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iREVIEWS

STATE-OF-THE-ART PAPER

Echo/Doppler Evaluation of Hemodynamics After Aortic Valve Replacement

Principles of Interrogation and Evaluation of High Gradients

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Echocardiography/Doppler is the standard clinical tool for the assessment of hemodynamics after aortic valve replacement. Analysis can include mean and peak transvalvular gradients, dimensionless valve index, effective orifice area from the continuity equation, geometric orifice area from planimetry, and energy loss coefficient. High gradients after aortic valve replacement can be, but are not necessarily, caused by left ventricular outflow obstruction; and not all left ventricular outflow obstruction after aortic valve replacement is due to prosthesis dysfunction. Understanding the methods by which echocardiography and Doppler are used to noninvasively assess aortic valve hemodynamics, and the caveats associated with those methods, can help the clinician distinguish obstructive from nonobstructive causes of high gradients, and prosthesis dysfunction from other causes of obstruction. (J Am Coll Cardiol Img 2010;3:296–304) © 2010 by the American College of Cardiology Foundation

Transvalvular gradients determined using Doppler echocardiography in general correlate well with invasive measures among patients with native aortic stenosis (AS) (1–3) and after aortic valve replacement (AVR) (4). The accuracy, noninvasive nature, broad availability, and absence of exposure to ionizing radiation have established echocardiography/Doppler as the standard for the clinical assessment of heart valve function (5,6), including the assessment of hemodynamics after AVR.

In general, echocardiography/Doppler assessment of prosthetic aortic valve (AV) hemodynamics is the same as the assessment of native AS. However, caveats exist for the noninvasive assessment of AV hemodynamics in general, and the presence of a prosthetic valve can introduce additional confounders. At a time when emphasis is (appropriately) being placed on prosthetic valve hemodynamics, an understanding of the data derived from their noninvasive assessment, including any inherent pitfalls and caveats, also is of importance. This review summarizes the means by which echocardiography/Doppler is used to assess hemodynamics after AVR and the potential caveats associated with its use, and proposes an algorithm for the clinical evaluation of high gradients after AVR.

Methods of Hemodynamic Assessment

Gradients. The most basic noninvasive method for the assessment of AV hemodynamics is with transvalvular gradients. By definition,

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pressure increases proximal to a restrictive orifice; the difference between the pressure proximal and distal to the orifice is a reflection of the degree of obstruction. Catheterization directly measures pressure proximal and distal to the AV; the difference between maximal pressures is the peak-to-peak gradient, and the average difference in pressures over the duration of flow is the mean pressure gradient. Of note, invasive techniques correct for any temporal delay between pressure increase in the aorta relative to the left ventricle (LV) by manually or electronically shifting the pressure traces to overlap.

Doppler echocardiography takes advantage of the acceleration of flow across a restrictive orifice, and the relationship defined by the Bernoulli equation between velocity and pressure, to assess gradients (7). Using the Bernoulli equation, the difference in pressure across a restrictive orifice is defined as:

$$\Delta P = P_1 - P_2 = 4\,(V_2^2 - V_1^2)$$

where P_1 and V_1 are the pressure and velocity, respectively, proximal to the restrictive orifice; and P_2 and V_2 are the pressure and velocity, respectively, distal to the orifice. The peak difference between pressures is the peak instantaneous gradient, and the average difference over the duration of flow is the mean gradient. Unlike the invasive measurement of pressure difference, Doppler echocardiography does not correct for the temporal shift in the timing of pressure in the aorta relative to the LV. Although there is good correlation between mean pressure gradients determined invasively and noninvasively (1–4), the two "peak" pressures determined by the two techniques are inherently different: the Doppler peak instantaneous gradient always is greater than the invasively determined peak-to-peak gradient.

Whether determined invasively or noninvasively, mean pressure gradient is a good measure of valve hemodynamics. However, gradients are sensitive to flow. The Gorlin equation demonstrates the relationship between gradient and flow, defining AV area as cardiac output (flow) divided by the product of heart rate, systolic ejection period, the Gorlin constant, and the square root of the mean gradient. For any fixed valve orifice area, gradient increases in a high-flow state and decreases in a low-flow state. Clinically, a high-flow state can occur because of pain, anxiety, or fever; or in association with medical conditions including anemia and hyperthyroidism. Significant aortic regurgitation also is associated with a high-flow state across the AV, even if forward cardiac output is not increased.

