cardiomyopathy with only mild or moderate AS but reduced calculated valve area (and severely reduced visible cusp separation) due to low cardiac output. Patients in the latter group would not benefit from aortic valve replacement surgery. Dobutamine stress echocardiography has been used to differentiate these two populations of heart failure patients. \(^{35}\) If the aortic valve area calculation remains in the severe range with a dobutamine-induced increase in stroke volume, the cardiomyopathy may be secondary to severe AS.
If ventricular contractile reserve is demonstrated, valve replacement surgery may be beneficial.

**Useful Values for Clinical Management**

Asymptomatic patients with isolated AS are managed medically and followed closely for signs of LV dysfunction or the emergence of symptoms. Surgery is the only effective therapy for symptomatic severe AS. With normal ventricular function and normal stroke volume, a peak aortic valve velocity of >4 m/s (64 mm Hg) is a clinically important value, because such patients likely have anatomically severe AS and are likely to reach a surgical end point within 2 years. Such patients should be monitored more frequently at this stage for the development of symptoms, at which point surgery would typically be indicated. For a given valve area, the measured aortic valve gradient is strongly influenced by heart rate, stroke volume, ejection duration, LV contractility, preload, and afterload. Therefore, peak and mean velocity and pressure gradient measurements in isolation are not helpful for clinical management. With normal LV contractility and heart rate, an aortic valve peak velocity ≤2.5 m/s almost always indicates mild AS, and an area calculation may not be necessary. When peak velocities are between 2.5–4.0 m/s, the valve area by continuity equation or by planimetry should be calculated with careful consideration given to the quality of the obtained data. As a general rule, a mean aortic valve gradient ≥50 mm Hg almost always indicates severe aortic valve stenosis, and an area calculation may not be necessary.

**FIGURE 21.11.** Dimensionless velocity index (DVI) method used to show an acceptably increased biologic aortic valve prosthesis gradient in the setting of increased stroke volume. (A) LVOT peak velocity, \( V_{LVOT} \) = 1.6 m/s. (B) Aortic valve biologic prosthesis peak velocity \( V_{AoV} \) = 3.6 m/s; DVI = 1.6 m/s ÷ 3.6 m/s = 0.44—no significant obstruction.

**FIGURE 21.12.** Dimensionless velocity index (DVI) demonstrates an obstructive gradient in a patient with a mechanical aortic valve prosthesis. (A) LVOT peak velocity, \( V_{LVOT} \) = 1.1 m/s. (B) Aortic valve mechanical prosthesis peak velocity \( V_{AoV} \) = 4.8 m/s; DVI = 1.1 m/s ÷ 4.8 m/s = 0.25—pathologic obstruction. Arrow indicates mechanical prosthesis disk opening and closure artifacts.
TABLE 21.1A. Severity of aortic stenosis (AS) by aortic valve (AoV) area and valve area index

<table>
<thead>
<tr>
<th>AS severity</th>
<th>AoV area (cm²)</th>
<th>AoV index (cm²/BSA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>&gt; 2.0</td>
<td>&gt; 1.0</td>
</tr>
<tr>
<td>Mild</td>
<td>1.6–2.0</td>
<td>0.90–1.10</td>
</tr>
<tr>
<td>Mild-to-moderate</td>
<td>1.3–1.6</td>
<td>0.75–0.90</td>
</tr>
<tr>
<td>Moderate</td>
<td>1.0–1.3</td>
<td>0.6–0.75</td>
</tr>
<tr>
<td>Moderate-to-severe</td>
<td>0.7–1.0</td>
<td>0.4–0.60</td>
</tr>
<tr>
<td>Severe</td>
<td>&lt; 0.70</td>
<td>&lt; 0.40</td>
</tr>
</tbody>
</table>

Note: Normal aortic valve area = 3.0–4.0. BSA, body surface area.

Aortic Valve Area

Valve area remains the best clinical indicator of AS severity. With a valve area of >1.0 cm², symptoms are probably not due to AS alone. At a valve area of approximately 1.0 cm², flow velocities and gradients increase significantly with physiologic flow. Valve areas in the range of 0.8 to 1.0 cm² represent a “gray zone” in which symptoms may indeed be due to AS, yet other clinical factors (concomitant AR, MR, coronary artery disease, patient body size, high output state, etc.) should be considered. Typical symptoms are almost always due to AS when the valve area is < 0.8 cm² [severe], in which case surgery is indicated. An aortic valve area index [valve area/body surface area [BSA]] may be of clinical utility in dealing with large or small patients. Table 21.1A, from the University of Chicago, and Table 21.1B, adapted from American College of Cardiology/American Heart Association [AHA] practice guidelines, each show slightly different grading systems. The ACC/AHA practice guidelines’ recommendations are less precise, and this may be more applicable across different laboratories, given the underlying margin for potential measurement error and varying patient sizes.

Chronic Aortic Regurgitation

Two-Dimensional and M-Mode Exam and Differential Diagnosis

The 2D exam is useful for determining the etiology of AR to the extent that cusp size, number, coaptation characteristics, and aortic root size can be evaluated. Native valve AR may be secondary to abnormalities of the aortic leaflets, the aortic root, or both. Understanding the mechanism for AR can be useful for surgical planning. Disease may be acquired or congenital. Any of the above-mentioned common causes of aortic stenosis [calcific degeneration, bicuspid valve [Fig. 21.13], rheumatic [Fig. 21.14]] can also be associated with either mixed disease or a predominant aortic regurgitation lesion due to cusp prolapse, malcoaptation of the cusp margins, or leaflet destruction from superimposed bacterial endocarditis [Fig. 21.15]. Chronic AR can be associated with aortic root enlargement, particularly in the elderly, but also in younger individuals with Marfan syndrome or anuulooaortic ectasia [Fig. 21.16] or in association with congenitally bicuspid aortic valves. Congenitally bicuspid aortic valves may exhibit obvious prolapse in the parasternal long axis view [reverse diastolic doming], which may be obvious [Fig. 21.13A]. Frequently, however, the degree of cusp prolapse is subtle and involves only one cusp, producing a highly eccentric AR jet, the severity of which can be difficult to assess and easily underestimated by color Doppler. Subtle congenital prolapse of an otherwise normal trileaflet valve also occasionally causes significant AR, as can focal fenestrations along the leaflet margins [tattered flag appearance on surgical inspection], which may be difficult to discern echocardiographically. Associated valve conditions include myxomatous degeneration, which can infrequently cause marked prolapse and regurgitation of the otherwise normal trileaflet aortic valve, although this condition is usually found in association with the more commonly affected mitral and tricuspid valves (see below). Characteristic 2D and M-mode rheumatic mitral valve abnormalities [MS and MR] strongly suggest a rheumatic etiology of associated AR. Other congenital abnormalities include unicuspid aortic valves, which may have significant AR, AS, or both, as is the case with bicuspid valves. Rare quadricuspid aortic valves are more often regurgitant than stenotic and frequently become “surgical” by the fourth or fifth decade. In congenital “fixed” subaortic membrane or tunnel stenosis, the subvalvular jet lesion can traumatize the aortic cusps, leading to aortic insufficiency. Supravalvar or subarterial ventricular septal defects [adjacent to the aortic annulus] can cause severe AR due to inadequate cusp support.

ECHOCARDIOGRAPHIC ASSESSMENT OF VALVULAR HEART DISEASE

A

B

C

D

E

F

G
Aortic Root Size

Aortic regurgitation is associated with the presence of aortic root enlargement, particularly if there is expansion (effacement) of the sinotubular junction (Fig. 21.16). The aortic root is measured using the 2D image at the maximum sinus of Valsalva diameter, perpendicular to the aortic long axis (Fig. 21.5A). Normal values for aortic root size, based on patient age ranges, have been published [American Society of Echocardiography (ASE) guidelines]. An aortic root diameter indexed for body surface area of $>2.1 \text{mm/m}^2$ is highly specific for aortic root enlargement.

M-Mode Measurements

M-mode measurements are pertinent for AR assessment when used for LV diameter assessment. Diastolic fluttering of the mitral valve’s anterior leaflet can be a sign of an eccentric AR jet striking this leaflet (Fig. 21.17).

Left Ventricular Size and Systolic Function Assessment

Increased left ventricular dimensions are a hallmark of chronic significant AR. Recognizing and following abnormal LV remodeling by echocardiography is important for clinical decision making. Parasternal long axis measurements of LV end-diastolic and end-systolic diameters (LVEDD and LVESD) can be highly reproducible and reflective of overall LV size and contractility in the absence of concomitant regional wall motion abnormalities from coronary artery disease or other cardiomyopathies.

In the past, echocardiography-derived LV volume and ejection fraction measurements have been thought to be difficult to reproduce, if not unreliable, because of variable imaging quality and technique. Because echocardiography technology has improved in recent years (improved 2D imaging and use of left-sided contrast agents), quantitative measures of LV size [volume and mass] and function [ejection fraction] are more reproducible and reliable. This improvement in technique has allowed for more accurate assessment of LV size and function, which is critical for the management of AR patients.
FIGURE 21.15. TEE: acute, severe AR. This 24-year-old man with a bicuspid aortic valve had mild AR by echocardiography 10 months earlier. (A) Linear whip-like vegetation (arrow) attached to a prolapsing cusp with early aortic root abscess formation (arrowhead); left atrium [LA], left ventricle [LV], aorta [Ao]. (B) Severe AR completely fills the LVOT by color Doppler imaging. (C) In a different similar case, color M-mode helps to display the diastolic timing of the turbulent AR jet during tachycardia [brackets]. (D) Pulsed Doppler, descending thoracic aorta shows sinusoidal appearance of forward systolic flow (arrow) and pan-diastolic reverse flow (double arrow). (E) Prominent diastolic mitral regurgitation [MR] by CW Doppler [upward arrow], a sign of acute severe AR, associated with premature mitral valve [MV] closure [downward arrow]. (F) TEE gastric window: AR deceleration slope [line] by CW Doppler quickly reaches baseline [short pressure half-time [PHT], <200ms] indicating almost equal aortic and ventricular diastolic pressure [severe acute AR].
fraction) have become increasingly reliable. Accordingly, recommendations for cardiac chamber quantification by echocardiography were recently updated.47 Although several methods for LV volume assessment exist47 and are applicable in varying situations, the biplane method of disks (modified Simpson’s rule48) is recommended when apical foreshortening can be eliminated and the endocardial surfaces are visible. The method uses summation of elliptical disk volumes derived from the apical four- and two-chamber views (Fig. 21.13F,G). When the LV cavity is relatively symmetrical and with uniform contractility, the method can also be applied to a single plane if either one of the two views is technically suboptimal.

