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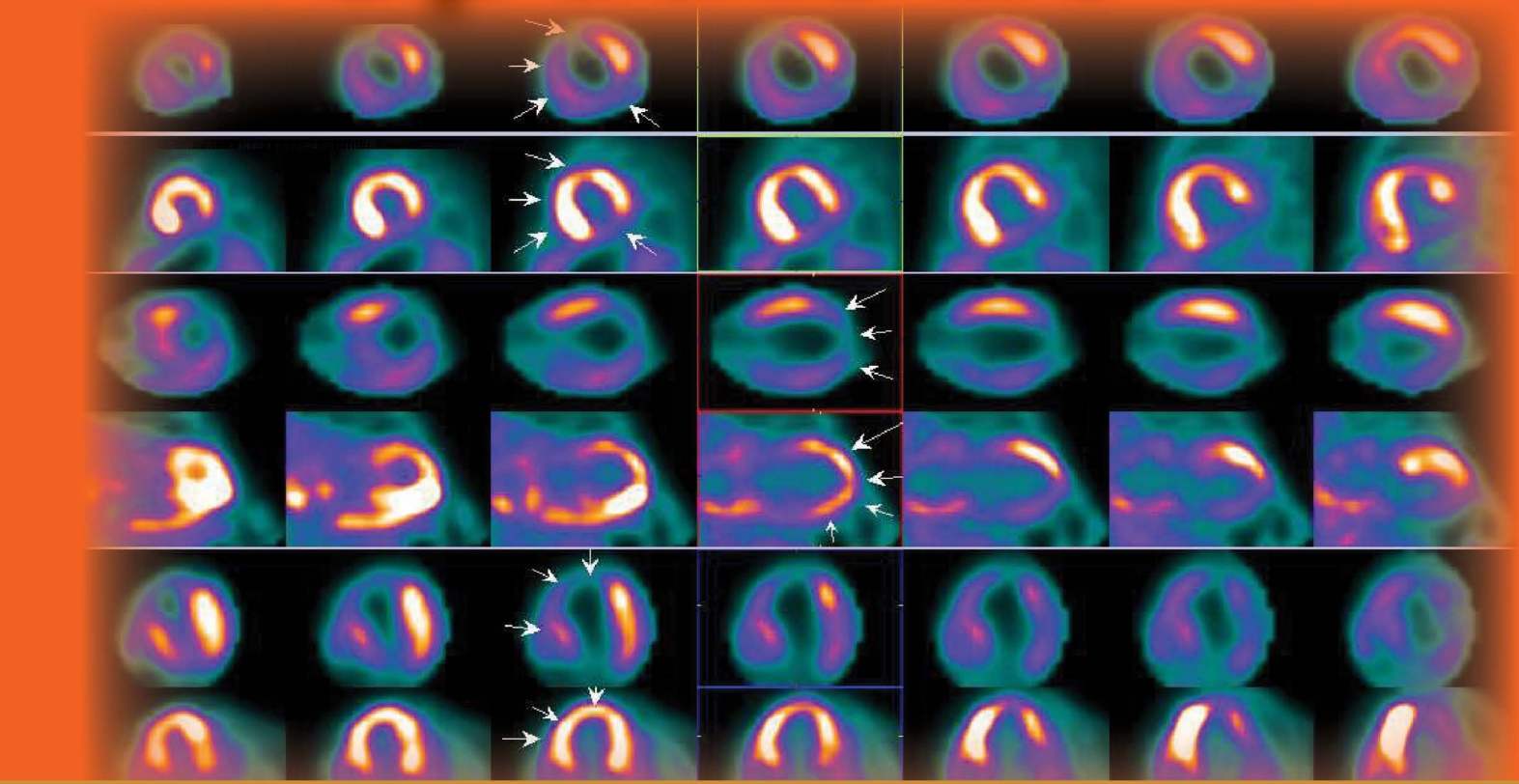
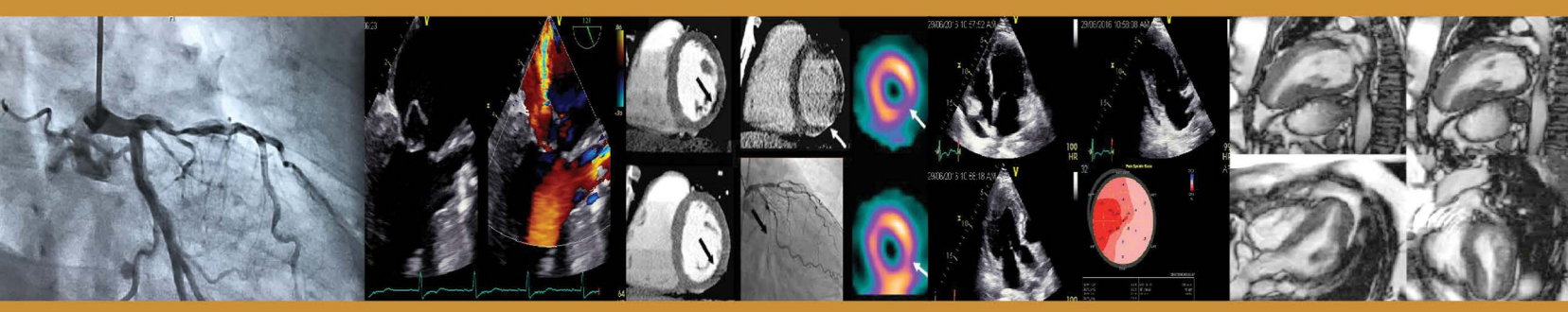
Cardiac Imaging Update 2017





