Chapter 30
Evaluation of Prosthetic Valves

Sabrina Phillips • Fletcher Miller

Replacement of dysfunctional native cardiac valves is very common in the practice of congenital heart disease. A subset of patients with congenital heart disease will require multiple “re-replacements” of valvular prostheses secondary to growth or prosthesis degeneration. Therefore, appropriate longitudinal evaluation of prosthetic valves is critical to clinical practice. Transthoracic echocardiography is the most useful tool in the routine evaluation of valve prostheses, but transesophageal echocardiography, cardiac catheterization, and fluoroscopy are all important adjuncts to the clinical evaluation of a dysfunctional prosthesis. As a reference for longitudinal follow-up, a transthoracic echocardiogram should be obtained early after valve implantation in all patients.

Prosthetic valves can be divided into two large categories: tissue and mechanical. The tissue prostheses include porcine or bovine heterografts and human allografts/homografts (Fig. 30.1). The mechanical prostheses include tilting disc prosthetics (e.g., the St. Jude bileaflet and the Medtronic-Hall single disc) and the ball-cage prosthesis (e.g., the Starr-Edwards) (Fig. 30.1). To accurately interpret echocardiographic findings, the exact type and size of the prosthesis should be known, as the various valve types demonstrate different hemodynamic profiles (Tables 30.1 through Table 30.4) and regurgitation characteristics. Prosthetic valve dysfunction may be caused by valve thrombosis, pannus formation, senescent changes (bioprostheses), and infection (Fig. 30.2).

EVALUATION OF THE MITRAL PROSTHESIS

Doppler interrogation of a mitral prosthesis is crucial in determining the functional status of the valve. However, the transthoracic echocardiographic evaluation of a mitral prosthesis should begin with a two-dimensional evaluation of valve stability and surrounding structures. Large vegetation, thrombus, and valve dehiscence can be identified. The leaflets of a bioprosthetic valve can be evaluated for symmetric excursion and evidence of calcification. The mechanical mitral prosthesis produces reverberation artifact, which limits the two-dimensional interrogation of the prosthesis itself and the surrounding structures, especially the left atrium (Fig. 30.3). Flow velocity through the prosthetic valve and determination of the maximal and mean pressure gradients should be obtained using continuous-wave Doppler interrogation. The pressure gradient can be elevated secondary to valve stenosis, regurgitation, or increased cardiac output. The pressure half-time measurement and the left ventricular outflow tract (LVOT) velocity can help distinguish between these two scenarios. The pressure half-time should be prolonged if the valve is obstructed, but normal or shortened if the increased pressure gradient across the valve is secondary to regurgitation. The LVOT velocity will be decreased if the increased velocity across the mitral prosthesis is secondary to severe regurgitation, as the forward flow across the LVOT will be decreased (Table 30.5). It is important to recall that the pressure half-time method used to determine the area of a native mitral valve will overestimate the area of a mitral prosthesis. If there is no significant aortic or mitral regurgitation, the continuity equation is a valid and more optimal method for determining the area of a mitral prosthesis. The continuity equation for this calculation is as follows:

\[ MP \text{ area} = \frac{LVOT \text{ area} \times (LVOT \text{ TVI})}{MP \text{ TVI}} \]

where MP is mitral prosthesis, LVOT TVI is the LVOT time-velocity integral, and MP TVI is the time-velocity integral of the mitral prosthesis inflow velocity obtained by continuous-wave Doppler.