TABLE 21.2. Left ventricular volume and volume index

<table>
<thead>
<tr>
<th></th>
<th>Normal range</th>
<th>Mildly dilated</th>
<th>Moderately dilated</th>
<th>Severely dilated</th>
</tr>
</thead>
<tbody>
<tr>
<td>A: Women</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diastolic</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDV [mL]</td>
<td>56–104</td>
<td>105–117</td>
<td>118–130</td>
<td>≥131</td>
</tr>
<tr>
<td>Indexed</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDV/BSA [mL/m²]</td>
<td>35–75</td>
<td>76–86</td>
<td>87–96</td>
<td>≥97</td>
</tr>
<tr>
<td>Systolic</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDV [mL]</td>
<td>19–49</td>
<td>50–59</td>
<td>60–69</td>
<td>≥70</td>
</tr>
<tr>
<td>Indexed</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDV/BSA [mL/m²]</td>
<td>12–30</td>
<td>31–36</td>
<td>37–42</td>
<td>≥43</td>
</tr>
<tr>
<td>B: Men</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diastolic</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDV [mL]</td>
<td>67–155</td>
<td>156–178</td>
<td>179–201</td>
<td>≥201</td>
</tr>
<tr>
<td>Indexed</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDV/BSA [mL/m²]</td>
<td>35–75</td>
<td>76–86</td>
<td>87–96</td>
<td>≥97</td>
</tr>
<tr>
<td>Systolic</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDV [mL]</td>
<td>22–58</td>
<td>59–70</td>
<td>71–82</td>
<td>≥83</td>
</tr>
<tr>
<td>Indexed</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDV/BSA [mL/m²]</td>
<td>12–30</td>
<td>31–36</td>
<td>37–42</td>
<td>≥43</td>
</tr>
</tbody>
</table>

Note: The LVEDV and LVESV indexes (mL/m²) are the most validated measures.

BSA, body surface area; LVEDV, left ventricular end diastolic volume; LVESV, left ventricular end systolic volume (not shown).

LV Ejection Fraction = (LVEDV − LVESV)/LVEDV

Reference limits for LV volumes are shown in Table 21.2, and reference limits for LV ejection fractions are shown in Table 21.3.

Doppler Evaluation for Aortic Regurgitation Severity Assessment

CONTINUOUS-WAVE DOPPLER

In the presence of AR, CW Doppler interrogation of the LVOT reveals a characteristic spectral Doppler velocity envelope produced by diastolic decay of the pressure gradient between the aortic root and aorta (Fig. 21.18). In some techni-
echocardiographic assessment of valvular heart disease

...difficult exams, detection of the characteristic appearance of AR by spectral Doppler may be the first or only clue to its presence. When the signal is well aligned with the interrogating beam, the density of the spectral Doppler signal can be used as a rough qualitative indicator of severity. Mild degrees of AR produce a faint signal, and moderate or severe jets produce a dense signal. This is because signal density correlates with the number of regurgitant red blood cells within the path of the interrogating beam. In many cases, the observer is unable to differentiate moderate from severe AR using jet density. Nonetheless, a dense jet by CW warrants further investigation.

**Pressure Half-Time**

The AR velocity by CW Doppler decays in a linear fashion according to the rate of pressure equilibration between the aortic root and left ventricle. The rate of pressure decay can be described by a deceleration time (DT), which is the time from initial peak velocity to zero velocity when the velocity slope is extended to the baseline. Pressure half-time (PHT) is the time for the AR velocity to decline to half of its original peak value. Both the DT and PHT are automatically calculated when the operator electronically defines the AR velocity deceleration slope (Fig. 21.18). When AR is severe, the aorta-to-LV pressure declines rapidly, producing a short PHT (Figs. 21.15F and 21.18B). When AR is mild, or mild-to-moderate (Fig. 21.18A), the PHT is long because the regurgitant volume is not sufficient to significantly affect either the aortic or ventricular diastolic pressures. A PHT <200 ms almost always indicates severe AR, and a PHT of >500 ms almost always indicates mild AR. The PHT for moderate AR usually lies within a wide range (200–500 ms). Mild and severe AR can also lie within this gray zone, depending on ventricular loading conditions.

**PHT Technical Notes and Pitfalls**

The PHT measurements are valid only when the CW interrogating beam is coaxial to flow and a clear linear spectral velocity envelope “edge” is present. Quality CW signals and acceptable PHT measurements are simply not possible with many eccentric or poorly aligned AR jets. The PHT can be relatively long (significantly >200 ms) with compensated chronic severe AR. This is because adaptive remodeling allows the LV to accept a large regurgitant volume without a significant rise in LV diastolic pressure. Another potential pitfall is that for a given degree of AR, the PHT will appear shortened in a patient with high LV filling pressures due to an underlying cardiomyopathy. In this situation, the LV diastolic pressure rises more rapidly for a given regurgitant volume, quickly reducing the aorta-to-LV diastolic pressure difference and shortening the PHT. Systemic vasodilator therapy can also shorten the PHT by decreasing the aortic pressure (low afterload).

**Pulsed Doppler-Derived Regurgitant Volume and Regurgitant Fraction**

Left ventricular outflow tract flow as measured by pulsed Doppler is increased in AR. This is because LVOT flow includes both the useful forward stroke volume and the “wasted” regurgitant volume. When a very high LVOT VTI measurement is encountered (e.g., LVOT VTI >30 cm), severe AR should be suspected (see Fig. 21.13D). The regurgitant volume (RV) can be obtained by subtracting a reference forward stroke volume (SV from mitral annulus or RV outflow tract) when there is no important concomitant reference valve regurgitation.

\[
RV_{AR} = SV_{LVOT} - SV_{ref\ valve}
\]

Details for calculating LVOT, mitral annulus, and RVOT stroke volume are found in Chapter 5. The regurgitant fraction (RF) is the regurgitant volume divided by the total LVOT stroke volume \(\times 100\).

\[
RF = \frac{RV}{SV_{LVOT}} \times 100
\]
The regurgitant fraction is a useful parameter when the total stroke volume is reduced because of cardiomyopathy. Reference values appear in Table 21.4. As an additional check (in the absence of significant MR), the total LV stroke volume can also be calculated using the modified Simpson’s rule.31 This type of quantitative analysis can be time-consuming, but it is useful in selected cases.

**Effective Regurgitant Orifice Area (EROA) Using Spectral Doppler**

After calculating the aortic regurgitant volume (see above), the EROA [a useful severity indicator] (Table 21.4) can then be calculated. Forward systolic stroke volumes or diastolic RV is the product of the cross-sectional area of the flow [EROA] and the VTI measured at the same site [by CW Doppler of the AR jet in this case].

\[
\text{EROA} = \frac{\text{RV}_{AR} \times \text{VTI}_{AR}}{\text{RV}}
\]

This method for EROA calculation is reliable only in regular rhythm and when there is a clear, well-aligned CW Doppler signal.

**Aortic Diastolic Flow Reversal by Pulsed Doppler**

As discussed in Chapter 5, the routine pulsed Doppler exam includes interrogation of the proximal descending thoracic aorta from the sternal notch window. In normal individuals [no AR], a normal low-velocity (<40 cm/s) early diastolic flow can be seen. In chronic, compensated severe AR, the early diastolic flow velocity peak is higher (usually >40 cm/s), and flow reversal becomes pan-diastolic (Fig. 21.13G). Unfortunately, the sternal notch window is not available in all subjects. Pan-diastolic flow reversal in the abdominal aorta, if present, is a more specific sign of severe AR. Pan-diastolic flow reversal is often more evident in acute severe AR [below], but it may be less evident in patients with poor aortic compliance.

**Aortic Regurgitation Severity by Color Doppler Jet Size**

Trace and mild AR are frequently detected by color Doppler in many otherwise normal, older individuals.30 Careful imaging of the entire proximal jet lesion by color Doppler, along with a corroborating CW Doppler signal typical of mild AR, may be all that is required to make the diagnosis in many such cases. As previously mentioned, there are many potential pitfalls for using color Doppler jet size as an indicator for regurgitation severity. With good imaging practices, however, a very small AR jet by color Doppler likely represents mild AR, and a very large AR jet likely represents severe AR [Table 21.4]. For jets that are neither clearly small nor clearly large, further evaluation is needed. When assessing color Doppler jet size, length and area measurements within the left ventricle can be misleading and unreliable indicators of severity. The height of jet within the LVOT, just below the aortic cusps and the more proximal vena contracta [VC] size57 [Fig. 21.19], can be a useful and readily measurable parameter (Table 21.4). The AR vena contracta and LVOT jet dimensions should not be based on one “freeze frame” but on the best assessment of the sustained jet during the diastolic period using a zoomed parasternal long axis image and a small color sector for high sample rate and improved color Doppler spatial resolution. Central AR jets are easier to measure [Fig. 21.19]. Eccentric AR jets can be difficult to measure. The area of the AR jet in short axis view at or slightly below the cusp coaptation zone can be very useful for assessing the 3D characteristics of the jet origin and the AR mechanism [e.g., central cusp malcoaptation, valve cusp perforation, valve extension along a cusp commissural zone, valve prolapse]. Cross-sectional area measurements can be challenging to interpret in rapidly expanding or eccentric jets and because of aortic root motion through the imaging plane. Cross-sectional area as a percentage of aortic root size may be difficult to interpret if the aortic root size is abnormal.

**Proximal Isovelocity Surface Area for Effective Regurgitant Orifice Area and Regurgitant Volume**

When a sizable proximal flow convergence zone is visualized [Figs. 21.16 and 21.20], severe AR should be suspected, and
this finding alone should prompt a more careful and detailed overall assessment using the other methods discussed. Although often technically challenging, because of calcific shadowing within the aortic root or eccentric jet, calculating the EROA and RV using PISA has been validated, using the formulas presented in the introductory part of this chapter.

\[
\text{EROA}_{\text{AR}} = 6.28r^2 \times \frac{V_a}{V_{pk,\text{AR}}} \\
\text{RV}_{\text{AR}} = \text{EROA} \times \text{VTI}_{\text{AR}}
\]

In summary, assessment of the severity of chronic aortic regurgitation can be one of the most challenging tasks for the echocardiographer. The final diagnosis may be the result of an integrated analysis of several supportive parameters without reliance upon only one or two severity indicators. When there are indirect signs of possible severe AR, but the aortic leaflets or the proximal jet cannot be clearly visualized by TTE, adjunctive TEE can play a valuable role (Figs. 21.14, 21.15, and 21.19). TEE, cardiac catheterization with aortic root injection, and, increasingly, cardiac magnetic resonance imaging (MRI) can be particularly useful in selected cases.

**Acute Severe Aortic Regurgitation**

Acute severe aortic regurgitation can be due to aortic valve endocarditis [Fig. 21.15], acute aortic dissection with propagation into an aortic cusp, an aortic cusp tear from a deceleration injury [uncommon], or partial sewing ring dehiscence or leaflet malfunction in a prosthetic aortic valve. Patients with true acute severe AR represent surgical emergencies and usually present with a relatively narrow pulse pressure and cardiogenic shock (pulmonary edema, tachycardia, low-output state). In hemodynamically unstable or intubated patients, surface echocardiography is frequently suboptimal for a variety of reasons, including tachycardia and signal attenuation. In the presence of cardiogenic shock, a TEE should be performed as soon as possible after endotracheal intubation and establishment of mechanical ventilation. Transesophageal echocardiography is used to firmly establish the diagnosis, rule out important associated valve disease (particularly with endocarditis), determine ventricular contractility, and assist with surgical planning. Patients with baseline poor LV compliance from severe AS or LV hypertrophy or other causes will demonstrate signs of hemodynamic collapse more quickly for a given acute AR volume load. This is because of a steeper LVEDV pressure-volume relationship curve.