GN Mahapatra • PC Manoria • Diwakar Jain

JAYPEE

Contents

SECTION 1: Cardiac Imaging

NONINVASIVE

2 The Intricacies in Echocardiographic Evaluation of Aortic Stenosis

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In the developing world, aortic stenosis (AS) represents as one of the most prevalent valvular heart disease. Severe aortic stenosis is transformation of the aortic valve in a severely restricted, thickened, calcific valve; however, the initiating process is less likely to be a degenerative one, but rather similar to atherosclerotic plaque formation.

In the last few years more efforts have been put to predict aortic valve events more accurately. Today aortic stenosis patients are older and have been found to have higher incidence of hypertension, coronary disease, and diastolic dysfunction. Thus, the proportion of patients with low stroke volume due to left ventricular systolic function, small chamber size, increased vascular (in addition to valvular) afterload, and due to impaired longitudinal shortening may represent one-third of cases.[1]

Echocardiography has become the main diagnostic tool in assessing AS patients **(Table 1)**. Beyond gradient and area values, it provides a comprehensive assessment of the aortic valve and aortic root morphology, which is of interest when planning the surgery, and of coexistent cardiac pathologies **(Table 1)**. It is also instrumental in assessing special subgroups of patients with low gradients or decreased LV contractility, or those who may be considered for AVR even if asymptomatic. The central role of echocardiography in the management of AS patients is acknowledged by the use of echocardiographic indices to define AS severity and indications for surgery.[2]

Table 1 Echocardiographic information in aortic stenosis patients

AV morphology: • Tricuspid or bicuspid • Severity of calcification
Aortic stenosis severity: • Gradient • Valve area
Aortic stenosis severity follow-up and progression of LV systolic function: Global (LVEF)
LV contractile reserve
Severity of LV hypertrophy
Response to exercise
Aortic dimensions and pathology
Co-existent valvular disease

Abbreviations: LV, left ventricle; LVEF, left ventricular ejection fraction

MAIN ECHOCARDIOGRAPHIC INDICES USED TO ASSESS AS SEVERITY

Flow Velocities and Gradients

- Both peak flow velocity (V_{max}) and mean gradient are obtained by Doppler interrogation of aortic flow. As such, good alignment of the Doppler line and the flow direction (<20°) is required for accurate and reproducible results.
- V_{max} is used rather than peak gradient to minimize the effect of small variations of velocity readings on the final result; this is less of a problem for the mean gradient that is obtained by integrating all the instantaneous gradients generated during ejection and is not a simple computation of mean velocity. For valvular jet velocities (V2) >3 m/s and subvalvular velocities (V1) <1.5 m/second, the latter can be ignored (simplified Bernoulli equation: $\Delta P = 4V2$), otherwise, both the proximal and the distal velocities have to be used (full Bernoulli equation). Furthermore, the cut-off values mentioned in **Table 2** are valid, if the LV ejection fraction (LVEF) is normal and there is no severe regurgitation across the valve.

