Evaluation of Prosthetic Heart Valves by Transesophageal Echocardiography: Problems, Pitfalls, and Timing of Echocardiography

Renee B. A. Van den Brink, MD

Transesophageal echocardiography (TEE) is especially suitable for examination of prosthetic valves because of the proximity of the esophagus to the heart and absence of interference with lungs and ribs. This article reviews normal and abnormal morphologic characteristics of prosthetic valves such as spontaneous echocontrast, microbubbles, strands, sutures, vegetations or thrombus. Doppler echocardiographic characteristics of normal and pathologic prosthetic valve function and the management of prosthetic valve pathology is discussed. Physicians taking care of patients with prosthetic valves should be familiar with the characteristics of normal and abnormally functioning prosthetic valves.

Transesophageal echocardiography (TEE) is especially suitable for detection of anatomic abnormalities of prosthetic valves. Because of the proximity of the esophagus to the heart and absence of interference with lungs and ribs, a very detailed image can be obtained of the atrial side of the mitral valve prosthesis and especially the posterior part of the aortic prosthesis. Acoustic shadowing from prosthetic material often obscures the anterior part of the aortic prosthesis.

Assessment of Anatomic Characteristics of Prosthetic Valves

Abnormal echoes that may be found in patients with prosthetic valves are spontaneous echo contrast (SEC), microbubbles or cavitations, strands, sutures, vegetations, and thrombus.[1]

- *Spontaneous echo contrast (SEC)* is defined as smoke-like echoes. SEC is caused by increased red cell aggregation that occurs in slow flow, for example, because of a low cardiac output, severe left atrial dilatation, atrial fibrillation, or due to pathologic obstruction of a mitral prosthesis. The prevalence of SEC is 7% to 53%.
- *Microbubbles* are characterized by a discontinuous stream of rounded, strongly echogenic, fast-moving transient echoes. Microbubbles occur at the inflow zone of the valve when flow velocity and pressure suddenly drop at the time of prosthetic valve closing, but may also be seen during valve opening. The microbubble potential seems to be correlated with valve design, occluder material, and the velocity of the leaflet closure.[2-4] Kaymaz et al[5] investigated mitral prosthetic valves and found microbubbles in 75% of the normal bileaflet valves compared with 39% of the tilting-disk valves (*P* < .0001). In prosthetic valves with thrombotic obstruction, microbubbles were found in only 6% (1/18), whereas they reappeared after successful thrombolytic treatment with relief of valvular obstruction in 69%. Microbubbles are probably due to carbon dioxide degassing. Degassing involves separation of the gas contained in the water (or blood). In the case of a transient drop in pressure, the gas separates out before redissolving in the water when normal pressure is re-established.[6] Microbubbles are not found in bioprosthetic valves.
- *Strands* are thin, mildly echogenic, filamentous structures that are several millimeter long and move independently from the prosthesis. They are often visible intermittently during the cardiac cycle but recur at the same site. They are usually located at the inflow side of the prosthetic valve (ie, the atrial side of a mitral prosthesis or the ventricular side of an aortic prosthesis). Strands are found in 6% to 45% of patients. The finding of prosthetic valve-associated strands represents a clinical dilemma, because the cause, and management of this finding is not clear. Prosthetic valve-associated strands are likely to have multiple causes; they

From the Department of Cardiology; Academic Medical Centre, Amsterdam; The Netherlands

Address reprint requests to Renee B. A. Van den Brink, Department of Cardiology; Academic Medical Centre; Meibergdreef 9, 1105 AZ Amsterdam; The Netherlands; e-mail: r.b.vandenbrink@amc.uva.nl

may have a fibrinous or a collagenous composition.[7-9] Strands have been found to be more common in patients undergoing TEE for evaluation of the source of embolism than in patients examined for other reasons.[10,11] Although this association may imply an embolic potential, prospective follow-up is limited and the therapeutic implications of prosthetic valve-associated strands remain unclear. Importantly, if strands consist of collagen, aggressive therapeutic anticoagulation is not likely to completely eliminate their embolic potential.

- *Sutures* are defined as linear, thick, bright, multiple, evenly spaced, usually immobile echoes seen at the periphery of the sewing ring of a prosthetic valve; they may be mobile when loose or unusually long.
- *Vegetations and thrombus* cannot be distinguished by echocardiography alone; the differential diagnosis of these sessile or pedunculated masses depends on the full clinical picture. They may be interpreted as vegetations in a febrile patient and as thrombus in a poorly anticoagulated patient.

Prosthetic valve integrity and motion can be evaluated accurately with TEE. For bioprostheses, evidence of leaflet degeneration can be recognized as leaflet thickening (cusps >3 mm in thickness), calcification (bright echoes of the cusps), or a tear (flail cusp). In mechanical valves, an impaired disc excursion or a stuck leaflet can be visualized. Prosthetic valve dehiscence is characterized by a rocking motion of the entire prosthesis. An annular abscess may be recognized as an echolucent, irregularly shaped area adjacent to the sewing ring of the prosthetic valve. Sometimes the abscess is echodense, and only a thickening of the wall adjacent to the annulus is visualized.

Assessment of Flow Characteristics of Prosthetic Valves

All normal functioning mechanical prosthetic valves cause:

- some obstruction to blood flow
- closure backflow (necessary to close the valve)
- leakage backflow (after valve closure)

The extent of normal obstruction and leakage of prosthetic valves depends on prosthetic valve de-

sign. The ball-in-cage prosthetic valve (Starr-Edwards, Edwards Lifescience) shows much obstruction and little leakage. The tilting disc prosthetic valve (Björk-Shiley; Omniscience; Medtronic Hall) shows less obstruction and more leakage. The bileaflet prosthetic valves (St. Jude Medical; Sorin Bicarbon; Carbomedics) show less obstruction and more leakage.