**Two-Dimensional and M-Mode Signs**

The aortic cusps and aortic root should be imaged first in long axis, short axis, and in oblique imaging planes for signs of vegetation, leaflet perforation or prolapse, aortic root abscess, aortic root dissection, prosthetic valve rocking motion, thrombosis, or prosthetic valve biologic cusp or mechanical disk opening or closure abnormalities in a pathology-directed exam. Expedient, systematic evaluation of the other valves should then be performed. In acute severe AI, the thoracic aorta may exhibit exaggerated pulsatility, though this clue may not be present in older patients with stiffer aortas. Evaluation of LV size and function and the pericardium is important. In aortic root dissection, pericardial effusion or myocardial ischemia may also be present if dissection involves a coronary ostium (more commonly the right). The LV size may not be dilated as in chronic AR. Left ventricle contractility may be hyperdynamic from a hyperadrenergic state and increased preload, unless there is also a complicating preexisting cardiomyopathy.

*Diastolic (premature) closure of the mitral valve* is a sign of steep increase in the left ventricular end diastolic pressure that exceeds the left atrial pressure. Occasionally, end diastolic (premature) opening of the aortic leaflets can be demonstrated by M-mode due to aortic and left ventricular pressure equalization. As with chronic AR, the mitral valve M-mode image may show reduced valve opening and high-frequency diastolic fluttering of the mitral anterior leaflet from eccentric AR impact. Because of a fast sample rate, color M-mode may be particularly useful in acute severe AR with tachycardia [Fig. 21.15C], in which case routine color Doppler of the LVOT [slower sample rate] may appear confusing because of turbulent, high-volume systolic and diastolic
flow. Color M-mode can quickly confirm the diastolic timing of the AR lesion and show an approximate jet height just below the aortic valve.

Doppler Signs

With severe AR, color Doppler may almost fill the LVOT [Fig. 21.15B]. The color Doppler flow convergence zone and the associated vena contracta should be displayed simultaneously, if possible, and correlated with leaflet abnormalities. The aortic valve PHT by CW Doppler obtained in a gastric view is short because of rapid aorta-to-left ventricular diastolic pressure equalization [Fig. 21.15D]. Diastolic MR, in the absence of prolonged PR interval, is another supportive sign of acute severe AR, and it can be demonstrated by CW Doppler60 [Fig. 21.15E]. Pan-diastolic flow reversal in the descending thoracic aorta is usually readily apparent by either surface echo or TEE [Fig. 21.15F].

Quantitative Measures

All of the above-mentioned semiquantitative methods can confirm the diagnosis of acute severe AR, particularly when the anatomic regurgitant orifice is clearly visualized and the mechanism has been discerned. Calculations of the aortic regurgitant volume and regurgitant fraction are often not feasible or necessary for the diagnosis of acute severe AR.

Acute AR Superimposed on Chronic Aortic Regurgitation

On some occasions, patients present with many of the features of acute severe AR [tachycardia, dyspnea, fever], though cardiogenic shock is not present on initial evaluation. In such cases, acute severe AR may have complicated underlying chronic AR [likely moderate] for which some degree of underlying physiologic adaptation and ventricular remodeling had previously occurred. A relatively wide pulse pressure may be present with some degree of LV dilatation. Preexisting valve abnormalities may be linked to the mechanism of acute severe AR (e.g., bicuspid aortic valve as a nidus for endocarditis or acute aortic dissection complicating a previously dilated aortic root with some degree of baseline AR).

Mitral Valve Disease

Mitral Stenosis

Two-Dimensional and M-Mode Examination and Differential Diagnosis

Mitral valve stenosis can be acquired or congenital. In adult echocardiography laboratories, the most common causes of acquired mitral stenosis [MS] are rheumatic and degenerative calcification of the native mitral valve. Mechanical prosthetic mitral valves can become obstructed [stenotic] because of thrombus or pannus formation. A biologic mitral prosthesis can undergo calcific degeneration with stenosis due to reduced cusp mobility. Echocardiographic findings of MS from right atrial tumor and congenital stenosis due to rheumatic mitral valve disease are usually present.

Rheumatic Mitral Stenosis

Stenosis is usually the predominant hemodynamic lesion in rheumatic mitral valve disease. However, varying degrees of mitral regurgitation are frequently present, with MR sometimes being the predominant lesion. Even in the early disease stage, when stenosis may be mild, several echocardiographic features of rheumatic mitral valve disease are usually present.

Rheumatic Leafl et Deformity. Classic rheumatic changes include leafl et thickening that is more prominent in the leafl et leading edges with relatively preserved mobility of the leafl et bases. Relatively preserved basal leafl et mobility and a reduced orific e area produce a characteristic diastolic “doming” appearance of the valve and a diastolic “hockey-stick deformity” of the anterior mitral leafl et in the parasternal long axis view [Fig. 21.20A]. Leafl et leading-edge inflammation causes commissural fusion, which is the primary mechanism for stenosis. Commisural fusion and thickened leafl et margins produce an ovoid or circular “fish-mouth” orific e, appreciated in the parasternal short axis view [Fig. 21.21]. Degenerative calcification can occur within the leafl et. In advanced disease, the entire leafl et can eventually become severely thickened and calcified with little apparent residual mobility.
**Submitral Apparatus.** Rheumatic inflammation extends into the chordae tendineae and often into the papillary muscles. A variable degree of chordal thickening, fusion, and shortening occurs. This deformity can be mild, moderate, or so extreme that the primary region of stenosis is actually within the fused chords and not at the leaflet level. Chordal shortening in advanced disease can be so extreme that discrete chords cannot be differentiated from the brightly echoic papillary muscles that seemingly fuse directly into the leaflets. Even in mild disease, posterior leaflet tethering and immobility relative to the anterior leaflet is a specific feature of rheumatic mitral valve disease. On M-mode examination, there is a characteristic and highly specific posterior leaflet “anterior diastolic motion” (Fig. 21.20B) because the tethered posterior leaflet must “track” the anterior leaflet instead of exhibiting normal diastolic leaflet separation (see Chapter 5).

**Rheumatic Left Atrium.** The left atrium is often severely dilated in rheumatic MS. Swirling left atrial spontaneous echocardiography contrast (“smoke”) and left atrial or left atrial appendage thrombus are common in rheumatic MS. Left atrial thrombus is usually apparent only by TEE. In some cases, calcification of the left atrial wall, probably related to pancarditis, results in even suboptimal TEE images in rheumatic heart disease.

**Percutaneous Balloon Mitral Valvuloplasty Assessment by Echocardiography.** In properly selected symptomatic rheumatic MS patients, a durable result, comparable to surgical mitral commissurotomy, can be achieved with percutaneous balloon mitral valvuloplasty (PBMV). Morphologic features, as evaluated by surface echocardiography, have been divided into four categories and rated on a scale of 1 to 4 for each feature (Table 21.5). It has been shown that the best PBMV results are achieved in young patients having a combined mitral stenosis or ≤8, mild-to-moderate MR, and no previous surgery. Other patients with less favorable valve anatomy (score >8) may also benefit from the procedure, but they may be more likely to have significant residual gradients or mitral regurgitation. The palliative mechanism of PBMV is separation of the fused mitral commissures. Focal calcification (not included in the above-mentioned scoring scheme) may also reduce the success rate and predispose to MR development. Its presence is usually discernible on the mitral valve (MV) short axis view. Percutaneous balloon mitral valvuloplasty carries a risk of stroke. A TEE is performed in advance of PBMV to ensure that LA or left atrial appendage (LAA) thrombus is not present. At some centers, TEE is used to guide the procedure.

**Mitral Annular Calcification**

This degenerative change is very commonly seen in the elderly and in chronic renal failure or hemodialysis patients and possibly in association with coronary artery disease [Figs. 21.3D and 21.22]. Some degree of associated MR is also

![FIGURE 21.22. Severe mitral annular calcification.](image)

**TABLE 21.5. Percutaneous balloon mitral valvuloplasty score**

<table>
<thead>
<tr>
<th>Feature</th>
<th>Grade 1</th>
<th>Grade 2</th>
<th>Grade 3</th>
<th>Grade 4</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mobility</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal mobility</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mob. limited to leaflet base</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Relatively immobile leaflets throughout</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Leaflet thickening</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Leaflets near normal</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mid-leaflets normal, thickened margins</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thickening throughout leaflets</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Marked thickening throughout leaflets</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Subvalvular thickening</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Minimal thickening just below leaflets</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thickening up to one third of chordal length</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thickening extending to the distal one third of chords</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Extensive thickening and shortening of the papillary muscles</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Calcification</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single area of increased echo brightness</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Scattered areas of brightness confined to leaflet margins</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brightness extending into the midportion of the leaflets</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Extensive brightness throughout much of the leaflet tissue</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Based on surface echocardiography examination.
Mitral annular calcification (MAC) is usually differentiated from rheumatic mitral disease by echocardiography because leaflet thickening, calcification, and immobility begin in the leaflet bases (Fig. 21.22), with relatively preserved mobility in the leaflet leading edges. With severe posterior annular calcification, the posterior leaflet can appear almost completely obscured on surface echocardiography. Relatively thin chordae tendineae, absence of mitral leaflet diastolic doming, or hockey-stick deformity (Fig. 21.22) can help to distinguish mitral calcific degeneration from rheumatic disease. Even in relatively advanced MAC, the hemodynamic severity of the associated mitral stenosis generally remains in the mild or mild-to-moderate range. Severe MAC-related mitral stenosis requiring surgery does occur, in which case the risk for periprosthetic MR is increased. Because commissural fusion is not the mechanism for stenosis in MAC, percutaneous balloon valvuloplasty is not a palliative option.

### Echocardiographic Parameters for Mitral Stenosis Severity

#### Valve Area by Planimetry in Rheumatic Mitral Stenosis

With good technique and adequate imaging windows (parasternal short axis view), direct mitral orifice planimetry can be the most accurate method for rheumatic mitral valve area assessment. Using fine adjustment of the imaging plane, the sonographer should make a concerted effort to image the smallest, most distal anatomic orifice. Because the rheumatic valve can be somewhat funnel-shaped, cutting through the body of the leaflets and not the leaflet margins is a common mistake that produces an erroneously large valve area. Ideally, three reproducible measurements are obtained. Newer 3D echocardiography techniques may be used to facilitate anatomic orifice location and planimetry. It should be noted that the planimetered MV area (anatomic orifice) may be slightly larger than the Doppler-derived mitral valve area (MVA) in the same patient since the latter is a measure of the physiologic orifice (site of vena contracta).

### Pitfalls

Planimetry, even by 3D imaging, may not be possible when there is shadowing artifact from severe calcification or image dropout from poor imaging windows. In some cases, the smallest apparent valve orifice area is disproportionately large relative to the transvalvular gradient by Doppler. This may be due to stenosis that occurs primarily in the subvalvular region because of chordal fusion. In such cases, other methods [below] must be applied. Note: Mitral valve sizes that cause clinical symptoms (rest or exercise pulmonary congestion) can vary markedly among patients depending on body size, cardiac output, and heart rate.

