VALVULAR HEART DISEASE

Doppler echocardiographic evaluation of prosthetic valve function

Philippe Pibarot,[1] Jean G Dumesnil[2]

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[1]Institut Universitaire de Cardiologie et de Pneumologie de Québec/Québec Heart & Lung Institute, Québec, Canada
[2]Department of Medicine, Laval University, Québec, Canada

Correspondence to
Dr Philippe Pibarot, Institut Universitaire de Cardiologie et de Pneumologie de Québec, 2725 Chemin Sainte-Foy, Québec G1V-4G5, Canada; philippe.pibarot@med.ulaval.ca

Owing to its versatile, non-invasive, radiation-free, and low cost nature, Doppler echocardiography is undoubtedly the method of choice to evaluate prosthetic valve function. This evaluation follows the same principles used for the evaluation of native valves, with some important specifics and caveats described in this article. A complete echocardiography includes two dimensional imaging of the prosthetic valve, evaluation of valve leaflet/occluder morphology and mobility, measurement of the transprosthetic gradients and valve effective orifice area (EOA), estimation of the degree of regurgitation, evaluation of left ventricular size and systolic function, and calculation of systolic pulmonary arterial pressure.[1]

TIMING OF ECHOCARDIOGRAPHIC FOLLOW-UP
Ideally, a baseline postoperative transthoracic echocardiography (TTE) study should be performed 3–12 weeks after surgery, when the chest wound has healed, ventricular function has improved, and anaemia with its associated hyperdynamic state has resolved.[1 w1 w2] However, if the patient is being transferred and/or may not return, it may be best to perform the study before hospital discharge, although image quality and reliability of measurements are often suboptimal. Annual echocardiography is recommended in patients with bioprosthetic valves after the first 5 years, whereas in patients with mechanical valves, routine annual echocardiography is not indicated in the absence of a change in clinical status.[1 w1 w2]

DETECTION AND QUANTIFICATION OF PROSTHETIC VALVE STENOSIS
The appearance of a new murmur or symptom in a patient with a prosthetic valve should prompt an urgent TTE study and, if indicated, trans-oesophageal echocardiography (TOE). However, the initial suspicion of prosthetic valve stenosis may be the incidental finding of abnormally high flow velocities and gradients detected during a routine examination. Table 1 presents the Doppler echocardiographic criteria for the detection and quantification of prosthetic valve stenosis.

Leaflet morphology and mobility
Prosthetic valve stenosis is generally associated with abnormal valve morphology and/or mobility (table 1, figure 1). TTE imaging of the valve occluder is often difficult to obtain because of reverberations and shadowing caused by the prosthetic valve

components. TOE can provide improved image quality and thereby improved detection of cusp calcification and thickening, valvular vegetations due to endocarditis, thrombus or pannus, and reduced leaflet/disc/ball mobility (figure 1).[2–4] In the case of mechanical prostheses, evaluation of occluder mobility can be attempted with some degree of success by TTE or TOE, but in our experience valve cine-fluoroscopy is definitely the best, most economical, and least invasive technique that can be used for this purpose.

Quantitative parameters
Quantitative parameters of prosthetic valve function include transprosthetic flow velocity and pressure gradients, valve EOA, and Doppler velocity index (DVI).

Transprosthetic velocity and gradient
The principles of interrogation and recording of flow velocity through prosthetic valves are similar to those used in evaluating native valve stenosis.[1] One should, however, bear in mind that the fluid dynamics of the mechanical valves may differ substantially from those of the native valve (figure 2). The flow is eccentric in the monoleaflet valves and composed of three separate jets in the bileaflet valves. Because the direction of the transprosthetic jet may be eccentric, multi-windows examination should be carefully performed to detect the highest velocity signal in prosthetic valves. Occasionally, an abnormally high jet gradient corresponding to a localised high velocity may be recorded by continuous wave (CW) Doppler interrogation through the smaller central orifice of the bileaflet mechanical prostheses in the aortic or mitral position (figure 2).[5] This phenomenon may lead to an overestimation of gradient and a false suspicion of prosthesis dysfunction.

It is important to keep in mind that high velocity or gradient alone is not proof of intrinsic prosthetic obstruction (table 1) and may be secondary to prosthesis–patient mismatch (PPM), high flow conditions, prosthetic valve regurgitation, or localised high central jet velocity in bileaflet mechanical valves. PPM is not an intrinsic dysfunction of the prosthesis, per se. This problem indeed occurs when the EOA of a normally functioning prosthesis is too small in relation to the patient's body size (and thus cardiac output requirements), resulting in abnormally high postoperative velocities and gradients.[6–8] Significant prosthetic valve regurgitation may also cause the flow rate across the prosthesis to increase, resulting in increased gradients.

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Table 1 Doppler echocardiographic criteria for detection and quantification of prosthetic valve stenosis

	Normal		Possible stenosis		Significant stenosis	
	Aortic	Mitral	Aortic	Mitral	Aortic	Mitral
Valve structure and motion						
Mechanical or bioprosthesis	Normal	Normal	Often abnormal**	Often abnormal**	Abnormal**	Abnormal**
Doppler quantitative parameters						
Peak velocity (m/s)* †	<3	<1.9	3—4	1.9—2.5	>4	>2.5
Mean gradient (mm Hg)* †	<20	≤5	20—35	6—10	>35	>10
Doppler velocity index* ¶	≥0.30	<2.2	0.25—0.29	2.2—2.5	<0.25	>2.5
Effective orifice area (cm²)* §	>1.2	≥2	0.8—1.2	1—2	<0.8	<1
Effective orifice area versus normal reference value* §	Reference±1SD	Reference±1SD	<Reference−1SD	<Reference−1SD	<Reference−2SD	<Reference−2SD
Contour of the transprosthetic jet‡	Triangular, early peaking	—	Triangular to intermediate	—	Rounded, symmetrical	—
Acceleration time (ms)‡	<80	—	80—100	—	>100	—
Pressure half time (ms)‡	—	<130	—	130—200	—	>200

Adapted in part from Zoghbi et al[1] with permission of the American Society of Echocardiography.
*The criteria proposed for these parameters are valid for near normal or normal stroke volume (ie, stroke volume 50—90 ml).
†These parameters are more affected by low or high flow states including low cardiac output and concomitant prosthetic valve regurgitation.
‡These parameters are influenced by chronotropy, left ventricular (LV) function and atrioventricular compliance.
§The effective orifice area of mitral prosthesis is not valid when more than mild concomitant aortic or mitral regurgitation is present. See table 2 to obtain the normal reference values of effective orifice area for the different models and sizes of prostheses.
¶This parameter is dependent on the size of the LV outflow tract.
**Abnormal mechanical valves: occluder that is immobile or with restricted mobility, thrombus or pannus; abnormal biologic valves: leaflet thickening/calcification, thrombus or pannus.