Bioprostheses normally show little or no leakage. Homografts, pulmonary autografts, and unstented bioprosthetic valves (Medtronic Freestyle, Toronto, Ontario, Canada) are almost unobstructive to blood flow. In contrast, stented bioprostheses (leaflets suspended within a frame) are rather obstructive to flow.

Transthoracic and transesophageal echocardiography are very suitable to determine if a prosthetic valve has pathologic obstruction or leakage and if so, the cause.

Doppler Assessment of Normal Obstruction of Prosthetic Valves

For a proper interpretation of Doppler echocardiographic data in the individual patient, one needs to know prosthesis size, heart rate, and body surface area (BSA).

Gradient measurement. For gradient measurement it is important to align the ultrasound beam as parallel as possible to the transprosthetic flow. Transprosthetic gradients across mitral prostheses are very easy to determine with TEE (mid-transesophageal level: 0°, 60°-90° or 120°). This is more difficult in an aortic prosthesis where one should use a transgastric position at 90° to 120° or a deep transgastric "upside-down view" at 0° to 20°.

A high transprosthetic gradient may be caused by:

- High stroke volume (slow heart rate or paravalvular leakage)
- Patient-Valve prosthesis mismatch (implantation of a valve prosthesis that is too small for the patient's body surface area)
- Obstruction by thrombus, tissue ingrowth (pannus), or vegetation

Valve area measurement. The valve area of an aortic valve prosthesis is determined by the continuity equation. The advantage of using the effective orifice area (EOA) as a measure of obstruction is that the transvalvular flow rate is

taken into account. The EOA of prosthetic valves is calculated as:

$$EOA \ (cm^2) = 0.785 \times (prosthesis \ size \ in \ cm)^2 \times VTI_{LVOT}/VTI_{Aortic \ prosthesis}.$$

VTI_{LVOT} is velocity time integral (VTI) of blood flow in the left ventricular outflow tract (LVOT), measured with pulsed Doppler in the LVOT just proximal from the aortic prosthesis (see gradient measurement for transducer position). The sample volume position is optimal if the signal contains a nice spectral envelope and the closing click of the prosthesis. The VTI is obtained by tracing the contour of the Doppler flow signal. Instead of the VTI, the maximum velocity may be used. $VTI_{Ao. \ prosthesis}$ is velocity time integral at the prosthetic orificium. This is recorded with continuous wave Doppler from the same (transgastric) transducer position.

In mitral prostheses, the time necessary for the initial transprosthetic gradient to decline to half of its initial value (P1/2 time) may be used to determine prosthetic valve obstruction to blood flow. It is important, however, to keep in mind that P1/2 time is not only related to the prosthetic valve area but also to the pressure gradient at the start of diastole and to left ventricular and left atrial compliance. Although it is tempting to calculate valve areas of a mitral prosthesis using the P1/2 time formula (ie, mitral valve area = 220 msec/P1/2 time of the patient), it is important to realize that this formula has been validated for native mitral valves only. It does not apply to mechanical valves.

Doppler Assessment of Pathologic Obstruction of Prosthetic Valves

Pathologic obstruction in an aortic prosthesis. In an aortic prosthesis, the gradient changes considerably with a change of stroke volume (this effect is much more pronounced than in the larger mitral prosthesis). Stroke volume is determined by the patient's BSA. Therefore, even in the same valve type and size of an aortic prosthesis with normal function, gradients differ widely among patients.[12] Thus, it is essential to perform in every patient a baseline transthoracic Doppler echocardiographic examination within 1 to 2 months after prosthetic valve implantation. As a rule of thumb, one may say that an increase of the mean gradient by 20 mm Hg or more between two Doppler echocardiographic examinations in the same patient is clinically relevant and may signify pathologic obstruction.

One should make sure, however, that the high gradient in an aortic prosthesis really originates at the prosthetic valve level and, in the case of an aortic prosthesis, is not caused by obliteration of the left ventricular lumen. Systolic obliteration of the left ventricle causes a dynamic gradient with the typical dagger-shaped flow signal with late systolic high velocities. This signal is in contrast to the early systolic high velocities that are seen in case of a fixed obstruction to flow (ie, a prosthetic valve). See Figure 1.

Pathologic obstruction in a mitral prosthesis. In a mitral prosthesis, a mean gradient of 10 mm Hg or more may signify pathologic obstruction. In case of a mitral prosthesis
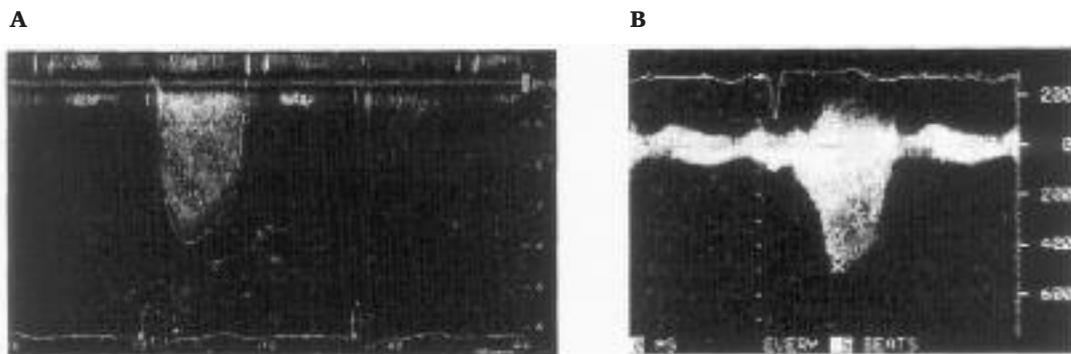

Figure 1. **(A)** Apical transducer position: fixed gradient in valvular aortic obstruction; note that the maximum gradient is reached very early in systole. **(B)** Apical transducer position: dynamic gradient; note that the maximum gradient is reached only in late systole, creating a typical dagger-shaped appearance of the continuous wave Doppler signal.

one should always interpret the transprosthetic gradient, taking into account the heart rate. An increase of heart rate occurs at the expense of the duration of diastole. This may have a profound influence on the gradient, especially in a mitral prosthesis.