### Doppler Evaluation

#### Continuous-Wave Doppler: Peak and Mean Gradients

Transmitral inflow velocity is measured from apical views using CW Doppler. Color Doppler guidance can help determine the best beam alignment for “directed” jets. Mitral stenosis is suspected by visual inspection of the spectral Doppler signal whenever the velocity remains substantially above the baseline throughout diastole, because at normal heart rates, the transmitral pressure gradient normally becomes zero, or very close to zero during mid-diastole. The valve peak and mean velocities and gradients are automatically derived by tracing the spectral signal’s outer edge (Fig. 21.23A). Table 21.7 shows mean resting gradients that typically accompany mild-, moderate-, and severe-range MS. The mean MV gradient is strongly influenced by heart rate. Tachycardia shortens the diastolic time available for transmitral pressure gradient

### Table 21.6. Mitral stenosis by valve area (cm²)

<table>
<thead>
<tr>
<th>Mitral Valve Area</th>
<th>Very Mild</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;2.0</td>
<td>&gt;1.5–2.0</td>
<td>&gt;1.0–1.5*</td>
<td>≤1.0</td>
<td></td>
</tr>
</tbody>
</table>

* Clinically severe MS can occur in this range because of variability of patient size, heart rate, and stroke volume at rest and with exercise.

### Notes

- Mitral valve sizes that cause clinical symptoms (rest or exercise pulmonary congestion) can vary markedly among patients depending on body size, cardiac output, and heart rate.

### Figure 21.23

Mitral stenosis assessment by CW Doppler, atrial fibrillation. Mean gradient varies (A), consistent PHT. Despite different RR intervals (B), the PHT measurement [green line] is the same in both cycles. See image for mitral valve area (MVA) calculation by PHT method.
21.23B) or concomitant MR. The pitfalls are as follows: Because the PHT is relatively independent of flow, this and PHT) by CW Doppler is easily obtained in many cases. The advantages because PHT

The DT. The advantages
due to patients with normal-range heart rates and stroke volumes. In irregular rhythm (atrial fibrillation) five to ten cycles should be averaged for gradient assessment. The peak gradient may be increased from high LV filling pressures not related to MS or from concomitant MR.

**Mitral Valve Area by Pressure Half-Time Method**

During diastole, there is a relatively linear decay of the LA-to-LV pressure gradient that is inversely proportional to the MV area. The time from MV peak velocity to zero velocity (extending the straight spectral Doppler velocity slope to baseline) is known as the deceleration time (DT). The DT can be visually estimated using the spectral Doppler image time scale. Mitral stenosis is also described by the PHT—time from initial peak pressure difference to half or the peak pressure difference. The PHT cannot be determined by visual inspection of the Doppler signal, but it is automatically calculated by echocardiography systems when the linear deceleration slope is electronically defined (Fig. 21.23B). One of the earliest and most clinically useful applications for the Doppler exam was validation of the following empirically derived formula:

\[
MVA = 220 / \text{PHT}
\]

Because PHT = (0.29) DT, MVA is also easily calculated using the DT. The advantages are as follows: The mitral inflow DT (and PHT) by CW Doppler is easily obtained in many cases. Because the PHT is relatively independent of flow, this method is useful in patients with atrial fibrillation (Fig. 21.23B) or concomitant MR. The pitfalls are as follows: With rapid heart rate (atrial fibrillation) or a large terminal A-wave, the deceleration slope may be too short to accurately define (Fig. 21.24C). The PHT method should be used with caution or not at all in patients with potentially significant left ventricular diastolic pressure elevation (e.g., advanced cardiomyopathy or significant concomitant AR). In such cases, progressive pressure rise in the receiving chamber hastens the decay of the LA-LV pressure gradient, and the calculated MV area will be erroneously large. The PHT may appear long secondary to impaired LV relaxation (LV diastolic dysfunction) in the absence of significant MS. In addition, the PHT method is not valid in the first day or two after percutaneous balloon valvuloplasty.

**Valve Area by Continuity Equation**

See aortic stenosis [above] for derivation of the continuity equation, which can also be applied to the mitral valve for area calculation (MVA). Stroke volume is the product of CSA and VTI. Flow across the mitral valve must equal flow across a reference valve or conduit, such as the LVOT:

\[
\text{CSA}_{MV} \times \text{VTI}_{MV} = \text{CSA}_{LVOT} \times \text{VTI}_{LVOT}
\]

\[
\text{MVA} = \left[ \frac{D^2 \times 0.785}{180} \right] \times \frac{\text{VTI}_{LVOT}}{\text{VTI}_{MV}}
\]

VTI_{MV} is derived by electronically outlining the mitral diastolic CW spectral Doppler signal. All variables for the continuity equation are obtained during a standard Doppler examination. Therefore, the method is easily applied. Reference flow in the right ventricular outflow tract (RVOT) can also be used provided that the RVOT diameter can be accurately measured. The continuity equation method is useful when there is chordal level stenosis, when valve planimetry is not possible, or if the PHT measurement is in doubt. The pitfalls of this method are as follows: It cannot be used when there is also significant MR because this will increase VTI_{MV} relative to the reference valve VTI, resulting in an erroneously small MV area calculation. The method cannot be used with significant reference valve regurgitation (i.e., AR or pulmonary regurgitation [PR]) because the increased VTI_{ref} relative to VTI_{MV} will result in an erroneously large MVA calculation. The method may be unreliable in irregular rhythms because the stroke volume will vary markedly from beat to beat, and simultaneous mitral and reference valve flows cannot be compared. Averaging multiple cycles (5 to 10) can be attempted or selecting cycles with a similar R-R interval, although this is somewhat more time-consuming.

**Proximal Isovelocity Surface Area Method**

A prominent PFC zone is often apparent on the atrial surface of the valve, particularly by TEE. The PISA formula for deriving valvular EROA was explained in the introduction (see above). The same formula can be used to calculate the MVA in MS using forward flow. Because the rheumatic MV inflow surface is frequently funnel-shaped and not flat, the PISA may not be hemispheric. Therefore, a correction factor (α/180) must be applied. Alpha (α) is the angle formed by lines running roughly parallel to the distal valve leaflets (Fig. 21.25).

\[
\text{MVA} = \frac{\alpha}{180} \times 6.28^2 \times V_d / V_{pk}
\]

The MV area by PISA is useful when other methods fail, and it is a reasonably accurate method in atrial fibrillation. The pitfalls of this method are as follows: The PFC zone may be difficult to visualize by surface echo. In addition, an “α measurement” tool is not provided on most analysis systems, though a reasonable estimate can sometimes be made by visual inspection.

**Tricuspid Regurgitation Velocity**

Pulmonary hypertension either at rest (in the absence of other causes) or during exercise (Fig. 21.24E), is a hallmark of clinically significant MS. An estimate of the systolic pulmonary artery pressure (SPAP) using the TR velocity by CW Doppler (see Chapter 5) is an important element of the exam.

**Exercise Doppler Echocardiography**

Exertional dyspnea, the cardinal clinical symptom of MS, can also be caused by a number of conditions not related to

### Table 21.7. Mitral stenosis by mean gradient (mm Hg)

<table>
<thead>
<tr>
<th>Severity</th>
<th>Mean gradient (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>&lt;5</td>
</tr>
<tr>
<td>Moderate</td>
<td>5–10</td>
</tr>
<tr>
<td>Severe</td>
<td>&gt;10</td>
</tr>
</tbody>
</table>

Note: Rough guidelines for “resting” Doppler assessment of MS. Gradients are strongly affected by heart rate and cardiac output.
FIGURE 21.24. Bicycle stress echocardiography in a large male with chronic obstructive pulmonary disease (COPD) and NY Heart Assoc. Functional Class III (NYHAFC III) exertional dyspnea. At rest (A–C) moderate mitral stenosis by valve area from PHT method and mean gradient (see Tables 21.6 and 21.7). Moderate pulmonary hypertension at rest, possibly from severe COPD. At low workload (25 W, bicycle ergometry, D,E), increased mean MV gradient = 15.5 mm Hg and peak systolic positive airway pressure (PAP) = 90 mm Hg (HR = 90 bpm), suggesting clinically severe MS. (NYHAFC I following percutaneous mitral balloon valvuloplasty.)

MS (e.g., primary pulmonary disease). A diagnostic dilemma arises when a patient with exertional dyspnea is also found to have MS with only mild-to-moderate range resting MV gradients or valve area. As in the cardiac catheterization laboratory, repeat hemodynamic measurements during exercise may clarify the situation. With modest exercise (staged supine bicycle protocol or immediately post treadmill), the mean mitral valve gradient and systolic pulmonary artery pressure will both increase markedly in clinically significant MS (Fig. 21.24), although the valve area calculation should remain constant. Dobutamine echocardiography in rheumatic MS has also been described.79

Conclusion
The MV area should be confirmed using two or three methods, given the number of potential pitfalls of each. The valve area may be mild by planimetry when there is severe chordal level...
echocardiographic assessment of valvular heart disease

The mean MV gradient may be low in severe MS with low cardiac output or bradycardia. The mean MV gradient may be high in mild MS with high cardiac output or with tachycardia. A shortened PHT is possible with severe MS with concomitant significant AR or cardiomyopathy. In mild MS, the PHT may appear long if there is impaired LV relaxation. The planimetry, PHT, and PISA methods are relatively reliable in atrial fibrillation. The PHT method should not be used early after mitral balloon valvuloplasty. When reporting gradients and when using the continuity equation in atrial fibrillation, measurements from five to ten cycles should be averaged. In problematic cases, an exercise Doppler examination may be helpful.

Mitral Regurgitation

Detection of mitral regurgitation [MR] by Doppler is relatively easy. However, accurate grading of MR severity can be technically challenging. As previously mentioned, no one method is sensitive and specific in all cases. This section reviews the 2D and Doppler supportive signs and semiquantitative and quantitative methods for diagnosing the degree of MR. When several of these findings are in agreement regarding lesion severity, an accurate integrated diagnosis is possible in most situations. Laboratories should ideally apply as many methods as possible in each case, both for confirmation and to maintain the skills needed for difficult cases.

Two-Dimensional and M-Mode Examination and Differential Diagnosis

The asymmetrical atrioventricular valves (mitral and tricuspid) are structurally and functionally more complex than the arterial valves (aortic and pulmonic). Normal mitral valve function depends on both normal structure and function of each component of the so-called mitral apparatus. The mitral apparatus consists of the mitral annulus and left atrium, the leaflets, chordae tendineae, the papillary muscles, and the left ventricle. Structural abnormalities of any component, visible by 2D or M-mode evaluation, raises the possibility for valve malfunction and provide clues as to etiology.

Left Ventricular and Left Atrial Size

A dilated left ventricle or left atrium should always heighten the suspicion for the presence of significant MR, because this condition imposes a volume load on both chambers. In patients with known moderate-to-severe or severe MR,
measurement of LV size and function are important for surgical timing. In chronic, compensated MR, without cardiomyopathy, the left ventricular end systolic volume should be normal and the left ventricular ejection fraction should be ≥60% due to low afterload. Left atrial enlargement accompanies chronic MR. In acute severe MR, the LA may be normal in size. Many comorbid conditions can also enlarge the left ventricle (e.g., cardiomyopathy, AR) or the left atrium (e.g., LV diastolic dysfunction, atrial fibrillation, etc.). See Tables 21.3 to 21.5 for left ventricular size and function by echocardiography.

