

Table 6 Parameters for evaluation of the severity of prosthetic aortic valve regurgitation

Parameter	Mild	Moderate	Severe
Valve structure and motion			
Mechanical or bioprosthetic	Usually normal	Abnormal [†]	Abnormal [†]
Structural parameters			
LV size	Normal [‡]	Normal or mildly dilated [‡]	Dilated [‡]
Doppler parameters (qualitative or semiquantitative)			
Jet width in central jets (% LVO diameter): color [*]	Narrow ($\leq 25\%$)	Intermediate (26%-64%)	Large ($\geq 65\%$)
Jet density: CW Doppler	Incomplete or faint	Dense	Dense
Jet deceleration rate (PHT, ms): CW Doppler [§]	Slow (>500)	Variable (200-500)	Steep (<200)
LVO flow vs pulmonary flow: PW Doppler	Slightly increased	Intermediate	Greatly increased
Diastolic flow reversal in the descending aorta: PW Doppler	Absent or brief early diastolic	Intermediate	Prominent, holodiastolic
Doppler parameters (quantitative)			
Regurgitant volume (mL/beat)	<30	30-59	>60
Regurgitant fraction (%)	<30	30-50	>50

PHT, Pressure half-time.

^{*}Parameter applicable to central jets and is less accurate in eccentric jets; Nyquist limit of 50 to 60 cm/s.

[†]Abnormal mechanical valves, for example, immobile occluder (valvular regurgitation), dehiscence or rocking (paravalvular regurgitation); abnormal biologic valves, for example, leaflet thickening or prolapse (valvular), dehiscence or rocking (paravalvular regurgitation).

[‡]Applies to chronic, late postoperative AR in the absence of other etiologies.

[§]Influenced by LV compliance.

is placed too close to the prosthetic valve, proximal acceleration may lead to overestimation of velocity and thus of regurgitant volume.

Although left-sided volume overload is expected in the presence of hemodynamically significant AR, LV volumes may reflect the preoperative state, especially in cases of early postoperative prosthetic regurgitation. However, if LV volumes fail to decrease after valve replacement for AR, a hemodynamically significant leak should be suspected among other factors.

3. Role of TEE in Prosthetic AR. The role of TEE in prosthetic AR is to better identify its site in technically difficult transthoracic echocardiographic studies (valvular vs paravalvular) and delineate the mechanism of regurgitation and associated complications such as endocarditis, abscess formation, masses, or thrombus that interfere with disc function.¹²⁸ Posterior paravalvular leaks that were not visible on surface imaging may be evident, and it may be possible to map the full extent of dehiscence leading to regurgitation.^{8,71} The long-axis view is useful for measuring jet width and the ratio of jet width to LVO tract width for the evaluation of severity. TEE may be limited in evaluating prosthetic AR in the midesophageal level because of acoustic shadowing anteriorly. Importantly, the presence of a concomitant mitral prosthesis will cause significant shadowing and obscure the LVO tract.^{129,130} In such cases it is critical to evaluate the prosthesis from transgastric transducer positions.

4. An Integrative Approach in Evaluating Prosthetic AR. Assessment of severity of AR is in general more difficult than in native valves because of the high prevalence of paravalvular regurgitation and eccentric jets. The process of grading AR should be comprehensive and integrative, using a combination of the qualitative and semi-quantitative parameters shown in Table 6. If the AR is definitely determined as mild or less using these parameters, no further measurement is required. If there are parameters suggestive of more than mild AR and the quality of the data lends itself to quantitation, it is desirable to measure quantitatively the degree of AR, including the regurgitant volume and fraction. When the evidence from the different parameters is concordant, it is easy to grade AR severity. When parameters are contradictory, one must look carefully for technical and physiologic reasons to explain these discrepancies and rely on

the components that have the best quality and are the most accurate, considering the presence of a prosthetic valve and the underlying clinical condition. TTE may be adequate for most of the qualitative and quantitative information needed to evaluate AR severity. TEE complements the transthoracic approach in technically difficult studies, in mapping the extent of annular involvement, and in evaluating the etiology of AR and associated complications.¹³¹

IV. EVALUATION OF PROSTHETIC MITRAL VALVES

A. Prosthetic Mitral Valve Function and Stenosis

1. Imaging Considerations. With the availability of several windows on TTE, recording of jet velocity across the prosthetic mitral valve is readily feasible to evaluate prosthetic valve function. However, a major consideration in the evaluation of prosthetic mitral valve function by echocardiography is the effect of acoustic shadowing by the prosthesis on assessment of MR¹³² (Figure 4). This problem is worse with mechanical valves than with bioprosthetic valves. On TTE, LV function is readily evaluated, but the left atrium is often obscured for imaging and Doppler interrogation. In contrast, TEE provides excellent visualization of the left atrium and MR, but acoustic shadowing limits visualization of the left ventricle (Figure 12). Thus, a comprehensive assessment of prosthetic mitral valve function often requires both transthoracic and transesophageal imaging when valve dysfunction is suspected clinically or on TTE.

a. Parasternal Views. In the parasternal long-axis view, the mitral valve prosthesis may obscure portions of the left atrium and its posterior wall.¹³² This may prevent detection of small degrees of MR or make it difficult to determine the precise origin or vena contracta of an MR jet (Figure 4). The parasternal long-axis view allows visualization the LVO tract, which can be impinged by higher profile prostheses. This can lead to LVO turbulence and at times significant LVO tract gradient. The short-axis view at the level of the prosthesis allows visualization of the leaflet excursion and sewing ring of a bioprosthetic mitral valve. It may allow determination of the circumferential extent of

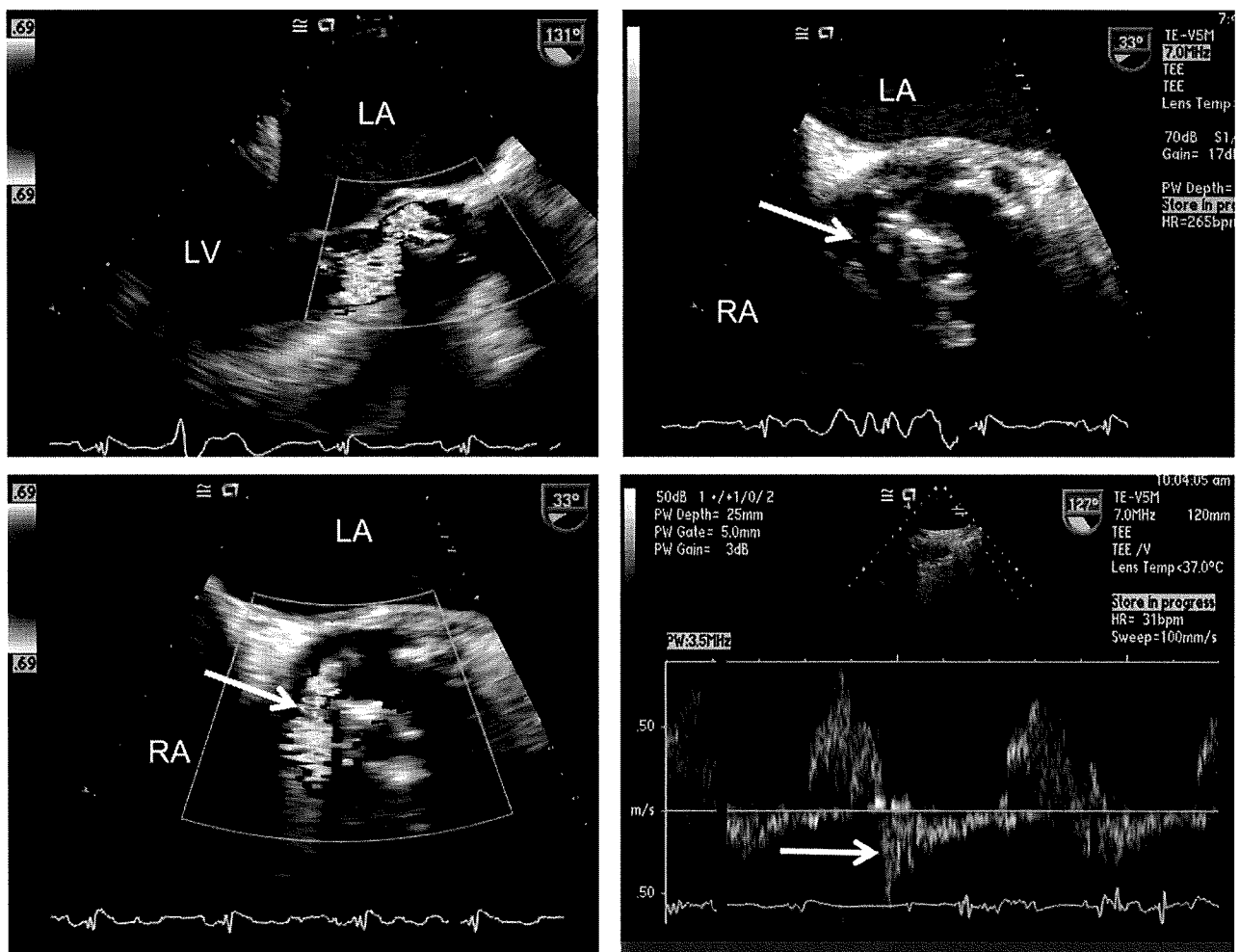


Figure 11 Transesophageal images of a patient with perivalvular significant AR demonstrating (arrows) the extent of dehiscence and regurgitation in cross-section and diastolic flow reversal in the descending aorta. Flow convergence in the aortic root, vena contracta, and an eccentric jet directed anteriorly in the LVO toward the septum (left upper panel) are seen. Because of jet eccentricity, measurement of jet width to LVO diameter is not advised in this case. See Videos 11 and 12. [View video clips online.](#)

a paravalvular leak by color flow mapping. For mechanical valves, the short-axis view is limited by acoustic shadowing of the posterior aspect of the valve sewing ring.

b. Apical Views. The apical views allow visualization of leaflet excursion for both bioprosthetic and mechanical valve prostheses. Muratori et al¹³³ showed a high concordance between leaflet excursion measurements by echocardiography (85% on TTE and 100% on TEE) and cinefluoroscopy for mitral valve prostheses. Apical views may allow the detection of thrombus or pannus that might limit leaflet excursion. Vegetations can be seen but often are masked by acoustic shadowing, which also limits the assessment of MR from apical windows.¹³² Despite this problem, paravalvular leaks may be seen because their origin is outside the sewing ring, and significant regurgitation is often suspected from the presence of proximal flow convergence on the LV side of the prosthesis.¹³⁴ The apical views almost always allow well-aligned parallel Doppler velocity recordings of forward flow through the prosthesis orifice. This yields important information about prosthetic valve function, such as peak velocity, peak and mean pressure gradient, and comparison of transmitral to LVO VTIs and derivation of their ratio (Figure 13). In certain normal valves or in obstructed valves, the inflow jet may be very eccentric

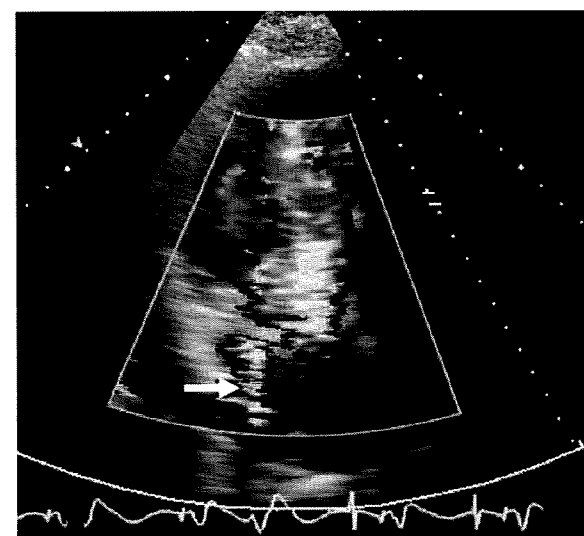
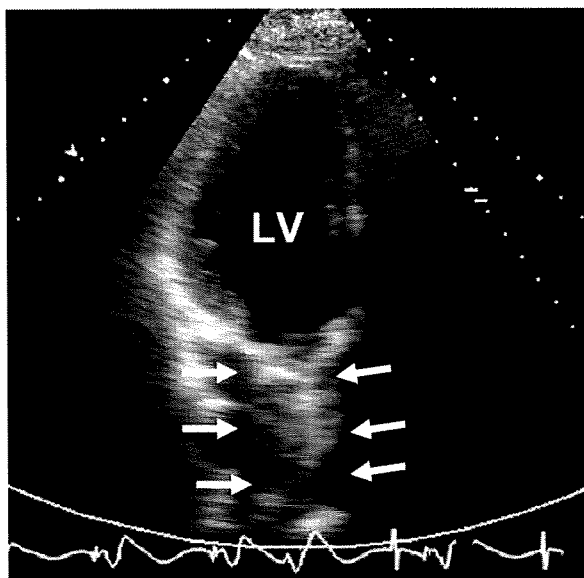
such that the least angulation with flow is obtained from a parasternal or lateral approach. Color flow Doppler is very helpful in evaluating the direction of flow into the left ventricle, thus further optimizing spectral Doppler recordings of jet velocity.

2. Doppler Parameters of Prosthetic Mitral Valve Function.

A complete examination should include peak early velocity, estimation of mean pressure gradient, heart rate, pressure half-time, and determination of whether regurgitation is present or suspected. A DVI and/or EOA may be determined, as needed, for further refinement. Other evaluation should include the determination of LV and RV size and function, LA size if possible, and an estimation of pulmonary artery systolic pressure (Table 7).

a. Peak Early Mitral Velocity. The peak E velocity is easy to measure and provides a simple screen for prosthetic valve dysfunction.¹³⁵ The peak velocity can be elevated in hyperdynamic states, tachycardia, small valve size, stenosis, or regurgitation. Tachycardia exerts a particularly important effect on velocity and gradient measurements in the mitral position because of the associated shortening of the diastolic filling period. In addition, inhomogeneous flow profiles across caged-ball and bileaflet prostheses can lead to Doppler velocity

Transthoracic



Transesophageal

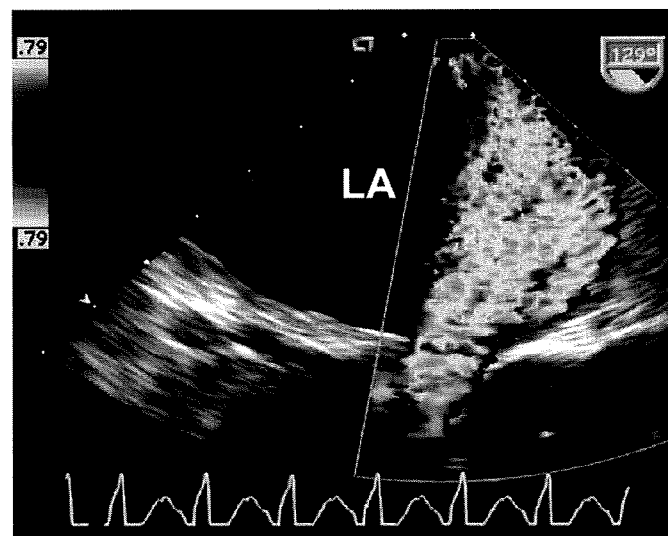
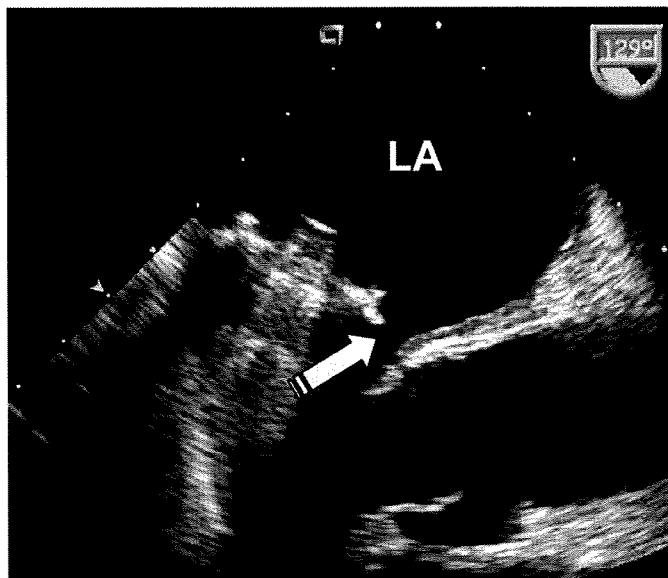