In addition, one should take into consideration the P1/2 time. A low P1/2 time in a patient with a high transprosthetic gradient is not a sign of pathologic obstruction but rather is a sign of a high transprosthetic flow rate, as can be found in severe leakage of the prosthetic valve (see Table 1).

The investigation of a patient who is suspected to have pathologic obstruction of a mitral prosthesis is delineated in Figure 2.

Table 1. Interpretation of P1/2 Time

	*Pmean**	*P1/2 time*
Normal	<11 mm Hg	<120 msec
Grey area	11-16 mm Hg	120-160 msec
Pathologic obstruction	>16 mm Hg	>160 msec

*Only applicable if the heart rate is 70-100 beats/min.

Doppler Assessment of Normal Leakage of Prosthetic Valves

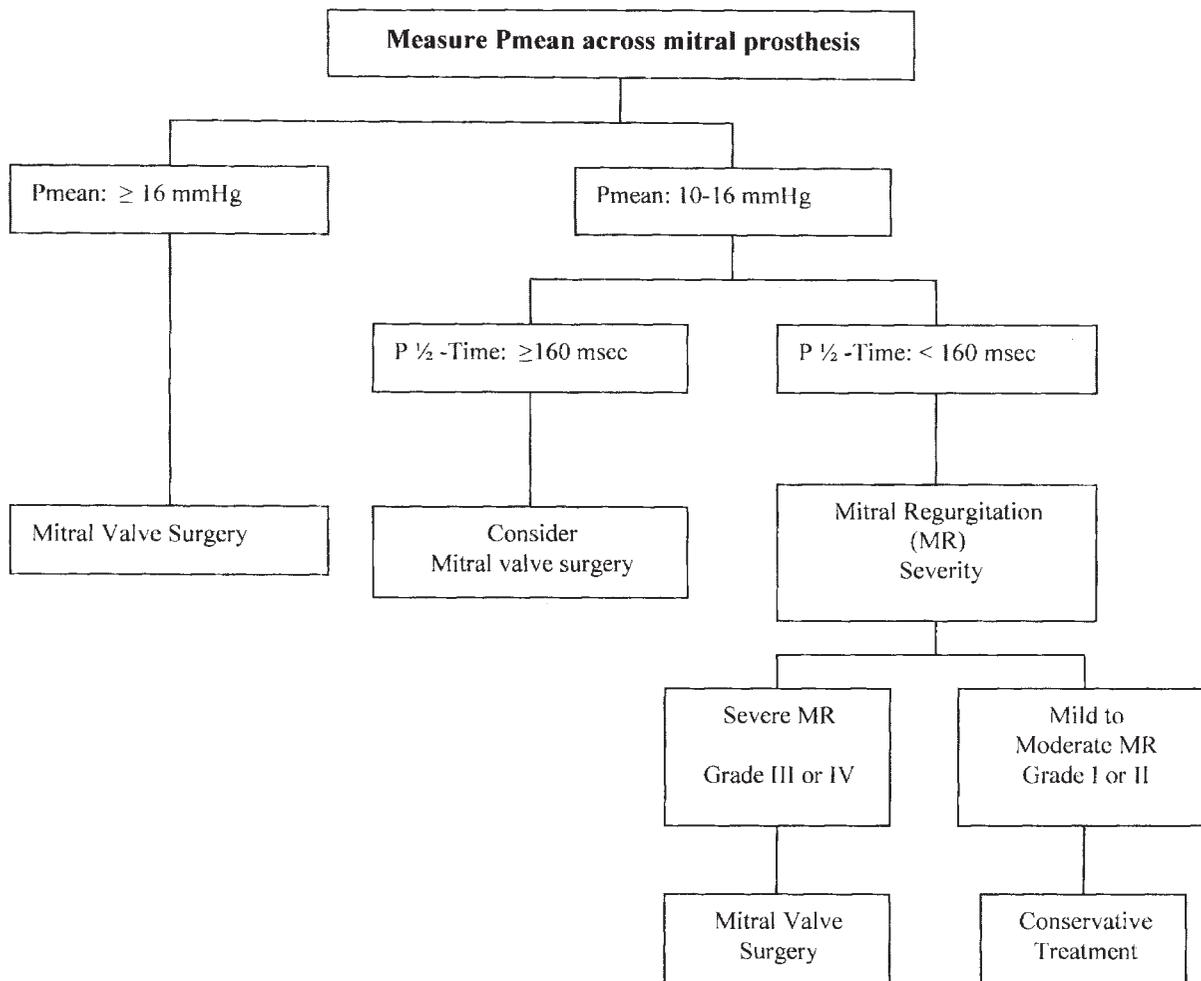Prosthetic valves can be categorized as mechanical or bioprosthetic. In vitro studies have demon-

Figure 2. Symptomatic patient suspected of mitral prosthesis dysfunction.

strated that mechanical prosthesis have closure backflow (necessary to close the valve) and leakage backflow (starting after valve closure). The closure and leakage backflow pattern is dependent on the prosthesis design (see Table 2 and Figure 3). For example, disc valves such as the St. Jude Medical and Medtronic Hall valves do not rest on a ledge of the orifice ring but fit inside the ring with a small space between the disc and ring or disc and pivot. Leakage backflow occurs through these small spaces and generates specific jet patterns within the left atrium. Ball-in-cage prostheses, however, consist of a poppet that rests on the ledge once the valve has been closed and leaves no space between ring and ball. Therefore, Starr-Edwards valves show only closure backflow and no leakage backflow.