MITRAL VALVE PROLAPSE AND MYXOMATOUS DEGENERATION

The mitral leaflet systolic coaptation zone normally lies on the ventricular side of an imaginary mitral annular plane, as visualized in the parasternal long axis view or apical three-chamber view. The mitral leaflets are said to “prolapse” when leaflet closure is followed by a late systolic excursion of one or both of the mitral leaflets beyond this imaginary plane, into the left atrial space (Fig. 21.26). The mitral annulus is not planar but “saddle-shaped.” Orientation of the valve in the four-chamber view can sometimes give the appearance of mitral valve prolapse (MVP) when there is none. Therefore, the four-chamber view is not used for diagnosing MVP. Mitral valve prolapse can be functional and transient because of hypovolemia and a hyperdynamic state (reduced LV and mitral annular size leading to leaflet redundancy) or myxomatous mitral valve degeneration. Mitral valve prolapse can be provoked or accentuated by performing a Valsalva maneuver during imaging. In myxomatous MV disease, prolapse usually results from combined leaflet redundancy and chordal elongation (Fig. 21.27). The degree of leaflet thickening can vary markedly among affected patients. Mitral annular dilatation is often present. A characteristic “myxomatous deformity” may include a hypermobile systolic bilowing or parachute-like appearance of the leaflets (Figs. 21.27 and 21.28). In advanced disease, the leaflets can be excessively thickened and calcified, which reduces leaflet mobility. Myxomatous changes can be generalized, involving both leaflets, or be highly localized, sometimes involving only a single segment of one leaflet. Therefore, careful sys-

**FIGURE 21.26.** Mitral valve prolapse (MVP), posterior leaflet. (A) Parasternal long axis view. The posterior leaflet (arrow) crosses the annular plane (red line). (B) M-mode shows late systolic hammock sign of MVP. (C) Color Doppler (same patient) suggests significant MR when a late systolic frame (see arrow, ECG tracing) is viewed in isolation. (D) CW Doppler demonstrates that late systolic MR (double arrows) is a transient event and unlikely to represent a significant regurgitant volume. Single arrow shows onset of systole (MV closure artifact).
tematic scanning across the entire mitral coaptation line is required to rule out MVP. Mitral valve prolapse can be a subtle and rapid event, particularly during tachycardia. Therefore, the M-mode exam (parasternal long axis views) can be helpful in making the diagnosis [Fig. 21.26B]. Associated tricuspid valve prolapse is not uncommon. Aortic valve myxomatous degeneration is occasionally seen.

Mitral valve prolapse can produce any degree of regurgitation (or none at all), depending on whether prolapse is mild, moderate, or severe and to what extent the prolapse is balanced between the two leaflets so that systolic coaptation is maintained. Myxomatous valve disease predisposes patients to chordal rupture, which can result in sudden, severe MR. Ruptured chords can sometimes be detected by surface echocardiography [Fig. 21.29] but are more easily identified by TEE [Figs. 21.30 and 21.31]. Highly eccentric MR is common in MVP and particularly when chordal rupture is present, though central MR can occur with balanced prolapse, usually without chordal rupture [Fig. 21.28]. Eccentric jet lesions generally flow away from the dysfunctional leaflet [i.e., posteriorly directed jet from anterior leaflet prolapse or anterior jet from posterior leaflet prolapse]. Chordal rupture is suspected when a leaflet flail segment is seen partially underriding the opposing leaflet, usually with an attached mobile linear echo density (the liberated chord).

**Surgical Repair of Mitral Valve Prolapse.** Use of the standard TTE and TEE views presented in Chapter 5 can identify all segments of both the posterior and anterior leaflets and the exact site of MR origin (Figs. 21.30 to 21.32). Anatomic imaging, combined with color Doppler, will show the primary site and mechanism of MR (e.g., leaflet
tethering, perforation, prolapse, or ruptured chord). Isolated posterior mitral leaflet prolapse or flail segment is generally most amenable to surgical repair (Fig. 21.30). Prolapse or flail segments of the anterior leaflet can be more difficult to correct surgically (Fig. 21.32). Many additional factors, including local experience, leaflet calcification, mobility, annular size, chordal length, and presence of coronary artery disease, go into the decision-making process. Intraoperative TEE is typically performed whenever MV repair is contemplated in order to confirm the diagnosis, and to assess the results in the operating room immediately after cardiopulmonary bypass (Fig. 21.30E).

**Mitral Annular Calcification**

Calcific degeneration of the mitral valve is common, particularly in the elderly, and can be present prematurely in chronic renal failure patients. Calcium deposits most commonly infiltrate the posterior aspects of the mitral annulus, though the process can become circumferential and begin to invade the bases of the leaflets where mobility is most notably impaired. The degree of MR can be challenging to diagnose because of shadowing of the left atrium. At least mild associated mitral stenosis is common, and this can affect mitral inflow gradient peak and mean velocities. As with prosthetic valve MR, TEE is frequently required for adequate MR evaluation in advanced calcific degeneration of the mitral valve.

**Rheumatic Mitral Regurgitation**

Although mitral stenosis is more commonly associated with rheumatic mitral valve disease, MR can be the predominant hemodynamic lesion in some cases. Rheumatic MR can result from scarred, restricted leaflets that appear almost frozen in a semiopen position or from additional leaflet destruction from superimposed endocarditis. Although rheumatic MS can produce severe left atrial enlargement, particularly with atrial fibrillation, historically it is rheumatic MR (usually with mixed MS) that has produced the largest of the left atria—“giant left atrium.” The giant left atrium is actually defined by chest x-ray [left atrium touching the right thoracic wall].

---

**FIGURE 21.29.** (A) Severe MR due to flail posterior leaflet segment [arrow] from chordal rupture, parasternal long axis view. (B) Massive highly eccentric MR jet by color Doppler directed away from the flail leaflet segment, encircling a severely enlarged left atrium [LA], apical three-chamber view. (C) Markedly increased mitral inflow E wave by pulsed Doppler = 1.5 cm/s (see Table 21.8) indicating severe MR. Other quantitative methods are not required. Note: chronic MR, patient relatively asymptomatic.
FIGURE 21.30. Intraoperative TEE, showing the mechanism for severe anteriorly directed eccentric MR due to myxomatous mitral valve disease primarily affecting the posterior leaflet with a flail middle scallop (P2) due to chordal rupture. (A) TEE five-chamber view. The flail myxomatous-appearing P2 segment and attached ruptured chorda tendina (arrow) resembles a duck’s head [a common appearance]. (B) TEE bicommissural view [see Chapter 5 for standard views] confirms isolated involvement of the P2 segment. (C) Posterior leaflet, P2 and the ruptured chord (arrow) override the mid portion of the relatively normal anterior mitral leaflet. (D) Large color Doppler proximal flow convergence (PFC) zone and a severe, wall-hugging left atrial jet [Coanda effect] are seen. This eccentric jet lesion is consistent with severe MR although the overall color Doppler jet areas is not as large as a comparable central jet lesion created by bileaflet prolapse [compare with Fig. 21.28B]. The PISA method for EROA quantitation cannot be used in this case due to the complex regurgitant orifice shape and a PFC zone that is “constrained” by the LV posterior wall. (E) Surgical specimen (excised P2 segment and ruptured chord [black arrow] in the same patient. (F) Intraoperative TEE immediately after successful quadratic P2 scallop resection with annular reduction [mitral annuloplasty ring seen in cross exaction [arrows] with no residual MR].
**Other Inflammatory Conditions**

Chordal thickening, shortening, and the resultant systolic leaflet tethering causing severe MR can be seen in a number of other inflammatory conditions (scleroderma, radiation valvulitis, anorexigenic- and ergotamine-induced valve disease; and hypereosinophilia). These other etiologies may be difficult, if not impossible, to distinguish echocardiographically (or even on surgical inspection) from rheumatic MR. Minimal or no stenosis and the lack of commissural fusion weigh against rheumatic disease.

**Endocarditis**

A variety of anatomic abnormalities may be present on the 2D or M-mode exam. On M-mode, a mobile echodensity adjacent or attached to the leaflets exhibiting independent motion suggests vegetation. Small vegetations are frequently visible by TTE, but this is highly dependent on imaging conditions. The presence of possible valve vegetations should raise suspicion for significant MR, and a confirmatory TEE may be indicated. Microbial pathogens (Staphylococcus aureus, most notably) can cause leaflet destruction from the release of collagenase and other destructive enzymes. Chordal rupture can occur, in which case mobile chords or flail leaflet segments may be difficult to distinguish from the vegetation material. Pseudoaneurysms of the mitral valve leaflets can perforate or impair leaflet function. Perforation may be visible as a segment of leaflet discontinuity or, if punctate, be apparent by turbulent color Doppler jet lesions arising from areas outside the normal coaptation zones. A perforated mitral annular abscess can produce an annular echo-free space and annular MR.

**Functional Mitral Regurgitation**

Functional MR exists when all elements of the mitral apparatus appear morphologically normal, but their function is impaired, usually because of dilated cardiomyopathy [Fig. 21.33]. The classic identifiable feature by 2D exam is incomplete closure of the mitral leaflet due to leaflet tethering due to papillary muscle displacement, although annular dysfunction may also play a role. Appreciated more recently is the role of LV contractile dyssynergy [detectable and quantifiable echocardiographically, see other chapters] in patients

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**FIGURE 21.31.** Intraoperative TEE showing the mechanism for severe posteriorly directed MR due to flail anterior leaflet segment A2 from chordal rupture [arrow].

**FIGURE 21.32.** TEE showing severe MR due to unusual cleft deformity [confirmed surgically] in the anteromedial commissure region [arrowhead]. Combined bicommissural [A] and gastric short axis view [B] imaging of the mitral valve confirms the regurgitant orifice location and lack of associated chordal rupture or prolapse.
with cardiomyopathy and left bundle branch block. In these patients, functional MR can sometimes be improved substantially by cardiac resynchronization therapy.83

Coronary Artery Disease.84

Left ventricular segmental wall motion abnormalities should heighten suspicion for MR. Ischemic MR is a common but not always easily defined entity. Ischemia can transiently affect papillary muscle function, leading to an eccentric MR jet from leaflet malcoaptation. Myocardial infarction can cause acute severe MR from a papillary muscle tear (leaflet prolapse) or complete avulsion (flail leaflets). Dysfunction of a scarred and retracted papillary muscle can produce severe MR from leaflet tethering.85 Extensive ventricular scarring can lead to adverse remodeling and functional MR instead of ischemic MR. A basal myocardial segment scar can distort mitral annular geometry and function.

Other Causes of Mitral Regurgitation Recognizable by Echocardiography

Systolic anterior motion (SAM) of the mitral valve can cause significant MR. Signs of this abnormality are covered in Chapter 35.

Doppler Evaluation of Mitral Regurgitation

Pulsed-Wave Doppler

As described in Chapter 5, the complete exam includes a pulsed wave (PW) Doppler signal acquired at the mitral leaflet tips, the mitral annulus, and the pulmonary vein. This simple routine provides important information for MR assessment by Doppler flow assessment.