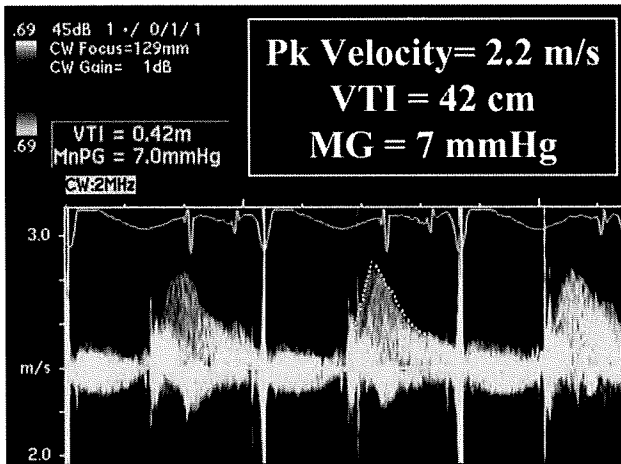
Figure 12 Transthoracic versus transesophageal echocardiographic and Doppler images in a patient with severe paravalvular MR. Shadowing on TTE of the left atrium (*arrows*) masked significantly the regurgitant jet by color Doppler (*single white arrow*). The extent of valvular dehiscence is shown by the *green arrow* on TEE as well as the severity of regurgitation by color Doppler. See Videos 13 to 16. [View video clips online.](#)

measurements that are elevated out of proportion to the actual measured gradient.^{18,136,137} For normally functioning bioprosthetic mitral valves, peak velocity can range from 1.0 to 2.7 m/s.^{138,139} In normally functioning bileaflet mechanical valves, the peak velocity is usually <1.9 m/s but can be up to 2.4 m/s.^{135,139,140} As a general rule, however, a peak velocity <1.9 m/s is likely to be normal in most patients with mechanical valves unless there is markedly depressed LV function. If the peak velocity is ≥ 1.9 m/s in a mechanical valve, one should consider a normally functioning valve with a high velocity versus prosthetic valve dysfunction (stenosis or regurgitation).^{135,139,140} This cut-off may be slightly higher in some bioprosthetic valves. If leaflet excursion is seen to be normal, there is no vegetation or thrombus, and there is no MR, it is likely the former. Because MR also increases transmitral flow velocity, patients with elevated peak E velocity may require TEE to exclude significant MR.

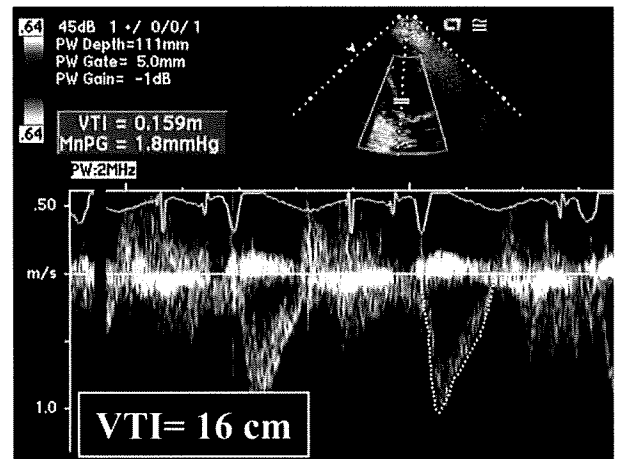
b. Mean Gradient. Mean gradient is also useful in assessing prosthetic mitral valve function and is normally <5 to 6 mm Hg.^{55,141} However, values up to 10 and 12 mm Hg have been reported in normally functioning Starr-Edwards and St Jude bileaflet prostheses, respectively,^{140,142} highlighting the need to compare serial values in the same patient over time. High mean gradients may be due to hyperdynamic states, tachycardia or PPM, regurgitation, or stenosis. The mean gradient is significantly affected by heart rate, so the heart rate at which the mean gradient is measured should always be reported.

c. Pressure Half-Time. The rate of blood flow across the mitral valve is dominated by the mitral orifice area in the presence of moderate or severe stenosis. However, when the mitral stenosis is only mild or there is a normally functioning valve, the rate of flow also

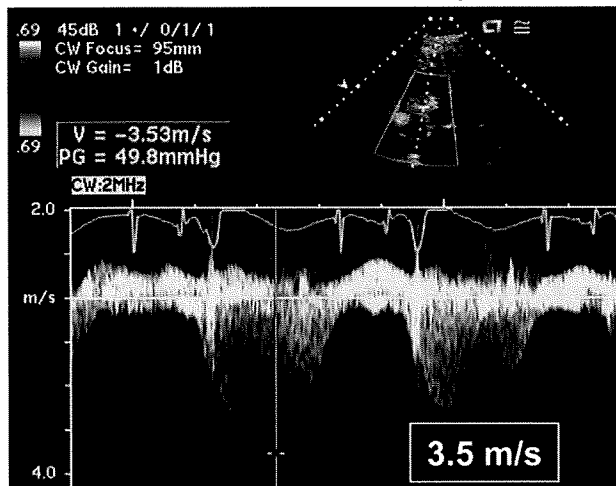
Prosthetic MV Jet



LVOT flow



TR Jet velocity



$$\frac{VTI_{PrMV}}{VTI_{LVO}} = \frac{42}{16} = 2.6$$

Figure 13 Transthoracic Doppler echocardiographic clues for significant mechanical MR. These recordings are for the same patient as in Figure 12. Peak early velocity, VTI of the jet, and mean gradient are higher than normal. In the presence of normal LV function, the VTI in the LVO tract is decreased with a resultant increase in the DVI. The TR jet velocity indicates pulmonary hypertension.

depends on atrial and ventricular compliance, ventricular relaxation, and the pressure difference at the start of diastole. Thus, a large rise in pressure half-time on serial studies or a markedly prolonged single measurement (>200 ms) may be a clue to the presence of prosthetic valve obstruction, because the pressure half-time seldom exceeds 130 ms across a normally functioning mitral valve prosthesis^{135,140} (Figure 14). However, minor changes in pressure half-time occur as a result of nonprosthetic factors, including loading conditions, drugs, or aortic insufficiency. Pressure half-time should not be obtained in tachycardic rhythms or first-degree atrioventricular block when E and A velocities are merged or the diastolic filling period is short.

d. EOA. Calculation of EOA from pressure half-time, as traditionally applied in native mitral stenosis, is not valid in prosthetic valves, because of its dependence on LV and LA compliance and initial LA pressure.^{34,55} Therefore, EOA calculation by the continuity equation is preferable to that measured by pressure half-time in mitral prostheses. In bileaflet valves, the smaller central orifice has a higher velocity than the larger outside orifices, which may lead to underestimation of EOA by the continuity equation.⁵⁵ Thus, the accuracy of EOA by the con-

tinuity equation may be better for bioprosthetic valves and single tilting disc mechanical valves. EOA is derived as stroke volume through the prosthesis divided by the VTI of the mitral jet velocity:

$$EOA_{PrMV} = \text{stroke volume} / VTI_{PrMV}$$

Stroke volume through the mitral valve is equated with that through the LVO when there is no significant MR or AR. Normative information on EOA and EOA indexed to body surface area is available for several types of prostheses in the mitral position.^{55,142-144} The use of effective areas is usually reserved for cases of discrepancy between information obtained from gradients and pressure half-time. Although derivation of EOA for prosthetic mitral valves is less often used, it is strongly advisable to note the VTI of the prosthetic valve, because it is much less dependent on heart rate compared with mean gradient. VTI of the prosthetic mitral jet is particularly useful in tachycardic and bradycardic states in which gradients may be misleading (high and low, respectively), and a derivation of EOA is readily obtained with the use of VTI_{PrMV} and an estimate of stroke volume (by echocardiography or Doppler).

Table 7 Echocardiographic and Doppler parameters in evaluation of prosthetic mitral valve function (stenosis or regurgitation)

Doppler echocardiography of the valve	Peak early velocity Mean gradient Heart rate at the time of Doppler Pressure half-time DVI*: VTI_{PrMV}/VTI_{LVO} EOA* Presence, location, and severity of regurgitation [†]
Other pertinent echocardiographic and Doppler parameters	LV size and function LA size [‡] RV size and function Estimation of pulmonary artery pressure

PrMV, Prosthetic mitral valve.

*These indices are used when further information is needed about valve function. EOA is calculated using the continuity equation.

[†]Often needs transeophageal echocardiographic evaluation because of acoustic shadowing.

[‡]May be difficult in the presence of shadowing or reverberation from the valve.

Recently, values of this ratio have been reported for a large number of patients with Carpentier-Edwards Duraflex bioprostheses (Edwards Lifesciences) and appear to be normally higher than for normal mechanical valves.¹⁴⁴

Because atrial fibrillation is frequent in patients with prosthetic mitral valves, close attention to matching cardiac cycles is crucial in the derivation of either EOA or the VTI_{PrMV}/VTI_{LVO} ratio, because both parameters are derived from different cardiac cycles. The preceding R-R interval to LVO velocity is matched to the R-R interval of mitral inflow velocity.

3. Diagnosis of Prosthetic Mitral Valve Stenosis. Significant valve obstruction may be obvious because of cusp thickening or reduced mobility. Failure of the color map to fill the orifice in all views is helpful if it is difficult to image the occluder. The initial impression will be corroborated by elevated peak E velocity and mean gradient, prolonged pressure half-time, and/or raised VTI_{PrMV}/VTI_{LVO} ratio. Table 8 lists the various Doppler parameters that are helpful in the evaluation of prosthetic mitral valve function, on the basis of available data from the literature and the consensus of the task force members. Cutoffs are probably different for bioprosthetic valves (Appendix B). When all parameters are normal, the probability of valve dysfunction is very low (0% stenosis, 2% regurgitation).¹³⁵ If the majority of

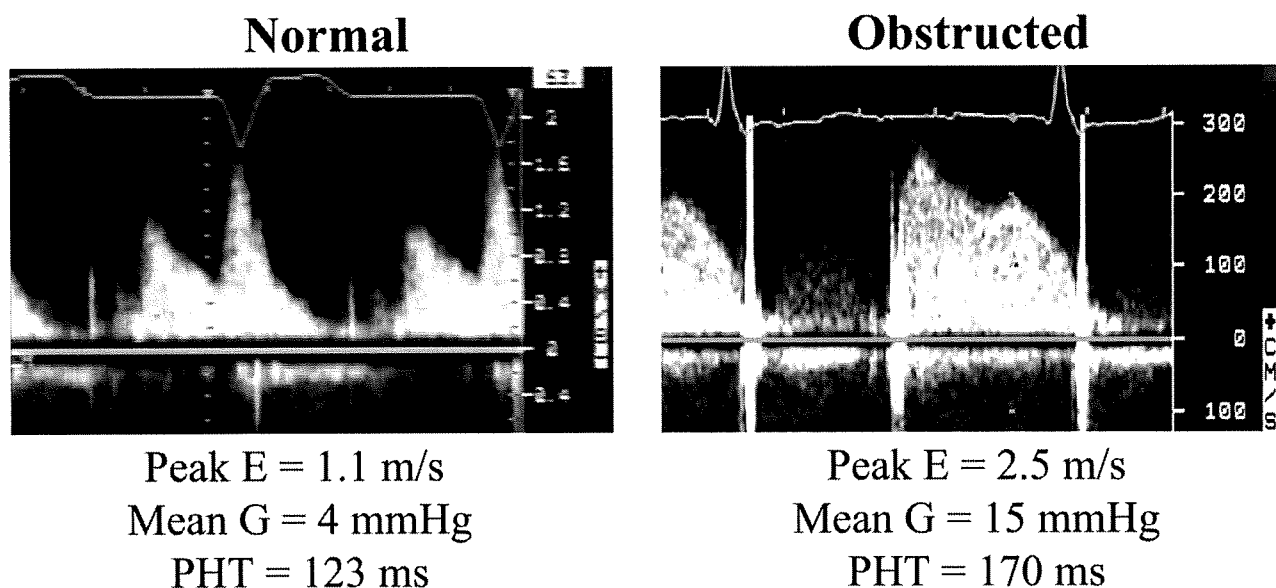


Figure 14 Doppler velocity patterns observed in a normal and an obstructed prosthetic valve in the mitral position. The velocity and gradients are elevated as well as pressure half-time in the obstructed valve.

e. DVI. Fernandes et al¹³⁵ proposed the use of the ratio of the VTIs of the mitral prosthesis to the LVO tract (VTI_{PrMV}/VTI_{LVO}) as an index of mechanical mitral prosthetic valve function. This DVI is the inverse of that proposed for prosthetic aortic valves (Figure 13). The concept is important in that elevated transmitral velocities can occur in the setting of prosthetic valve stenosis, regurgitation, or high output states. In high output states, the ratio is unchanged, because the increase in velocity occurs across both the LVO and the prosthetic valve. However, the VTI_{PrMV}/VTI_{LVO} ratio would be elevated either in stenosis (increased velocity across the valve) or regurgitation (increased velocity across the valve and decreased velocity in the LVO). In mechanical valves, a $VTI_{PrMV}/VTI_{LVO} < 2.2$ is most often normal.¹³⁵ Higher values should prompt consideration of prosthetic valve dysfunction.

parameters are abnormal, the predictive value for abnormal valvular function is 100%. An increased pressure half-time (or decreased EOA) in the presence of other abnormal parameters of elevated velocity and gradients points to valve stenosis as opposed to regurgitation.¹³⁵ When quantitative Doppler measures are of uncertain significance or somewhat discordant, one must determine whether the abnormal values reflect true prosthetic valve dysfunction or are altered despite normal prosthetic valve function because of situations such as high output states, tachycardia, or PPM. Thus, knowledge of the size of the implanted valve and its baseline Doppler parameters or previous TEE for serial comparison cannot be overemphasized. It is also important to examine the anatomy of the leaflets for normal excursion, vegetations, pannus, or thrombus, as well as to look for

Table 8 Doppler parameters of prosthetic mitral valve function

	Normal*	Possible stenosis [†]	Suggests significant stenosis* [‡]
Peak velocity (m/s) ^{† §}	<1.9	1.9-2.5	≥2.5
Mean gradient (mm Hg) ^{† §}	≤5	6-10	>10
VTI _{PrMV} /VTI _{LVO} ^{† §}	<2.2	2.2-2.5	>2.5
EOA (cm ²)	≥2.0	1-2	<1
PHT (ms)	<130	130-200	>200

PHT, Pressure half-time; PrMV, prosthetic mitral valve.

*Best specificity for normality or abnormality is seen if the majority of the parameters listed are normal or abnormal, respectively.

†Slightly higher cutoff values than shown may be seen in some bioprosthetic valves.

‡Values of the parameters should prompt a closer evaluation of valve function and/or other considerations such as increased flow, increased heart rate, or PPM.

§These parameters are also abnormal in the presence of significant prosthetic MR.

rocking or dehiscence of the sewing ring or a echo-free space adjacent to the sewing ring consistent with pseudoaneurysm or abscess.¹⁴⁵⁻¹⁴⁷ TEE should be considered because of its outstanding visualization of the structure and function of mitral valve prostheses of all types to make the diagnosis of obstruction when this is uncertain after TTE and in all cases to differentiate the possible causes of obstruction, particularly if thrombolysis is contemplated.⁸⁶

B. Prosthetic Mitral Valve Regurgitation

1. Imaging Considerations. Given that direct detection of prosthetic MR is often not possible with transthoracic Doppler techniques, particularly in mechanical valves, one must rely on indirect signs suggestive of significant MR on TTE (Table 9). These include a hyperdynamic LV with low systemic output, an elevated mitral E velocity, an elevated VTI_{PrMV}/VTI_{LVO} ratio, a dense CW regurgitant jet with early systolic maximal velocity, a large zone of systolic flow convergence toward the prosthesis seen in the LV, or a rise in pulmonary artery pressure compared with an earlier study¹⁴⁸ (Figure 13). Pressure half-time is often normal in prosthetic MR unless there is concomitant stenosis.^{135,148} Of the findings listed, the most accurate are those that reflect an increase in flow through the prosthesis (peak early velocity ≥ 1.9 m/s in mechanical valves, mean gradient ≥ 6 mm Hg), particularly when the high flow is not proportional to the flow ejected systemically (VTI_{PrMV}/VTI_{LVO} > 2.2).^{135,148} The presence of any of these findings in a patient with appropriate clinical symptoms represents a clear indication for TEE.