Transprosthetic jet contour and acceleration time

In a normal valve, the contour of the CW flow velocity through the prosthesis generally has a triangular shape, with early peaking of the velocity and a short acceleration time (ie, the time from the onset of flow to maximal velocity (table 1)). With prosthetic valve obstruction, a more rounded velocity contour is seen, with the velocity peaking almost in mid-ejection, and the acceleration time being prolonged.[1] These parameters should be interpreted with caution as they are also highly influenced by left ventricular (LV) chronotropy and function.

Effective orifice area

The EOA of prosthetic aortic valves is calculated with the continuity equation, similar to native aortic valve EOA:[7 9] $EOA = (CSA_{LVOT} \times TVI_{LVOT})/TVI_{PrAv}$, where CSA_{LVOT} is the cross-sectional area of the LV outflow tract (LVOT) diameter, TVI_{LVOT}, the time—velocity integral obtained by pulsed wave Doppler in the LVOT, and TVI_{PrAv}, the time—velocity integral obtained by CW Doppler through the aortic prosthesis. When measuring the EOA of prosthetic aortic valves, a few specific caveats should be taken into consideration. (1) The cross-sectional area of the LVOT is derived from the diameter measurement just underneath the prosthesis from the parasternal long axis zoomed view assuming a circular geometry. This measurement is often difficult because of the reverberations and artefacts caused by the prosthesis stent or sewing ring. Particular attention should be paid not to mistake the inner border of the prosthesis stent/ring for the inner edge of the LVOT. It is important to emphasise that the substitution of the LVOT diameter by the labelled prosthesis size in the continuity equation is not a valid method to determine the EOA of aortic prostheses.[10] (2) For the recording of LVOT velocity signal, care should be exercised in locating the pulsed wave Doppler sample volume adjacent to the prosthesis while avoiding the region of subvalvular acceleration (this usually requires a position 0.5—1 cm below the sewing ring towards the apex). (3) The continuity equation method is also valid in the presence of concomitant prosthetic aortic valve regurgitation.

The EOA of mitral prostheses is calculated by the continuity equation using the stroke volume measured in the LVOT.[7] However, this method cannot be applied when there is more than mild concomitant mitral or aortic regurgitation. The pressure half-time method is not valid to estimate the valve EOA of mitral prostheses.[8 11] Nonetheless, the pressure half-time may be useful if it is significantly delayed (table 1) or shows significant lengthening from one follow-up visit to the other despite similar heart rates.

The cut-off values of EOA proposed in the American Society of Echocardiography guidelines to identify prosthetic valve stenosis[1] have an important limitation, given that they overlap substantially with the normal reference values of EOA of several prostheses models. In our experience, the recognition of prosthetic valve stenosis is better achieved by comparing the measured EOA to the normal reference value of EOA for the model and size of prosthesis implanted in the patient (table 2), rather than applying fixed cut-off values to all patients regardless of the characteristics of their prosthesis.[12] If the measured EOA is less than the reference EOA −1 SD, one should suspect possible stenosis (tables 1 and 2). If the measured EOA is less than reference EOA −2SD, there is a high likelihood of significant stenosis.

Doppler velocity index

The DVI is a dimensionless ratio of the proximal flow velocity in the LVOT to the flow velocity through the aortic prosthesis: $DVI = V_{LVOT}/V_{PrAv}$. Time—velocity time integrals may also be used in place of peak velocities to calculate $DVI = TVI_{LVOT}/TVI_{PrAv}$. In the case of prosthetic mitral valves, the DVI is calculated by dividing the time—velocity
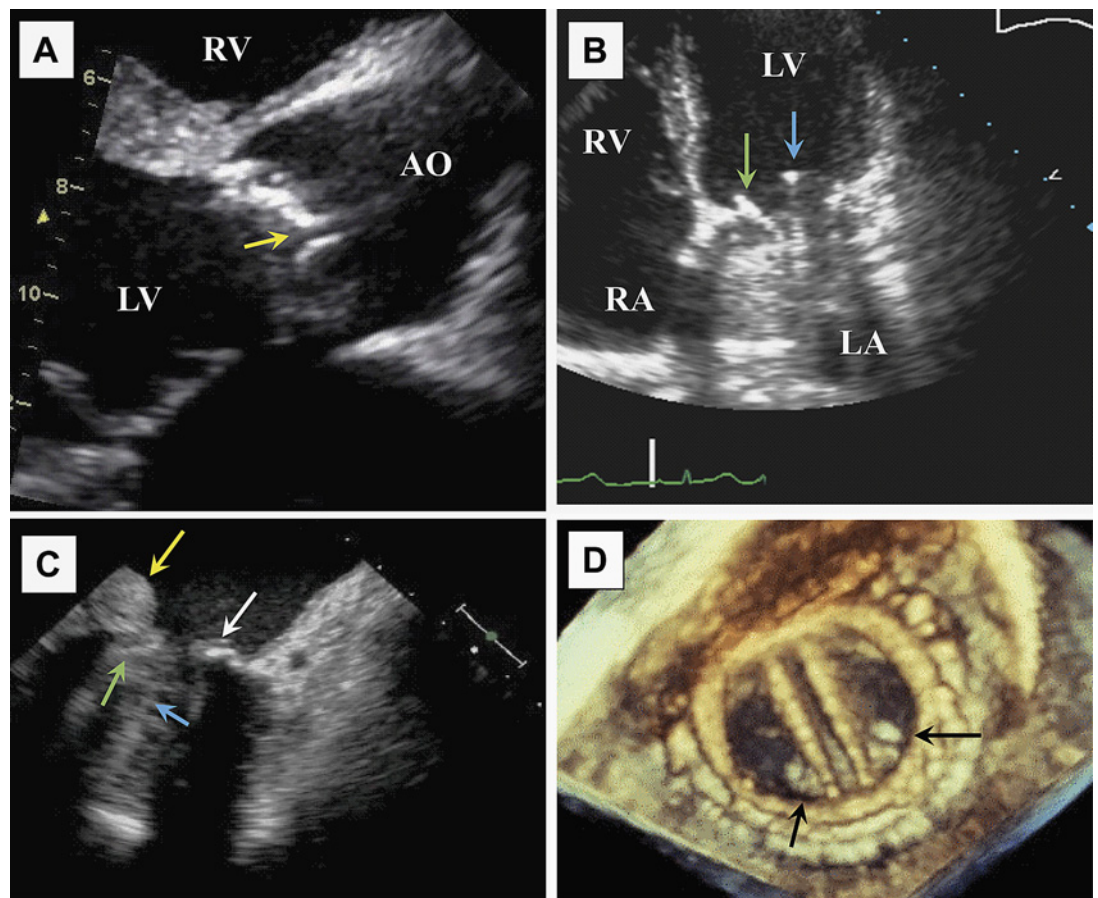
Figure 1 Evaluation of prosthetic valve leaflet morphology and mobility. (A) Transthoracic echocardiographic (TTE) systolic image of a patient with thickening and reduced mobility of aortic bioprosthetic valve cusps (yellow arrow). (B) TTE view of mitral bileaflet mechanical prosthesis in diastole with a fixed leaflet (green arrow); the other leaflet is still mobile (blue arrow). (C) Transoesophageal echocardiographic (TOE) diastolic image of a patient with obstructed mitral bileaflet mechanical valve (yellow arrow: large size thrombus; white arrow: pannus; blue arrow: reverberation caused by the mobile leaflet; green arrow: immobile leaflet). (D) Three dimensional TOE diastolic view of a mitral bileaflet mechanical valve from the left atrium. This image shows thrombi (black arrows) attached to the hinge mechanism of the valve; the motion of the leaflets is not impaired. Courtesy of Dr John Chambers, Guy's and St Thomas Hospitals, London UK (panel A) and Dr Steven A Goldstein, Washington Hospital Centre (panel C). AO, aorta; LA, left atrium; RA, right atrium; LV: left ventricle; RV: right ventricle.