In contrast to mean gradient, peak gradient (peak-to-peak or peak instantaneous) is a less reliable measure of valve hemodynamics, owing to the substantial influence of LV contractility in addition to the influence of transvalvular flow. The peak pressure gradient is an especially unreliable indicator of hemodynamics in the setting of a prosthetic AV, where high velocities are commonly observed immediately after valve opening.

Characteristics of the spectral Doppler envelope beyond simple velocity quantification also can provide information pertinent to the severity of AS. The Doppler envelope associated with normal prosthetic AV function is triangular in shape, and peaks in early systole. With significant AV obstruction, the Doppler envelope becomes more rounded in contour and peaks later in systole; with longer acceleration time and ejection time, and a higher ratio of acceleration time to ejection time (8).

Effective orifice area. Effective orifice area (EOA) is determined using echocardiography Doppler and the continuity equation, and is a reflection of the minimal cross-sectional area of the outflow jet (the *vena contracta*) (7). EOA is calculated as the product of the cross-sectional area of the LV outflow tract (from its diameter, measured in the parasternal long-axis view) and the LV outflow tract velocity time integral (using pulsed-wave Doppler from an apical window), divided by the AV velocity-time integral (using continuous-wave Doppler from an apical, right parasternal, or suprasternal window). Unlike gradients, EOA provides an accurate assessment of stenosis severity independent of flow in most hemodynamic states.

Dimensionless valve index. The dimensionless valve index (DVI) is a unitless ratio of the velocity proximal to and through the AV (8). Typically expressed as the ratio of velocity-time integrals, DVI also can be expressed as the ratio of peak velocities. Similar to EOA, DVI should reflect hemodynamics independent of flow, and can be especially useful for serial assessment when the diameter of the LV outflow tract cannot be reliably measured. In general, DVI < 0.25 suggests significant obstruction.

ABBREVIATIONS AND ACRONYMS

AS = aortic stenosis
AV = aortic valve
AVR = aortic valve replacement
DVI = dimensionless valve index
EOA = effective orifice area
GOA = geometric orifice area
LV = left ventricle/ventricular
PG_{max} = maximal pressure gradient
PG_{net} = net pressure gradient
PPM = prosthesis patient mismatch

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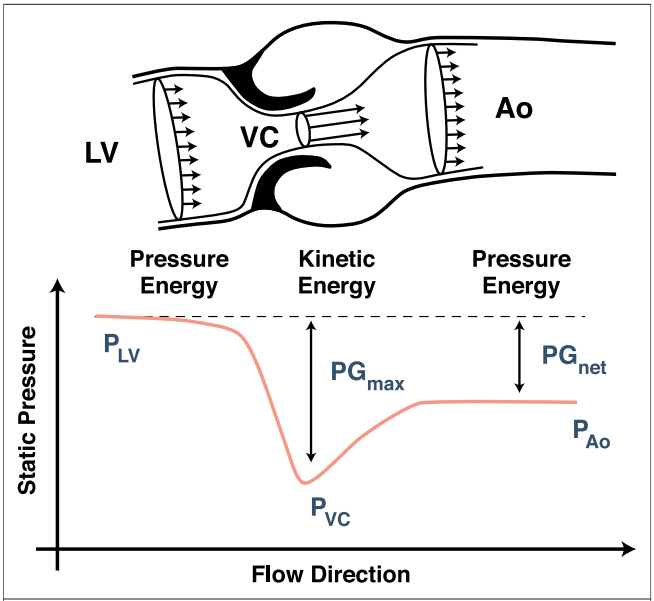

Figure 1. Pressure Recovery Distal to the AV

High static pressure in the left ventricle (LV) is converted to kinetic energy, maximal at the *vena contracta* (VC). In the ascending aorta (Ao), kinetic energy can be either lost as thermal energy or recovered as pressure energy. Pressure recovery is more prominent when the ascending aorta is not dilated. The maximal pressure gradient (PG_{max}) is the difference in static pressures between the left ventricle (P_{LV}) and the *vena contracta* (P_{VC}), and correlates with the Doppler gradient. The net pressure gradient (PG_{net}) is the difference in static pressures between the left ventricle and the ascending aorta (P_{Ao}) after pressure recovery has occurred, and correlates with the catheterization gradient. AV = aortic valve.