Obviously, if one uses TEE, valve leakage from a mitral prothesis is visualized much easier than valve leakage from an aortic prosthesis because of the position of the mitral prosthesis in relation to the esophagus.

Pathologic regurgitation can be distinguished from normal backflow by the color Doppler appearance of the jets. Normal closure and leakage backflow jets are low-velocity non-aliasing jets encoded in a homogeneous color (red in mitral valve prostheses). In contrast, pathologic jets are more turbulent and extensive. They are often eccentric (crescent shaped) and adherent to the left atrial wall (see Figure 4A).

Pathologic regurgitation in mechanical valves may be caused by prosthetic valve dehiscence or by interference of structures, such as thrombus or vegetations, with disc closure. In bioprosthetic valves, pathologic regurgitation may be caused by prosthesis dehiscence or leaflet degeneration (calcification or tear).

Pathologic regurgitation is categorized as paravalvular and valvular regurgitation. Evaluation of a prosthetic valve for regurgitation is done by centering the prosthetic valve in the mid-esophageal four-chamber view. Then the sewing ring is imaged in full by rotation of the imaging plane from 0° to 180°, keeping the sewing ring in the center of the image and making small adjustments of the transducer tip (see Figure 4B).[13] Anatomic landmarks for localization of paravalvular leakage and for communication with the surgeon are the aorta and left atrial appendage.

Doppler Assessment of Pathologic Leakage of Prosthetic Valves

The severity of pathologic regurgitation in aortic prostheses is difficult to assess by TEE.

A jet width/LVOT diameter of 40% or more and a vena contracta diameter of 0.6 cm or more are signs of severe aortic regurgitation.

The severity of pathologic regurgitation in mitral prostheses is assessed by measurement of the jet area; in free jets, a jet area of 8 cm² or more means severe mitral regurgitation. Jet area measurement in eccentric jets hugging the left atrial

Table 2. Normal Patterns of Back Flow in Prosthetic Valves[13]

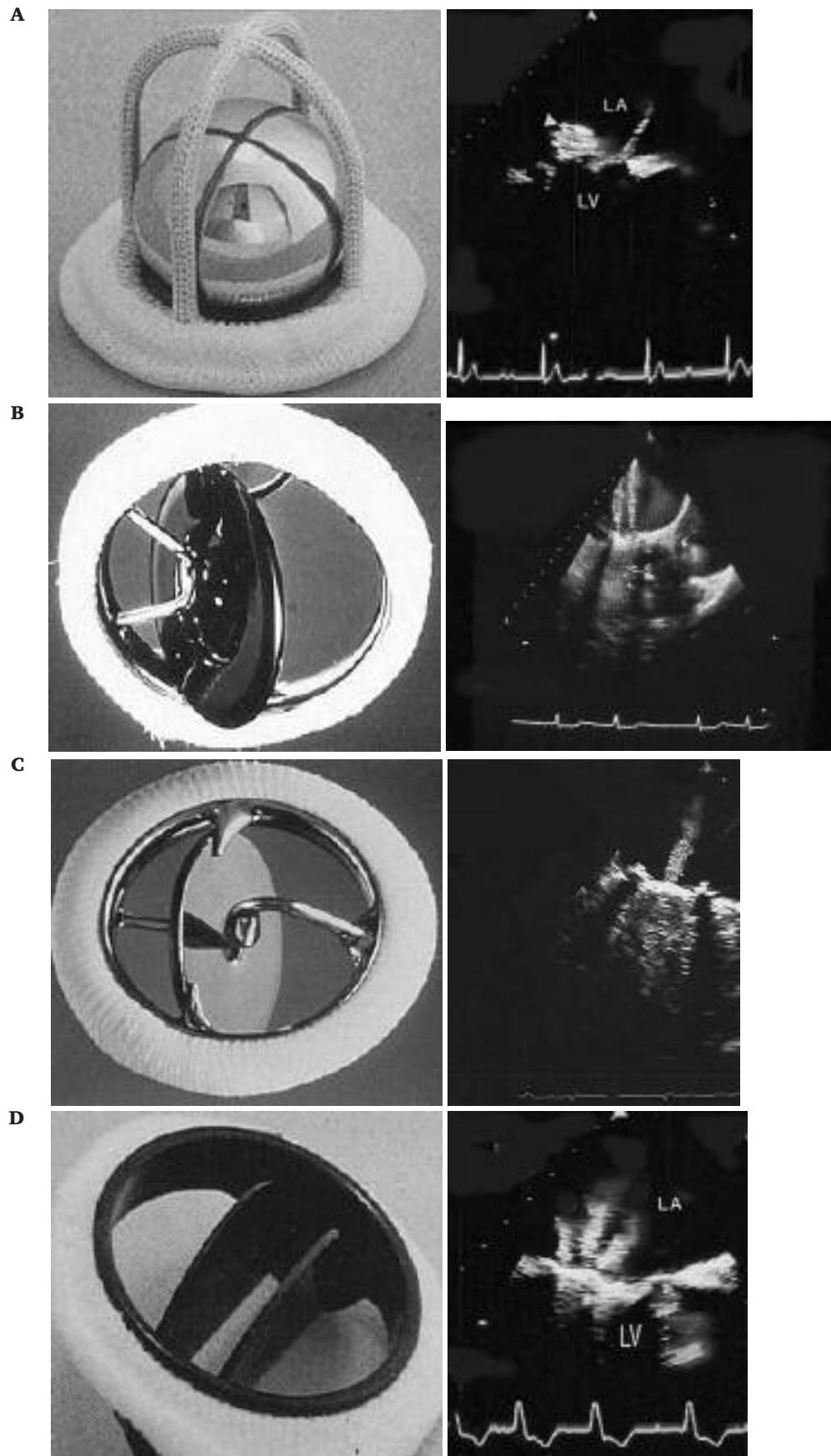|	*Duration*	*Central Jets*	*Peripheral Jets*	*Backflow Volume (mL per cycle)*
Mechanical prostheses				
Starr Edwards	Early systole	0	2, confluent	4
Björk Shiley	Holosystolic	0	2	8
Medtronic Hall	Holosystolic		2	5.5
	Mid + late	1		
	Systolic			
St. Jude Medical	Holosystolic	1-2	2	4.5
Bioprostheses				
Stented	Early	1	0	2
Stentless	Early	1	0	2
Homografts		0	0	—