2. Role of TEE. TEE is highly sensitive and specific in detecting prosthetic MR and assessing its mechanism.¹⁴⁹⁻¹⁵¹ However, the assessment of severity is still achieved best by combining TEE with transthoracic examination. The sensitivity of TEE is high, such that the "built-in" trivial MR needs to be differentiated from pathologic MR. Paravalvular leaks on color Doppler have a typical appearance of a jet that passes from the left ventricle into the left atrium outside the surgical ring and often projects into the atrium in an eccentric direction (Figure 12). Because the regurgitation may be present anywhere around the circumference of the ring, it is essential that the valve be inspected in multiple planes. It is also crucial to show the origin of the jet as it passes through the area of dehiscence, its flow convergence and vena contracta. In patients with prosthetic valve endocarditis,

a perivalvular abscess may create a fistula between the left ventricle and left atrium that functions like a paravalvular leak. In addition to interrogation of the jet by CW Doppler, TEE allows a better recording of the pulmonary venous flow to corroborate the assessment of MR severity. Systolic flow reversal is specific for severe MR,¹⁶ provided that the MR jet is not directed into the interrogated vein.

3. Assessment of Severity of Prosthetic MR. Assessment of severity of prosthetic MR can be difficult at times because of the lack of a single quantitative parameter that can be applied consistently in all patients. The only methodology that one could apply to compute a regurgitant volume consists of deriving a total LV stroke volume by 2D echocardiography and subtracting from it the stroke volume across the LVO (or RV outflow). However, this method relies on an accurate determination of LV volumes by 2D echocardiography, and to date, it has not been properly validated. Three-dimensional echocardiography may facilitate this approach.¹⁵² Consequently, the best methodology at this time is to integrate several findings from both TTE and TEE that together suggest a given severity of regurgitation (Table 10).

A well preserved LV ejection fraction > 60% with normal size or enlarged left ventricle, along with a relative reduction in LVO or RV outflow stroke volume, should raise the possibility that the MR is significant. TEE is often needed to complete the assessment of prosthetic MR severity and complement the transthoracic echocardiographic findings. Distinction of mild from moderate or severe prosthetic MR is usually possible with the findings below. By contrast, it is more difficult to discriminate moderate from severe MR. As with native MR, regurgitant jet area reflects MR severity when the jets are central in origin, as seen with tissue valve degeneration, and works best at the extremes (ie, mild versus severe). A small thin jet (jet area < 4 cm²) in the left atrium usually reflects mild MR, while a large, wide jet (≥ 8 cm²) reflects a moderate or severe lesion.¹⁵³ Maximal width of the vena contracta is the index that best relates with angiographic assessment of prosthetic MR, particularly in paravalvular regurgitation; mild, moderate, and large leaks have been defined as widths of 1 to 2, 3 to 6, and ≥ 6 mm, respectively.¹⁵⁴ In a study involving 96 consecutive patients, 80% of patients with small (1-2 mm) regurgitation were asymptomatic, whereas 62% of those with large leaks were in New York Heart Association class III or IV.¹⁵⁵ As with native MR, the behavior of the jet in the left atrium, particularly significant swirling within the atrium, is specific for significant MR, as is the presence of retrograde systolic flow in one or more of the pulmonary veins. Likewise, the radius of the proximal flow convergence can be used in combination with recordings of MR velocity by CW Doppler to estimate effective regurgitant orifice area. However, because of the eccentric nature of many of these lesions, effective regurgitant orifice area is often overestimated; thus, the cutoff used to detect severe MR is ≥ 0.5 cm².¹⁵⁴ More studies are needed to further substantiate these observations. The more concordant the parameters are in their normality or abnormality, the more confident is the evaluation of the severity of regurgitation.

V. EVALUATION OF PROSTHETIC PULMONARY VALVES

A. Prosthetic Pulmonary Valve Function

1. Imaging Considerations. Because the pulmonary valve is located anteriorly and superiorly, it is often difficult to fully visualize by either TTE or TEE. Typically, the pulmonary valve can be visualized using the RV outflow tract view from the parasternal window (modified

Table 9 Transthoracic echocardiographic findings suggestive of significant prosthetic MR in mechanical valves with normal pressure half-time

Finding	Sensitivity	Specificity	Comments
Peak mitral velocity ≥ 1.9 m/s*	90%	89%	Also consider high flow, PPM
$VTI_{PrMV}/VTI_{LVO} \geq 2.5^*$	89%	91%	Measurement errors increase in atrial fibrillation due to difficulty in matching cardiac cycles; also consider PPM
Mean gradient ≥ 5 mmHg*	90%	70%	At physiologic heart rates; also consider high flow, PPM
Maximal TR jet velocity > 3 m/s*	80%	71%	Consider residual postoperative pulmonary hypertension or other causes
LV stroke volume derived by 2D or 3D imaging is $>30\%$ higher than systemic stroke volume by Doppler	Moderate sensitivity	Specific	Validation lacking; significant MR is suspected when LV function is normal or hyperdynamic and VTI_{LVO} is <16 cm
Systolic flow convergence seen in the left ventricle toward the prosthesis	Low sensitivity	Specific	Validation lacking; technically challenging to detect readily

PrMV, Prosthetic mitral valve.

*Data from Olmos et al.¹⁴⁸ When both peak velocity and VTI ratio are elevated with a normal pressure half-time, specificity is close to 100%.

Table 10 Echocardiographic and Doppler criteria for severity of prosthetic MR using findings from TTE and TEE

Parameter	Mild	Moderate	Severe
Structural parameters			
LV size	Normal*	Normal or dilated	Usually dilated [†]
Prosthetic valve	Usually normal	Abnormal [¶]	Abnormal [¶]
Doppler parameters			
Color flow jet area [#]	Small, central jet (usually <4 cm ² or $<20\%$ of LA area)	Variable	Large central jet (usually >8 cm ² or $>40\%$ of LA area) or variable size wall-impinging jet swirling in left atrium
Flow convergence**	None or minimal	Intermediate	Large
Jet density: CW Doppler	Incomplete or faint	Dense	Dense
Jet contour: CW Doppler	Parabolic	Usually parabolic	Early peaking, triangular
Pulmonary venous flow	Systolic dominance [§]	Systolic blunting [§]	Systolic flow reversal [†]
Quantitative parameters^{††}			
VC width (cm)	<0.3	0.3-0.59	≥ 0.6
R vol (mL/beat)	<30	30-59	≥ 60
RF (%)	<30	30-49	≥ 50
EROA (cm ²)	<0.20	0.20-0.49	≥ 0.50

EROA, Effective regurgitant orifice area; RF, regurgitant fraction; R vol, regurgitant volume; VC, vena contracta.

*LV size applied only to chronic lesions.

[†]Pulmonary venous systolic flow reversal is specific but not sensitive for severe MR.

[‡]In the absence of other etiologies of LV enlargement and acute MR.

[§]Unless other reasons for systolic blunting (eg, atrial fibrillation, elevated LA pressure).

^{||}Parameter may be best evaluated or obtained with TEE, particularly in mechanical valves.

[¶]Abnormal mechanical valves, for example, immobile occluder (valvular regurgitation), dehiscence or rocking (paravalvular regurgitation); abnormal biologic valves, for example, leaflet thickening or prolapse (valvular), dehiscence or rocking (paravalvular regurgitation).

[#]At a Nyquist limit of 50 to 60 cm/s.

**Minimal and large flow convergence defined as a flow convergence radius <0.4 and ≥ 0.9 cm for central jets, respectively, with a baseline shift at a Nyquist limit of 40 cm/s; cutoffs for eccentric jets may be higher.

^{††}These quantitative parameters are less well validated than in native MR.

from the parasternal short axis at the aortic level) or, in young patients, from the subcostal view. Unfortunately, the limited acoustic windows may limit the ability to fully assess prosthetic valve function.

Because the RV outflow is "funnel" shaped, it makes application of the continuity equation difficult; the RV outflow diameter changes dramatically as it approaches the pulmonary valve, making the accurate calculation of stroke volume in the RV outflow difficult. This compromises the accuracy of the calculation of EOA.

Branch pulmonary arterial stenosis is often associated with conditions that cause pulmonary valve stenosis. After replacement of the pulmonary valve, the branch stenosis may still be present and may interfere with the assessment of the replaced valve. For example, CW Doppler across the prosthetic valve may also record the peak velocity across the branch pulmonary artery stenosis. In this situation, PW Doppler may be a preferred method to assess the transprosthetic gradient rather than CW Doppler, especially because right-sided prosthetic valves often

have lower gradients. PW Doppler should be used only if aliasing does not occur.

Pulmonary valve replacements are sometimes inserted as pulmonary valve conduits. Although the valve may be functioning well without stenosis, the conduit may develop edge stenosis, again causing corruption of the CW Doppler signal. Thus, an elevated CW Doppler velocity should prompt further 2D visualization of the valve and conduit as well as PW Doppler interrogation to determine if branch pulmonary or conduit stenosis is present.

2. Evaluation of Pulmonary Valve Function. There is a paucity of data on prosthetic valves in the pulmonary position. Most of the data come from small sets of patients, mostly in the pediatric population with underlying congenital heart disease. Placement of the prosthetic valve and conduit in an anatomically aberrant position and the presence of RV structural abnormalities make standardization of velocities and gradients difficult. Evaluation of leaflet structure and mobility when feasible is helpful.

Suggested Doppler echocardiographic parameters for evaluation of prosthetic pulmonary valve function are presented in Table 11. Characterization of pulmonary valve prostheses is limited to pulmonary homograft valve conduits¹⁵⁶ or xenografts¹⁵⁷ in patients with congenital or systemic disease affecting the pulmonary valve or to cryopreserved pulmonary homografts in patients undergoing the Ross procedure.¹⁵⁸⁻¹⁶⁰ The types of xenografts used are variable (Carpentier-Edwards, Hancock, Ionescu-Shiley), with resultant variability in normal values. Reported parameters are limited to Doppler velocities and gradients; only a few studies have derived EOA.¹⁶¹

Findings that raise the question of prosthetic valve dysfunction are listed in Table 12 (Figure 15). In general, normal homografts have a peak velocity < 2.5 m/s (mean gradient < 15 mm Hg), and normal xenografts have a peak velocity < 3.2 m/s (mean gradient < 20 mm Hg). Reports on mechanical prostheses in the pulmonary position are limited, making general recommendations difficult.^{157,162,163} Another indirect approach to assess for the presence of valve stenosis that complements direct estimation of pulmonary valve gradient is to evaluate RV systolic pressure. If there is new RV systolic hypertension, prosthetic pulmonary stenosis should be considered and excluded. Direct visualization of the leaflets with full excursion and periodic, clinically indicated repeat echocardiography in the same patient for serial comparison remains the best method to rule out stenosis.

B. Prosthetic Pulmonary Valve Regurgitation

There are limited data regarding the echocardiographic assessment of prosthetic PR. In patients with severe PR, volume overload of the right ventricle is present, with resultant flattening of the interventricular septum in diastole and resultant paradoxical motion.^{164,165} With Doppler, the severity of regurgitation is usually subjectively graded, using an integrative approach similar to native PR,¹⁶ with few modifications particularly relating to the eccentricity of some regurgitant lesions (Table 13). The advantages and limitations of each of these parameters have been previously discussed in detail.¹⁶

Using color Doppler, the severity of PR is graded on the basis of the components of the jet, including regurgitant jet width, vena contracta, and its penetration depth into the RV outflow.¹⁶⁶⁻¹⁷³ Some authors have assessed the severity by jet width, in a manner analogous to that described for AR.¹⁷⁴ A thin narrow jet <25% of the pulmonary annulus is generally considered mild, and a wide jet >50% of the pulmonary annulus is severe. In paravalvular jets or other eccentric jets, however, these parameters become less reliable for assessment of PR severity and may underestimate the significance of the lesion. Rever-

Table 11 Imaging and Doppler parameters in evaluation of prosthetic pulmonary valve function

Doppler echocardiography of the valve	Peak velocity/peak gradient Mean gradient DVI* EOA* Presence, location, and severity of regurgitation
Related cardiac chambers	RV size, function, and hypertrophy; RV systolic pressure [†]

*Theoretically possible to calculate; few data exist.

†The RV dimensions are helpful only for patients who had normal right ventricles prior to valve replacement (ie, Ross procedure).

Table 12 Findings suspicious for prosthetic pulmonary valve stenosis

Cusp or leaflet thickening or immobility
Narrowing of forward color map
Peak velocity through the prosthesis >3 m/s or >2 m/s through a homograft*
Increase in peak velocity on serial studies [†]
Impaired RV function or elevated RV systolic pressure

*Suspicious but not diagnostic of stenosis.

†More reliable parameter.

sal of flow in the distal main pulmonary artery is indicative of at least moderate regurgitation.

Other supportive signs of PR severity include spectral Doppler recordings. A rapid deceleration rate of the jet velocity recording by CW or PW Doppler can be consistent with more severe regurgitation. However, this deceleration is also influenced by several factors, including RV diastolic properties.¹⁶ In severe PR, a rapid equalization of RV and pulmonary artery pressures can occur before the end of diastole. In this case, a "to and fro" flow signal in the shape of a sine wave, with termination of flow in mid to late diastole, can be seen. The density of the PR Doppler signal also reflects the regurgitation severity.¹⁶

Quantitative parameters of regurgitation severity can be used in principle, with derivation of regurgitant volume and regurgitant fraction from the difference between pulmonary and systemic flow.¹⁶ Although this measurement has not been validated for this purpose in PR, the concept is valid¹⁶ but may be difficult to apply in practice. Because of the presence of a pulmonary prosthesis, pulmonary stroke volume determination is performed best in the RV outflow, just proximal to the valve, and compared with flow at the aortic or mitral annulus. In general, a regurgitant fraction <30% is usually mild, whereas a regurgitant fraction >50% is severe.^{170,175} The RV outflow and pulmonary valve are anterior structures, offering a clear advantage of TTE in evaluating PR. Thus, although these structures can be visualized with TEE, the role of TEE in evaluating PR is limited.