Geometric orifice area. Geometric orifice area (GOA) is the minimal cross-sectional area of the AV orifice. Using transthoracic or transesophageal echocardiography, GOA is determined by planimetry of the valve in short-axis. It cannot be determined in vivo for a mechanical prosthesis. For a tissue valve, its accuracy is affected by how well the valve orifice is visualized, and whether the funnel-shaped valve is visualized (and traced) at its minimal cross-sectional area. The GOA and EOA of a valve are the same only if the minimal cross-sectional area of the flow jet (the *vena contracta*) happens to coincide with the cross-sectional area of the valve.

Pressure recovery and energy loss coefficient. The pressure recovery phenomenon can be responsible for Doppler gradients that are substantially higher, and valve area that is substantially lower, than those determined invasively (9–13). When blood accelerates across a restrictive orifice, pressure energy proximal to the stenosis is converted to kinetic energy. Distal to the stenosis, blood decelerates. (In a patient with severe AS, the peak velocity across the AV approaches 5 m/s; however, blood flow in the descending aorta typically is no more than 1 m/s.) Because energy is neither created nor destroyed within the circulatory system, kinetic energy gained during flow acceleration must be converted to another form of energy during flow deceleration. In practice, kinetic energy is variably converted to thermal energy (heat) or is recovered as pressure energy (termed *pressure recovery*) (Fig. 1). Conversion of kinetic energy to heat dominates if there is turbulent flow and a dilated ascending aorta—both typical in the setting of significant native AS. In contrast, pressure recovery predictably occurs, and may predominate, in the setting of laminar transvalvular flow and a normal-caliber proximal ascending aorta (10–13).

Because continuous-wave Doppler resolves maximum velocity anywhere along the axis of interrogation, instantaneous flow acceleration is detected, and converted to the pressure gradient using the Bernoulli equation. In contrast, pressure determined during heart catheterization typically is assessed several centimeters distal to the AV, at a location after flow deceleration and pressure recovery have occurred (Fig. 1) (9). Clinically, the *net* pressure gradient (PG_{net}) between the LV and the ascending aorta (after pressure recovery) is more representative of the actual hemodynamic burden placed on the LV than is the *maximal* pressure gradient (PG_{max}) determined using Doppler echocardiography.

The effect of pressure recovery on EOA and valve gradients can be estimated, and should be accounted for in patients with proximal ascending aorta diameter ≤3.0 cm (11–13). The energy loss coefficient is an expression of valve area (in square centimeters) after correction for pressure recovery, and is more representative of the hemodynamic burden of AV obstruction than is the EOA (12):

$$E_LCo = (EOA \times Ao_A)/(Ao_A - EOA)$$

where E_LCo is the energy loss coefficient and Ao_A is the cross-sectional area of the proximal ascending aorta (typically at the sinotubular junction or proximal tubular ascending aorta, determined from diameter). Similarly, the contribution of pressure recovery to the Doppler gradient can be estimated (10):

$$\text{Pressure recovery (mm Hg)} = PG_{max} \times 2 \times (EOA \times Ao_A) \times (1 - [EOA/Ao_A])$$

where PG_{max} represents the Doppler-derived gradient. PG_{net} (after pressure recovery) can be noninvasively estimated:

$$PG_{net} = PG_{max} - \{PG_{max} \times 2 \times (EOA \times Ao_A) \times (1 - [EOA/Ao_A])\}.$$

Caveats

Doppler gradients. Doppler gradients can underestimate the severity of AS in 3 circumstances. First, failure to align the Doppler beam parallel with the highest velocity jet results in underestimation of the velocity by a factor of the cosine of the angle between the jet and the interrogating Doppler beam. Since the highest velocity jet direction cannot be reliably predicted, AV velocities should be carefully interrogated from multiple transducer positions. Second, gradients vary with flow, and in a low-flow state, gradients underestimate the severity of AS. Low-flow low-gradient AS typically is associated with patients with low LV ejection fraction (14). However, there has been recent appreciation of an important subset of patients with low-flow low-gradient AS despite a normal LV ejection fraction (15). The latter can be mediated by systolic dysfunction despite a normal EF, or low cardiac output owing to a small LV cavity size and small stroke volume despite preserved LV contractility (16). Finally, gradients can underestimate the severity of AS in patients with elevated systemic blood pressure (17).