integral of the transprosthetic flow by that of the LVOT flow: $DVI = TVI_{PrMv}/TVI_{LVOT}$. These parameters can be helpful to screen for valve obstruction, particularly when the cross-sectional area of the LVOT cannot be obtained (table 1).[13]

DETECTION AND QUANTIFICATION OF PROSTHETIC VALVE REGURGITATION

Assessment of severity of prosthetic aortic valve regurgitation is generally much more complex than in native valves because of the high prevalence of paravalvular regurgitation and eccentric jets (figure 3). Care is needed to separate physiologic from pathologic prosthesis regurgitation. Mechanical prostheses have a normal regurgitant volume known as leakage backflow. As opposed to the pathologic regurgitant jets, the normal leakage backflow jets are characterised by being short in duration, narrow, and symmetrical (figure 3). In the case of pathologic regurgitation, it is also important to localise the origin of the regurgitant jet(s) in order to distinguish paravalvular from transvalvular regurgitation.

Imaging considerations

TTE generally provides a good visualisation of the LVOT and of prosthetic aortic regurgitation. TOE may be useful to identify the origin (paravalvular vs transvalvular) of the regurgitant jets in technically difficult TTE studies and to identify the mechanism of regurgitation and the associated complications such as flail bioprosthetic cusp, presence of pannus, thrombus, vegetations or masses interacting with occluder closure, abscess formation, or prosthesis dehiscence.[2] Assessment of prosthetic mitral regurgitation by TTE is problematic because the left atrium is largely occulted by the acoustic shadowing due to the metallic components of the prosthesis (figure 4).[1] This problem is more frequent in mechanical valves than in bioprosthetic valves. Hence, TOE should be systematically performed when there is a clinical or TTE suspicion of pathologic mitral regurgitation.[2] TOE is indeed superior to TTE in detecting prosthetic mitral regurgitation and determining its localisation and mechanism.[4 14]
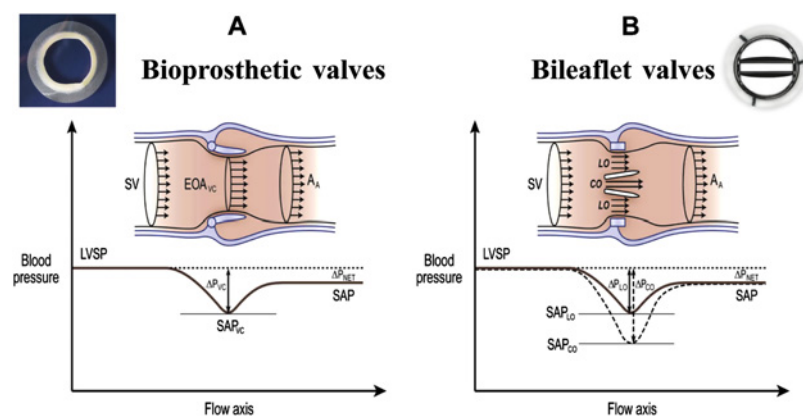
Figure 2 Schematic representation of velocity and pressure changes from the left ventricular outflow tract (LVOT) to the ascending aorta (A_A) in the presence of a stented bioprosthesis (left) and a bileaflet mechanical valve (right), illustrating the phenomena of pressure recovery and localised high gradient. Because of pressure recovery, velocities are lower and systolic arterial pressure (SAP) is higher at the distal aorta than at the level of the vena contracta (VC). This is further exaggerated in the case of a bileaflet valve, in which the velocity is locally higher in the central orifice (CO) and thus pressure drop (ΔP) is higher at that level. Doppler gradients are estimated from maximal velocity at the level of the vena contracta and represent the maximal pressure drop, whereas invasive estimation of gradients usually reflect net pressure difference (ΔP_{NET}) between LV systolic pressure (LVSP) and ascending aorta. LO, lateral orifice; SV, stroke volume in LVOT. Adapted from Zoghbi et al[1] with permission of the American Society of Echocardiography.