VI. EVALUATION OF PROSTHETIC TRICUSPID VALVES

A. Prosthetic Tricuspid Valve Function

1. Imaging Considerations. The transthoracic approach allows a multitude of windows for visualization and flow interrogation of

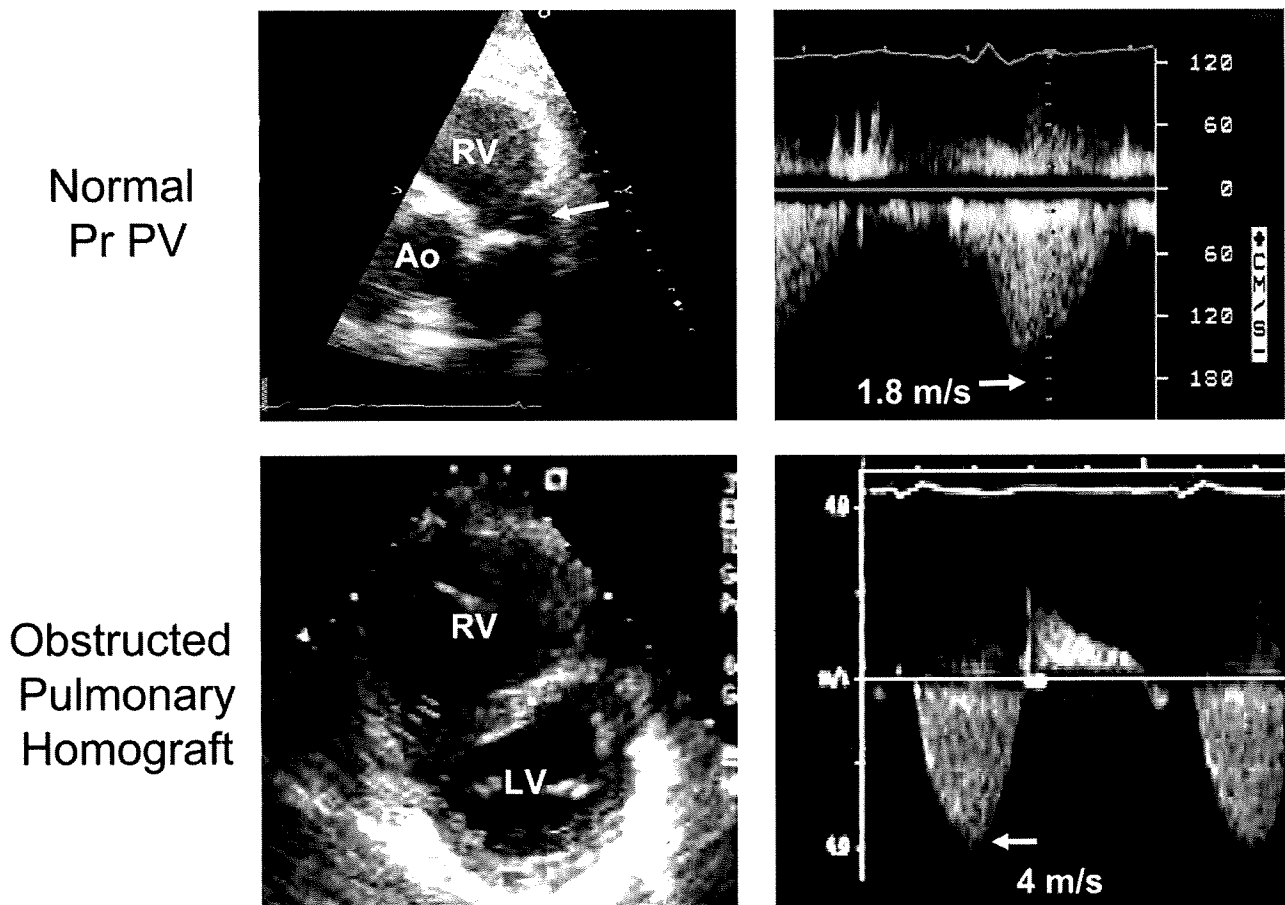


Figure 15 Examples of a normal prosthetic pulmonary valve and that of an obstructed pulmonary homograft showing dilatation of the right ventricle and a deformed septum. The obstructed homograft had a maximal gradient of 64 mm Hg.

prosthetic tricuspid valves. These include the parasternal, low parasternal, apical, and subcostal transducer positions. Forward flow hemodynamics are measured using CW Doppler from the various transducer positions to obtain the highest velocity measurements.

2. Doppler Parameters of Tricuspid Prosthetic Valve Function. The tricuspid prosthesis velocity varies not only with cycle length but also with respiration. Several cardiac cycles are therefore recorded by Doppler. A minimum of 5 cardiac cycles are averaged, whether the patient is in sinus rhythm or in atrial fibrillation; alternatively, measurements could be performed in midexpiratory apnea. Measurements include peak E velocity, peak A velocity (for patients in sinus rhythm), pressure half-time, mean gradient, and VTI. Similar to mitral prostheses, the average heart rate during the Doppler evaluation of the prosthesis should be noted in the report. When possible, and particularly when there is concern about valve obstruction, the prosthesis EOA can be calculated, although few data exist for the tricuspid valve. This is most often accomplished by dividing the stroke volume measured in the LVO tract by the prosthesis VTI, keeping in mind that the continuity principle is not in effect if there is more than mild TR or AR. If there is significant prosthetic TR, there is currently no convenient method for measuring the prosthesis EOA. In cases of significant AR without significant TR, the stroke volume can be measured at the level of the pulmonary annulus, because it represents the true systemic output. By analogy to mitral prostheses, it is likely that there is a cutoff for the DVI, the ratio of tricuspid pros-

thesis VTI divided by LVO tract VTI that, in combination with normal pressure half-time, will indicate a likelihood of significant tricuspid prosthesis regurgitation. However, to date, this cutoff has not been established in the literature. Of note, the EOA should not be calculated using the formula $220/\text{pressure half-time}$, because the constant of 220 has not been validated for tricuspid prostheses.

3. Diagnosis of Prosthetic Tricuspid Valve Stenosis. Echocardiographic and Doppler parameters that need to be obtained in the evaluation of prosthetic tricuspid valve function are listed in Table 14. Prosthetic tricuspid obstruction may be obvious on imaging from thickening and reduced opening of the biologic cusps or reduced opening of the mechanical occluder. A narrowed inflow color map is a helpful corroborative sign. Obstruction is also suggested on CW Doppler by an E velocity > 1.7 m/s, mean gradient > 6 mm Hg, or pressure half-time > 230 ms^{176,177} (Figure 16). Indirect, nonspecific signs are an enlarged right atrium and engorged inferior vena cava.

Suggested cutoffs of Doppler parameters for considering tricuspid prosthesis dysfunction are shown in Table 15. These cutoffs were selected using combined normal range data from the 3 pertinent published studies. These studies included a total of only 121 patients. Forty-seven patients had older-generation xenograft tricuspid prostheses, and 78 had mechanical prostheses with valve sizes ranging from 25 to 35 mm.¹⁷⁶⁻¹⁷⁸ It is anticipated that these cutoffs may change as larger series for newer model tricuspid valve prostheses emerge.

Table 13 Evaluation of severity of prosthetic pulmonary valve regurgitation

Parameter	Mild	Moderate	Severe
Valve structure	Usually normal	Abnormal or valve dehiscence	Abnormal or valve dehiscence
RV size	Normal*	Normal or dilated	Dilated [†]
Jet size by color Doppler (central jets) [‡]	Thin with a narrow origin; jet width $\leq 25\%$ of pulmonary annulus	Intermediate; jet width 26%-50% of pulmonary annulus	Usually large, with a wide origin; jet width $>50\%$ of pulmonary annulus; may be brief in duration
Jet density by CW Doppler	Incomplete or faint	Dense	Dense
Jet deceleration rate by CW Doppler	Slow deceleration	Variable deceleration	Steep deceleration [§] , early termination of diastolic flow
Pulmonary systolic flow vs systemic flow by PW Doppler [†]	Slightly increased	Intermediate	Greatly increased
Diastolic flow reversal in the pulmonary artery	None	Present	Present

Adapted from Zoghbi et al.¹⁶

*Unless other cause of RV dilatation exists, including residual postsurgical dilatation.

†Cutoff values for regurgitant volume and fraction are not well validated.

‡Unless there are other reasons for RV enlargement. Acute PR is an exception. RV volume overload is usually accompanied with typical paradoxical septal motion.

§Steep deceleration is not specific for severe PR.

||At a Nyquist limit of 50 to 60 cm/s; parameter applies to central jets and not eccentric jets.

For the 121 patients in the currently available series, the mean tricuspid E velocity was 1.3 ± 0.2 m/s, with all patients having E velocities of ≤ 1.7 m/s. For patients in sinus rhythm, the mean A velocity was 1.0 ± 0.3 m/s. The mean gradient for patients with normal St Jude Medical tricuspid prostheses was 2.7 ± 1.1 mm Hg. It was 3.2 ± 1.1 mm Hg for patients with normal xenograft tricuspid prostheses and 3.1 ± 0.8 mm Hg for those with normal caged-ball tricuspid prostheses. All 121 patients with normal tricuspid prostheses had mean gradients ≤ 5.5 mm Hg.

The mean pressure half-times for patients with normal xenograft prostheses (146 ± 39 ms) and normal caged-ball prostheses (144 ± 46 ms) were greater than that for patients with normal St Jude Medical tricuspid prostheses (108 ± 32 ms). All but 1 of these 121 patients had a pressure half-time < 200 ms (the single exception was a patient with a normal tricuspid caged-ball prosthesis with a pressure half-time of 230 ms).

To date, there are no data from a large series of patients with normal tricuspid prostheses that include measurement of EOA by the continuity equation or of the DVI (VTI_{PrTV}/VTI_{LVO}) akin to that of prosthetic mitral valves.

B. Prosthetic TR

Although several studies have addressed the issue of structural failure in tricuspid bioprostheses, the focus has been placed on increased gradients, with little mention of TR. Therefore, the present guidelines are based on expert recommendation rather than on data from clinical studies. The suggested criteria for assessing severity of prosthetic TR are proposed to be similar to those for native tricuspid valves,¹⁶ with few modifications (Table 16).

1. Imaging Considerations. A combination of parasternal, apical, and subcostal views is needed for the optimal assessment of tricuspid valve function and cardiac adaptation. In significant TR, right atrial and RV dilatation with diastolic septal flattening occurs in association with dilatation of the inferior vena cava and hepatic veins. The size of the cardiac chambers, however, should be interpreted with caution, because many if not all of these adaptations could be due to the underlying pathology and changes that occurred before tricuspid

Table 14 Echocardiographic and Doppler parameters in evaluation of prosthetic tricuspid valve function

Doppler echocardiography of the valve	Peak early velocity Mean gradient Heart rate at time of Doppler assessment Pressure half-time Pressure half-time VTI_{PrTV}/VTI_{LVO} * EOA* Presence, location, and severity of TR
Related cardiac chambers, inferior vena cava and hepatic veins	RV size and function Right atrial size Size of inferior vena cava and response to inspiration Hepatic vein flow pattern

PrTV, Prosthetic tricuspid valve.

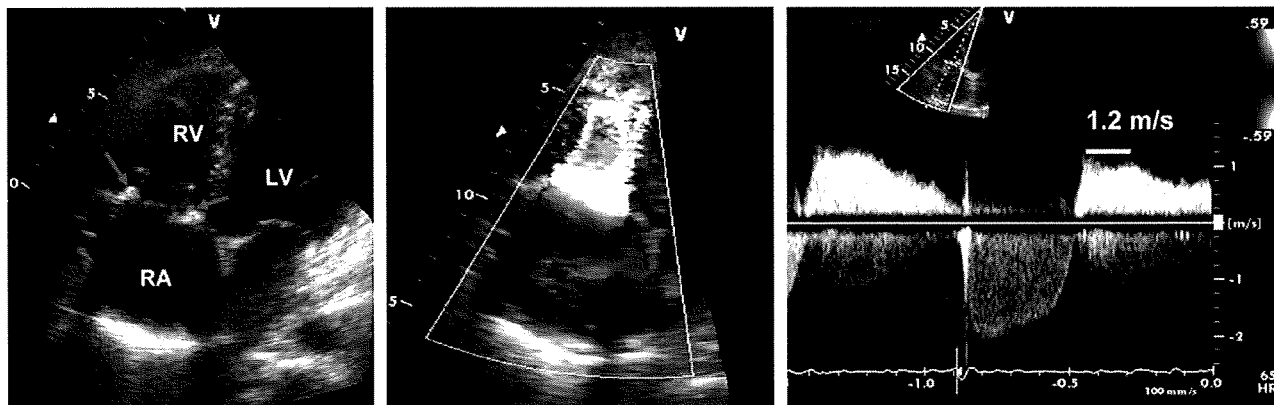
*Feasible measurements of valve function, similar to mitral prostheses, but no large series to date.

valve implantation. The absence of these findings, however, argues against severe TR.

2. Doppler Parameters of Tricuspid Prosthetic Valve Regurgitation. TTE with Doppler is a good screening test for TR but is limited by attenuation, particularly in patients with mechanical valves. The best views may be the RV inflow or subcostal views. Quantitative color Doppler techniques used in native valvular regurgitation have a limited role in prosthetic regurgitation. However they are part of the global examination of suspected severe TR. For instance, a large flow convergence or vena contracta usually means severe TR, and its location may help in assessing the origin of the regurgitation.

For spectral Doppler, screening with CW Doppler is better than PW Doppler. Both imaging and nonimaging CW Doppler probes should be used, the latter having a superior penetration. Clues from spectral CW Doppler that suggest severe regurgitation include a dense spectral recording with a triangular, early peaking velocity as well as elevated peak and mean tricuspid diastolic pressure gradients.

Normal Tricuspid Prosthetic Valve



Tricuspid Stenosis

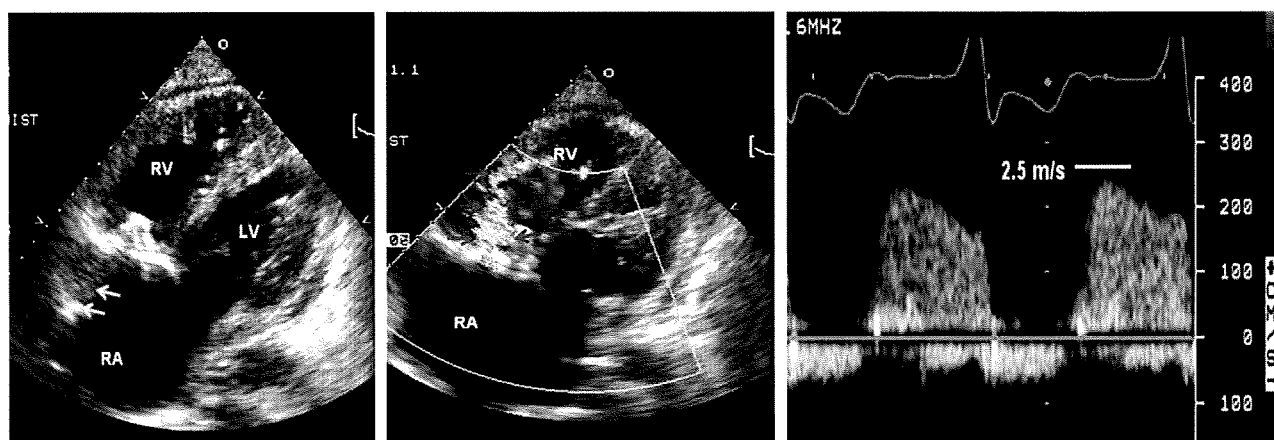


Figure 16 Transthoracic echocardiographic and Doppler images in a patient with normal prosthetic tricuspid valve and another with prosthetic stenosis. The case with normal prosthetic valve function had mild TR and a large central inflow jet in diastole. The patient with tricuspid valve stenosis had an eccentric narrow jet with an elevated velocity and mean gradient.

Table 15 Doppler parameters of prosthetic tricuspid valve function

	Consider valve stenosis*
Peak velocity†	>1.7 m/s
Mean gradient†	≥6 mm Hg
Pressure half-time	≥230 ms
EOA and VTI _{PrTV} /VTI _{LVO}	No data yet available for tricuspid prostheses

PrTV, Prosthetic tricuspid valve.

*Because of respiratory variation, average ≥5 cycles.

†May be increased also with valvular regurgitation.

The presence of a prosthetic valve with its inherent restriction to flow influences the flow pattern in the hepatic veins. Even when the prosthetic valve functions normally, some degree of systolic blunting can be expected. Marked systolic blunting is more sensitive for significant TR but is not specific and can be seen in patients with elevated central venous pressure of any etiology or in patients with atrial fibrillation. In general, holosystolic reversal of hepatic venous flow indicates severe TR.

3. TEE for Prosthetic Tricuspid Valves. TEE should be considered for all patients with clinical and/or transthoracic echocardiographic evidence of tricuspid prosthesis obstruction. The study focuses on delineating the motion of prosthetic leaflets or occluder and on identification of masses attached to the prosthesis. Imaging with TEE, however, may be technically suboptimal because of shadowing of the prosthesis from the interatrial septum or cardiac crux. Doppler angulation with the transesophageal echocardiographic approach may not be as favorable as with TTE, with a resultant underestimation of the velocity and gradient across the valve.

TEE should also be considered for patients with suspected prosthetic TR. The examination focuses on identifying the jet or jets as paravalvular or transvalvular. Semiquantitation of severity is performed by identifying the extent to which the color jet of regurgitation fills the right atrium and, when possible, evaluation of hepatic vein flow using a transgastric approach. If a zone of flow convergence can be identified on the ventricular side of the prosthesis and is not significantly distorted by adjacent structures, TR can be quantitated according to the proximal isovelocity surface area method.¹⁶ TEE should be considered as an adjunct to TTE for all patients with high clinical suspicion of endocarditis. Here the examination would focus not only on identification of vegetations but also on evidence of

Table 16 Echocardiographic and Doppler parameters used in grading severity of prosthetic tricuspid valve regurgitation

Parameter	Mild	Moderate	Severe
Valve structure	Usually normal	Abnormal or valve dehiscence	Abnormal or valve dehiscence
Jet area by color Doppler, central jets only (cm ²)	<5	5-10	>10
VC width (cm)*	Not defined	Not defined, but <0.7	>0.7
Jet density and contour by CW Doppler	Incomplete or faint, parabolic	Dense, variable contour	Dense with early peaking
Doppler systolic hepatic flow	Normal or blunted	Blunted	Holosystolic reversal
Right atrium, right ventricle, IVC	Normal [†]	Dilated	Markedly dilated

IVC, Inferior vena cava; VC, vena contracta.