Doppler gradients can overestimate the severity of AS in 5 circumstances, 3 of which involve errors in measurement. First, contamination of the continuous-wave Doppler signal by mitral regurgitation, or mistaking mitral regurgitation for LV outflow, can lead to a scenario in which the LV-to-left atrial pressure gradient is mistaken for the LV-to-aorta gradient. Because mitral regurgitation velocity typically is ~5 m/s, a substantial AV gradient could be mistakenly reported. However, the duration of the spectral Doppler jet can be useful in differentiating AS from mitral regurgitation. Because mitral regurgitation starts earlier and lasts longer than LV outflow, a continuous-wave Doppler envelope that is appreciably longer than the pulsed-wave Doppler envelope of the LV outflow tract should be suspected of including mitral regurgitation. Second, attempting to correct for the angle of Doppler interrogation relative to the direction of blood flow (the angle theta) can lead to overestimation of gradients by a factor of 1/(cosine theta); as a rule, angle correction is discouraged because the actual direction of the highest velocity flow vector in a turbulent jet cannot be reliably predicted. Third, the spectral Doppler envelope can be overtraced, such that more area is included under the traced curve used to calculate gradient than is actually defined by the modal velocity profile (7). Fourth, any high-flow state can be associated with gradients that are out of proportion to the actual degree of LV outflow obstruction. Finally, as discussed above, the pressure recovery phenomenon can be responsible for a Doppler gradient (PG_{max}) that is substantially higher than the invasively determined pressure gradient (PG_{net}).

EOA. EOA is an excellent means to describe in vivo AV hemodynamics relatively independent of flow. Caveats associated with its use relate to potential pitfalls in its calculation, failure to incorporate the effects of pressure recovery, and failure to correct for body size. There are two potential pitfalls in the calculation of EOA using the continuity equation. First, EOA is calculated from 3 different echocardiography/Doppler modalities utilizing at least 2 transthoracic windows. Diameter and pulsed-wave Doppler interrogation of the LV outflow tract theoretically are determined at the same exact location, but use different echocardiographic views; error is introduced with failure to measure at the same location. Second, the calculation of LV outflow tract area involves squaring the outflow tract radius, introducing a potentially large error if the diameter is measured inaccurately. For an LV outflow tract diameter of 2.0 cm, a 10% error in measurement (1.8 cm) results in a 19% error in calculated EOA.

Although EOA reflects AV hemodynamics relatively independent of flow, an exception can occur in the setting of very low flow, in which the AV may fail to open to its full potential. Finally, patient body size affects the interpretation of AV area determined by any method. Because the AV normally is larger in a large individual and smaller in a small individual, a small AV has relatively more hemodynamic impact (and reflects a greater severity of AS) in a large compared with a small individual. To account for this, valve area should be indexed to body surface area; severe AS typically is taken as a valve area index <0.6 cm^2/m^2 (5,6).

GOA. AV planimetry for GOA can be as accurate as is the visualization of the valve orifice. This is affected by the imaging modality (transthoracic vs. transesophageal echocardiography), and by operator experience in establishing on-axis imaging and recognizing the minimal valve orifice area of a potentially funnel-shaped valve. However, attenuation and reverberation artifact can make accurate visualization of the valve orifice difficult in association with native or bioprosthetic valve cusp calcification, and impossible in association with a mechanical prosthesis.

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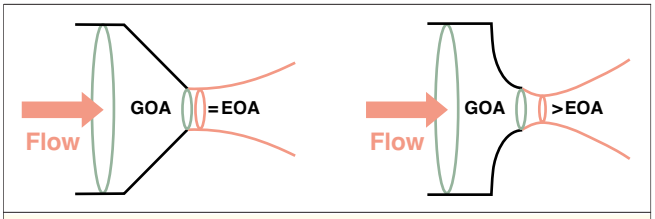

Figure 2. Effect of Valve Shape on EOA

With a more gradually narrowed orifice, the minimal jet area (*vena contracta*) is the same as the minimal valve orifice area **(left)**. With more abrupt narrowing and a flatter valve, the flow jet continues to accelerate and contract distal to the restrictive orifice **(right)**. EOA = effective orifice area; GOA = geometric orifice area.