Parameters of prosthetic valve regurgitation severity

The same principles and methods used for grading severity of native valve regurgitation[w3–w5] can be used for prosthetic valve regurgitation (tables 3 and 4).[1] It must be remembered, however, that there are very limited data on the application and validation of quantitative parameters such as the width of the regurgitant jet or the vena contracta, the effective regurgitant orifice area, and the regurgitant volume in the context of prosthetic valves.[15 16] Given that all parameters of prosthetic valve regurgitation have important limitations and may be subject to measurement errors, a comprehensive, multi-parametric approach is highly recommended (tables 3 and 4).[1]

Aortic prosthetic valve regurgitation

The ratio of regurgitant jet diameter/LVOT diameter may be overestimated in the case of eccentric or crescent shaped jets and underestimated in the case of jets impinging the wall of the LVOT or the anterior mitral valve (table 3).[1 w3–w5] In contrast to native valves, the width of the vena contracta may be difficult to measure accurately in the long axis view due to the shadowing caused by the prosthesis ring or stent.

For semi-quantitative evaluation of the severity of paravalvular regurgitation, careful imaging of the

Table 2 Normal reference values of effective orifice areas for the prosthetic valves

Prosthetic aortic valves							
Prosthetic valve size (mm)	**19**	**21**	**23**	**25**	**27**	**29**	**Reference**
Stented bioprosthetic valves							
Mosaic	1.1±0.2	1.2±0.3	1.4±0.3	1.7±0.4	1.8±0.4	2.0±0.4	w8
Hancock II	–	1.2±0.2	1.3±0.2	1.5±0.2	1.6±0.2	1.6±0.2	w8
Carpentier-Edwards Perimount	1.1±0.3	1.3±0.4	1.50±0.4	1.80±0.4	2.1±0.4	2.2±0.4	w8
Carpentier-Edwards Magna	1.3±0.3	1.5±0.3	1.8±0.4	2.1±0.5	–	–	w9, w10
Biocor (Epic)	1.0±0.3	1.3±0.5	1.4±0.5	1.9±0.7	–	–	w11, w12
Mitroflow	1.1±0.2	1.2±0.3	1.4±0.3	1.6±0.3	1.8±0.3	–	w13
Stentless bioprosthetic valves							
Medtronic Freestyle	1.2±0.2	1.4±0.2	1.5±0.3	2.0±0.4	2.3±0.5	–	w8
St Jude Medical Toronto SPV	–	1.3±0.3	1.5±0.5	1.7±0.8	2.1±0.7	2.7±1.0	w8
Mechanical valves							w8
Medtronic-Hall	1.2±0.2	1.3±0.2	–	–	–	–	w8
St Jude Medical Standard	1.0±0.2	1.4±0.2	1.5±0.5	2.1±0.4	2.7±0.6	3.2±0.3	w8
St Jude Medical Regent	1.6±0.4	2.0±0.7	2.2±0.9	2.5±0.9	3.6±1.3	4.4±0.6	w14
MCRI On-X	1.5±0.2	1.7±0.4	2.0±0.6	2.4±0.8	3.2±0.6	3.2±0.6	w15
Carbomedics Standard and Top Hat	1.0±0.4	1.5±0.3	1.7±0.3	2.0±0.4	2.5±0.4	2.6±0.4	w8
ATS Medical*	1.1±0.3	1.6±0.4	1.8±0.5	1.9±0.3	2.3±0.8	–	w16

Prosthetic mitral valves						
Prosthetic valve size (mm)	**25**	**27**	**29**	**31**	**33**	**Reference**
Stented bioprosthetic valves						
Medtronic Mosaic	1.5±0.4	1.7±0.5	1.9±0.5	1.9±0.5	–	w17, w18
Hancock II	1.5±0.4	1.8±0.5	1.9±0.5	2.6±0.5	2.6±0.7	w19
Carpentier-Edwards Perimount	1.6±0.4	1.8±0.4	2.1±0.5	–	–	w18
Mechanical valves						
St Jude Medical Standard	1.5±0.3	1.7±0.4	1.8±0.4	2.0±0.5	2.0±0.5	w18
MCRI On-X†	2.2±0.9	2.2±0.9	2.2±0.9	2.2±0.9	2.2±0.9	w15,w18, w20