Figure 3. Normal patterns of back flow in prosthetic valves. Note that the closure and leakage backflow pattern is dependent on the prosthesis design. **(A)** Starr Edwards prosthesis *(left)* and leakage backflow *(right)*. **(B)** Björk-Shiley prosthesis *(left)* and leakage backflow *(right)*; **(C)** Medtronic Hall valve *(left)* and leakage backflow *(right)*; and **(D)** Saint Jude Medical valve *(left)* and leakage backflow *(right)*. Printed with permission of: Van den Brink RBA, et al. Am J Cardiol 63:1471-1474, 1989 (reference 12).
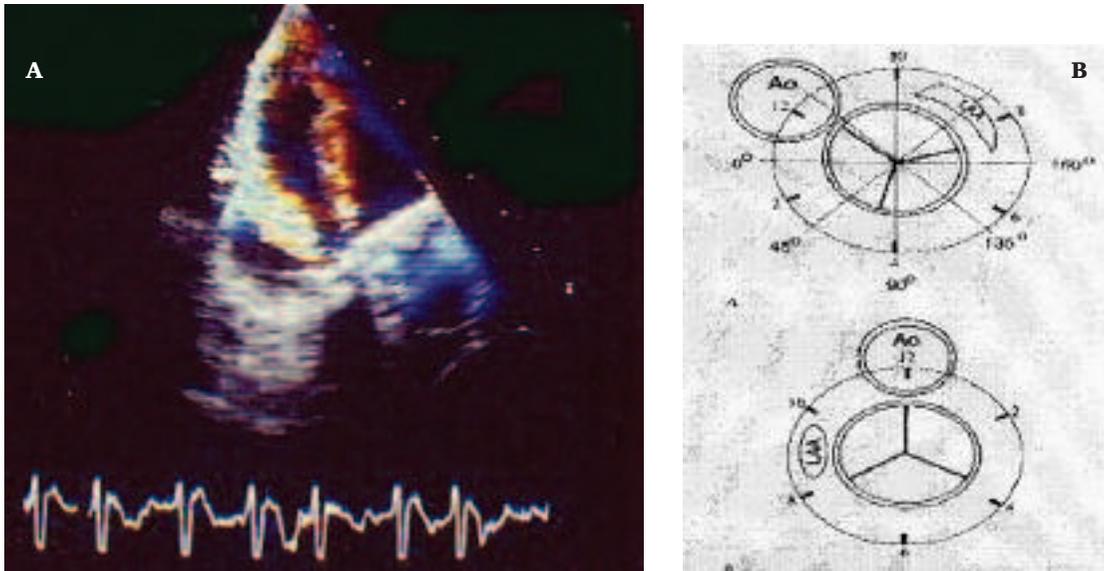
Figure 4. Pathologic leakage of prosthetic valves. Note the difference of jet characteristics between pathologic leakage and normal leakage backflow. **(A)** Normal backflow low-velocity non-aliasing jet encoded in a homogeneous color and pathologic turbulent crescent-shaped jet encoded to the left atrial wall.[12] Printed with permission of: Van den Brink RBA, et al. Am J Cardiol 63:1471-1474, 1989 (reference 12). **(B)** *A,* Reference view displaying the prosthetic mitral valve and its relationship to the aortic root *(Ao)* and left atrial appendage *(LAA)* as seen from the left ventricular apex. The hours of a clock face corresponding to those shown in the surgical perspective, have been overlaid. *B,* Surgical view of prosthetic mitral valve and its relationship to the aortic root.[13] Printed with permission of: Foster GP, et al. Ann Thorac Surg 65:1025-1031, 1998 (reference 13).

wall may underestimate the severity of regurgitation because of the Coanda effect (spreading of the jet along the atrial wall). If such an eccentric jet extends to the posterior left atrial wall, mitral regurgitation is deemed to be severe. In addition, systolic flow reversal in the pulmonary vein, diastolic forward transprosthetic flow with a Vmax exceeding 2 m/sec (in combination with a short P1/2 time), and a diameter of the neck of the mitral regurgitation jet at the regurgitant orificium (vena contracta) of 6 mm or more are all signs of severe mitral regurgitation.

Problems in Prosthetic Valves

Valve Prosthesis-Patient Mismatch

It is very important to prevent valve prosthesis-patient mismatch, that means, a prosthetic valve that is too small for the patient. Valve prosthesis-patient mismatch can be prevented by measurement of the LVOT diameter (=aortic annulus diameter) in a 120° long-axis view and warning the surgeon if this diameter seems to be less than 23 mm in a normal adult patient. If the LVOT is small, for example, a stentless valve may be used or even an annuloplasty can be performed.

Pibarot et al[14] has described three easy steps to prevent aortic valve prosthesis-patient mismatch (AVP-PM)

Step I: Calculate the patient's BSA: (Weight in kg)$^{0.425}$ × (Length in cm)$^{0.725}$ × 0.007184.