Table 2 Stages of valvular aortic stenosis[33]

Stage	*Definition*	*Valve anatomy*	*Valve hemodynamics*	*Hemodynamic consequences*
A	At risk of AS	• Bicuspid aortic valve(or other congenital valve anomaly) • aortic valve sclerosis	Aortic V_{max} <2 m/s	None
B	Progressive AS	• Mild to moderate leaflet calcification of a bicuspid or trileaflet valve with some reduction in systolic motion or • Rheumatic valve changes with commissural fusion	• Mild AS: Aortic V_{max} 2.0–2.9 m/s or mean pressure <20 mm Hg • Moderate AS: Aortic V_{max} 3.0–3.9 m/s or mean pressure 20–39 mm Hg	• Early LV diastolic dysfunction may be present • Normal LVEF
C: Asymptomatic Severe AS				
C1	Asymptomatic Severe AS	Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening	Aortic V_{max} ≥4 m/s or mean pressure ≥40 mm Hg • AVA typically is <1.0 cm^2 (or AVAi <0.6 cm^2/m^2 Very severe AS is an aortic V_{max} >5 m/s or mean pressure >60 mm Hg	• LV diastolic dysfunction • Mild LV hypertrophy • Normal LVEF
C2	Asymptomatic Severe AS with LV dysfunction	Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening	Aortic V_{max} >4 m/s or mean pressure >40 mm Hg AVA typically <1.0 cm^2 (or AVA <0.6 cm^2/m^2)	LVEF <50%
D: Symptomatic Severe AS				
D1	Symptomatic severe high gradient AS	Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening	Aortic V_{max} >4 m/s or mean pressure >40 mm Hg AVA typically <1.0 cm^2 (or AVA <0.6 cm^2/m^2) but may be larger with mixed AS/AR	• LV diastolic dysfunction • LV hypertrophy pulmonary hypertension may be present
D2	Symptomatic severe low-flow/ low gradient AS with reduced LVEF	Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening	AVA<1.0 cm^2 with resting aortic V_{max} 4 m/s or mean pressure <40 mm Hg Dobutamine stress echocardiography shows AVA <1.0 cm^2 with V_{max} >4 m/s at any flow rate	• LV diastolic dysfunction • LV hypertrophy • LVEF <50%
D3	Symptomatic Severe low gradient AS with normal LVEF or paradoxical low-flow severe AS	Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening	AVA <1.0 cm^2 with aortic V_{max} <4 m/s or mean DP <40 mm Hg • Indexed AVA< 0.6 cm^2/m^2 and • Stroke volume index <35 mL/m^2 • Measured when patient is normotensive (systolic BP <140 mm Hg)	• Increased LV relative wall thickness – Small LV chamber with low stroke volume – Restrictive diastolic filling – LVEF ≥50%

Sources of Error for Gradient Calculations

- Gradient underestimation is due to:
  - Malalignment of the Doppler line with the main flow direction; and
  - Missing the transducer position/window providing the optimal signal.
- Gradient overestimation is due to–
  Confusion with a different, higher-velocity systolic flow (mitral regurgitation [MR], LV outflow tract [LVOT]

obstruction); and inclusion of a beat following a long diastole in measurements.

AORTIC VALVE AREA

Aortic valve area (AVA) is usually calculated using the continuity equation. All modern echocardiographic machines have incorporated analysis software to calculate AVA from the traced VTI and the measured LVOT diameter **(Fig. 1)**. An alternative method to obtain the AVA is by direct planimetry of the valve orifice in parasternal short-axis view, using either transthoracic or transesophageal echocardiography (TEE).

Sources of Error for Aortic Valve Area Calculations

The continuity equation method is subject to gradient error calculations and inaccurate LV outflow tract measurements, for which inter- and intra-observer variability may reach 8%.[2] This dimension, being squared in the continuity equation formula, can result in significant error and underestimation of valve area. The direct planimetry of the aortic valve orifice requires good-quality images, occasionally available only with TEE. Even with TEE, aortic valve direct planimetry may be inaccurate with a heavily calcified valve and is therefore considered to be an acceptable alternative when Doppler measurements are unreliable, but it is not a primary method to assess AVA.

DIMENSIONLESS VELOCITY INDEX

It is ratio between subvalvular (LVOT level) and valvular peak velocities, it is a version of the continuity equation that ignores LVOT diameter and thus is not subject to errors related to its measurements. A dimensionless velocity index (DVI) <0.25 is indicative of severe AS, with a valvular area of 25% of the expected normal valve area for the patient's body size. DVI does not provide a valve area but rather confirms or weakens the qualitative diagnosis of severe AS. The velocity index is also useful to differentiate between high valvular gradients due to truly severe AS and mild AS with increased velocities due to high-flow conditions such as sepsis or hyperthyroidism, when the DVI remains >0.3 as both the valvular and subvalvular velocities are high.