Mitral $E$ Velocity. In severe MR, the mitral forward flow is markedly increased due to the additive forward stroke volume and the “wasted” regurgitant volume. The increased diastolic flow increases the mitral $E$ velocity. In patients with normal ejection fraction, a mitral leaflet tip E-wave velocity $\geq1.2\,\text{cm/s}$ [Fig. 21.29C] is a sensitive and moderately specific sign of severe MR.86 Increased mitral $E$ velocity is not a reliable sign in patients with cardiomyopathy. However, when the mitral inflow pattern is A-wave dominant, there is little likelihood of severe MR.
Pulmonary Vein Systolic Flow Reversal. By TTE and TEE [Fig. 21.34], the PW Doppler sample volume is placed approximately 1 cm into the right superior pulmonary vein (TTE, apical four-chamber view) or in all TEE-visualized pulmonary veins. For a time, based primarily on early TEE reports, pulmonary vein systolic flow reversal was thought to be a highly sensitive and specific sign of severe MR. Later reports have shown that pulmonary vein systolic flow reversal is neither highly sensitive nor highly specific for severe MR. Directed jets that are not severe may enter a pulmonary vein. In chronic severe MR, the remodeled LA may accommodate severe MR without actual pulmonary vein systolic flow reversal. Nonetheless, pulmonary vein inflow systolic blunting or systolic flow reversal should raise the question of possibly severe MR, which should be confirmed using other methods.

Pulsed Doppler of the Left Atrial Appendage. Occasionally, a highly eccentric severe MR jet travels directly into the LAA, where its energy is largely absorbed. Left atrial appendage MR jets may be due to paravalvular leaks in the adjacent prosthetic valves sewing ring or unusual cases of native valve MVP or endocarditis. This possibility should be borne in mind during TEE evaluation.

CONTINUOUS-WAVE DOPPLER
Continuous-wave CW Doppler density (brightness of the spectral Doppler signal) is proportional to the number of regurgitant red blood cells within the path of the interrogating beam. The faint signal of trivial or mild MR can be distinguished from the dense signal of moderate to severe MR. The bright density of all forward flow in the mitral valve (above the baseline) is a useful benchmark for determining the relative regurgitant density (Fig. 21.35). With MVP or functional MR, the signal may be dense only in late or in early stoke, respectively. In these two situations, the CW signal can be useful for demonstrating that the MR is not sustained and is unlikely to be severe, even if dense (Fig. 21.26D). The CW signal can exhibit a V-wave “cut-off” in severe MR with cardiomyopathy and a high left atrial pressure in acute severe MR (see below). The pitfalls are as follows: CW density is a helpful supportive sign and not a true quantitative measure. The CW Doppler signal can be shadowed in calcified or prosthetic valves. The CW beam may not be accurately placed within the jet, potentially resulting in a faint jet despite severe MR. In curvilinear, eccentric jets, CW Doppler cannot be brought into coaxial alignment, so that jet CW Doppler density and peak velocity may be low, even in severe lesions.

LEFT ATRIAL JET AREA BY COLOR DOPPLER
Overall jet size [maximum area] within the left atrium is useful only in extreme circumstances. The area generally includes only the zone of sustained “mosaic” color Doppler. A mosaic is the appearance of alternating, colored, adjacent pixels, signifying a zone of high-velocity turbulent flow. A very small left atrial jet usually means mild MR. A very large jet that almost fills the left atrium usually means severe MR (Table 21.8). The pitfalls are as follows: A common pitfall is an inappropriate Nyquist limit setting. The color Doppler Nyquist limit setting should be in the range of 50 to 60 m/s. Lower or higher settings increase or decrease the mosaic jet area, respectively. Many other factors (described earlier) affect the size and appearance of the jet. Mild-to-moderate volume “high-energy” central jets can entrain surrounding blood and appear severe by color Doppler. Severe MR can appear mild to moderate by color Doppler if hypertension or high left atrial pressure (unobstructed MR) are present. Severe eccentric MR with a “wall-hugging” jet may appear only mild to moderate based on area assessment.99 The tendency for high-energy jets to “cling” to an adjacent solid boundaries surface is known as the Coanda effect (Figs. 21.29 to 21.31). In the future, left atrial jet volume assessment using 3D echocardiography may be of incremental value.

VENA CONTRACTA WIDTH BY COLOR DOPPLER
When the sonographer confirms that only a single predominant MR jet is present, the VC height can be a valuable and relatively easy-to-obtain linear measure of regurgitation severity that does not vary significantly with hemodynamic loading conditions. Details of accurate VC imaging techniques have been described9 and are shown in Figures 21.33 and 21.36. Accurate measurement requires optimized zoomed views of the jet origin (PVC zone, anatomic orifice, and VC region), without regard to the appearance of the distal expanding jet lesion. Measurement is made ideally in the parasternal long axis view to take advantage of the relatively high axial resolution of color Doppler. The VC width is a direct linear measurement, and any error is not propagated due to squaring. The pitfalls are as follows: The VC may not be visible because of leaflet calcification. The method is not valid for multiple jets. The VC width may be overestimated in apical views because of lower color Doppler lateral resolution relative to axial resolution.

FIGURE 21.34. TEE, pulmonary vein systolic flow reversal [arrows below baseline]. Diastolic dominant pulmonary vein forward flow [arrow above baseline] in a patient with significant MR.
echocardiographic assessment of valvular heart disease

FIGURE 21.35. Mitral regurgitation by CW Doppler, surface echo, four-chamber view. (A) Trivial or trace MR with faint, incomplete signal. (B) Mild MR with faint but complete spectral Doppler envelope. (C) Moderate MR with complete envelope and moderately dense signal intensity. (D) Severe MR. Mitral infl ow (above baseline) can be used as a reference for signal intensity. Note: Signal profiles and intensity can be markedly diminished with eccentric jets, shadowing artifacts or with poor interrogating beam alignment.

**TABLE 21.8. Mitral regurgitation Doppler and quantitative parameters**

<table>
<thead>
<tr>
<th></th>
<th>Mild</th>
<th>Moderate</th>
<th>Moderate-to-Severe</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jet area† [color Doppler]</td>
<td>Small, central jet&lt;4cm² or &lt;20% LA area</td>
<td>Variable</td>
<td>Variable</td>
<td>Large, central jet (&gt;10cm² or &gt;40% LA area)</td>
</tr>
<tr>
<td>Mitral infl ow [PW]</td>
<td>A-dominant*</td>
<td></td>
<td></td>
<td>E-dominant, &gt;1.2cm/s*</td>
</tr>
<tr>
<td>MR jet density [CW]</td>
<td>Faint/incomplete</td>
<td>Dense</td>
<td>Dense</td>
<td>Early-peaking or V-wave cut-off (triangular)</td>
</tr>
<tr>
<td>MR jet contour [CW]</td>
<td>Parabolic</td>
<td>Parabolic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>VC width [cm]</td>
<td>&lt;0.3</td>
<td>0.3–0.69</td>
<td>≥0.7</td>
<td></td>
</tr>
<tr>
<td>RV [mL/beat]</td>
<td>&lt;30</td>
<td>260</td>
<td>≥50</td>
<td></td>
</tr>
<tr>
<td>RF (%)</td>
<td>&lt;30</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EROA [cm²]</td>
<td>&lt;0.20</td>
<td>0.20–0.29</td>
<td>0.30–0.39</td>
<td>≥0.40</td>
</tr>
</tbody>
</table>

PW, pulsed-wave Doppler; CW, continuous-wave Doppler; VC, vena contracta; RV, regurgitant volume; RF, regurgitant fraction; EROA, effective regurgitant orifice area.

* In the absence of other causes of increased LA pressures, individuals >50yrs.
†Jet area for eccentric jets is not predictable by color Doppler.

**Proximal Isovelocity Surface Area Method for Effective Regurgitant Orifice Area and Right Ventricle**

This method works best for central jets and may be particularly useful for demonstrating central jets that appear severe by color Doppler jet area alone. The concepts of proximal flow convergence and PISA were discussed earlier. In TTE, images are obtained in apical views in zoomed mode (Fig. 21.36E). In TEE (Fig. 21.37), the best coaxially aligned views are used. Whether by TTE or TEE, a standard practice is to shift the color Doppler baseline toward the direction of the regurgitant jet, so that the lower Nyquist limit ($V_a$) is reduced to approximately 40cm/s or lower. This produces a homogeneous PFC zone color appearance within the PISA border. $V_a$...
FIGURE 21.36. MR quantitation using three methods in the same patient in a routine exam. [A] LVOT diameter measurement. [B] Zoom mode, for vena contracta (VC) measurement. [C] MV short axis with color Doppler confirms single MR orifice. [D] Moderately large central jet, apical four-chamber view [color Nyquist limit >50 and <60]. [E] “Zoomed” view of proximal flow convergence; reduction of color Doppler Nyquist limit in direction of the jet lesion; PISA radius measurement (r). [F] Mid-diastolic mitral annulus diameter measurement. [G] Pulsed Doppler at the mitral annulus level, MV annulus VTI measurement. [H] VTI and peak velocity of MR jet by CW Doppler. [I] Pulsed Doppler for VTI of the LVOT. See images for variables. VC method: \( VC = 0.5 \text{ cm} \) [simple, direct linear measurement]. PISA method: \( EROA = 6.28 r^2 \times \frac{V_a}{V_{pk}} = 0.22 \text{ cm}^2 \); RV = EROA \times VTI_{MR} = 41.6 \text{ mL}; SV = 6.28 \left( r^2 \right) \times V_r = 118 \text{ mL}; RF = RV/SV = 0.35 [moderate MR, see Table 21.8]. Doppler flow method: \( SV_{LVOT} = 0.785 \times D_{LVOT}^2 \times VTI_{LVOT} = 37.7 \text{ mL}; SV_{MVann} = 0.785 \times D_{MVann}^2 \times VTI_{MVann} = 73.2 \text{ mL}; RV = SV_{MVann} \times VTI_{MR} = 35.5 \text{ mL}; RF = RV/SV_{MVann} = 0.48. All methods are in rough agreement for a diagnosis of moderate MR [see text for formulas and Table 21.8 for reference values].

FIGURE 21.37. PISA method for MR quantitation by TEE. A nearly perfectly hemispheric PISA is formed by a punctate fenestration (arrow) in the mitral anterior leaflet base (A, simultaneous 2D and color Doppler image). The radius is easily measured in zoom mode (B). Aliasing velocity \( (V_a) \) is reduced in direction of jet lesion. (C) CW Doppler of the MR jet. Calculations: see images for variables. EROA = 6.28 \left( r^2 \right) \times \frac{V_a}{V_{pk}} = 0.22 \text{ cm}^2; RV = EROA \times VTI_{MR} = 15.7 \text{ mL}; SV = 6.28 \left( r^2 \right) \times V_r = 114.5 \text{ mL}; RF = RV/SV = 0.13. All consistent with mild MR [see text for Table 21.8].
overestimated EROA. Despite these limitations, the presence of one or more areas of prominent proximal flow convergence (at standard color Doppler Nyquist settings) is a sign of possibly significant MR that warrants further investigation. Therefore, PFC (PISA) imaging attempts are never wasted.

**Mitral Annular Flow and Right Ventricle by Pulsed Doppler**

The standard exam includes pulsed Doppler within the mitral annulus. The mitral annular diameter can be measured, and mitral annular stroke volume determined \( SV = \text{CSA} \times \text{VTI} \) (see example calculation in Figure 21.36). When mitral regurgitation is present, the forward diastolic mitral flow includes the useful forward stroke volume in addition to the added “wasted” RV. The mitral valve RV can be determined by subtracting the stroke volume obtained at a competent reference valve (LVOT or RVOT) from the increased mitral annular total SV:

\[
\text{Total SV}_{\text{MV,annulus}} = \text{SV}_{\text{reference valve}} - \text{RV}_{\text{MV}}
\]

See Table 21.8 for clinically useful RV values. When the overall forward stroke volume is reduced (cardiomyopathy), the regurgitant volume may be misleadingly low, and the regurgitant fraction (RF) should be used instead (see Table 21.8 for reference values):

\[
\text{RF}_{\text{MV}} = \frac{\text{RV}}{\text{SV}_{\text{MV}}}
\]

**Pitfalls.** The method is difficult to apply in irregular rhythms (e.g., atrial fibrillation) because of variable stroke volumes beat to beat. Because annular diameters are squared, measurement errors are compounded. Significant reference valve regurgitation invalidates the method. Accurate mitral annular measurement can be difficult or impossible with severe mitral annular calcification (Fig. 21.22) or other morphologic deformity.