Effective orifice area is expressed as mean values ±SD available in the literature.
Adapted from Pibarot et al[12] with permission of the American Heart Association.
*For the ATS medical valve, the label valve sizes are: 18, 20, 22, 24, 26 mm.
†The On-X valve has just one size for 27–29 mm and 31–33 mm prostheses. In addition, the strut and leaflets are identical for all sizes (25–33 mm); only the size of the sewing cuff is different.
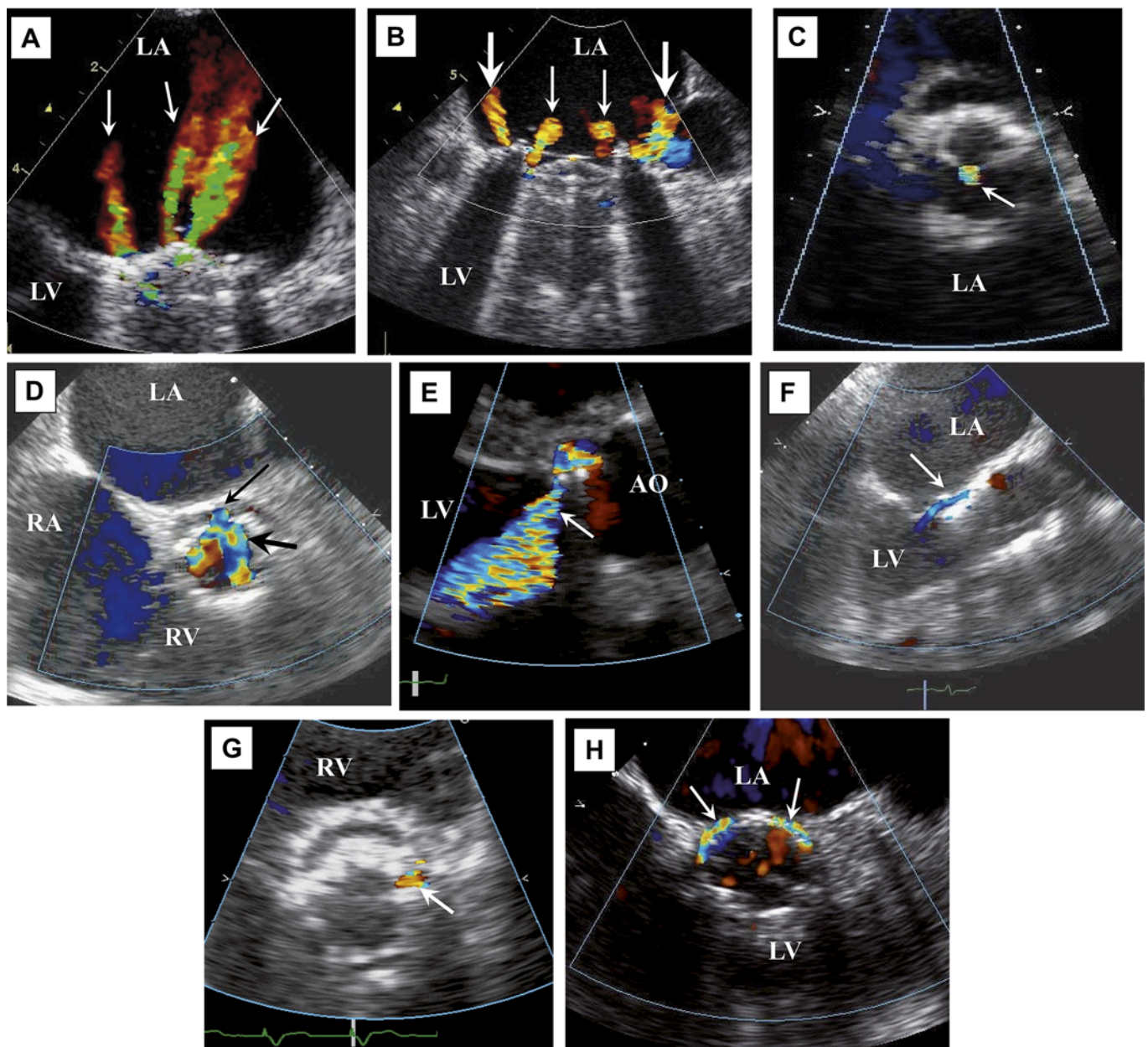
Figure 3 Colour Doppler images of transvalvular and paravalvular regurgitation. The white or black arrows indicate the regurgitant jet(s). (A, B) Transoesophageal echocardiographic (TOE) views of normal physiological regurgitant jets (thin white arrows; A and B) and paravalvular regurgitant jets (thick white arrows; B) in mitral bileaflet mechanical valves. (C) Transthoracic echocardiographic (TTE) short-axis view of mild transvalvular regurgitation in a stented bioprosthetic aortic valve. (D) TOE short axis view of severe transvalvular regurgitation (thick black arrow; one of the cusps is blocked in open position) and mild paravalvular regurgitation (thin black arrow) in a transcatheter bioprosthetic aortic valve. (E) TTE long axis view of moderate paravalvular eccentric regurgitant jet in a stented bioprosthetic aortic valve. (F) TOE long axis view of a mild paravalvular regurgitation in a transcatheter bioprosthetic aortic valve. (G) TTE short axis view of a mild paravalvular regurgitation (one single jet occupying <10% of circumference) in a stented aortic bioprosthetic valve. (H) TOE short axis view of a severe paravalvular regurgitation (two jets occupying >20% of circumference) in a transcatheter bioprosthetic aortic valve. Courtesy of Dr John Chambers, Guy's and St Thomas Hospitals, London (panels A and E) and Dr Arsène Basmadjian, Montreal Heart Institute (panel B). AO, aorta; LA, left atrium; LV, left ventricle; RA, right atrium.

neck of the jet in a short axis view, at the level of the prosthesis sewing ring (surgically implanted prostheses) or stent (transcatheter bioprostheses), allows determination of the circumferential extent of regurgitation in the case of paravalvular regurgitation (table 3, figure 3).[1 17 18] The entrainment of the regurgitant (paravalvular or transvalvular) jet in the LVOT may lead to rapid broadening of the jet just after the vena contracta and thus to over-estimation of regurgitant severity when using colour Doppler parameters. Moreover, the estimation of regurgitation severity becomes more complex and less reliable when multiple jets are present.

Mitral prosthetic valve regurgitation

At TTE examination, the presence of 'occult' mitral prosthesis regurgitation should be suspected when the following signs are present: presence of flow

convergence on the LV side of the prosthesis during systole; increased mitral peak E wave velocity, gradient, and/or DVI; unexplained or new worsening of pulmonary arterial hypertension; and a dilated and hyperkinetic LV (table 4).[13] TOE should be systematically performed when there is a clinical or TTE suspicion of occult mitral regurgitation (figure 4).[2]

Because prosthetic regurgitation is often characterised by eccentric and/or multiple jets (figure 3), the proximal isovelocity surface area (PISA) method is difficult to achieve and may grossly under- or overestimate regurgitation severity. Given these important limitations, the volumetric method is often preferred to the PISA method for quantitation of prosthesis regurgitation[1]

SPECIFIC CONSIDERATIONS FOR PARTICULAR TYPES OF VALVE SUBSTITUTES
Stentless aortic valve substitutes

The stentless substitutes (stentless bioprostheses, aortic homografts, and pulmonary autografts) are more likely than stented valves to have minor transvalvular regurgitant jets. In the early postoperative period, the stentless bioprostheses, particularly if implanted by the subcoronary technique, may exhibit higher than expected gradients, which often regress within the next 3 months most likely due to regression of the oedema between the prosthesis and the aortic wall and/or remodelling of the LVOT. In the late postoperative follow-up, the stentless substitutes are more likely than stented

Figure 4 Effect of the position of the mechanical prosthetic valve and of the echocardiographic imaging view on shadowing and masking of a regurgitation jet by Doppler. A higher effect from transthoracic imaging is seen on prostheses in the mitral position compared to the aortic position. Reproduced from Zoghbi et al[1] with permission of the American Society of Echocardiography.

bioprostheses or mechanical valves to develop 'functional' central aortic insufficiency due to dilation of the aortic root. It is thus recommended to measure regularly (ie, every 2 years) the dimensions of the aortic root in patients with a stentless aortic valve substitute.