Step II: Determine the minimal requirement for prosthetic valve effective orifice area to prevent AVP-PM. See Table 3.

Step III: Choose a prosthesis using reference values for effective AVA of different types and sizes of prostheses.[15]

Valve prosthesis-patient mismatch should be suspected if in a normal adult (BSA ≥ 1.7 m²), an aortic prosthesis smaller than 23 mm is inserted or a mitral prosthesis smaller than 27 mm. Valve prosthesis-patient mismatch is found much more often with aortic than with mitral valve protheses.
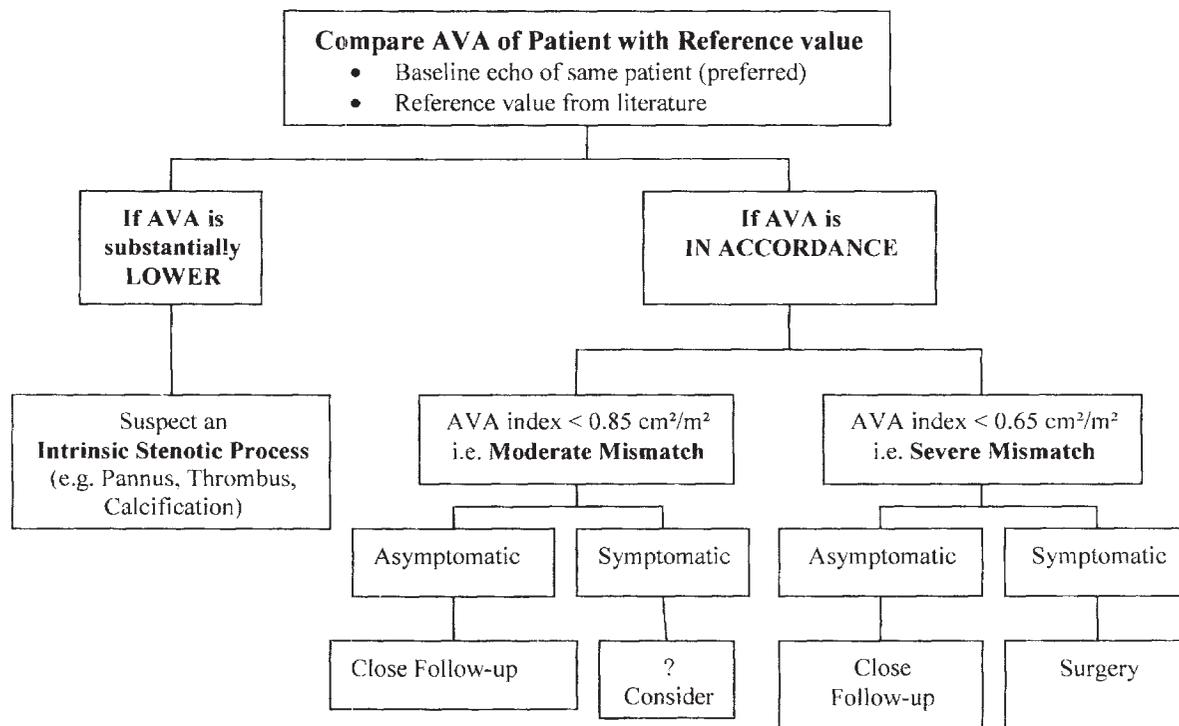
AVP-PM has been defined as follows:

- mild AVP-PM is said to be present if the effective prosthetic orifice area index is 0.85 to 1.75 cm²/m²,

Table 3. Effective Valve Area of an Aortic Prosthesis According to BSA, Necessary to Prevent Clinically Important Aortic Prosthesis-Patient Mismatch

Patient BSA (m²)	Minimal Valve EOA (cm²) for indexed EOA >0.85 cm²/m² (Ideal)	Minimal Valve EOA (cm²) for indexed EOA >0.80 cm²/m²	Minimal Valve EOA (cm²) for indexed EOA >0.75 cm²/m²
1.50	1.28	1.20	1.13
1.55	1.32	1.24	1.16
1.60	1.36	1.28	1.20
1.65	1.40	1.32	1.24
1.70	1.45	1.36	1.28
1.75	1.49	1.40	1.31
1.80	1.53	1.44	1.35
1.85	1.57	1.48	1.39
1.90	1.62	1.52	1.43
1.95	1.66	1.56	1.46
2.00	1.70	1.60	1.50
2.05	1.74	1.64	1.54
2.10	1.79	1.68	1.58

BSA, body surface area; *EOA,* effective orifice area.

Figure 5. Suggestion for management of a patient with valve prosthesis-patient mismatch.

- moderate AVP-PM if it is 0.65 to 0.85 cm²/m², and
- severe AVP-PM exists if the effective prosthetic valve area index is 0.65 cm²/m² or less.

Patient-valve mismatch with an intrinsically normal functioning prosthetic valve should be distinguished from intrinsic obstruction of the prosthetic valve. Differential diagnosis and management of these problems are depicted in Figure 5.

Intrinsic Stenotic Process Leading to Pathologic Prosthetic Valve Obstruction

It is difficult, if not impossible, to distinguish pathologic sources of prosthetic valve obstruction, such as pannus, thrombus, or vegetations, solely by their echocardiographic characteristics.[16,17] How can pannus be distinguished from thrombus? Pannus has an insidious clinical course of symptoms and increase of gradient, whereas thrombus often has a short history of rapidly increasing symptoms, absent or muffled opening or closure clicks, and a history of inadequate anticoagulation.