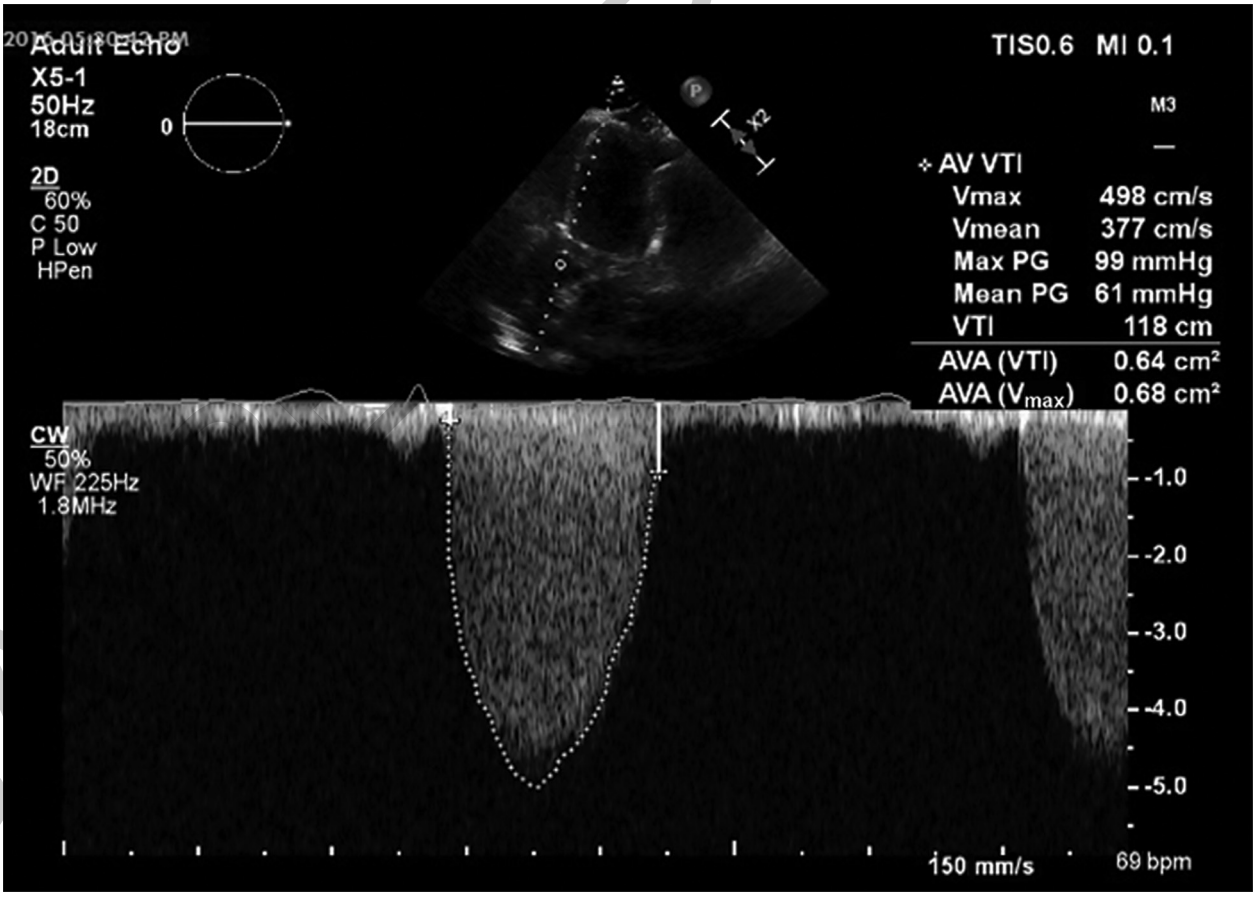

Fig. 1: Severe aortic stenosis by continuity equation

Real Dilemmas in the Diagnosis of Aortic Stenosis

The limitations and possible errors described above are generally well-known and, although occasionally confusing, their avoidance by appropriate technique and awareness is expected from a good echo study.