**Mitral Regurgitation Assessment by Transesophageal Echocardiography**

Tranmsoesophageal echocardiography can be an ideal modality for assessing mitral leaflet morphology and MR severity. The left atrial blood pool provides an excellent acoustic medium for imaging, usually with no interposed structures that could cause signal attenuation. Tranmsoesophageal echocardiography is very useful when surface echocardiography is technically difficult for any reason, particularly when there is left atrial shadowing from calcification or a mitral valve prosthesis. Any of the above-mentioned quantitative measures or indicators can be applied to TEE, potentially with improved accuracy in some cases. Many times, surface echocardiography excels at gathering spectral Doppler data because of an increased number of potential imaging windows necessary for obtaining coaxial jet alignment. Transesophageal echocardiography generally excels at providing a higher resolution detailed look at leaflet anatomy. One must bear in mind that mild MR to moderate-range jets will appear more impressive by TEE than by TTE because of improved image clarity and decreased imaging depths. Therefore, the other factors regarding jet severity (jet density by CW Doppler, VC, PISA analysis, etc.) must be kept in mind to avoid overcalling MR severity. Tranmsoesophageal echocardiography is ideally used as a complementary imaging modality when the surface exam is inconclusive or when surgery is being considered (endocarditis with potential abscess, possible mitral valve repair, prosthetic valve dysfunction).

**Acute Severe Mitral Regurgitation**

Acute severe MR should be considered in the differential diagnosis of patients with cardiogenic shock. Acute severe MR can be inadvertently missed on review of a surface echocardiogram without a high index of suspicion. As mentioned above, the CW and color Doppler signals may be inconspicuous in torrential MR (e.g., acute chordal or papillary muscle rupture) because of systemic hypotension and high left atrial pressure (low LV-to-LA pressure gradient) due to almost unobstructed (nonturbulent) regurgitant flow. Color Doppler signs for low-velocity acute severe MR may also be lacking in this situation due to tachycardia with relatively slow color Doppler sample rate. The MR “V-wave cut-off” sign by CW Doppler (Fig. 21.39) is usually present, however. Other clues...
to the diagnosis include normal or hyperdynamic LV contractility with a reduced LVOT stroke volume by LVOT PW Doppler. Acute severe MR is a potential surgical emergency. Confirmatory TEE for both severity and mechanism assessment should be performed expeditiously when the diagnosis is suspected.

**Tricuspid Valve Disease**

**Tricuspid Stenosis**

**Two-Dimensional Imaging and Differential Diagnosis**

Tricuspid stenosis (TS) is an uncommon diagnosis that is suspected in the clinical setting of systemic venous hypertension with a prolonged y-descent as observed in the jugular vein, visually, by RA pressure tracing, or by hepatic vein pulsed Doppler. Rheumatic TS, carcinoid heart disease, tumor obstruction, and obstructed tricuspid valve (TV) prosthesis are all important but uncommon conditions that can be recognized by echocardiography.

**Rheumatic Tricuspid Stenosis**

Rheumatic TV changes that are evident by echocardiography mirror those seen in rheumatic mitral valve disease: leaflet leading edge thickening, with reduced mobility, commissural fusion, and leaflet doming and leaflet tethering from chordal shortening and fusion. Rheumatic TS rarely if ever occurs in the absence of rheumatic MV disease. Associated TR is common.

**Carcinoid Heart Disease**

Carcinoid heart disease is suspected by 2D echocardiography when the TV leaflets and submitral apparatus appear markedly thickened and fully or partially frozen in a semiopen position with marked systolic leaflet malcoaptation [Fig. 21.40]. Tricuspid stenosis is usually in the mild to moderate range with associated torrential TR. Associated pulmonary stenosis (PS) and PR (due to similar leaflet deformity) are the rule in carcinoid heart disease, whereas rheumatic PV disease is a rare diagnosis, particularly in Western countries.

Tricuspid stenosis rarely occurs with obstructive atypical right atrial myxomas or other, usually malignant, tumors. A prosthetic TV [either biologic or mechanical] can develop forward flow obstruction [Fig. 21.41].

**Tricuspid Stenosis Assessment by Doppler**

Pulse-wave Doppler of the hepatic vein inflow demonstrates a prolonged y-descent due to delayed diastolic RA emptying. Because complicating TS can be easily overlooked in the setting of dramatic associated TR (rheumatic or carcinoid TV disease), the diastolic tricuspid inflow velocities should always be carefully observed by CW Doppler. Values for pathologic TV gradients are much lower than for MS, since systemic venous hypertension is very poorly tolerated. Clinically apparent TS [lower extremity edema, ascites, early satsiety, etc.] can occur whenever the mean resting TV gradient is >5 mm Hg. After TV repair, intraoperative TEE is useful for evaluating successful treatment of TR and also for ensuring that excessive annular reduction has not created TS.
Tricuspid Regurgitation

Two-Dimensional Examination and Differential Diagnosis

Hemodynamically significant TR is commonly seen in adult echocardiography laboratories. Moderate and even severe-range TR is often tolerated without clinical symptoms for long periods, as long as the pulmonary artery pressure (forward flow resistance) remains normal [low] and right ventricle systolic function is normal. Echocardiography is important not only for diagnosis of TR but also for detecting signs of eventual right ventricle failure due to volume overload.

Tricuspid Valve Prolapse

Tricuspid valve prolapse, often associated with myxomatous degeneration of the mitral valve, is recognizable by 2D imaging, though obtaining views that are in true long or short axis alignment with the TV annulus can be difficult. In any of a number of views, one or both leaflets may be seen underriding the opposing leaflet or appearing to cross that annular plane. Flail leaflet segments and severe TR can result from chordal rupture, related to myxomatous degeneration, endocarditis [Fig. 21.42], endomyocardial biopsy,96,97 or deceleration injury.

Posttraumatic Tricuspid Regurgitation

Deceleration injuries [e.g., fall from a ladder, car crashes] can result in chordal rupture or anterior leaflet tear, most commonly. The anatomic clue for leaflet tear may be the lack of other valve pathology and an odd appearance to the jet origin [e.g., a basal leaflet jet origin] with the trauma even sometimes having occurred years or decades before diagnosis.

Rheumatic Tricuspid Regurgitation

The association with rheumatic MV disease is high, with clinically significant rheumatic TR being much more common than TR. The degree of rheumatic TR should be routinely excluded prior to MV surgical repair, since postoperative severe TR is often not well tolerated, particularly in older patients. Recognizable TV leaflet malcoaptation due to leaflet overtethering from chordal thickening, and shortening and secondary annular dilatation are usually apparent echocardiographically due to increased leaflet and subvalvular echogenicity.

Functional Tricuspid Regurgitation

When the right ventricle is chronically exposed to volume or pressure overload, for whatever reason, annular dilatation

![Figure 21.42. Signs of severe “unobstructed TR” from TV endocarditis. (A) Flail leaflets (arrows) with unimpressive color Doppler jet lesion [potentially overlooked laminar flow]. (B) Low-velocity TR jet by CW Doppler with “v-wave cut-off” [double arrows] with increased TV inflow velocity (>1 cm/s, single arrow]. (C) Large hepatic vein systolic wave by pulsed Doppler. (D) Paradoxical septal motion by M-mode, parasternal long axis view with inferolateral and septal walls moving in unison.](image)
and possibly papillary muscle displacement can cause severe leaflet malcoaptation. Acute pressure overload (acute pulmonary embolus) can also cause acute functional TR, as can acute right ventricle dilatation and right ventricle dysfunction from right ventricle infarction. The TV annulus is less fibromuscular than the MV annulus. The looser TV annulus, therefore, is particularly subject to functional TR that is frequently at least partially reversible with medical or other treatment of the underlying cause of right ventricle pressure and or volume overload. However, chronic severe right ventricle volume overload from any cause (PR, atrial septal defect, and TR itself) begets more functional TR and eventually leads to right ventricle failure. This process can be accelerated by pulmonary hypertension. Paradoxical septal motion by M-mode (Fig. 21.42A) or 2D inspection (Fig. 21.43) is a sign of right ventricle volume overload that should prompt a search of significant TR, PR, and ASD—if not all three—in some cases. Paradoxical septal motion occurs because of equalized right ventricle and LV diastolic pressure, causing the septum paradoxically to move toward the LV central axis during diastole.

**Doppler Evaluation of Tricuspid Regurgitation Severity**

**Color Doppler**

Severe TR can be missed by color Doppler when there is nearly unobstructed and nonturbulent regurgitant flow (Fig. 21.42), particularly when associated with low pulmonary artery pressure (low right ventricle to right atrium peak pressure difference). With unobstructed TR, a turbulent color Doppler jet lesion may not be present at all (Fig. 21.42A). On the other hand, when the leaflets are reasonably intact, a color Doppler jet lesion can be very prominent (Fig. 21.44A),
tractus usually signify clinically significant TR. Eccentric Doppler criteria are needed. Color Doppler alone can be misleading in severe TR, other TV leaflets are not always clearly visualized by TTE, and TR jets may be underestimated by color jet area. Because the modified Bernoulli equation does not apply.

Regurgitant flow is relatively unobstructed, in which case the Doppler may be unreliable in torrential TR because the size.

Which may influence interpretation of the color Doppler jet can be used to estimate systolic PA pressure (see Chapter 5), (Figs. 21.42B and 21.44B). The TR velocity by CW Doppler of Fig. 21.45. Severe stenosis of a pulmonary valve homograft prosthesis (horizontal arrow) with moderate PR (downward arrow) with relatively steep deceleration slope.

Important note: Systolic PA pressure assessment by CW Doppler may be unreliable in torrenal TR because the regurigant flow is relatively unobstructed, in which case the modified Bernoulli equation does not apply.

PULSED DOPPLER OF THE HEPATIC VEIN, SUBCOSTAL VIEW
A large hepatic vein systolic wave by pulsed Doppler [Fig. 21.42C] can be an important sign of severe TR, particularly when the TV is not well visualized either by 2D exam or by color Doppler and when correlated with an enlarged right ventricle with paradoxical septal motion. The hepatic vein systolic wave, however, may become less prominent with severe right ventricle failure and decreased overall right ventricle stroke volume.