GOA is not synonymous with EOA; GOA describes the anatomic orifice, and EOA describes in vivo flow. Valve shape is known to influence the relationship between EOA and GOA (18,19). In contrast to a pliable, domed AV, a relatively flat AV (typical of degenerative AS or bioprosthetic AS) is associated with a flow jet that continues to accelerate and narrow after it passes through the restrictive orifice, and the *vena contracta* is distal to and smaller than the GOA (Fig. 2). The coefficient of contraction, defined as the ratio of EOA to GOA, is a predictable function determined by valve shape. Clinically, the coefficient of contraction varies from 0.90 to 0.71 (18), defining up to a 29% difference between the EOA and GOA. Of note, the GOA of a prosthetic valve does not correlate with observed in vivo hemodynamics (20).

Pressure recovery. Pressure recovery can cause substantially higher gradients and substantially lower valve area on echocardiography/Doppler compared with invasively derived measurements (Fig. 1). In adult patients with significant native AS, transvalvular flow is turbulent, and the ascending aorta typically is dilated. Both factors preclude substantial recovery of pressure energy distal to the stenosis, and compensation for pressure recovery is unnecessary (1–3). However, pressure recovery can play a greater role after AVR (9,13). Pressure recovery has been well documented especially in association with bileaflet mechanical AV prostheses (9); pressure recovery also is observed after bioprosthetic AVR (13). Although the ascending aorta often is dilated in patients undergoing AVR, concomitant aortic root repair serves to fix the ascending aorta at a normal caliber. As such, combined AVR and aortic root repair can be associated with substantial pressure recovery. As an example of the impact of pressure recovery related to aorta size, a 21-mm AV prosthesis with Gorlin valve area (and energy loss coefficient) 1.3 cm^2 would have an EOA of only 1.0 cm^2 in a patient with an ascending aorta diameter of 2.4 cm, a 23% error and suggestive of borderline severe AS. In the same patient, pressure recovery would be responsible for 34% of the Doppler gradient.

Evaluation of High Gradients After Aortic Valve Replacement

High gradients in the absence of obstruction. High gradients after AVR can be due to 1 or more of several potential etiologies. Some, but not all, are associated with obstruction to LV outflow (Table 1).

High gradients after AVR can occur without LV outflow obstruction in the setting of measurement error, high-flow states, and pressure recovery. Measurement error and pressure recovery are more fully discussed above. In a high-flow state, valve appearance, EOA, DVI, and the contour of the spectral Doppler envelope all should remain normal.

High gradients in the presence of obstruction. High gradients after AVR can be caused by obstruction at the level of the valve due to prosthesis dysfunction, pannus, or prosthesis–patient mismatch (PPM); or due to obstruction above or below the level of the AV. Prosthesis dysfunction can occur gradually, with progressive sclerosis and calcification late after implantation of a bioprosthesis. Similar to native AS, it is recognized echocardiographically with visualization of the bioprosthesis cusps. Conversely, prosthesis dysfunction can occur abruptly. Valve thrombosis, or formation of a vegetation in association with infective endocarditis, can result in

Table 1. High Gradients After AVR

No LV outflow obstruction
Measurement error
Signal contamination or confusion with MR
Correction for "cosine theta"
Over-tracing spectral Doppler envelope
High-flow state
Fever, pain, anemia, hyperthyroidism, anxiety, significant aortic regurgitation
Pressure recovery
LV outflow obstruction
Obstruction at the aortic valve
Prosthesis dysfunction
Bioprosthesis calcification
Thrombus or vegetation
Pannus overgrowth
Prosthesis–patient mismatch
Subvalvular or supravalvular obstruction

AVR = aortic valve replacement; LV = left ventricle; MR = mitral regurgitation.

incomplete opening of a mechanical valve occluder. Less commonly, a large thrombus or vegetation can compromise the orifice of either a mechanical or a tissue prosthesis. Although it is possible in some circumstances to echocardiographically assess mechanical occluder mobility, fluoroscopic interrogation can be more reliable for a prosthesis in the aortic position. Both thrombus and vegetation appear echocardiographically as a soft-tissue density mass or masses associated with a prosthesis; although some echocardiographic features can help distinguish the two, differentiation often is made from clinical rather than from echocardiographic features.

Pannus overgrowth can occur after mechanical or bioprosthetic AVR. LV outflow obstruction and increased gradient can occur if the orifice area is sufficiently compromised, or if pannus overgrowth compromises systolic opening of a mechanical occluder. Pannus overgrowth of clinical significance typically occurs late—many years, or decades—after AVR, but paradoxically can occur within months of surgery. Pannus is difficult to detect echocardiographically, owing to its occurrence directly adjacent to the prosthetic sewing cuff, with associated attenuation and reverberation artifact; the echocardiographic diagnosis of pannus overgrowth often is a diagnosis of exclusion.