Pannus may grow over the valve housing and pivot guards and restrict the opening of the valve without restricting the closing of the leaflet. Pannus consists of echodense material mainly on the inflow side of the prosthetic valve with approximately the same echo intensity as the valve housing. Pannus is often difficult to visualize with two-dimensional (2D) echocardiography but might be suspected if (1) compared with the baseline echo, there is an important increase of the mean gradient and decrease of valve area or velocity index without another obvious cause and (2) a restricted valve opening at cinefluoroscopy.[18]

Lin et al[16] found that independent predictors for thrombus were (1) an increased gradient (Pmax Aortic ≥ 50 mm Hg, Pmean of mitral prosthesis ≥ 10 mm Hg), (2) a mobile mass at the prosthetic valve, (3) a mass attached to the occluder, and (4) an international normalized ratio of 2.5 or less.

Thromboembolism in a Patient with Prosthetic Valve thrombosis

If a patient presents with a transient ischemic attack or ischemic cerebrovascular accident, a thorough transthoracic and transesophageal echocar-diogram should be performed. At 2D echocardiography, one may find spontaneous echocontrast, echostrands, or even a thrombus at the prosthetic valve or in the left atrial appendage.

Management depends on the presence or absence of a clot on the prosthetic valve and the extent of the clot (Table 4, Figure 6). If a clot is found at echocardiographic examination, a choice should be made between surgery, thrombolysis, or conservative treatment, such as with warfarin and heparin.

Studies on thrombolysis in prosthetic valve thrombosis demonstrate a mortality that compares well with surgery; however, there is a substantial morbidity, including embolism, stroke and recurrence rate, which is higher than if surgery is performed (Table 5).

The overall mortality of surgery in prosthetic valve thrombosis is 12.3%. However, this high mortality is caused by a very high 17.5% mortality of patients in New York Heart Association (NYHA) class IV, whereas mortality in NYHA classes I to III is substantially lower at 4.7%.[19]

Table 4. Management if No Clot is Found at Transthoracic and Transesophageal Echocardiography

If INR was inadequate before the event
- Optimize anticoagulation level
- If the patient is not taking aspirin, add aspirin (80 or 100 mg/d)

If INR was adequate before the event
- If the patient is not taking aspirin, add aspirin (80 or 100 mg/d) ***or***
- Increase anticoagulation level of target INR by 0.5 or 1 point (INR ≤ 4.5)

INR, international normalized ratio.

Table 5. Meta-analysis of Studies on Thrombolysis in Prosthetic Valve Thrombosis

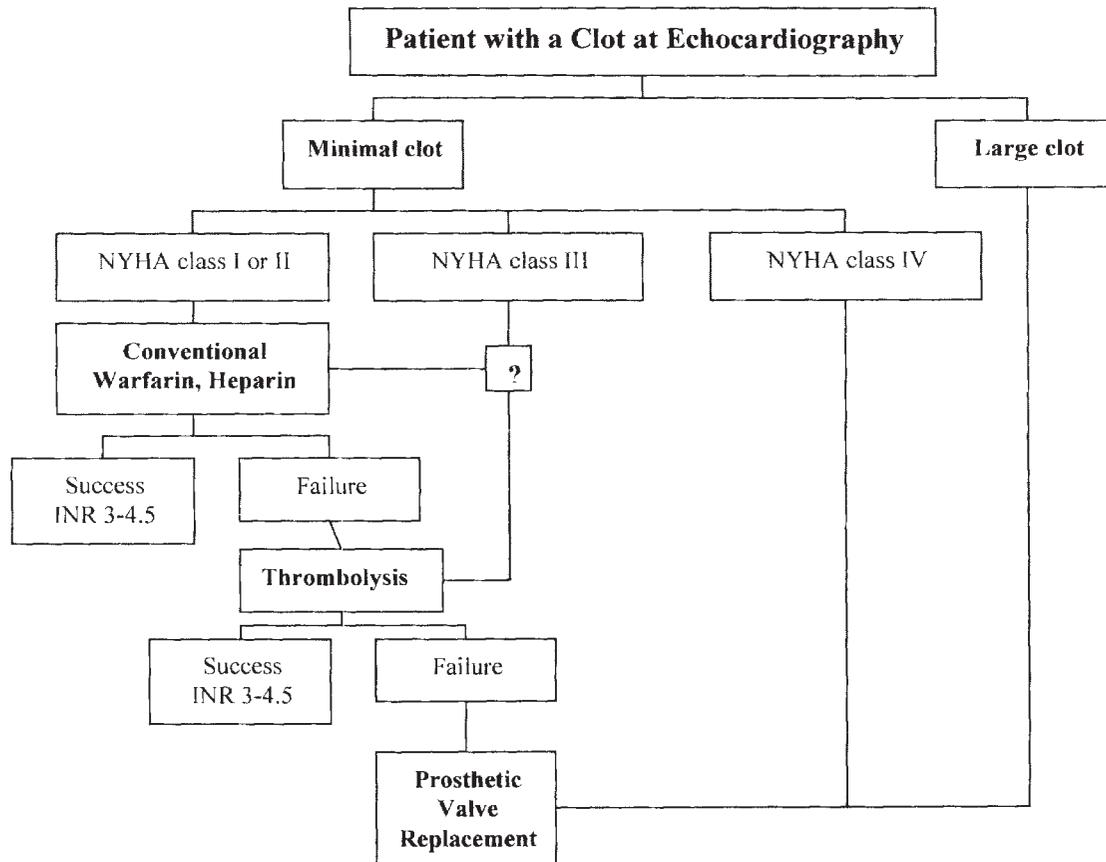|	*Mitral Prosthesis (n=122)(%)*	*Aortic Prosthesis (n=55)(%)*
Success	81	86
Recurrence	18	16
Death (%)	7	6
Embolism	10	10
Stroke	4	2
Nondisabling bleed	11	8

Figure 6. Management if a clot is found at transthoracic or transesophageal echocardiography.[19] This flowchart is based on the results of a systematic review of the literature on thrombolysis in prosthetic valve thrombus. (Reprinted with permission.)