The echocardiographic uncertainties one may encounter in the diagnosis of AS relate mainly to contradictory results and lack of concordance between accepted echocardiographic indices of AS severity or between echocardiographic and catheterization results. The challenge of appropriate quantification of AS in patients with decreased LV function is well recognized. However, even patients with good LV contractility exhibit a mixture of hemodynamic patterns. In their review of 3,483 echocardiographic studies of patients with various degrees of AS and normal EF, Minners, et al. found in 30% of cases a lack of concordance between the different criteria of AS severity. Moreover, V_{max} and gradient were in the range of severe AS in 40–45% of patients, while by AVA 69–76% of them were diagnosed as having severe AS.[3]

Aortic stenosis severe by gradient but mild to moderate by aortic valve area. Occasionally, high (>4 m/s) peak velocities and elevated mean gradients (>30–40 mm Hg) are found in patients whose AVA by continuity equation is only in the mild to moderate range (>1 cm^2).

Frequently, the reasons for this discordance are related to execution errors of the echocardiographic study:

- Incorrect positioning of the pulsed-wave Doppler sample volume too close to the aortic valve, so that the LVOT signal is contaminated by the high-velocity valvular flow; and
- Erroneous measurement resulting in overestimation of the LVOT diameter. Real discrepancies, i.e. high velocities and gradients in the absence of significant AS, can occur in patients with a high cardiac output state, such as sepsis, hyperthyroidism, anemia or with AV fistulas. Awareness of the patient's clinical condition and a DVI >0.25 should clarify this condition. Aortic stenosis mild to moderate by gradient but severe by aortic valve area faced with this discrepancy, the first step is to establish the contractility of the left ventricle and dichotomise these low-gradient severe AS patients into those with reduced or normal EF.

Low-flow-Low-gradient Aortic Stenosis (LF/LGAS) with LV Systolic Dysfunction

It is defined as a combination of AVA <1 cm^2 (0.6 cm^2/m^2), mean gradient <40 mm Hg and LVEF <40%, and is described in 5–10% of patients with AS.[4,5] can be present in AS patients as a result of either concomitant pathology (coronary artery disease or cardiomyopathy) or of long-standing severe AS.

Other criteria which have been proposed in the literature to define the LF state in AS, include a cardiac index 3.0 L/min/m^2 and a stroke volume index 35 mL/m^2.[2,16,22] Given that the gradient essentially depends on the flow per beat (i.e. the stroke volume) rather than on the flow per minute (i.e. the cardiac output), the former is the most frequently used parameter in this context.[22,25-27]

The main diagnostic challenge in LF-LGAS with low LVEF is to distinguish true severe from pseudosevere AS.

In the former, the primary culprit is deemed to be the valve disease, and the LV dysfunction is a secondary or concomitant phenomenon. Conversely, the predominant factor in pseudosevere AS is deemed to be myocardial disease, and AS severity is overestimated due to incomplete opening of the valve in relation to the LF state. Distinction between these two entities is essential because patients with true severe AS generally benefit from aortic valve replacement (AVR), whereas those with pseudosevere AS may not benefit.

ASSESSING LEFT VENTRICLE CONTRACTILE AND/OR FLOW RESERVE

The term "flow reserve" is utilized rather than "contractile reserve" because several mechanisms not necessarily related to intrinsic contractility may contribute to the lack of stroke volume increase during DSE, including: (1) afterload mismatch due to an imbalance between the severity of the stenosis and myocardial reserve;[29] (2) inadequate increase of myocardial blood flow due to associated CAD; and/or (3) irreversible myocardial damage due to previous myocardial infarction or extensive myocardial fibrosis.

deFilippi et al.[15] were the first to demonstrate that low-dose dobutamine stress echocardiography (DSE) may be used in these patients to assess the presence of LV flow reserve and to distinguish true versus pseudo severe aortic stenosis and to risk-stratify the patient in terms of perioperative risk and possible benefit of AVR. The use of DSE for this purpose has received a Class IIa (Level of Evidence: B) recommendation in the American College of Cardiology/American Heart Association-European Society of Cardiology (ACC/AHA-ESC/EACTS) guidelines.[22-24]

The accepted approach is to perform low-dose dobutamine stress echo study (DSE) and to quantify the inotropic response and the changes in AVA and transvalvular gradient.