Pulmonary Valve Disease

Pulmonary Stenosis

TWO-DIMENSIONAL EVALUATION AND DIFFERENTIAL DIAGNOSIS
Duo to a relatively anterior and immediately retrosternal position and a posteriorly directed orientation, the pulmonary valve can be difficult to visualize by both surface and transesophageal echocardiography. The normal, thin semilunar cusps are almost identical in morphology to the normal aortic valve cusps, making them frequently difficult to fully visualize under normal circumstances. Acquired native valve PS is distinctly uncommon, with rheumatic heart disease only rarely involving this valve. Calcific PS of a native congenitally normal trileaflet valve is essentially not a described entity, although calcification and stenosis of a pulmonary valve biologic prosthesis can occur (Fig. 21.45), and residual or very slowly progressive obstruction of previously ballooned congenital PS can be seen in adult echocardiography laboratories. As previously mentioned, carcinoid heart disease (also unusual) is characterized by mixed PS and PR, the stenosis component of which is usually mild to moderate in severity. An important feature of severe RVOT obstruction (PS) is that the right ventricle can produce systemic-level systolic pressures when the pressure rise has been gradual. A sign of this is a D-shaped ventricular septum in the parasternal short axis view of the left ventricle. This is different from the paradoxical septal motion of right ventricle volume overload (see above) in that the septum remains “flattened” during diastole and systole because the high right ventricle pressure no longer allows the left ventricle systemic pressure to determine systolic septal curvature. Interventricular septal flattening, however, is a nonspecific finding since it will also occur in other conditions leading to right ventricle pressure overload (pulmonary hypertension from any cause).

DOPPLER EVALUATION
Although the pulmonary valve may be difficult to fully visualize by 2D imaging, it is often well aligned for Doppler evaluation. A routine component of the TTE is CW Doppler of the right ventricle outflow tract/pulmonary valve. This generally detects either valvular or subvalvular obstruction as in the case of the failed pulmonary homograft prosthesis in Figure 21.45. Dynamic infundibular RVOT obstructive gradients are usually late-peaking and can be superimposed upon the fixed valve lesion. This phenomenon is not uncommon in congenital forms of PS.

Pulmonary Regurgitation
A trivial to mild degree of PR is present in most normal subjects. This is usually detectable by color Doppler on routine exam since the valve lies very close to the transducer.
in the parasternal short axis view, and the PR jet is usually ideally aligned for detection by Doppler. Even mild PR lesions, therefore, can appear somewhat prominent by CW Doppler, as in Figure 21.46A,B. With small PR regurgitant volumes, the deceleration time of the PR jet is long, and some degree of persistent PV end-diastolic pressure differential exists, which is even more evident in the setting of pulmonary hypertension (increased PR velocity throughout diastole by CW Doppler). Severe PR, on the other hand, produces a dense but steep deceleration time by CW Doppler with no significant residual end-diastolic pressure differential (Fig. 21.46). Unobstructed PR can be due to endocarditis, congenital or surgical absence of the valve leaflets, failed prosthesis, or carcinoid heart disease. Endocarditis is the least likely of these to produce mixed PS and PR. In “free” PR, the brief, low-velocity, severe PR jet can be easily missed by color Doppler. So careful attention to other signs of right ventricular volume overload can be important clues for the diagnosis [paradoxic septum, right ventricle enlargement, functional TR, high forward RVOT VTI by pulsed Doppler and the typical CW Doppler profile (Fig. 21.46D)].

**Prosthetic Valve Dysfunction**

Prosthetic valves may be placed in any of the four valve positions. In addition, the mitral, tricuspid, and pulmonary valves can be repaired in favorable circumstances. A clinical history of prosthetic valve surgery may not be available for the sonographer or interpreting physician, although 2D and Doppler signs of prosthetic valves can be recognized.

**Mechanical Prostheses: Recognition by Two-Dimensional Imaging**

Although many old-generation prosthetic valve types remain in the population, they are now much less commonly encountered in echocardiography laboratories. The most common type of new-generation *mechanical valve prosthesis* (in usage for >20 years) consists of two symmetrical durable pyrolytic carbon tilting disks, suspended within a rigid ring. The mechanical valve ring typically produces a small shadowing artifact in the far field. The disks, particularly in the closed
position, produce a prominent and usually easily recognizable reverberation artifact. Reverberation consists of additional artifactual echo reflections in the far field, distinguishing it from shadowing, which is the lack of echoes in the far field. These imaging artifacts aid in recognition of mechanical prosthetic valves, but they also make obstructive or insufficiency lesions and their mechanisms difficult to identify (Fig. 21.47).

Biologic Valves: Recognition by Two-Dimensional Imaging

Modern biologic valves can be divided into three subtypes that can be recognized echocardiographically: (1) “Stented” valves (used in any position) consist of trileaflet semilunar cusps constructed from pericardium or from porcine aortic valve xenograft origin, suspended with three rigid supporting “struts.” (2) “Stentless” valves (used in the aortic position) are porcine aortic valve xenografts supported by a fabric backing instead of struts. (3) Pulmonary autografts or homografts (used in the aortic or pulmonary positions) consist of the intact pulmonary annular and leaflet structures. Stentless valves and pulmonary homografts or autografts can be difficult to differentiate echocardiographically from a native valve, although on detailed inspection annular thickening is seen, and for stentless valves the backing material can often be distinguished by careful inspection. The stented biologic valve, on the other hand, is easily recognized because of the protruding echogenic struts (Fig. 21.48). The biologic leaflets should exhibit opening and closure characteristics typical of any normal aortic valve. Over time, however, reduced leaflet motion, calcification and even cusp prolapse or flail segments can sometimes be identified by surface imaging as shadowing and reverberation artifacts are less problematic.

Doppler Evaluation

In comparison with normal native valves, normally functioning prosthetic valves (and repaired valves) typically exhibit mild obstructive gradients at rest. Normal prosthetic valve flow velocities and gradients vary among patients according to valve type, patient size, and other hemodynamic variables. Therefore, it is important to conduct a baseline echocardiography examination, usually within 6 to 8 weeks after valve implantation surgery for future reference. For questionable valves and when baseline data are not available, reference tables may be consulted for normal valve velocities and gradients.100

FIGURE 21.47. Bileaflet mechanical valve prosthesis, mitral position. (A) TTE, four-chamber view. Prosthetic valve reverberation artifacts obscure the left atrium (LA). (B) TEE, four-chamber view. Prosthetic valve reverberation artifacts obscure the left ventricle (LV). (C) TEE shows normal degree of central bileaflet mechanical prosthetic MR. Jets typically originate from the central margin of the sewing ring as shown. Sewing ring shadowing artifact (absent far field echoes, double arrow, left side); reverberation artifact (additional far field echoes, double arrows, right side).
Many of the same principles that apply for native valve obstructive lesions can be used for evaluating prosthetic valve forward flow. A mechanical valve can become obstructed due to pannus (organized thrombus) or recent thrombus formation within the sewing ring. Unfortunately, thrombus and pannus formation can be difficult to visualize, although TEE may be of significant incremental benefit for making this diagnosis and guiding therapy. Perivalvular leaks can result from technical failure of a suture or from inadequacy of the underlying anchoring connective tissue (degenerative changes or destruction from endocarditis). Prosthetic valves can become dysfunctional due to obstructive lesions or regurgitation. As already alluded to, biologic valves undergo degeneration over time, which can produce obstructive, insufficiency, or mixed lesions.

The CW Doppler signal is usually diagnostic for a bileaflet mechanical valve because of the prominent opening and closure ultrasound artifacts produced by the disks (Fig. 21.49). Prosthetic valve forward flow gradients can increase markedly from either obstructive or regurgitation lesions. Whenever a prosthetic valve gradient is more than mildly increased, a thorough Doppler investigation is warranted. Regardless of valve position, the sonographer should systematically image all aspects of the valve sewing ring with color Doppler, in an attempt to identify sites of abnormal proximal flow convergence in the sewing ring, which can be a sign of a perivalvular leak since the jet lesion (particularly with perivalvular MR on surface echo) may be hidden by shadowing and reverberation artifacts. CW Doppler should "explore" the prosthetic ring in case a focal, dense regurgitation jet is encountered. Mechanical valves always exhibit a normal amount of central regurgitation that originates inside the sewing ring and generally extends no more than 1 to 2 cm in jet length by color Doppler from the valve (Fig. 21.48C). These small jets are normal in the LV outflow tract with a normally functioning aortic valve prosthesis. More extensive LVOT jets may require further investigation. Although there is no normal amount of central leak for a biologic prosthesis, mild central regurgitation is sometimes seen even immediately after implantation. Baseline documentation and routine follow-up by surface echocardiography are all that is indicated if the forward flow characteristics and leaflet motion are otherwise normal.

A high peak forward flow gradient across the mitral or tricuspid valve prosthesis and a relatively steep [short] deceleration time suggest prosthetic valve regurgitation. A high
peak and mean gradient and lengthened mitral deceleration time usually suggests prosthetic valve obstruction [Fig. 21.41], although combined obstruction and regurgitation can occur. Whenever prosthetic valve dysfunction is suspected, comprehensive TTE and TEE are frequently needed in order to address the situation.91,103,104 Figures 21.50 and 21.51 show TEE evaluations of dysfunctional mechanical mitral and aortic valve prostheses, respectively.

FIGURE 21.49. Normal forward flow velocities by CW Doppler of a mechanical aortic valve prosthesis [A] and mechanical mitral valve prosthesis [B] in the same patient. The isovolumic contraction [IC] and isovolumic relaxation [IR] periods are clearly demarcated by the characteristic mechanical leaflet opening and closure artifacts in this patient with “double” mechanical prosthetic valves.

FIGURE 21.50. TEE evaluation of severe perivalvular MR in a patient with prosthetic valve bacterial endocarditis. [A] The prosthetic leaflets appear in a typical closed position. Mitral annular abscess with associated vegetation [arrow]. [B] In another imaging plane, same patient, a localized region of sewing ring dehiscence [upward arrow] is visible. Sewing ring [downward arrow], linear reverberations, mechanical disks in open position [arrowhead], left atrial appendage [LAA]. (C) Dolor Doppler shows severe perivalvular MR (from outside the sewing ring) entering the left atrial appendage.
required. In a significant number of other cases, significant findings, and rigorous quantitative methods are not mild or they are clearly severe, based on a few specific chambers. As mentioned throughout this chapter, there are only be applied when significant valve lesions are first recognized, and then categorized as far as hemodynamic severity with analysis of the effects on the sending or receiving chambers. As mentioned throughout this chapter, there are advantages and disadvantages for each of the methods that have been discussed. In many cases, lesions are clearly mild or they are clearly severe, based on a few specific findings, and rigorous quantitative methods are not required. In a significant number of other cases, significant lesions may be missed altogether, or misdiagnosed if one or more quantitative methods or a combination of supporting features of lesion severity are not integrated into the final diagnosis.

**Summary**

Echocardiographic assessment of valvular heart disease is a cornerstone of modern general cardiology practice. Echocardiographic analysis of valve disease may be divided into two complementary categories: (1) the two-dimensional exam that may lead one to suspect valve pathology and etiology and severity, based on anatomic features; and (2) the Doppler examination. These components of the transthoracic examination are absolutely complementary. Clinical management guidelines for valvular heart disease can only be applied when significant valve lesions are first recognized and then categorized as far as hemodynamic severity with analysis of the effects on the sending or receiving chambers. As mentioned throughout this chapter, there are advantages and disadvantages for each of the methods that have been discussed. In many cases, lesions are clearly mild or they are clearly severe, based on a few specific findings, and rigorous quantitative methods are not required. In a significant number of other cases, significant lesions may be missed altogether, or misdiagnosed if one or more quantitative methods or a combination of supporting features of lesion severity are not integrated into the final diagnosis.

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