PPM is defined as an inadequate valve orifice area for an individual patient despite normal function of the prosthesis. An indexed EOA <0.85 cm^2/m^2 typically is taken as moderate PPM, and an indexed EOA <0.65 cm^2/m^2 as severe PPM (21). PPM after AVR is associated with excess short-term (22) and long-term (23) mortality, with a strong interaction with LV systolic dysfunction. PPM typically can be avoided through the use of a prosthesis with a predicted EOA taken from a reliable source (8,24,25), indexed to patient body surface area, that is ≥ 0.85 cm^2/m^2. PPM is suggested on echocardiography/Doppler in the setting of high transvalvular gradients but an echocardiographically normal-appearing prosthesis, and an EOA that falls within tolerance of anticipated.

Although supravalvular LV outflow obstruction can occur in Williams syndrome and in atypical aortic coarctation, it is an unlikely cause of high transvalvular gradient after AVR, because the ascending aorta is directly visualized at the time of aortotomy. In contrast, subvalvular LV outflow obstruction can and does occur after AVR (26). Subvalvular LV outflow obstruction after AVR probably occurs most commonly as a result of LV hypertrophy secondary to valvular AS, with systolic compromise of the LV outflow tract. Less commonly, subvalvular LV outflow obstruction after AVR can occur in the setting of concomitant hypertrophic obstructive cardiomyopathy. Because subvalvular obstruction often is not recognized prior to AVR, it can first present with increased gradients after surgery. Subvalvular LV outflow obstruction after AVR typically is not associated with the "dagger"-shaped spectral Doppler envelope that is seen in hypertrophic obstructive cardiomyopathy (26).

Chronicity. The chronicity of high gradients after AVR can offer an important clue to the underlying etiology (Table 2). Among the potential causes of high gradients that are not associated with LV outflow obstruction, pressure recovery would be anticipated early after surgery and on subsequent echocardiograms. In contrast, measurement error and high-flow states are transient, and would not be anticipated on all post-operative echocardiograms. Among the potential causes of high gradients that are associated with LV outflow obstruction, PPM and subvalvular obstruction should be present early after surgery and on all subsequent echocardiograms; whereas prosthesis dysfunction and pannus overgrowth are unlikely early after surgery, and typically are evident only on later post-operative echocardiograms. The ability to compare later post-operative gradients to those early after AVR is an important reason to perform routine baseline echocardiography/Doppler relatively early after surgery, at a time when hemodynamics and echocardiography windows have returned to normal, and when normal prosthesis function is still relatively certain (5,6).

Diagnostic algorithm. In evaluating a patient after AVR, the practitioner typically has access to clinical and echocardiography/Doppler data. If Doppler gradients are high, dominant questions typically are

Table 2. Chronicity as a Clue to Etiology of High Gradients After AVR

High Gradients Early After AVR	High Gradients Acquired Later After AVR
No outflow obstruction	
Pressure recovery	Measurement error
	High-flow state
Outflow obstruction	
Prosthesis–patient mismatch	Prosthesis dysfunction
Subvalvular or supravalvular obstruction	Pannus overgrowth

AVR = aortic valve replacement.

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whether the patient has actual LV outflow obstruction, and if so, whether prosthesis dysfunction is responsible.

Actual LV outflow obstruction should be considered clinically more likely in a patient with symptoms or physical findings suggestive of hemodynamically significant AS. When basing an evaluation on echocardiography/Doppler data alone, the algorithm shown in Figure 3 may be useful. In general, measurement error should be excluded, and the valve should be carefully interrogated for abnormal appearance that could herald prosthesis dysfunction. If the echocardiogram is performed late after AVR, comparison with an earlier postoperative echocardiogram is useful to evaluate for interval change that could suggest prosthesis dysfunction or pannus overgrowth. For high gradients observed on an echocardiogram performed early after surgery, or on an echocardiogram without an earlier study available for comparison, both echocardiographic and clinical data should be scrutinized for evidence of a high-flow state. In the setting of high gradients due to increased flow, the triangular shape and early-peaking contour of the spectral Doppler envelope typically is preserved. Barring evidence of a high-flow state, the echocardiogram should be scrutinized for evidence of subvalvular obstruction, and an ascending aorta diameter ≤3.0 cm should lead to consideration for pressure recovery as a factor contributing to high gradients. If the valve manufacturer and size are known, the anticipated EOA should be indexed to the patient's body surface area to assess for PPM.