Adapted from Zoghbi et al.¹⁶

*For a valvular TR jet, extrapolated from native TR; unknown cutoffs for paravalvular TR.

†If no other reason for dilatation.

perivalvular extension of the infection such as ring abscess, valve dehiscence, or fistula formation.

VII. ECHOCARDIOGRAPHIC EVALUATION OF PROSTHETIC VALVES IN THE PEDIATRIC POPULATION

Although the prevalence of prosthetic valves is much less common in the pediatric population, their presence has obvious implications for individual patients. To date, a paucity of published information exists concerning the appropriate evaluation of prosthetic valves in the young.¹⁷⁹⁻¹⁸¹ Equally important is the lack of studies detailing normal Doppler echocardiographic values across prosthetic valves in the pediatric population.¹⁷⁹ Hence, to date, much of the information is gleaned from the evaluation of adult patients with prosthetic valves. Fortunately, many of the important principles concerning hemodynamics, echocardiographic imaging, and other considerations are similar. Thus, the evaluation of prosthetic valve function, as discussed earlier, can and should be readily applied to the pediatric population. The following will not reiterate the principles of echocardiographic evaluation already discussed but will highlight the differences particularly germane to the pediatric population.

A. Prosthetic Valves Are Uncommon in Pediatrics

Prosthetic valve placement is naturally avoided in pediatric patients, the most important aspect being that growth of the patient will lead to inevitable PPM. Additionally, tissue valves, especially when placed in the systemic circulation, can result in rapid calcification and subsequent degeneration. Thus, before the early adult age group, metallic valves are generally used in the systemic circulation, with the inherent difficulties of anticoagulation in the pediatric age group. This had led to widespread use of the Ross procedure, especially for aortic valve replacement. The translocated pulmonary valve and root does not require anticoagulation, and studies have documented tissue growth, commensurate with that of the patient. However, the Ross procedure is associated with the need for early percutaneous or surgical interventions either for pulmonary conduit stenosis or regurgitation. Additionally, aortic root and annular dilation can occur, with associated progressive AR.

B. Aspects of Pediatric Congenital Heart Disease Alter the Standard Approach to Echocardiographic Prosthetic Valve Evaluation

Much emphasis has been placed on anatomic imaging of prosthetic valves in adult patients. However, even in young children, the echo-

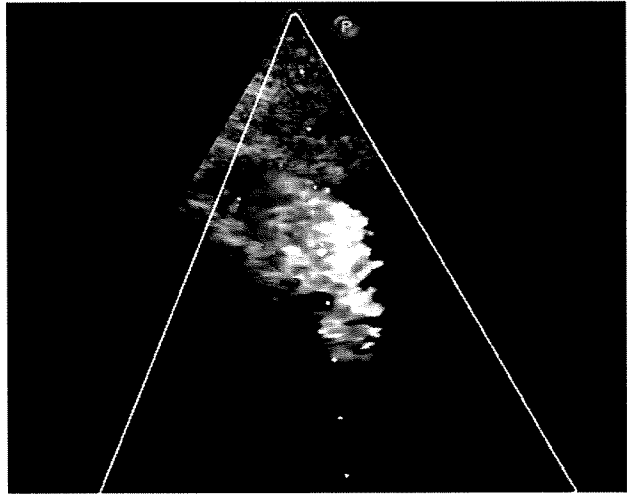
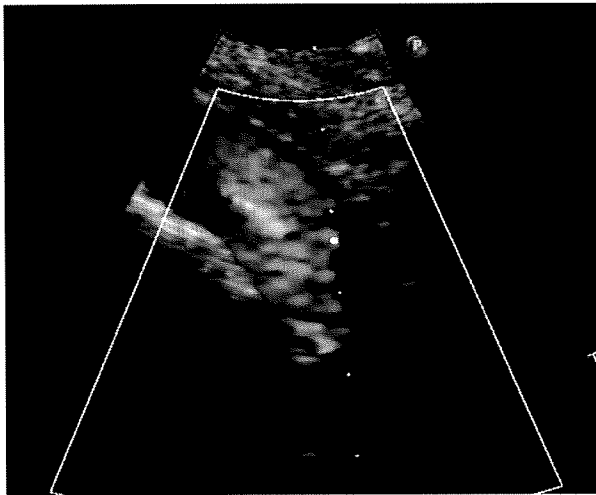
cardiographic images may be suboptimal because of multiple surgeries, chest wall deformities, and so on. In particular, imaging of the valve leaflets may be difficult to perform by 2D imaging. This is related in part to the low frame rates, in relation to higher heart rates in pediatric patients. M-mode echocardiography, with much higher frame rates, may yield some improvement in examining leaflet motion. Often, standard fluoroscopy is also used to evaluate metallic leaflet motion and position.

A significant contributor to the difficulty in echocardiographic evaluation of prosthetic valves in the pediatric age group is the coexistence of multiple levels of obstruction. For example, patients with Shone's syndrome may have supra-aortic mitral ring, parachute mitral valve, subaortic stenosis, bicuspid aortic valve, and aortic coarctation. If a prosthetic valve is placed in the aortic position, associated subaortic stenosis will not allow application of the continuity equation to determine EOA. Additionally, associated coarctation may directly affect the pressure gradients across a prosthetic aortic valve. On the right side of the heart, multiple levels of obstruction across a conduit, especially if stenosis extends to the right or left pulmonary artery, will directly affect pressure measurements across the valve.

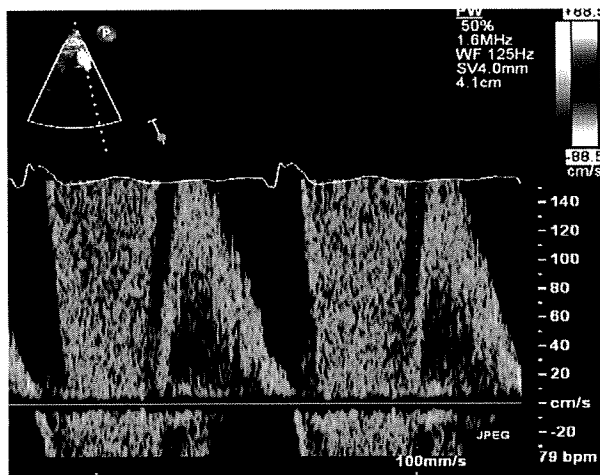
Another example of the differences between pediatric and adult patients relates to placement of prosthetic valves in the supra-annular position. In the rare event that an infant with a small mitral valve annulus requires placement of a prosthetic mitral valve, surgeons may choose to place the valve in the supra-annular position. This is associated with a significant elevation in LA mean pressure related to high "v" waves, even in the absence of valve dysfunction.¹⁸² Doppler recording in this situation will show an elevated transmitral E velocity and mean pressure gradient. This phenomenon has been attributed to alterations in atrial compliance.

The use of pulmonary artery conduits after repair of a multitude of congenital heart defects is much more common in pediatric patients (Figure 17). This includes neonatal repair of truncus arteriosus, tetralogy of Fallot with pulmonary atresia, and Rastelli operation for transposition of the great arteries with LVO tract obstruction. With advances in surgical techniques, RV-to-pulmonary artery conduits are being placed for the Sano revision of the stage I operation for hypoplastic left-heart syndrome or for the Yasui surgery for interrupted aortic arch. The increasing use of the Ross procedure in pediatric patients of all ages has resulted in increased numbers of these conduits. RV-to-pulmonary artery conduits are now being placed with increasing frequency in adolescents and young adults who have severe PR after primary repair of tetralogy of Fallot. Early reports had demonstrated that Doppler can accurately measure the gradients across RV-to-pulmonary conduits. Doppler maximal instantaneous gradients may closely approximate those obtained at catheterization

RV- PA Conduit



Pulsed Doppler



CW Doppler

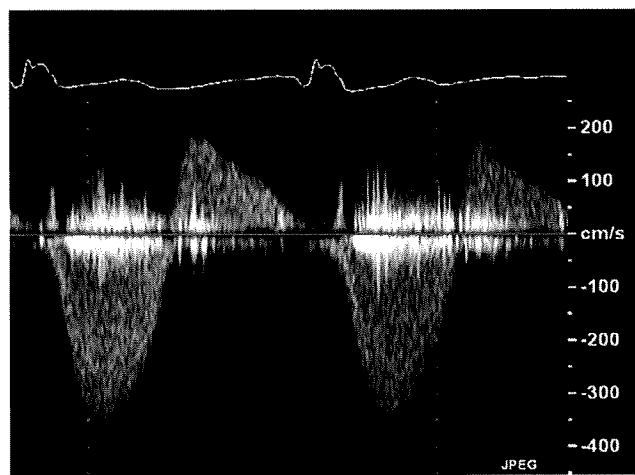


Figure 17 Two-dimensional echocardiographic evaluation of a RV-to-pulmonary artery homograft valve conduit after repair of a truncus arteriosus, interrogated from the left parasternal view. Forward flow (blue) and retrograde flow (red) of pulmonary regurgitation is seen. Pulsed Doppler shows aliasing signals of stenosis and a retrograde signal of regurgitation at the level of the valve. CW Doppler signal from the valve conduit shows significant stenosis and regurgitation.

when there is a discrete region of narrowing, such as calcified valve or discrete obstruction at the insertion site of the proximal conduit to the right ventricle. However, in other situations, Doppler may underestimate the severity of disease. This most often occurs when significant stenosis occurs at the conduit-to-pulmonary artery anastomosis. The jet lesions in these conduits are quite eccentric and difficult to interrogate, even with dedicated CW Doppler probes. However, in the presence of associated peripheral pulmonary stenosis, CW Doppler interrogation of these areas of obstruction may obfuscate the measurement of proximal obstruction. Moreover, these velocities often exceed the gradients at catheterization. In such situations, evaluation of the TR jet to assess RV pressure cannot be overemphasized.

C. Importance of PPM in Pediatrics

Mitral and or aortic valve stenosis in infants or children is associated with annular hypoplasia, often resulting in placement of a smaller prosthetic valve than would be appropriate for the patient's size.

This problem is magnified with pediatric patients' growth. Surgeons may attempt to enlarge the aortic annular region by either a Konno or Manugian procedure, yet still a suboptimal sized valve will be placed. When high velocities are interrogated across prosthetic valves in young patients, the algorithms as presented in this document should be applied. The cutoff values for the peak and mean velocities and gradients must be considered in the context of the patient's size; PPM and pressure recovery need to be taken into account.

A very significant contributor to progressive valvar obstruction in the pediatric age group is pannus formation. Moreover, severe left-sided obstruction is often associated with endocardial fibroelastosis, a fibrous scarring that may incite subsequent pannus formation.¹⁸³ The obstructive fibrous tissue may be difficult to image by standard transthoracic imaging. TEE may result in improved imaging, but the pannus may be difficult to differentiate from the actual sewing ring. On the other hand, thrombus formation may be an acute cause of a sudden increase in pressure gradients and/or development of

3 D Echocardiography

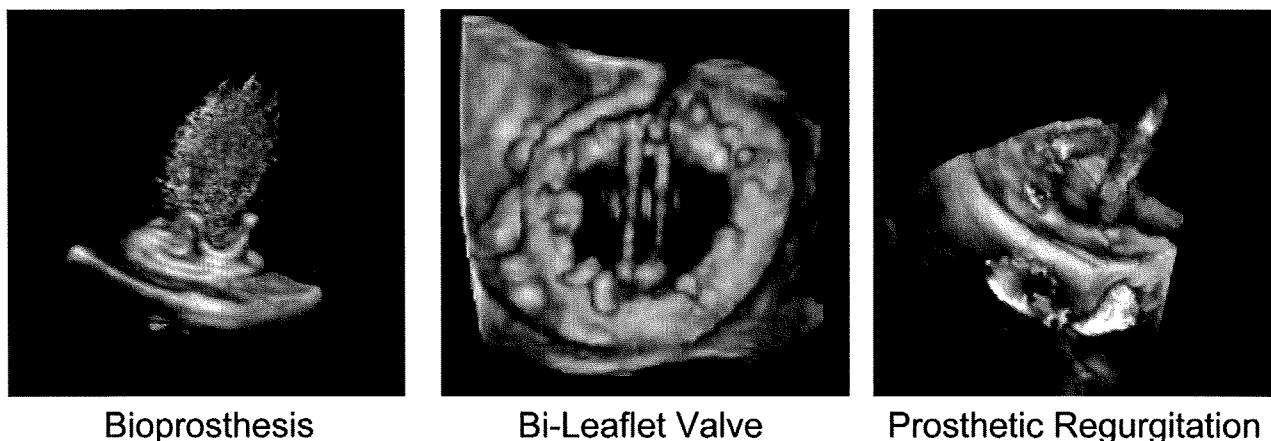


Figure 18 Examples of 3D echocardiography and Doppler images of a bioprosthetic valve (*left*), bileaflet mechanical valve in the mitral position from the LA view (Video 17, [video clips online](#)), and a prosthetic valve with regurgitation.

regurgitation. The valve may be fixed in position, culminating in prosthetic valve stenosis and regurgitation, which would be unlikely to be seen with either pannus formation or PPM.

D. Potential Pitfalls in the Measurement of Prosthetic Valve EOA in Pediatrics

The evaluation of prosthetic valve function often requires measurement of EOA. In pediatric patients, associated shunts will affect flow and hence pressure gradients. This includes the presence of an atrial septal defect, which potentially decreases flow across a prosthetic mitral and/or aortic valve, or a patent ductus arteriosus, which may increase flow. Equally important, PPM necessitates calculation of indexed EOA.

To date, EOA by the continuity equation has not been readily applied in the pediatric population. As mentioned, the most difficult aspect for accurate derivation of EOA has been the determination of the preprosthetic valve VTI and area. Several factors make this measurement more difficult in pediatric patients. First, the area is presumed to be circular. This may not be true in patients with associated LVO tract or RV outflow tract disease. Second, for area measurement, the radius is raised to the second power, and thus even small discrepancies in diameter measurement will result in large errors in area calculation. This potential error will be magnified in smaller patients. Third, the preobstruction flow velocity pattern will not be laminar in pediatric patients with subaortic or subpulmonary stenosis. Fourth, translational motion of the heart impedes the ability to place the Doppler sample volume precisely in the area of the preprosthetic valve. This problem again seems to be more evident in younger pediatric patients.

A few studies in small numbers of patients have reported on the optimal manner to determine the presence of PPM in pediatric patients. One study examined 32 infants and children with placement of prosthetic St Jude Medical or Carbomedics valves in the mitral position. The Doppler measurement that correlated best to the manufacturer-derived EOA was the peak Doppler velocity, not the EOA. Potentially, some of the problems of the calculation of EOA may be overcome by use of the DVI. The index has been related to severity of disease in adult patients, but application of this index has not been reported in pediatric patients. Despite the potential pitfalls,

attempts to use EOA and/or DVI should become part of the standard evaluation of pediatric patients with prosthetic valves.

E. Evaluation of Corresponding Atrial and Ventricular Size and Function

Equally important to the evaluation of prosthetic valve function is the analysis of the corresponding effects on atrial and ventricular size and function. In pediatric patients, normative values for 2D echocardiographic evaluations for LV size and function, using z-score values, have been developed. These values can be tracked over time, therefore incorporating changes relative to growth and development. Unfortunately, echocardiographic normative values for RV volume and mass or left or right atrial volumes have not been established but are under way. Indices of ventricular systolic performance should be used to account for loading conditions. Such echocardiographic parameters include indices of wall stress to velocity of circumferential fiber shortening or wall stress to fractional shortening. Although echocardiographic evaluation of prosthetic valve function includes indices of ventricular systolic performance, evaluation of ventricular diastolic dysfunction should also be explored.