Transcatheter aortic valves
For the measurement of stroke volume and valve EOA of transcatheter bioprosthetic valves, the

Table 3 Doppler echocardiographic criteria for severity of prosthetic aortic valve regurgitation (central and paravalvular)

	Mild	Moderate	Severe
Valve structure and motion			
Mechanical or bioprosthesis	Usually normal	Usually abnormal†	Usually abnormal†
Doppler parameters (qualitative or semi-quantitative)			
Vena contracta width (mm)*	<3	3—6	>6
Jet width in central jets (% LVOT diameter): colour Doppler*	Narrow (≤25)	Intermediate (26—64)	Large (≥65)
Jet density: CW Doppler	Incomplete or faint	Dense	Dense
Jet deceleration rate (PHT, ms): CW Doppler‡	Slow (>500)	Variable (200—500)	Steep (<200)
LV outflow versus RV outflow ratio: PW Doppler (ratio of stroke volumes or time—velocity integrals)	Slightly increased (>1.2)	Intermediate (>1.5)	Greatly increased (>1.8)
Diastolic flow reversal in the ascending aorta: PW Doppler	Absent or brief early diastolic	Intermediate	Prominent holodiastolic (end-diastolic velocity >18 cm/s)
Circumferential extent of paravalvular regurgitation (%)¶	<10	10—20	>20
Doppler parameters (quantitative)			
Regurgitant volume (ml/beat)	<30	30—59**	≥60
Regurgitant fraction (%)	<30	30—49	≥50
Indirect signs			
LV size§	Normal	Normal/mildly dilated	Dilated

Adapted in part from Zoghbi et al[1] with permission of the American Society of Echocardiography.
*Parameter applicable to central jets and is less accurate in eccentric jets and paravalvular jets.
†Abnormal mechanical valves: immobile occluder, dehiscence or rocking (paravalvular regurgitation); abnormal biologic valves: leaflet thickening/calcification or prolapse, dehiscence or rocking (paravalvular regurgitation).
‡This parameter is influenced by left ventricular compliance.
§Applies to chronic, late postoperative prosthetic aortic valve regurgitation in the absence of other aetiologies.
¶Applies only to paravalvular regurgitation.
**The moderate regurgitation group can be further subdivided into two subclasses: mild-to-moderate regurgitation when regurgitant volume is between 30—44 ml, and moderate-to-severe when it is between 45—59 ml.[w3]
CW, continuous wave; LV, left ventricular; LVOT, LV outflow tract; PHT, pressure half-time; PW, pulsed wave; RV, right ventricular.

Table 4 Doppler echocardiographic criteria for severity of prosthetic mitral valve regurgitation (central and paravalvular)

	Mild	Moderate	Severe
Valve structure and motion			
Mechanical or bioprosthesis	Usually normal	Usually abnormal†	Usually abnormal†
Doppler parameters (qualitative or semi-quantitative)			
Colour flow jet area	Small, central jet (usually <4 cm² or <20% of LA area)	Variable	Large central jet (usually >8 cm² or >40% of LA area) or variable size wall impinging jet swirling in LA
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: PW Doppler	Systolic dominance	Systolic blunting	Systolic flow reversal
Doppler velocity index: PW Doppler	<2.2	2.2–2.5	>2.5
Doppler parameters (quantitative)			
Vena contracta width (mm)	<3	3–6	>6
Regurgitant volume (ml/beat)	<30	30–59**	≥60
Regurgitant fraction (%)	<30	30–49	≥50
Effective regurgitant orifice area (mm²)	<20	20–39**	≥40
Indirect signs			
LV size§	Normal	Normal/mildly dilated	Dilated
LA size	Normal	Normal/mildly dilated	Dilated
Pulmonary hypertension (SPAP ≥50 mm Hg at rest and ≥60 mm Hg at exercise)	Generally absent	Variable	Generally present

Adapted in part from Zoghbi et al[1] with permission of the American Society of Echocardiography.
*Parameter applicable to central jets and is less accurate in eccentric jets.
†Abnormal mechanical valves: immobile occluder, dehiscence or rocking (paravalvular regurgitation); abnormal biologic valves: leaflet thickening/calcification or prolapse, dehiscence or rocking (paravalvular regurgitation).
§Applies to chronic, late postoperative prosthetic aortic valve regurgitation in the absence of other aetiologies.
**The moderate regurgitation group can be further subdivided into two subclasses: (1) mild-to-moderate regurgitation when regurgitant volume is between 30–44 ml or effective regurgitant orifice area (EROA) between 20–29 mm²; and (2) moderate-to-severe regurgitation when the regurgitant volume is between 45–59 ml or EROA between 30–39 mm².[w4]
CW, continuous wave; LA, left atrial; LV, left ventricular; LVOT, LV outflow tract; PHT, pressure half time; PW, pulsed wave; RV, right ventricular; SPAP, systolic pulmonary artery pressure.

LVOT diameter and velocity should be measured immediately proximal to the apical border of the stent. However, if the border of the stent sits low in the LVOT, which may occur more frequently with self-expandable prostheses (such as the CoreValve), it may be preferable to measure the LVOT diameter and velocity within the proximal portion of the stent at approximately 5–10 mm below the bioprosthetic valve leaflets. Paravalvular regurgitation is more common following transcatheter aortic valve implantation versus standard valve replacement (figure 3). Recent studies indeed report incidences of regurgitation of 30–80% with 5–14% being moderate or severe.[17 19 w6] The severity of paravalvular regurgitations appears to remain stable during follow-up. Although very rare, some cases of delayed migration and embolisation of the prosthesis have been reported following transcatheter valve implantation. The distance between the ventricular end of the prosthesis stent and the hinge point of the mitral valve measured in the parasternal long axis view can be used to monitor the position of the prosthesis during follow-up.