If no identifiable cause of high gradients is found, pannus overgrowth (not usually visualized on echocardiographic imaging) should be considered. Consideration also should be given to additional testing (including fluoroscopy, transesophageal imaging, computed tomography, or cardiac magnetic resonance) to address possible prosthesis dysfunction. In the asymptomatic patient, simple follow-up imaging may be appropriate. Notably, causes of elevated gradients that are not associated with actual LV outflow obstruction should be carefully evaluated and excluded, and echocardiography/Doppler findings should be taken in clinical context with

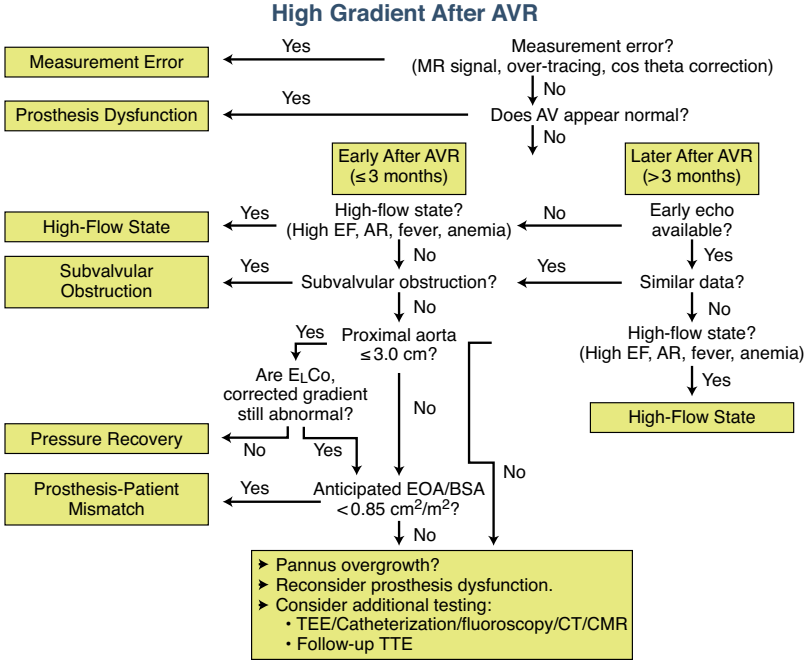

Figure 3. Algorithm for Evaluation of High Gradients After AVR

Measurements should be scrutinized for error. The valve should be interrogated for anatomic evidence of prosthesis dysfunction. Evidence of a high-flow state and subvalvular obstruction should be sought. Correction for pressure recovery should be performed if the ascending aorta diameter is ≤3.0 cm. Prosthesis–patient mismatch should be considered based on anticipated valve area indexed to patient body surface area. AR = aortic regurgitation; AV = aortic valve; AVR = aortic valve replacement; BSA = body surface area; CMR = cardiac magnetic resonance; CT = computed tomography scan; Echo = echocardiogram; EF = ejection fraction; E_LCo = energy loss coefficient; EOA = effective orifice area; MR = mitral regurgitation; TEE = transesophageal echocardiography; TTE = transthoracic echocardiography.

patient symptoms and physical findings, before making a clinical determination that there is important LV outflow obstruction after AVR.

Conclusion

High gradients after AVR can be, but are not necessarily, caused by LV outflow obstruction. Understanding the methods by which echocardiography and Doppler are used to assess AV hemodynamics, and the associated caveats, can help the clinician distinguish obstructive from nonobstructive causes of high gradients, and prosthesis dysfunction from other causes of obstruction. Measurement error, a high-flow state, and the presence of pressure recovery are common factors that can contribute to elevated Doppler gradients in the absence of LV outflow obstruction. Prosthesis dysfunction, pannus overgrowth, subvalvular obstruction, and PPM can be responsible for high Doppler gradients due to actual LV outflow obstruction. Echocardiography/Doppler findings should be taken in clinical context, and possible causes of high gradients that are not associated with LV outflow obstruction should be carefully evaluated and excluded, before making a clinical determination that there is important LV outflow obstruction after AVR.