Persisting Fever in a Patient with a Prosthetic Valve: Is it Endocarditis?

Endocarditis is a clinical diagnosis, but it can be confirmed by 2D echocardiography. Echocardiographic signs of endocarditis in prosthetic valves are vegetation, abscess, prosthetic valve dehiscence, and ruptured abscess or fistula (see Figure 7).

TEE plays an important role in the timing of surgical intervention in prosthetic valve endocarditis by the finding of:

• Large vegetations in a patient with systemic thromboembolic complications. A cerebrovascular accident is not a contraindication for heart surgery provided that there is no cerebral hemorrhage and the time between the embolic event and surgery is short, preferably less than 72 hours, so that the blood-brain barrier can be expected not to be significantly disturbed.
• Prosthetic valve dehiscence with rocking motion of the prosthetic valve in patients with active endocarditis. This is an indication for early surgical intervention.
• Paravalvular leakage. This can progress very rapidly because of further tearing loose of sutures.
• A periprosthetic abscess. This can often not be cured with antibiotic treatment alone. If the patient is treated with antibiotics alone, serial TEE examinations should be performed to monitor the extent of the abscess to optimize the timing of surgical intervention.
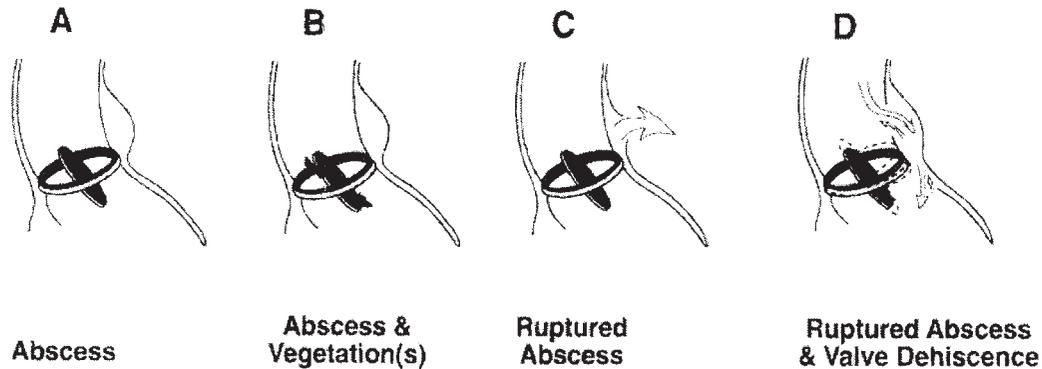
Figure 7. Prosthetic valve infections. Printed with permission of: *The Practice of Clinical Echocardiography*, edited by C. M. Otto, page 812; WB Saunders Company, 1997.
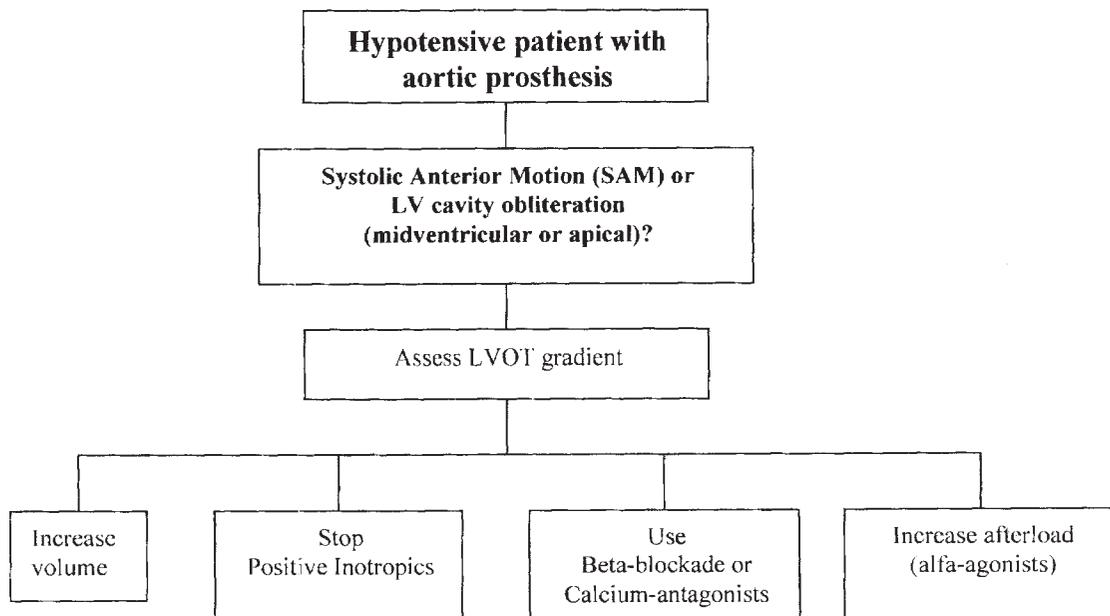

Figure 8. Treatment of patients with a dynamic gradient.

Hypotension in a Patient With an Aortic Prosthesis

In patients with an aortic prosthesis and hypotension, always be prepared for a dynamic gradient. Because patients with an aortic valve prosthesis quite often have a small concentric hypertrophic left ventricle, they are prone to develop high dynamic gradients in case of hypovolemia. See Figure 1. If a dynamic gradient is found in a hypotensive patient with an aortic prosthesis, one should increase volume, stop positive inotropic drugs, use β-blockers or calcium antagonists, or if these measures, fail increase afterload (α-agonists). See Figure 8.

Conclusion

Physicians who take care of patients with a prosthetic valve should be familiar with the characteristics of normal and abnormally functioning prosthetic valves.

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