Regurgitation should be evaluated with color-flow imaging, spectral Doppler interrogation, and two-dimensional evaluation of the valve. If there is regurgitation, it is important to distinguish periprosthetic regurgitation from prosthetic regurgitation (Fig. 30.4), as the mechanism for each is different. The degree of regurgitation should then be assessed. The degree of prosthetic regurgitation can be assessed both by semiquantitative methods and quantitative methods (proximal isovelocity surface area [PISA]) if the jet is well seen. Severe regurgitation is indicated by the following:

1. Increased mitral inflow peak velocity 2.5 m/s or greater with normal mitral inflow pressure half-time (≤150 m/s)
2. Dense mitral regurgitant continuous-wave Doppler signals
3. Regurgitant fraction 55% or greater
4. Systolic Doppler flow reversals in the pulmonary vein

While prosthetic regurgitation can be detected by color flow imaging, the mechanical mitral prosthesis poses special challenges. The artifact produced in the left atrium may limit the ability to detect even significant degrees of prosthetic and periprosthetic regurgitation. In this situation, indirect indicators of significant regurgitation should be used. These include two-dimensional evidence of valve instability or periprosthetic tissue changes, increased left ventricular dimension, increased inflow gradient with a normal or reduced pressure half-time, and decreased LVOT velocity. Transesophageal echocardiography is very useful in defining the presence and degree of mechanical mitral prosthesis regurgitation (Fig. 30.5), as the posterior-to-anterior direction of the ultrasound beam allows visualization of the left atrium without imaging artifact.

When evaluating a prosthesis for regurgitation, it must be remembered that a small degree of prosthetic regurgitation is inherent for most mechanical prosthesis. The Doppler fingerprint of this regurgitation varies by prosthesis type. A Medtronic-Hall prosthesis...
has one central jet of regurgitation, the St. Jude Medical mecha-
nical prosthesis has two side and one central jet, the Starr-Edwards
prosthesis has two curved side jets, and the Björk-Shiley prosthesis
has two unequal side jets (Fig. 30.6). These normal regurgi-
tant jets should be small, with a jet area less than 2 cm² and a jet
length of less than 2.5 cm. Periprosthetic regurgitation is always abnor-
mal. It may be a small amount of regurgitation related to a gap
in the sutures placed at the anastomosis, or it may be secondary
to an infectious process with perivalvular extension. Periprosthetic
regurgitation that is significant may require treatment. Surgical
intervention has traditionally been required, but transcatheter
placement of devices to eliminate periprosthetic leakage is now pos-
sible in selected patients without evidence of active infection who
are not surgical candidates.

### EVALUATION OF THE AORTIC PROSTHESIS

An aortic valve prosthesis should be evaluated with an approach
similar to the evaluation of the mitral prosthesis, with a few caveats.
The spectral Doppler assessment of the left ventricular outflow tract
and the aortic valve are usually best obtained with transesophageal
imaging. Transesophageal imaging often does not allow optimal
alignment of the Doppler beam to obtain the most accurate veloci-
ties through the valve and outflow tract. Left ventricular size, wall
thickness, and function are also often most reliably measured dur-
ing transthoracic imaging. Regurgitation can be demonstrated by
both transthoracic and transesophageal imaging, but anterior peri-
prosthetic jets are best seen on transthoracic images and posterior
jets are best demonstrated by transesophageal images secondary to
the location of the transducer and the artifact produced by the valve.
Transesophageal images in general are better for the detection of
vegetation.

Obstruction of an aortic prosthesis results in increased flow
velocity across the prosthesis and is best quantitated by Doppler
echocardiography. Doppler interrogation should be performed in
multiple imaging windows to obtain the maximal velocity across
the prosthesis. To completely evaluate the prosthesis, continu-
ous-wave Doppler should be obtained. From the highest velocity
signal, a maximum instantaneous pressure gradient and a mean
pressure gradient should be calculated. Pulsed-wave Doppler
interrogation of the LVOT and measurement of the LVOT TVI is
critical in determining the etiology of the increased blood flow
velocity across the aortic prosthesis. Care should be taken to
place the Doppler sample volume for this measurement below
the area of flow acceleration. If the valve is truly obstructed, the
LVOT velocity should not be increased. If the increased velocity
across the valve is secondary to aortic regurgitation or a high out-
put state, the LVOT velocity will be increased. The ratio of LVOT
velocity or TVI versus AV velocity or TVI is helpful. If the prosthesis
is obstructed, the ratio decreases (LVOT TVI/AV TVI ≤ 0.2 with
normal ≥ 0.3) (Table 30.2).