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REFERENCES

1. Hatle L, Angelsen BA, Tomsdal A. Non-invasive assessment of aortic stenosis by Doppler ultrasound. Br Heart J 1980;43:284–92.
2. Hegrenæs L, Hatle L. Aortic stenosis in adults. Non-invasive estimation of pressure differences by continuous wave Doppler echocardiography. Br Heart J 1985;54:396–404.
3. Currie PJ, Seward JB, Reeder GS, et al. Continuous-wave Doppler echocardiographic assessment of severity of calcific aortic stenosis: a simultaneous Doppler-catheter correlative study in 100 adult patients. Circulation 1985; 71:1162–9.
4. 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.
5. Bonow RO, Carabello B, Chatterjee K, et al. ACC/AHA 2006 guidelines for the management of valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Develop Guidelines for the Management of Patients With Valvular Heart Disease). J Am Coll Cardiol 2006;48: e1–e148.
6. Vahanian A, Baumgartner H, Bax J, et al. Guidelines on the management of valvular heart disease: the Task Force on the Management of Valvular Heart Disease of the European Society of Cardiology. Eur Heart J 2007; 28:230–68.
7. 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.
8. Zoghbi WA, Chambers JB, Dumesnil JG, et al. Recommendations for evaluation of prosthetic valves with echocardiography and Doppler ultrasound. J Am Soc Echocardiogr 2009;22:975–1014.
9. Baumgartner H, Khan S, DeRobertis M, Czer L, Maurer G. Discrepancies between Doppler and catheter gradients in aortic prosthetic valves. Circulation 1990;82:1467–75.
10. 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.
11. Baumgartner H, Stefenelli 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.
12. Garcia D, Pibarot P, Dumesnil JG, Sakr F, Durand LG. Assessment of aortic stenosis severity. A new index based on the energy loss concept. Circulation 2000;101:765–71.
13. Garcia D, Dumesnil JG, Durand LG, Kadem L, Pibarot P. Discrepancies between catheter and Doppler estimates of valve effective orifice area can be predicted from the pressure recovery phenomenon. Practical implications with regard to quantification of aortic stenosis severity. J Am Coll Cardiol 2003;41:435–42.
14. Monin JL, Quéré JP, Monchi M, et al. Low-gradient aortic stenosis. Operative risk stratification and predictors for long-term outcome: a multicenter study using dobutamine stress hemodynamics. Circulation 2003;108: 319–24.
15. Hachicha Z, Dumesnil JG, Bogaty P, Pibarot P. Paradoxical low-flow, low-gradient severe aortic stenosis despite preserved ejection fraction is associated with higher afterload and reduced survival. Circulation 2007; 115:2856–64.
16. Carabello B. Aortic stenosis. Two steps forward, one back. J Am Coll Cardiol 2007;115:2799–800.
17. Kadem L, Dumesnil JG, Rieu R, Durand LG, Garcia D, Pibarot P. Impact of systemic hypertension on the assessment of aortic stenosis. Heart 2005;91:354–61.
18. Gilon D, Cape EG, Handschumacher MD, et al. Effect of three-dimensional valve shape on the hemodynamics of aortic stenosis: three-dimensional echocardiographic stereolithography and patient studies. J Am Coll Cardiol 2002;40:1479–86.
19. Garcia D, Pibarot P, Landry C, et al. Estimation of aortic valve effective orifice area by Doppler echocardiography: effects of valve inflow shape and flow rate. J Am Soc Echocardiogr 2004;17:756–65.
20. Pibarot P, Dumesnil JG, Cartier PC, Métras J, Lemieux MD. Patient-prosthesis mismatch can be predicted at the time of operation. Ann Thorac Surg 2001;71:S265–8.

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21. Pibarot P, Dumesnil JG. Hemodynamic and clinical impact of prosthesis-patient mismatch in the aortic position and its prevention. J Am Coll Cardiol 2000;36:1131–41.
22. Blais C, Dumesnil JG, Baillot 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.
23. Mohty D, Dumesnil JG, Echahidi N, et al. Impact of prosthesis-patient mismatch on long-term survival after aortic valve replacement. J Am Coll Cardiol 2009;53:39–47.
24. 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.
25. 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.
26. Bach DS. Subvalvular left ventricular outflow obstruction for patients undergoing aortic valve replacement for aortic stenosis: echocardiographic recognition and identification of patients at risk. J Am Soc Echocardiogr 2005; 18:1155–62.

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