INTERPRETATION OF HIGH TRANSPROSTHETIC GRADIENTS

PPM is the most frequent cause of a high gradient after valve replacement, the other potential causes being a central jet artefact in bileaflet prostheses (figure 2), intrinsic valve dysfunction, high flow states or technical errors.[1 12] The following algorithm can be used to interpret these high gradients and make the differential diagnosis (figure 5).[20] The first step in the algorithm is to simply calculate the projected indexed EOA—that is, EOA/body surface area, using the normal reference value of EOA for the type and size of prosthesis having been implanted (table 2). If the result is <0.85 cm²/m² in the aortic position or <1.2 cm²/m² in the mitral position, one can then surmise that PPM is present and that, depending on its degree of severity, it may be partially or totally responsible for the high gradient. PPM is considered to be severe when the indexed EOA is ≤0.65 cm²/m² and ≤0.9 cm²/m² in the aortic and mitral positions, respectively. It is also important to keep in mind that both phenomena—that is, PPM and intrinsic dysfunction—may coexist.

If PPM is not present or not severe enough to explain totally the increased gradient, the next crucial step would be to evaluate leaflet morphology and mobility thoroughly. In the case of mechanical valves, TTE and even TOE often do not allow adequate visualisation of leaflet(s) motion and cine-fluoroscopy should be performed in addition to echocardiography. If leaflet mobility is abnormal (figure 1), dysfunction should be
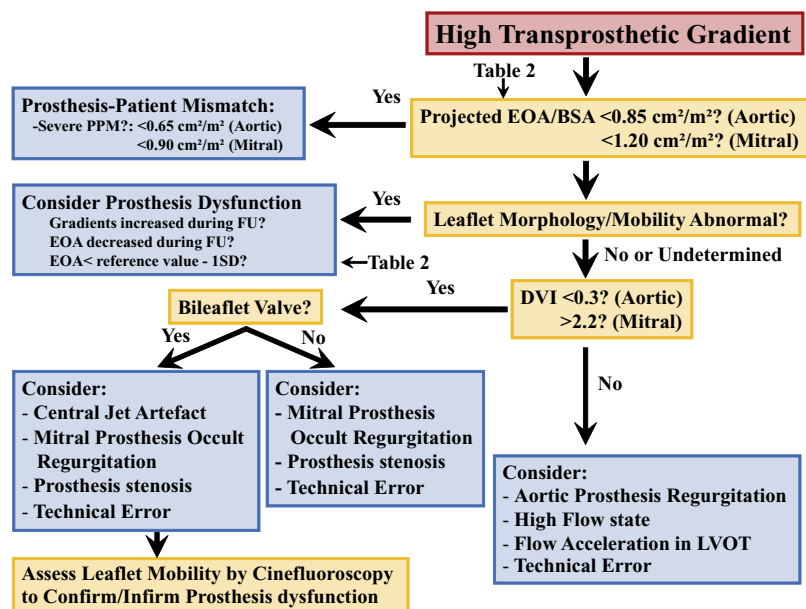
Figure 5 Algorithm for interpreting abnormally high transprosthetic pressure gradients after aortic or mitral valve replacement. BSA, body surface area; DVI, Doppler velocity index; EOA, effective orifice area; FU, follow-up; PPM, prosthesis—patient mismatch. Adapted from Dumesnil and Pibarot,[20] with permission of Springer.

suspected; this can be corroborated if the EOA actually measured by echocardiography is much lower than the normal reference value for the same type and size of prosthesis (table 2) and/or has decreased progressively during follow-up. If, on the other hand, there is still uncertainty with regards to possible abnormal leaflet mobility, one can then proceed to calculate the DVI. If the DVI is

<0.30 in the aortic position or >2.2 in the mitral position and the prosthesis is a bileaflet valve with normal leaflet mobility, the high gradient is likely due to a central jet artefact—that is, a localised high jet velocity through the central orifice (figure 2). On the other hand, if the valve is not bileaflet, intrinsic dysfunction should be reconsidered as above and the possibility of a technical error should also be envisioned. Finally, if the DVI is normal, possible aetiologies for the high gradient include high flow states, aortic regurgitation, flow acceleration in the LVOT, and again a technical error. It should be noted that technical errors, particularly with regards to measurements in the LVOT, are frequent and that this possibility should never be neglected. When confronted with a patient with increased gradient across a mitral prosthesis, abnormal DVI, and normal or undetermined leaflet mobility, one should also consider the possibility of occult trans- or paravalvular regurgitation (figure 5).

PROSTHETIC VALVE THROMBOSIS

If prosthetic valve thrombosis (PVT) is suspected, a comprehensive TTE is performed first, with particular attention directed at evaluation of leaflet mobility and valve haemodynamic function and identification of thrombus and/or pannus (figure 1).[21] In the rare case of massive PVT with haemodynamic instability, TOE will not be needed, as this dramatic presentation constitutes a surgical emergency.[21] In most other cases, TOE will provide important incremental diagnostic information, which will also guide therapeutic management. It is important to differentiate thrombi from a fibrous pannus, which is usually annular in location and typically appears as a very dense immobile echo (figure 1). Pannus formation is more frequent on aortic than on mitral prostheses and, when observed on mitral prosthetic valves, they most often occur on the atrial side of the prosthesis.

The most important factors to consider for therapeutic decision making (figure 6) are: (1) the presence of prosthetic valve obstruction; (2) the size and mobility of the thrombus; and (3) the clinical and haemodynamic condition of the patient. In non-obstructive left-sided PVT confirmed by TTE or TOE, first line treatment comprises a short course of intravenous heparin with close echocardiographic follow-up plus adjustment of warfarin treatment and addition of aspirin (100 mg).[21] However, if the medical treatment is unsuccessful, surgery should be considered in patients with large or mobile thrombi, and thrombolysis with urokinase, streptokinase, or recombinant tissue plasminogen activator is recommended in other patients.[21 22 w1 w2]