The area of the aortic prosthesis can be estimated by the conti-
nuity equation as follows:

\[
AP \text{ area} = \frac{LVOT \text{ area} \times (\text{LVOT TVI}/\text{AP TVI})}{SROD^2 \times 0.785 \times (\text{LVOT TVI}/\text{AP TVI})}
\]

where \(AP\) is aortic prosthesis and \(SROD\) is sewing ring diameter.
Evaluation of aortic prosthetic regurgitation should include two-
dimensional evaluation of the valve and surrounding structures,
flow-color imaging to determine the jet characteristics and location

### Table 30.2 DOPPLER HEMODYNAMIC PROFILES OF 456 NORMAL MITRAL VALVE PROSTHESES

<table>
<thead>
<tr>
<th>Type of Prosthesis</th>
<th>No. of Prostheses</th>
<th>Peak Velocity (m/s)</th>
<th>Mean Gradient (mm Hg)</th>
<th>Effective Area (cm²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heterograft</td>
<td>150</td>
<td>1.6 ± 0.3</td>
<td>4.1 ± 1.5</td>
<td>2.3 ± 0.7</td>
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<tr>
<td>Ball-cage</td>
<td>161</td>
<td>1.8 ± 0.3</td>
<td>4.9 ± 1.8</td>
<td>2.4 ± 0.7</td>
</tr>
<tr>
<td>Björk-Shiley</td>
<td>79</td>
<td>1.7 ± 0.3</td>
<td>4.1 ± 1.6</td>
<td>2.6 ± 0.6</td>
</tr>
<tr>
<td>St. Jude Medical</td>
<td>66</td>
<td>1.6 ± 1.0</td>
<td>4.0 ± 1.8</td>
<td>3.0 ± 0.8</td>
</tr>
</tbody>
</table>


### Table 30.3 DOPPLER HEMODYNAMIC PROFILES OF 82 NORMAL TRICUSPID VALVE PROSTHESES

<table>
<thead>
<tr>
<th>Type of Prosthesis</th>
<th>No. of Prostheses</th>
<th>Peak Velocity (m/s)</th>
<th>Mean Gradient (mm Hg)</th>
<th>Pressure Half-time (ms)</th>
</tr>
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<tbody>
<tr>
<td>Heterograft</td>
<td>41</td>
<td>1.3 ± 0.2</td>
<td>13.3 ± 1.1</td>
<td>146 ± 39</td>
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<tr>
<td>Ball-cage</td>
<td>33</td>
<td>1.3 ± 0.2</td>
<td>3.1 ± 0.8</td>
<td>144 ± 46</td>
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<tr>
<td>St. Jude Medical</td>
<td>7</td>
<td>1.2 ± 0.3</td>
<td>2.7 ± 1.1</td>
<td>108 ± 32</td>
</tr>
<tr>
<td>Björk-Shiley</td>
<td>1</td>
<td>1.3</td>
<td>2.2</td>
<td>144</td>
</tr>
<tr>
<td>Total</td>
<td>82</td>
<td>1.3 ± 0.2</td>
<td>3.1 ± 1.0</td>
<td>142 ± 42</td>
</tr>
</tbody>
</table>