F. The Need for More Research in the Pediatric Population

Perhaps the most difficult aspect concerning the evaluation of prosthetic valves in pediatric patients is the paucity of published data. The deleterious outcomes related to PPM in adults have been demonstrated, but the effect of PPM is unknown in pediatric patients. Few studies have reported on the outcomes of pediatric patients with prosthetic valves. Therefore, pediatric cardiologists rely on guidelines established for adult patients. The importance for additional research and analysis in a large number of pediatric patients cannot be overemphasized.

VIII. CONCLUSIONS AND FUTURE DIRECTIONS

Echocardiography with Doppler is currently the modality of choice for evaluation and management of prosthetic heart valves as well as native cardiac valves. Imaging of the prosthesis in addition to the related cardiac chambers is crucial in evaluating overall prosthetic valve function and assessment of the extent of reverse remodeling of the

cardiac chambers after surgery. Doppler interrogation with color and spectral modalities plays a central role in evaluating prosthetic valve function and related complications because of limitations of imaging alone, particularly in mechanical valves. In patients with suspected prosthetic valvular dysfunction, TEE is frequently needed for identification of the mechanism of obstruction or regurgitation, particularly in mechanical valves.

In general, evaluation of prosthetic valve function is more challenging, on the basis of the variability of inherent mild obstruction observed with the wide range of prosthetic valve types and sizes. Thus, the cardiac history plays a major role in the echocardiographic evaluation by documenting the type and size of the inserted valve or conduit. Serial comparison with a baseline postoperative study is also essential in facilitating accurate assessment of valve function. More research is needed on normative values of various parameters of valvular function and their prognostic impact in the pediatric population.

Recent advances in real-time 3D imaging from the transthoracic and, more important, from the transesophageal approach offer an important additional dimension in the echocardiographic evaluation of prosthetic valve function¹⁸⁴ (Figure 18). Three-dimensional imaging provides a powerful tool to image, for the first time with ultrasound, the motion of the entire valve apparatus and its annulus. This will undoubtedly enhance our appraisal of prosthetic valve function and the differentiation of PPM from valvular obstruction in the same setting. The incorporation of color Doppler will enhance measurements of flow convergence, vena contracta, and the extent of the jet in the receiving chamber for improved quantitation of prosthetic valvular regurgitation. Furthermore, preliminary experience¹⁸⁵ has shown that future applications of real-time 3D TEE will most likely include guidance of percutaneous interventions in high-risk patients with para-valvular regurgitation.

Supplementary data

Supplementary data associated with this article can be found in the online version, at doi:10.1016/j.echo.2009.07.013

REFERENCES

1. Bonow RO, Carabello BA, Kanu C, et al. ACC/AHA 2006 guidelines for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 1998 Guidelines for the Management of Patients With Valvular Heart Disease): developed in collaboration with the Society of Cardiovascular Anesthesiologists: endorsed by the Society for Cardiovascular Angiography and Interventions and the Society of Thoracic Surgeons. *Circulation* 2006;114:e84-231.
2. Jamieson WR. Current and advanced prostheses for cardiac valvular replacement and reconstruction surgery. *Surg Technol Int* 2002;10:121-49.
3. Christakis GT, Butth KJ, Goldman BS, et al. Inaccurate and misleading valve sizing: a proposed standard for valve size nomenclature. *Ann Thorac Surg* 1998;66:1198-203.
4. Chambers JB, Oo L, Narracott A, Lawford PM, Blauth CI. Nominal size in six bileaflet mechanical aortic valves: a comparison of orifice size and biologic equivalence. *J Thorac Cardiovasc Surg* 2003;125:1388-93.
5. Grube E, Schuler G, Buellesfeld L, et al. Percutaneous aortic valve replacement for severe aortic stenosis in high-risk patients using the second- and current third-generation self-expanding CoreValve prosthesis: device success and 30-day clinical outcome. *J Am Coll Cardiol* 2007;50:69-76.
6. Khambadkone S, Coats L, Taylor A, et al. Percutaneous pulmonary valve implantation in humans: results in 59 consecutive patients. *Circulation* 2005;112:1189-97.
7. Webb JG, Chandavimol M, Thompson CR, et al. Percutaneous aortic valve implantation retrograde from the femoral artery. *Circulation* 2006;113:842-50.
8. Moss R, Ivens E, Pasupati S, et al. Role of echocardiography in percutaneous aortic valve implantation. *J Am Coll Cardiol Cardiovasc Imaging* 2008;1:15-24.
9. Gammie JS, Brown JW, Brown JM, et al. Aortic valve bypass for the high-risk patient with aortic stenosis. *Ann Thorac Surg* 2006;81:1605-10.
10. Lang RM, Bierig M, Devereux RB, et al. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. *J Am Soc Echocardiogr* 2005;18:1440-63.
11. Alam M, Goldstein S, Lakier JB. Echocardiographic changes in the thickness of porcine valves with time. *Chest* 1981;79:663-8.
12. Effron MK, Popp RL. Two-dimensional echocardiographic assessment of bioprosthetic valve dysfunction and infective endocarditis. *J Am Coll Cardiol* 1983;2:597-606.
13. Del Rizzo DF, Goldman BS, Christakis GT, David TE. Hemodynamic benefits of the Toronto stentless valve. *J Thorac Cardiovasc Surg* 1996;112:1431-45.
14. Walther T, Autschbach R, Falk V, et al. The stentless Toronto SPV bioprosthesis for aortic valve replacement. *Cardiovasc Surg* 1996;4:536-42.
15. Quinones MA, Otto CM, Stoddard M, Waggoner A, Zoghbi WA. Recommendations for quantification of Doppler echocardiography: a report from the Doppler Quantification Task Force of the Nomenclature and Standards Committee of the American Society of Echocardiography. *J Am Soc Echocardiogr* 2002;15:167-84.
16. Zoghbi WA, Enriquez-Sarano M, Foster E, et al. Recommendations for evaluation of the severity of native valvular regurgitation with two-dimensional and Doppler echocardiography. *J Am Soc Echocardiogr* 2003;16:777-802.
17. Burstow DJ, Nishimura RA, Bailey KR, et al. Continuous wave Doppler echocardiographic measurement of prosthetic valve gradients. A simultaneous Doppler-catheter correlative study. *Circulation* 1989;80:504-14.
18. Baumgartner H, Khan S, DeRobertis M, Czer L, Maurer G. Discrepancies between Doppler and catheter gradients in aortic prosthetic valves in vitro. A manifestation of localized gradients and pressure recovery. *Circulation* 1990;82:1467-75.
19. Baumgartner H, Khan S, DeRobertis M, Czer L, Maurer G. Effect of prosthetic aortic valve design on the Doppler-catheter gradient correlation: an in vitro study of normal St. Jude, Medtronic-Hall, Starr-Edwards and Hancock valves. *J Am Coll Cardiol* 1992;19:324-32.
20. Baumgartner H, Schima H, Tulzer G, Kuhn P. Effect of stenosis geometry on the Doppler-catheter gradient relation in vitro: a manifestation of pressure recovery. *J Am Coll Cardiol* 1993;21:1018-25.
21. Baumgartner H, Stefanelli T, Niederberger J, Schima H, Maurer G. "Overestimation" of catheter gradients by Doppler ultrasound in patients with aortic stenosis: a predictable manifestation of pressure recovery. *J Am Coll Cardiol* 1999;33:1655-61.
22. Chafizadeh ER, Zoghbi WA. Doppler echocardiographic assessment of the St. Jude Medical prosthetic valve in the aortic position using the continuity equation. *Circulation* 1991;83:213-23.
23. Assey ME, Zile MR, Usher BW, Karavan MP, Carabello BA. Effect of catheter positioning on the variability of measured gradient in aortic stenosis. *Cathet Cardiovasc Diagn* 1993;30:287-92.
24. Chambers JB, Spriggs DC, Cochrane T, et al. Continuity equation and Gorlin formula compared with directly observed orifice area in native and prosthetic aortic valves. *Br Heart J* 1992;67:193-9.
25. Garcia D, Pibarot P, Dumesnil JG, Sakr F, Durand LG. Assessment of aortic valve stenosis severity: a new index based on the energy loss concept. *Circulation* 2000;101:765-71.
26. Heinrich RS, Fontaine AA, Grimes RY, et al. Experimental analysis of fluid mechanical energy losses in aortic valve stenosis: importance of pressure recovery. *Ann Biomed Eng* 1996;24:685-94.

27. Heinrich RS, Marcus RH, Ensley AE, Gibson DE, Yoganathan AP. Valve orifice area alone is an insufficient index of aortic stenosis severity: effects of the proximal and distal geometry on transaortic energy loss. *J Heart Valve Dis* 1999;8:509-15.
28. Levine RA, Jimoh A, Cape EG, McMillan S, Yoganathan AP, Weyman AE. Pressure recovery distal to a stenosis: potential cause of gradient "overestimation" by Doppler echocardiography. *J Am Coll Cardiol* 1989;13:706-15.
29. Levine RA, Cape EG, Yoganathan AP. Pressure recovery distal to stenoses: expanding clinical applications of engineering principles. *J Am Coll Cardiol* 1993;21:1026-8.
30. Levine RA, Schwammenthal E. Stenosis is in the eye of the observer: impact of pressure recovery on assessing aortic valve area. *J Am Coll Cardiol* 2003;41:443-5.
31. Niederberger J, Schima H, Maurer G, Baumgartner H. Importance of pressure recovery for the assessment of aortic stenosis by Doppler ultrasound. Role of aortic size, aortic valve area, and direction of the stenotic jet in vitro. *Circulation* 1996;94:1934-40.
32. Marcus RH, Heinrich RS, Bednarz J, et al. Assessment of small-diameter aortic mechanical prostheses: physiological relevance of the Doppler gradient, utility of flow augmentation, and limitations of orifice area estimation. *Circulation* 1998;98:866-72.
33. Dumesnil JG, Honos GN, Lemieux M, Beauchemin J. Validation and applications of indexed aortic prosthetic valve areas calculated by Doppler echocardiography. *J Am Coll Cardiol* 1990;16:637-43.
34. Dumesnil JG, Honos GN, Lemieux M, Beauchemin J. Validation and applications of mitral prosthetic valvular areas calculated by Doppler echocardiography. *Am J Cardiol* 1990;65:1443-8.
35. Dumesnil JG, Yoganathan AP. Valve prosthesis hemodynamics and the problem of high transprosthetic pressure gradients. *Eur J Cardiothorac Surg* 1992;6(suppl):S34-7.
36. Pibarot P, Dumesnil JG. Hemodynamic and clinical impact of prosthesis-patient mismatch in the aortic valve position and its prevention. *J Am Coll Cardiol* 2000;36:1131-41.
37. Hanayama N, Christakis GT, Mallidi HR, et al. Patient prosthesis mismatch is rare after aortic valve replacement: valve size may be irrelevant. *Ann Thorac Surg* 2002;73:1822-9.
38. Blais C, Dumesnil JG, Baillet R, Simard S, Doyle D, Pibarot P. Impact of valve prosthesis-patient mismatch on short-term mortality after aortic valve replacement. *Circulation* 2003;108:983-8.
39. Milano AD, De CM, Mecozzi G, et al. Clinical outcome in patients with 19-mm and 21-mm St. Jude aortic prostheses: comparison at long-term follow-up. *Ann Thorac Surg* 2002;73:37-43.
40. Tasca C, Brunelli F, Cirillo M, et al. Impact of valve prosthesis-patient mismatch on left ventricular mass regression following aortic valve replacement. *Ann Thorac Surg* 2005;79:505-10.
41. Blackstone EH, Cosgrove DM, Jamieson WR, et al. Prosthesis size and long-term survival after aortic valve replacement. *J Thorac Cardiovasc Surg* 2003;126:783-96.
42. Koch CG, Khandwala F, Estafanos FG, Loop FD, Blackstone EH. Impact of prosthesis-patient size on functional recovery after aortic valve replacement. *Circulation* 2005;111:3221-9.
43. Dumesnil JG, Pibarot P. Prosthesis-patient mismatch and clinical outcomes: the evidence continues to accumulate. *J Thorac Cardiovasc Surg* 2006;131:952-5.
44. Pibarot P, Dumesnil JG. Prosthesis-patient mismatch: definition, clinical impact, and prevention. *Heart* 2006;92:1022-9.
45. Mohty D, Malouf JF, Girard SE, et al. Impact of prosthesis-patient mismatch on long-term survival in patients with small St Jude Medical mechanical prostheses in the aortic position. *Circulation* 2006;113:420-6.
46. Ruel M, Al-Faleh H, Kulik A, Chan KL, Mesana TC, Burwash IG. Prosthesis-patient mismatch after aortic valve replacement predominantly affects patients with preexisting left ventricular dysfunction: effect on survival, freedom from heart failure, and left ventricular mass regression. *J Thorac Cardiovasc Surg* 2006;131:1036-44.
47. Pibarot P, Dumesnil JG, Lemieux M, Cartier P, Metras J, Durand LG. Impact of prosthesis-patient mismatch on hemodynamic and symptomatic status, morbidity and mortality after aortic valve replacement with a bio-prosthetic heart valve. *J Heart Valve Dis* 1998;7:211-8.
48. Castro LJ, Arcidi JM Jr, Fisher AL, Gaudiani VA. Routine enlargement of the small aortic root: a preventive strategy to minimize mismatch. *Ann Thorac Surg* 2002;74:31-6.
49. Pibarot P, Dumesnil JG, Cartier PC, Metras J, Lemieux MD. Patient-prosthesis mismatch can be predicted at the time of operation. *Ann Thorac Surg* 2001;71:S265-8.
50. Rahimtoola SH, Murphy E. Valve prosthesis-patient mismatch. A long-term sequela. *Br Heart J* 1981;45:331-5.
51. Lam BK, Chan V, Hendry P, et al. The impact of patient-prosthesis mismatch on late outcomes after mitral valve replacement. *J Thorac Cardiovasc Surg* 2007;133:1464-73.
52. Li M, Dumesnil JG, Mathieu P, Pibarot P. Impact of valve prosthesis-patient mismatch on pulmonary arterial pressure after mitral valve replacement. *J Am Coll Cardiol* 2005;45:1034-40.
53. Magne J, Mathieu P, Dumesnil JG, et al. Impact of prosthesis-patient mismatch on survival after mitral valve replacement. *Circulation* 2007;115:1417-25.
54. Mohty D, Dumesnil JG, Echahidi N, et al. Impact of prosthesis-patient mismatch on long-term survival after aortic valve replacement: influence of age, obesity, and left ventricular dysfunction. *J Am Coll Cardiol* 2009;53:39-47.
55. Bitar JN, Lechin ME, Salazar G, Zoghbi WA. Doppler echocardiographic assessment with the continuity equation of St. Jude Medical mechanical prostheses in the mitral valve position. *Am J Cardiol* 1995;76:287-93.
56. Ionescu A, Fraser AG, Butchart EG. Prevalence and clinical significance of incidental paraprosthetic valvar regurgitation: a prospective study using transoesophageal echocardiography. *Heart* 2003;89:1316-21.
57. O'Rourke DJ, Palac RT, Malenka DJ, Marrin CA, Arbuckle BE, Plehn JF. Outcome of mild periprosthetic regurgitation detected by intraoperative transesophageal echocardiography. *J Am Coll Cardiol* 2001;38:163-6.
58. Practice guidelines for perioperative transesophageal echocardiography. A report by the American Society of Anesthesiologists and the Society of Cardiovascular Anesthesiologists Task Force on Transesophageal Echocardiography. *Anesthesiology* 1996;84:986-1006.
59. Grewal KS, Malkowski MJ, Piracha AR, et al. Effect of general anesthesia on the severity of mitral regurgitation by transesophageal echocardiography. *Am J Cardiol* 2000;85:199-203.
60. Kubitz JC, Annecke T, Kemming GI, et al. The influence of positive end-expiratory pressure on stroke volume variation and central blood volume during open and closed chest conditions. *Eur J Cardiothorac Surg* 2006;30:90-5.
61. McKinlay KH, Schinderle DB, Swaminathan M, et al. Predictors of inotropes use during separation from cardiopulmonary bypass. *J Cardiothorac Vasc Anesth* 2004;18:404-8.
62. Schroeder RA, Mark JB. Is the valve OK or not? Immediate evaluation of a replaced aortic valve. *Anesth Analg* 2005;101:1288-91.
63. Reeves ST, Glas KE, Eltzhig H, et al. Guidelines for performing a comprehensive epicardial echocardiography examination: recommendations of the American Society of Echocardiography and the Society of Cardiovascular Anesthesiologists. *J Am Soc Echocardiogr* 2007;20:427-37.
64. Rizzoli G, Guglielmi C, Toscano G, et al. Reoperations for acute prosthetic thrombosis and pannus: an assessment of rates, relationship and risk. *Eur J Cardiothorac Surg* 1999;16:74-80.
65. Doenst T, Borger MA, David TE. Long-term results of bioprosthetic mitral valve replacement: the pericardial perspective. *J Cardiovasc Surg (Torino)* 2004;45:449-54.
66. Kilian E, Oberhoffer M, Gulbins H, Uhlig A, Kreuzer E, Reichart B. Ten years' experience in aortic valve replacement with homografts in 389 cases. *J Heart Valve Dis* 2004;13:554-9.
67. Zoghbi WA. Echocardiographic recognition of unusual complications after surgery on the great vessels and cardiac valves. In: Otto CM, editor. *The practice of clinical echocardiography*. New York: Elsevier; 2007: 605-26.
68. Birmingham GD, Rahko PS, Ballantyne F III. Improved detection of infective endocarditis with transesophageal echocardiography. *Am Heart J* 1992;123:774-81.