Urgent or emergent surgery (ie, thrombectomy or valve replacement) is the treatment of choice in patients with obstructive valve thrombosis (figure 6).[21 22] However, rescue thrombolysis should be considered in patients unlikely to survive surgery or when surgical treatment is unavailable and the patient cannot be transferred.[21 w1 w2]
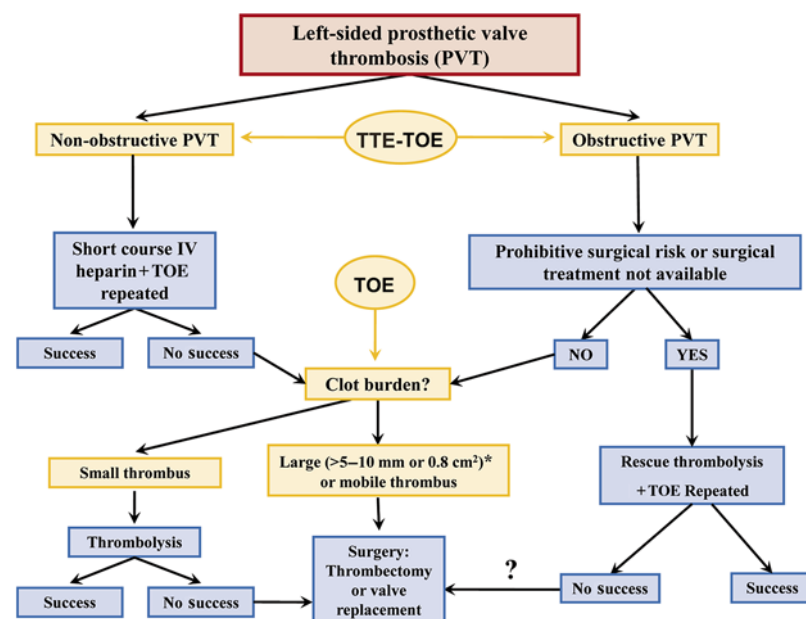

Figure 6 Algorithm for the management of patients with left-sided prosthetic valve thrombosis (PVT). TOE, transoesophageal echocardiography; TTE, transthoracic echocardiography. Adapted from Dumesnil and Pibarot[12] with permission of American Heart Association and from Pibarot and Dumesnil[w21] with permission of Elsevier. *Largest length and area of the thrombus measured from any TOE view.[22]

Doppler echocardiographic evaluation of prosthetic valve function: key points

- ► A comprehensive multiparametric approach that integrates several direct and indirect Doppler echocardiographic indices of valve function is key to detect, quantitate, and manage prosthetic valve dysfunction appropriately.
- ► Transthoracic echocardiography (TTE) may underestimate the presence and severity of prosthetic mitral valve thrombosis and/or regurgitation because of shadowing caused by the valve components. Transoesophageal echocardiography (TOE) is useful to unmask these 'occult' abnormalities.
- ► Assessment of severity of prosthetic aortic valve regurgitation is generally much more complex than in native valves because of the high prevalence of paravalvular regurgitation and eccentric jets.
- ► A high transprosthetic gradient or velocity does not necessarily imply prosthetic valve dysfunction and, vice versa, a low gradient/velocity does not necessarily indicate absence of valve dysfunction.
- ► Evaluation of prosthetic leaflet morphology and mobility by TTE, TOE, or other imaging modalities is fundamental to make the differential diagnosis between the three main potential aetiologies of high transprosthetic gradient: prosthesis—patient mismatch, prosthetic valve dysfunction, and localised high gradient.

Thrombolysis may also be considered in patients with mild to moderate prosthetic valve obstruction who are haemodynamically stable and have a small clot burden.

PROSTHETIC VALVE ENDOCARDITIS

Prosthetic valve endocarditis represents one of the most frequent and severe forms of infective endocarditis.[23] TOE is mandatory in prosthetic valve endocarditis because of its better sensitivity and specificity for the detection of vegetations, abscesses, and paravalvular lesions in this setting.[2] When compared with native valve endocarditis, prosthetic valve endocarditis is characterised by a lower incidence of vegetations and higher incidence of abscesses and paravalvular complications. In mechanical valves, the infection usually involves the junction between the sewing ring and the annulus, leading to paravalvular abscess, dehiscence, pseudoaneurysms, and fistula, whereas in bioprosthetic valve endocarditis, infection is more frequently located on the leaflets, leading to cusp tear, perforation, and vegetations.

The sensitivity and specificity of both TTE and TOE are lower in prosthetic valve endocarditis than in native valve endocarditis.[23] Indeed, the components of the prosthetic valve may hinder the detection of vegetations and abscesses. Moreover, it may be difficult to differentiate a thrombus from a vegetation or bioprosthetic valve degeneration from infective lesions. Consequently, a negative echocardiogram is often observed in prosthetic valve endocarditis and does not rule out the diagnosis of infective endocarditis. If the initial TOE is negative but suspicion for infective endocarditis remains, one should repeat the TOE within 7—10 days and/or use an alternative imaging modality such as multislice computed tomography (CT) and positron emission tomography (PET).

Surgery should be considered in the following situations: failure of medical treatment, haemodynamically significant prosthesis regurgitation or obstruction, especially if associated with deterioration of LV function, large vegetations, paraprosthetic complications, and development of intracardiac fistulas.[24 w7]

FUTURE PERSPECTIVES

One of the most difficult challenges in the Doppler echocardiographic assessment of prosthetic valve function is the quantitation of prosthetic valve regurgitation, particularly when it is paravalvular, eccentric, and/or multi-jets. The analysis of three dimensional colour Doppler images and/or of the backscattered Doppler power analysis may provide interesting avenues for future research in this regard. Real-time three dimensional TOE also enables en-face visualisation of the prosthesis (figure 1D), which may improve detection and localisation of thrombus/pannus, and of trans- or paravalvular regurgitation. Further studies are also needed to promote the development and rational utilisation of multimodality imaging for the evaluation of prosthetic valve function. Indeed, these modalities may nicely complement Doppler echocardiography for assessment of prosthetic valve obstruction (cine-fluoroscopy and multislice CT), regurgitation (MRI), and endocarditis (PET).

CONCLUSION

The particular context of prosthetic valves poses some major challenges in terms of imaging and flow dynamics that push Doppler echocardiography to its limits. A comprehensive approach that integrates several direct and indirect parameters of valve function measured by TTE and TOE is key to assessing prosthetic valve function

appropriately, and other imaging modalities are often needed to complement or confirm the information obtained by Doppler echocardiography.

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Philippe Pibarot and Jean G Dumesnil

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Notes