### Table 30.4 DOPPLER ECHOCARDIOGRAPHIC DATA FOR PULMONARY VALVE PROSTHESES

<table>
<thead>
<tr>
<th>Type of Prosthesis</th>
<th>No.</th>
<th>Size (mm)</th>
<th>Peak Velocity (m/s)</th>
<th>Mean Gradient (mm Hg)</th>
<th>Trivial/Mild Prosthetic Regurgitation (No.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carpenter-Edwards</td>
<td>24</td>
<td>26.5 ± 1.8</td>
<td>2.4 ± 0.5</td>
<td>12.1 ± 5.3</td>
<td>7</td>
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<tr>
<td>Pulmonary homograft</td>
<td>17</td>
<td>24.2 ± 1.8</td>
<td>1.8 ± 0.6</td>
<td>8.4 ± 4.8</td>
<td>15</td>
</tr>
<tr>
<td>Aortic homograft</td>
<td>3</td>
<td>22.3 ± 0.6</td>
<td>2.5 ± 0.4</td>
<td>14.4 ± 4.3</td>
<td>3</td>
</tr>
<tr>
<td>Hancock</td>
<td>3</td>
<td>26.0 ± 3.0</td>
<td>2.4 ± 0.5</td>
<td>14.0 ± 5.7</td>
<td>1</td>
</tr>
<tr>
<td>Ionescu-Shiley</td>
<td>2</td>
<td>25.0 ± 0.0</td>
<td>2.4 ± 0.4</td>
<td>12.5 ± 3.5</td>
<td>2</td>
</tr>
<tr>
<td>St. Jude Medical</td>
<td>1</td>
<td>25</td>
<td>2.6</td>
<td>12.0</td>
<td>1</td>
</tr>
<tr>
<td>Björk-Shiley</td>
<td>1</td>
<td>25</td>
<td>2.0</td>
<td>7.0</td>
<td>1</td>
</tr>
</tbody>
</table>

(prosthetic versus periprosthetic), pressure half-time of the regurgitant jet, mitral inflow pattern, diastolic reversal flow in the descending thoracic aorta, and regurgitation fraction (if the jet is suitable for quantification). The normal regurgitation patterns of mechanical prosthetic valves should be kept in mind so as not to be confused with pathologic regurgitation. Severe aortic valve regurgitation is indicated by:

1. Pressure half-time of regurgitant jet of 250 ms or less
2. Restrictive mitral inflow pattern (if the aortic regurgitation is acute)
3. Holodiastolic reversal in the descending thoracic aorta Doppler
4. Regurgitant fraction of 55% or greater

**EVALUATION OF TRICUSPID AND PULMONARY PROSTHESES**

Tricuspid and pulmonary valve replacements are much more common in patients with congenital heart disease. The evaluation of these prostheses is analogous to the evaluation of the aortic and mitral prosthetic valves with a few exceptions. The tricuspid and pulmonary valves are usually anterior structures. Therefore, imaging of these valves is often best with transthoracic imaging. The posterior location of the echo transducer in transesophageal imaging can diminish the ability to evaluate these anterior structures. If transesophageal imaging is used, transgastric images are usually best for evaluation.

Several forms of congenital heart disease require placement of an extraanatomic conduit and valve to establish continuity between the subpulmonary ventricle and the pulmonary arteries. These conduits are best evaluated with transthoracic images. Unique imaging windows must be obtained to completely determine the functional status of these prostheses. Imaging windows can often be found by palpation of the chest wall. Since the valve prosthesis is very close to the chest wall in these situations, flow through the valve is often felt as a vibratory sensation or “thrill.” Placing the transducer at the site of the thrill may provide the best visualization of the valve. Often, the valve prosthesis itself is not seen and one must rely on Doppler interrogation and other indirect findings to determine the status of the prosthesis. Indirect findings include calculation of the right ventricular systolic pressure from the tricuspid valve regurgitation velocity using the modified Bernoulli equation: $P = \frac{4V^2}{H}$. The velocity (and hence the calculated pressure gradient) across the pulmonary conduit cannot exceed the velocity through the tricuspid valve. Other indirect findings include right ventricular size, wall thickness, and function. The interventricular septal motion may also provide clues to right ventricular pressure or volume overload. If the septum flattens only in diastole, volume overload should be suspected. Septal flattening in both systole and diastole is indicative of right ventricular pressure overload.