69. Daniel WG, Mugge A, Grote J, et al. Comparison of transthoracic and transesophageal echocardiography for detection of abnormalities of prosthetic and bioprosthetic valves in the mitral and aortic positions. *Am J Cardiol* 1993;71:210-5.
70. Karalis DG, Bansal RC, Hauck AJ, et al. Transesophageal echocardiographic recognition of subaortic complications in aortic valve endocarditis. Clinical and surgical implications. *Circulation* 1992;86:353-62.
71. Afridi I, Apostolidou MA, Saad RM, Zoghbi WA. Pseudoaneurysms of the mitral-aortic intervalvular fibrosa: dynamic characterization using transesophageal echocardiographic and Doppler techniques. *J Am Coll Cardiol* 1995;25:137-45.
72. Daniel WG, Mugge A, Martin RP, et al. Improvement in the diagnosis of abscesses associated with endocarditis by transesophageal echocardiography. *N Engl J Med* 1991;324:795-800.
73. Mugge A. Echocardiographic detection of cardiac valve vegetations and prognostic implications. *Infect Dis Clin North Am* 1993;7:877-98.
74. Baddour LM, Wilson WR, Bayer AS, et al. Infective endocarditis: diagnosis, antimicrobial therapy, and management of complications: a statement for healthcare professionals from the Committee on Rheumatic Fever, Endocarditis, and Kawasaki Disease, Council on Cardiovascular Disease in the Young, and the Councils on Clinical Cardiology, Stroke, and Cardiovascular Surgery and Anesthesia, American Heart Association: endorsed by the Infectious Diseases Society of America. *Circulation* 2005;111:e394-434.
75. Lowry RW, Zoghbi WA, Baker WB, Wray RA, Quinones MA. Clinical impact of transesophageal echocardiography in the diagnosis and management of infective endocarditis. *Am J Cardiol* 1994;73:1089-91.
76. Ali AS, Trivedi V, Lesch M. Culture-negative endocarditis—a historical review and 1990s update. *Prog Cardiovasc Dis* 1994;37:149-60.
77. Bayer AS. Infective endocarditis. *Clin Infect Dis* 1993;17:313-20.
78. Shively BK, Gurule FT, Roldan CA, Leggett JH, Schiller NB. Diagnostic value of transesophageal compared with transthoracic echocardiography in infective endocarditis. *J Am Coll Cardiol* 1991;18:391-7.
79. Gueret P, Vignon P, Fournier P, et al. Transesophageal echocardiography for the diagnosis and management of nonobstructive thrombosis of mechanical mitral valve prosthesis. *Circulation* 1995;91:103-10.
80. Isada LR, Torelli JN, Stewart WJ, Klein AL. Detection of fibrous strands on prosthetic mitral valves with transesophageal echocardiography: another potential embolic source. *J Am Soc Echocardiogr* 1994;7:641-5.
81. Orsinelli DA, Pearson AC. Detection of prosthetic valve strands by transesophageal echocardiography: clinical significance in patients with suspected cardiac source of embolism. *J Am Coll Cardiol* 1995;26:1713-8.
82. Barbetseas J, Nagueh SF, Pitsavos C, Toutouzas PK, Quinones MA, Zoghbi WA. Differentiating thrombus from pannus formation in obstructed mechanical prosthetic valves: an evaluation of clinical, transthoracic and transesophageal echocardiographic parameters. *J Am Coll Cardiol* 1998;32:1410-7.
83. Dzavik V, Cohen G, Chan KL. Role of transesophageal echocardiography in the diagnosis and management of prosthetic valve thrombosis. *J Am Coll Cardiol* 1991;18:1829-33.
84. Hurrell DG, Schaff HV, Tajik A. Thrombolytic therapy for obstruction of mechanical prosthetic valves. *Mayo Clin Proc* 1996;71:605-13.
85. Lengyel M, Fuster V, Keltai M, et al. Guidelines for management of left-sided prosthetic valve thrombosis: a role for thrombolytic therapy. Consensus Conference on Prosthetic Valve Thrombosis. *J Am Coll Cardiol* 1997;30:1521-6.
86. Tong AT, Roudaut R, Ozkan M, et al. Transesophageal echocardiography improves risk assessment of thrombolysis of prosthetic valve thrombosis: results of the international PRO-TEE registry. *J Am Coll Cardiol* 2004;43:77-84.
87. Zoghbi WA, Desir RM, Rosen L, Lawrie GM, Pratt CM, Quinones MA. Doppler echocardiography: application to the assessment of successful thrombolysis of prosthetic valve thrombosis. *J Am Soc Echocardiogr* 1989;2:98-101.
88. Donatelli F, Triggiani M, Mariani MA, et al. Rest and exercise hemodynamics of stentless porcine bioprostheses in aortic position. *Cardiologia* 1994;39:41-7.
89. Eriksson MJ, Brodin LA, Dellgren GN, Radegran K. Rest and exercise hemodynamics of an extended stentless aortic bioprosthesis. *J Heart Valve Dis* 1997;6:653-60.
90. Fries R, Wendler O, Schieffer H, Schafers HJ. Comparative rest and exercise hemodynamics of 23-mm stentless versus 23-mm stented aortic bioprostheses. *Ann Thorac Surg* 2000;69:817-22.
91. Jaffe WM, Coverdale HA, Roche AH, Whitlock RM, Neutze JM, Barratt-Boyes BG. Rest and exercise hemodynamics of 20 to 23 mm allograft, Medtronic Intact (porcine), and St. Jude Medical valves in the aortic position. *J Thorac Cardiovasc Surg* 1990;100:167-74.
92. Pibarot P, Dumesnil JG, Jobin J, Cartier P, Honos G, Durand LG. Hemodynamic and physical performance during maximal exercise in patients with an aortic bioprosthetic valve: comparison of stentless versus stented bioprostheses. *J Am Coll Cardiol* 1999;34:1609-17.
93. Chambers J, Rimington H, Rajani R, Hodson F, Blauth C. Hemodynamic performance on exercise: comparison of a stentless and stented biological aortic valve replacement. *J Heart Valve Dis* 2004;13:729-33.
94. Reis G, Motta MS, Barbosa MM, Esteves WA, Souza SF, Bocchi EA. Dobutamine stress echocardiography for noninvasive assessment and risk stratification of patients with rheumatic mitral stenosis. *J Am Coll Cardiol* 2004;43:393-401.
95. Castaneda-Zuniga W, Nicoloff D, Jorgensen C, Nath PH, Zollikofer C, Amplatz K. In vivo radiographic appearance of the St. Jude valve prosthesis. *Radiology* 1980;134:775-6.
96. Mehlman DJ. A pictorial and radiographic guide for identification of prosthetic heart valve devices. *Prog Cardiovasc Dis* 1988;30:441-64.
97. White AF, Dinsmore RE, Buckley MJ. Cineradiographic evaluation of prosthetic cardiac valves. *Circulation* 1973;48:882-9.
98. Cohn LH, Collins JJ Jr, DiSesa VJ, et al. Fifteen-year experience with 1678 Hancock porcine bioprosthetic heart valve replacements. *Ann Surg* 1989;210:435-42.
99. Sands MJ Jr, Lachman AS, O'Reilly DJ, Leach CN Jr, Sappington JB, Katz AM. Diagnostic value of cinefluoroscopy in the evaluation of prosthetic heart valve dysfunction. *Am Heart J* 1982;104:622-7.
100. de Mol BA, Kallewaard M, McLellan RB, van Herwerden LA, Defauw JJ, van der GY. Single-leg strut fractures in explanted Bjork-Shiley valves. *Lancet* 1994;343:9-12.
101. Guit GL, van Voorthuisen AE, Steiner RM. Outlet strut fracture of the Bjork-Shiley mitral valve prosthesis. *Radiology* 1985;154:298.
102. Hiratzka LF, Kouchoukos NT, Grunkemeier GL, Miller DC, Scully HE, Wechsler AS. Outlet strut fracture of the Bjork-Shiley 60 degrees Convexo-Concave valve: current information and recommendations for patient care. *J Am Coll Cardiol* 1988;11:1130-7.
103. Odell JA, Durandt J, Shama DM, Vythilingum S. Spontaneous embolization of a St. Jude prosthetic mitral valve leaflet. *Ann Thorac Surg* 1985;39:569-72.
104. Piers LH, Dikkers R, Boonstra PW. Visualisation of a St. Jude prosthetic mitral valve using electron beam tomography. *Heart* 2007;93:302.
105. Numata S, Okada H, Kitahara H, Kawazoe K. Four-dimensional evaluation of implanted mechanical valve with 64-row multi-detector computed tomography. *Eur J Cardiothorac Surg* 2007;31:934.
106. Leborgne L, Renard C, Tribouilloy C. Usefulness of ECG-gated multi-detector computed tomography for the diagnosis of mechanical prosthetic valve dysfunction. *Eur Heart J* 2006;27:2537.
107. Teshima H, Hayashida N, Enomoto N, Aoyagi S, Okuda K, Uchida M. Detection of pannus by multidetector-row computed tomography. *Ann Thorac Surg* 2003;75:1631-3.
108. Teshima H, Aoyagi S, Hayashida N, et al. Dysfunction of an ATS valve in the aortic position: the first reported case caused by pannus formation. *J Artif Organs* 2005;8:270-3.
109. Lembecke A, Hein PA, Enzweiler CN, Hoffmann U, Klessen C, Dohmen PM. Acute myocardial ischemia after aortic valve replacement: a comprehensive diagnostic evaluation using dynamic multislice spiral computed tomography. *Eur J Radiol* 2006;57:351-5.
110. Morgan-Hughes GJ, Owens PE, Roobottom CA, Marshall AJ. Three dimensional volume quantification of aortic valve calcification using multislice computed tomography. *Heart* 2003;89:1191-4.

111. Melina G, Horkaew P, Amrani M, Rubens MB, Yacoub MH, Yang GZ. Three-dimensional in vivo characterization of calcification in native valves and in Freestyle versus homograft aortic valves. *J Thorac Cardiovasc Surg* 2005;130:41-7.
112. Rashtian MY, Stevenson DM, Allen DT, et al. Flow characteristics of four commonly used mechanical heart valves. *Am J Cardiol* 1986;58:743-52.
113. Falicov RE, Walsh WF. Retrograde crossing of aortic Bjork-Shiley prosthesis. *Am J Cardiol* 1979;43:1062-4.
114. Karsh DL, Michaelson SP, Langou RA, Cohen LS, Wolfson S. Retrograde left ventricular catheterization in patients with an aortic valve prosthesis. *Am J Cardiol* 1978;41:893-6.
115. Kober G, Hilgermann R. Catheter entrapment in a Bjork-Shiley prosthesis in aortic position. *Cathet Cardiovasc Diagn* 1987;13:262-5.
116. Dunn M. Is transeptal catheterization necessary? *J Am Coll Cardiol* 1985;5:1393-4.
117. Horstkotte D, Haerten K, Seipel L, et al. Central hemodynamics at rest and during exercise after mitral valve replacement with different prostheses. *Circulation* 1983;68:1161-8.
118. Schoenfeld MH, Palacios IF, Hutter AM Jr, Jacoby SS, Block PC. Underestimation of prosthetic mitral valve areas: role of transeptal catheterization in avoiding unnecessary repeat mitral valve surgery. *J Am Coll Cardiol* 1985;5:1387-92.
119. Ben Zekry S, Saad RM, Little SH, Zoghbi WA. Flow acceleration time: a novel diagnostic parameter for prosthetic aortic valve stenosis [abstract]. *Circulation* 2008;118: S1069.
120. Chambers J, Rajani R, Hankins M, Cook R. The peak to mean pressure decrease ratio: a new method of assessing aortic stenosis. *J Am Soc Echocardiogr* 2005;18:674-8.
121. Rothbart RM, Castriz JL, Harding LV, Russo CD, Teague SM. Determination of aortic valve area by two-dimensional and Doppler echocardiography in patients with Normaland stenotic bioprosthetic valves. *J Am Coll Cardiol* 1990;15:817-24.
122. Otto CM, Pearlman AS, Comess KA, Reamer RP, Janko CL, Huntsman LL. Determination of the stenotic aortic valve area in adults using Doppler echocardiography. *J Am Coll Cardiol* 1986;7:509-17.
123. Zoghbi WA, Farmer KL, Soto JG, Nelson JG, Quinones MA. Accurate noninvasive quantification of stenotic aortic valve area by Doppler echocardiography. *Circulation* 1986;73:452-9.
124. Zabalgoitia M, Herrera CJ, Chaudhry FA, Calhoun JH, Mehlmán DJ, O'Rourke RA. Improvement in the diagnosis of bioprosthetic valve dysfunction by transesophageal echocardiography. *J Heart Valve Dis* 1993;2:595-603.
125. Saad RM, Barbetseas J, Olmos L, Rubio N, Zoghbi WA. Application of the continuity equation and valve resistance to the evaluation of St. Jude Medical prosthetic aortic valve dysfunction. *Am J Cardiol* 1997;80:1239-42.
126. Rajani R, Mukherjee D, Chambers JB. Doppler echocardiography in normally functioning replacement aortic valves: a review of 129 studies. *J Heart Valve Dis* 2007;16:519-35.
127. Rallidis LS, Moysakias IE, Ikonomidis I, Nihoyannopoulos P. Natural history of early aortic paraprosthetic regurgitation: a five-year follow-up. *Am Heart J* 1999;138:351-7.
128. Kupferwasser I, Mohr-Kahaly S, Erbel R, et al. Improved assessment of pathological regurgitation in patients with prosthetic heart valves by multiplane transesophageal echocardiography. *Echocardiography* 1997;14:363-74.
129. Mohr-Kahaly S, Kupferwasser I, Erbel R, Oelert H, Meyer J. Regurgitant flow in apparently normal valve prostheses: improved detection and semiquantitative analysis by transesophageal two-dimensional color-coded Doppler echocardiography. *J Am Soc Echocardiogr* 1990;3:187-95.
130. Mohr-Kahaly S, Kupferwasser I, Erbel R, et al. Value and limitations of transesophageal echocardiography in the evaluation of aortic prostheses. *J Am Soc Echocardiogr* 1993;6:12-20.
131. Bach DS. Transesophageal echocardiographic (TEE) evaluation of prosthetic valves. *Cardiol Clin* 2000;18:751-71.
132. Sprecher DL, Adamick R, Adams D, Kisslo J. In vitro color flow, pulsed and continuous wave Doppler ultrasound masking of flow by prosthetic valves. *J Am Coll Cardiol* 1987;9:1306-10.
133. Muratori M, Montorsi P, Teruzzi G, et al. Feasibility and diagnostic accuracy of quantitative assessment of mechanical prostheses leaflet motion by transthoracic and transesophageal echocardiography in suspected prosthetic valve dysfunction. *Am J Cardiol* 2006;97:94-100.
134. Cohen GI, Davison MB, Klein AL, Salcedo EE, Stewart WJ. A comparison of flow convergence with other transthoracic echocardiographic indexes of prosthetic mitral regurgitation. *J Am Soc Echocardiogr* 1992;5:620-7.
135. Fernandes V, Olmos L, Nagueh SF, Quinones MA, Zoghbi WA. Peak early diastolic velocity rather than pressure half-time is the best index of mechanical prosthetic mitral valve function. *Am J Cardiol* 2002;89:704-10.
136. Baumgartner H, Schirra H, Kuhn P. Discrepancies between Doppler and catheter gradients across bileaflet aortic valve prostheses. *Am J Cardiol* 1993;71:1241-3.
137. Yoganathan AP, Chauv A, Gray RJ, et al. Bileaflet, tilting disc and porcine aortic valve substitutes: in vitro hydrodynamic characteristics. *J Am Coll Cardiol* 1984;3:313-20.
138. Goetze S, Brechtken J, Agler DA, Thomas JD, Sabik JF III, Jaber WA. In vivo short-term Doppler hemodynamic profiles of 189 Carpentier-Edwards Perimount pericardial bioprosthetic valves in the mitral position. *J Am Soc Echocardiogr* 2004;17:981-7.
139. Rosenhek R, Binder T, Maurer G, Baumgartner H. Normal values for Doppler echocardiographic assessment of heart valve prostheses. *J Am Soc Echocardiogr* 2003;16:1116-27.
140. Malouf JF, Ballo M, Connolly HM, et al. Doppler echocardiography of 119 normal-functioning St Jude Medical mitral valve prostheses: a comprehensive assessment including time-velocity integral ratio and prosthesis performance index. *J Am Soc Echocardiogr* 2005;18:252-6.
141. Panidis IP, Ross J, Mintz GS. Normal and abnormal prosthetic valve function as assessed by Doppler echocardiography. *J Am Coll Cardiol* 1986;8:317-26.
142. Malouf JF, Ballo M, Hodge DO, Herges RM, Orszulak TA, Miller FA Jr. Doppler echocardiography of normal Starr-Edwards mitral prostheses: a comprehensive function assessment including continuity equation and time-velocity integral ratio. *J Am Soc Echocardiogr* 2005;18:1399-403.
143. Blauwet LA, Malouf JF, Connolly HM, et al. Doppler echocardiography of 79 normal CarboMedics mitral prostheses: a comprehensive assessment including time-velocity integral ratio and prosthesis performance index. *J Am Soc Echocardiogr* 2009;20:1125-30.
144. Blauwet LA, Malouf JF, Connolly HM, et al. Doppler echocardiography of 240 normal Carpentier-Edwards Duraflex porcine mitral bioprostheses: a comprehensive assessment including time velocity integral ratio and prosthesis performance index. *J Am Soc Echocardiogr* 2009;22:388-93.
145. Davila-Roman VG, Waggoner AD, Kennard ED, et al. Prevalence and severity of paravalvular regurgitation in the Artificial Valve Endocarditis Reduction Trial (AVERT) echocardiography study. *J Am Coll Cardiol* 2004;44:1467-72.
146. Montorsi P, Cavoretto D, Parolari A, Muratori M, Alimento M, Pepi M. Diagnosing prosthetic mitral valve thrombosis and the effect of the type of prosthesis. *Am J Cardiol* 2002;90:73-6.
147. Ronderos RE, Portis M, Stoermann W, Sarmiento C. Are all echocardiographic findings equally predictive for diagnosis in prosthetic endocarditis? *J Am Soc Echocardiogr* 2004;17:664-9.
148. Olmos L, Salazar G, Barbetseas J, Quinones MA, Zoghbi WA. Usefulness of transthoracic echocardiography in detecting significant prosthetic mitral valve regurgitation. *Am J Cardiol* 1999;83:199-205.
149. Alton ME, Pasierski TJ, Orsinelli DA, Eaton GM, Pearson AC. Comparison of transthoracic and transesophageal echocardiography in evaluation of 47 Starr-Edwards prosthetic valves. *J Am Coll Cardiol* 1992;20:1503-11.

150. Daniel LB, Grigg LE, Weisel RD, Rakowski H. Comparison of transthoracic and transesophageal assessment of prosthetic valve dysfunction. *Echocardiography* 1990;7:83-95.
151. Flachskampf FA, Hoffmann R, Franke A, et al. Does multiplane transesophageal echocardiography improve the assessment of prosthetic valve regurgitation? *J Am Soc Echocardiogr* 1995;8:70-8.
152. Hung J, Lang RM, Flachskampf FA, et al. 3D echocardiography: a review of the current status and future directions. *J Am Soc Echocardiogr* 2007;20:213-33.
153. Fischer CH, Campos FO, Moises VA, et al. Quantification of mitral prosthesis failure by different methods of evaluation by transesophageal echocardiography. *Arq Bras Cardiol* 1998;71:741-5.
154. Vitarelli A, Conde Y, Cimino E, et al. Assessment of severity of mechanical prosthetic mitral regurgitation by transoesophageal echocardiography. *Heart* 2004;90:539-44.
155. Genoni M, Franzen D, Tavakoli R, et al. Does the morphology of mitral paravalvular leaks influence symptoms and hemolysis? *J Heart Valve Dis* 2001;10:426-30.
156. Meliones JN, Snider AR, Bove EL, et al. Doppler evaluation of homograft valved conduits in children. *Am J Cardiol* 1989;64:354-8.
157. Novaro GM, Connolly HM, Miller FA. Doppler hemodynamics of 51 clinically and echocardiographically normal pulmonary valve prostheses. *Mayo Clin Proc* 2001;76:155-60.
158. da Costa F, Haggi H, Pinton R, Lenke W, Adam E, Costa IS. Rest and exercise hemodynamics after the Ross procedure: an echocardiographic study. *J Card Surg* 1998;13:177-85.
159. Moidl R, Simon P, Kupilik N, et al. Increased pulmonary flow velocities in oversized homografts in patients after the Ross procedure. *Eur J Cardiothorac Surg* 1997;12:569-72.
160. Ward KE, Elkins RC, Overholt ED, et al. Evaluation of cryopreserved homografts in the right ventricular outflow tract after the Ross procedure: intermediate-term follow up. *J Heart Valve Dis* 1997;6:130-3.
161. Briand M, Pibarot P, Dumesnil JG, Cartier P. Midterm echocardiographic follow-up after Ross operation. *Circulation* 2000;102:III10-4.
162. Rosti L, Murzi B, Colli AM, et al. Mechanical valves in the pulmonary position: a reappraisal. *J Thorac Cardiovasc Surg* 1998;115:1074-9.
163. Waterbolk TW, Hoendermis ES, den H I, Ebels T. Pulmonary valve replacement with a mechanical prosthesis. Promising results of 28 procedures in patients with congenital heart disease. *Eur J Cardiothorac Surg* 2006;30:28-32.
164. Cooper J. Conventional and color Doppler assessment of right-sided valvular regurgitation. In: Nanda NK, editor. *Textbook of color Doppler echocardiography*. Philadelphia: Lea & Febiger; 1989:160-7.
165. Rao PS, Galal O, Patnana M, Buck SH, Wilson AD. Results of three to 10 year follow up of balloon dilatation of the pulmonary valve. *Heart* 1998;80:591-5.
166. Allen BS, El-Zein C, Cuneo B, Cava JP, Barth MJ, Ilbawi MN. Pericardial tissue valves and Gore-Tex conduits as an alternative for right ventricular outflow tract replacement in children. *Ann Thorac Surg* 2002;74:771-7.
167. Kanter KR, Budde JM, Parks WJ, et al. One hundred pulmonary valve replacements in children after relief of right ventricular outflow tract obstruction. *Ann Thorac Surg* 2002;73:1801-6.
168. Kanter KR, Fyfe DA, Mahle WT, Forbess JM, Kirshbom PM. Results with the freestyle porcine aortic root for right ventricular outflow tract reconstruction in children. *Ann Thorac Surg* 2003;76:1889-94.
169. Koh M, Yagihara T, Uemura H, et al. Long-term outcome of right ventricular outflow tract reconstruction using a handmade tri-leaflet conduit. *Eur J Cardiothorac Surg* 2005;27:807-14.
170. Marx GR, Hicks RW, Allen HD, Goldberg SJ. Noninvasive assessment of hemodynamic responses to exercise in pulmonary regurgitation after operations to correct pulmonary outflow obstruction. *Am J Cardiol* 1988;61:595-601.
171. Settepani F, Kaya A, Morshuis WJ, Schepens MA, Heijmen RH, Dossche KM. The Ross operation: an evaluation of a single institution's experience. *Ann Thorac Surg* 2005;79:499-504.
172. Sievers HH, Dahmen G, Graf B, Stierle U, Ziegler A, Schmidtke C. Midterm Results of the Ross procedure preserving the patient's aortic root [abstract]. *Circulation* 2003;108:55-60.
173. Weyman AE. Right ventricular outflow tract. In: Weyman AE, editor. *Principles and practices of echocardiography*. Philadelphia: Lea & Febiger; 1994:863-900.
174. Chan KC, Fyfe DA, McKay CA, Sade RM, Crawford FA. Right ventricular outflow reconstruction with cryopreserved homografts in pediatric patients: intermediate-term follow-up with serial echocardiographic assessment. *J Am Coll Cardiol* 1994;24:483-9.
175. Goldberg SJ, Allen HD. Quantitative assessment by Doppler echocardiography of pulmonary or aortic regurgitation. *Am J Cardiol* 1985;56:131-5.
176. Connolly HM, Miller FA Jr., Taylor CL, Naessens JM, Seward JB, Tajik AJ. Doppler hemodynamic profiles of 82 clinically and echocardiographically normal tricuspid valve prostheses. *Circulation* 1993;88:2722-7.
177. Kobayashi Y, Nagata S, Ohmori F, Eishi K, Nakano K, Miyatake K. Serial Doppler echocardiographic evaluation of bioprosthetic valves in the tricuspid position. *J Am Coll Cardiol* 1996;27:1693-7.
178. Aoyagi S, Nishi Y, Kawara T, Oryoji A, Kosuga K, Ohishi K. Doppler echocardiographic evaluation of St. Jude Medical valves in the tricuspid position. *J Heart Valve Dis* 1993;2:279-86.
179. Kocis KC, Snider AR, Lupinetti FM, Bove EL. Doppler forward flow profiles of St. Jude Medical prosthetic valves in pediatric patients. *Am J Cardiol* 1994;74:77-9.
180. Masuda M, Kado H, Tatewaki H, Shiokawa Y, Yasui H. Late results after mitral valve replacement with bileaflet mechanical prosthesis in children: evaluation of prosthesis-patient mismatch. *Ann Thorac Surg* 2004;77:913-7.
181. Karamlou T, Jang K, Williams WG, et al. Outcomes and associated risk factors for aortic valve replacement in 160 children: a competing-risks analysis. *Circulation* 2005;112:3462-9.
182. Adatia I, Moore PM, Jonas RA, Colan SD, Lock JE, Keane JF. Clinical course and hemodynamic observations after supraannular mitral valve replacement in infants and children. *J Am Coll Cardiol* 1997;29:1089-94.
183. Dinarevic S, Redington A, Rigby M, Sheppard MN. Left ventricular pannus causing inflow obstruction late after mitral valve replacement for endocardial fibroelastosis. *Pediatr Cardiol* 1996;17:257-9.
184. Sugeng L, Shernan SK, Weinert L, et al. Real-time three-dimensional transesophageal echocardiography in valve disease: comparison with surgical findings and evaluation of prosthetic valves. *J Am Soc Echocardiogr* 2009;21:1347-54.
185. Little SH, Ben Zekry S, Zoghbi WA, Kleiman NS, Guthikonda S. Three-dimensional transesophageal echocardiogram provides real-time guidance during percutaneous paravalvular mitral repair. *Cir Heart Fail* 2009;1:293-4.

Appendix A. Normal Doppler Echocardiographic Values for Prosthetic Aortic Valves*

Valve	Size	Peak gradient (mm Hg)	Mean gradient (mmHg)	Effective orifice area (cm ²)
ATS <i>Bileaflet</i>	19	47.0± 12.6	25.3± 8.0	1.1± 0.3
	21	23.7± 6.8	15.9± 5.0	1.4± 0.5
	23		14.4± 4.9	1.7± 0.5
	25		11.3± 3.7	2.1± 0.7
	27		8.4± 3.7	2.5± 0.1
	29		8.0± 3.0	3.1± 0.8
ATS AP <i>Bileaflet</i>	18		21.0± 1.8	1.2± 0.3
	20	21.4± 4.2	11.1± 3.5	1.3± 0.3
	22	18.7± 8.3	10.5± 4.5	1.7± 0.4
	24	15.1± 5.6	7.5± 3.1	2.0± 0.6
Baxter Perimount <i>Stented bovine pericardial</i>	26		6.0± 2.0	2.1± 0.4
	19	32.5± 8.5	19.5± 5.5	1.3± 0.2
	21	24.9± 7.7	13.8± 4.0	1.3± 0.3
	23	19.9± 7.4	11.5± 3.9	1.6± 0.3
	25	16.5± 7.8	10.7± 3.8	1.6± 0.4
	27	12.8± 5.4	4.8± 2.2	2.0± 0.4
Biocor <i>Stented porcine</i>	23	30.0± 10.7	20± 6.6	1.3± 0.3
	25	23.0± 7.9	16± 5.1	1.7± 0.4
	27	22.0± 6.5	15.0± 3.7	2.2± 0.4
Extended Biocor <i>Stentless</i>	19-21	17.5± 6.5	9.6± 3.6	1.4± 0.4
	23	14.7± 7.3	7.7± 3.8	1.7± 0.4
	25	14.0± 4.3	7.4± 2.5	1.8± 0.4
Bioflo <i>Stented bovine pericardial</i>	19	37.2± 8.8	26.4± 5.5	0.7± 0.1
	21	28.7± 6.2	18.7± 5.5	1.1± 0.1
	21	38.9± 11.9	21.8± 3.4	1.1± 0.3
Bjork-Shiley <i>Single tilting disc</i>	23	28.8± 11.2	15.7± 5.3	1.3± 0.3
	25	23.7± 8.2	13.0± 5.0	1.5± 0.4
	27		10.0± 2.0	1.6± 0.3
Carbomedics Reduced <i>Bileaflet</i>	19	43.4± 1.2	24.4± 1.2	1.2± 0.1
Carbomedics Standard <i>Bileaflet</i>	19	38.0± 12.8	18.9± 8.3	1.0± 0.3
	21	26.8± 10.1	12.9± 5.4	1.5± 0.4
	23	22.5± 7.4	11.0± 4.6	1.4± 0.3
	25	19.6± 7.8	9.1± 3.5	1.8± 0.4
	27	17.5± 7.1	7.9± 3.2	2.2± 0.2
	29	9.1± 4.7	5.6± 3.0	3.2± 1.6
Carbomedics Tophat <i>Bileaflet</i>	21	30.2± 10.9	14.9± 5.4	1.2± 0.3
	23	24.2± 7.6	12.5± 4.4	1.4± 0.4
	25		9.5± 2.9	1.6± 0.32
Carpentier Edwards <i>Pericardial</i> <i>Stented bovine pericardial</i>	19	32.1± 3.4	24.2± 8.6	1.2± 0.3
	21	25.7± 9.9	20.3± 9.1	1.5± 0.4
	23	21.7± 8.6	13.0± 5.3	1.8± 0.3
	25	16.5± 5.4	9.0± 2.3	
Carpentier Edwards <i>Standard</i> <i>Stented porcine</i>	19	43.5± 12.7	25.6± 8.0	0.9± 0.2
	21	27.7± 7.6	17.3± 6.2	1.5± 0.3
	23	28.9± 7.5	16.1± 6.2	1.7± 0.5
	25	24.0± 7.1	12.9± 4.6	1.9± 0.5
	27	22.1± 8.2	12.1± 5.5	2.3± 0.6
Carpentier Supra-Annular <i>Stented porcine</i>	29		9.9± 2.9	2.8± 0.5
	19	34.1± 2.7		1.1± 0.1
	21	28.0± 10.5	17.5± 3.8	1.4± 0.9
	23	25.3± 10.5	13.4± 4.5	1.6± 0.6
	25	24.4± 7.6	13.2± 4.8	1.8± 0.4
27	16.7± 4.7	8.8± 2.8	1.9± 0.7	