Pulmonary valve prosthetic regurgitation may be brief in duration. The spectral Doppler pattern may be helpful in determining the degree of regurgitation. Rapid equalization of pulmonary artery and right ventricular diastolic pressure will occur if the degree of regurgitation is severe. This will result in a Doppler pattern of regurgitation that returns to the baseline before the end of diastole (Fig. 30.7). This pattern does not always signify significant regurgitation, however. If the right ventricle has poor compliance, smaller volumes of regurgitation cause greater changes in the diastolic pressure and can lead to a similar Doppler pattern. Significant non-compliance may result in diastolic forward flow with atrial contraction (Fig. 30.8).

Tricuspid prosthetic valves are often large and have lower flow velocity across them. Therefore, a lower calculated pressure
Echocardiography in Pediatric and Adult Congenital Heart Disease

FIGURE 30.5. Mechanical mitral prosthesis visualized by transesophageal echocardiography. Note that there is no artifact obscuring the left atrium (A), allowing better visualization of the periprosthetic regurgitation jet (B).

FIGURE 30.6. Appearance of normally functioning mechanical mitral prosthesis. (A) Medtronic-Hall prosthesis with a single central jet of normal regurgitation. (B) St. Jude prosthesis with three jets of normal regurgitation.

Table 30.5

<table>
<thead>
<tr>
<th>Mitral Prosthesis</th>
<th>PHT</th>
<th>LVOT Velocity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obstruction</td>
<td>↑</td>
<td>↔, ↓</td>
</tr>
<tr>
<td>Regurgitation</td>
<td>↔, ↓</td>
<td>↓</td>
</tr>
<tr>
<td>High output</td>
<td>↔</td>
<td>↑</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Aortic Prosthesis</th>
<th>Mitral Inflow</th>
<th>LVOT Velocity</th>
<th>LVOT/AV TVI Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obstruction</td>
<td>↔</td>
<td>↔, ↓</td>
<td>↓</td>
</tr>
<tr>
<td>Regurgitation</td>
<td>↔, ↓</td>
<td>↑</td>
<td>↔</td>
</tr>
<tr>
<td>High output</td>
<td>↑</td>
<td>↑</td>
<td>↔</td>
</tr>
</tbody>
</table>

AV, aortic valve; LVOT, left ventricular outflow tract; PHT, pressure half-time; TVI, time-velocity integral. ↑, increased; ↓, decreased; ↔, no change.
gradient is expected compared with the mitral valve prosthesis. Respiratory variation in transtricuspid flow is greater than that in transmитral flow. Flow is increased across the tricuspid prosthesis during inspiration and decreased during expiration. Measurement of the prosthetic gradient either should be obtained over several cardiac cycles and averaged or should be obtained during held expiration.

Tricuspid valve prosthetic regurgitation, when severe, may be laminar. Systolic flow reversals in the hepatic veins are a marker of severe tricuspid prosthetic regurgitation but are not always present. If the right atrium is very large, the systolic flow reversals in the hepatic veins are diminished or absent.

**PATIENT–PROSTHESIS MISMATCH**

Prosthetic valves are manufactured in multiple different diameters. At surgery, the largest possible prosthesis should be implanted to provide the largest effective orifice area. If a prosthesis has an effective orifice area that is too small in relation to the patient’s body surface area, an increased gradient will be present without inherent stenosis of the valve. This is known as patient–prosthesis mismatch. Patient–prosthesis mismatch is determined by calculating the effective orifice area of the prosthesis and dividing that area by the body surface area of the patient to obtain an indexed effective orifice area. If the indexed effective orifice area is greater than 0.85 cm²/m², the degree of mismatch is mild. Severe patient–prosthesis mismatch is defined as an indexed effective orifice area of 0.6 cm²/m² or less. Patient–prosthesis mismatch has been shown in several studies to correlate with poor patient outcomes.

**SUGGESTED READING**