Dobutamine Stress Echo Study in Low-gradient Aortic Stenosis

The accurate assessment of aortic valve area in patients with a reduced stroke volume is difficult because the calculated valve area is proportional to stroke volume and the constant of the Gorlin equation varies with transvalvular flow.[12,13] As a result, some patients with AS and a low transvalvular pressure gradient have a reduced valve area because of inadequate stroke volume in the presence of thickened valve leaflets rather than a fixed, anatomic stenosis. Cannon et al[14] described 8 such patients who were identified as having severe AS using

Table 3 Hemodynamic response patterns to dobutamine in patients with low-flow-low-gradient aortic stenosis

Increase in stroke volume	*Gradient*	*Aortic valve area*	*Conclusion*
>20%	⇔	Increased >1–1.2 cm^2	Contractile reserve present, pseudo-severe AS
>20%	Increased	⇔	Contractile reserve present, true severe AS
<20%	⇔	⇔	No contractile reserve ? AS

⇔ = no significant change

the Gorlin equation but only mild AS during inspection of the valve at the time of surgery. These individuals were thought to have "pseudo-AS"; i.e., their aortic valve had thickened leaflets, which opened in direct relation to systolic blood flow. If the stroke volume was small, the leaflets opened poorly, resulting in a demonstrable transvalvular pressure gradient and a small calculated valve area. As the stroke volume increased, the leaflets opened more effectively, resulting in a larger valve area.

Dose of dobutamine required is in range of 5–20 μ/kg/minute, and although the dose-response to dobutamine is unpredictable and the inotropic response does not necessarily parallel the chronotropic and blood pressure response,[6] an increase in heart rate is generally taken as proof of dopaminergic stimulation sufficient to elicit an inotropic response. CR is considered to be present if the dobutamine infusion results in ≥20% increase in cardiac output.[4,7] In subjects, who show an increase in peak velocity (0.6 m/s), stroke volume (20%), or mean transvalvular pressure gradient (10 mm Hg) with DSE have LV contractile reserve. Patients with true severe AS and evidence of CR have a clear indication for AVR. The possible response patterns to dobutamine in patients with LF/LGAS are summarized in **Table 3**.

DeFilippi et al.[15] demonstrated that DSE could be used to distinguish individuals with fixed AS from those with pseudo-AS. In patients with fixed AS, dobutamine induced an increase in peak velocity, mean transvalvular pressure gradient, and valve resistance and no change in valve area. In contrast, in those with pseudo-AS, dobutamine caused a considerable increase in valve area (0.3 cm^2) without a substantial change in peak velocity, mean transvalvular pressure gradient, or valve resistance.

DISTINGUISHING BETWEEN TRUE SEVERE AND PSEUDOSEVERE AORTIC STENOSIS

The evaluation of the changes in EOA and gradient during dobutamine infusion are also helpful in differentiating true severe from pseudosevere AS. Typically, pseudosevere AS shows an increase in EOA and relatively little increase in gradient in response to increasing flow, whereas true severe AS is characterized by little or no increase in EOA and an increase in gradient that is congruent with the relative increase in flow. Several parameters and criteria have been proposed in the literature to identify patients with pseudosevere AS during DSE, including a peak stress mean gradient 30 or 40 mm Hg depending on studies, a peak stress EOA 1.0 or 1.2 cm^2, and/or an absolute increase in EOA 0.3 cm^2;[15,16,22,26,28,30] thus, the optimal cut-off values remain to, be determined. The prevalence of pseudosevere AS is reported to be between 20% and 30%.[15,26,28,31]

Some patients may nonetheless have an ambiguous response to DSE due to variable increases in flow [15,26,27] and interpreting the changes in EOA and gradients without considering the relative changes in flow may often be problematic. Hence, to overcome this limitation, the investigators of the TOPAS (Truly or Pseudo-Severe Aortic Stenosis) study proposed to calculate the projected EOA that would have occurred at a standardized flow rate of 250 mL/s (EOAProj) [26,27] and this new parameter has been shown to be more closely related to actual AS severity, impairment of myocardial blood flow, LV flow reserve, and survival than the traditional DSE parameters.[5,26,27,32] Patients with no increase in stroke volume may nonetheless have an increase in mean flow rate sufficient to allow a reliable measurement of EOAProj; this is due to shortening of LV ejection time in relation to an increase in heart rate.[26,27] However, there are 10–20% of patients in whom the increase in flow rate is insufficient to allow calculation of EOAProj. In such cases or those with ambiguous results during DSE, quantification of valve calcification by multislice computed tomography may also be useful.

Therefore, DSE clearly can help to differentiate patients with fixed low-gradient AS from those with pseudo-AS.

In the study of Quere et al.[17] published in Circulation, the operative mortalities for those with and without LV contractile reserve were 6% and 33%, respectively.

From a previously reported French multicenter trial,[16] Quere et al.[17] identified 66 patients with symptomatic AS, a mean transvalvular pressure gradient 40 mm Hg, and an LVEF 40% who survived valve replacement surgery and underwent an evaluation of functional status and LVEF postoperatively. It was found that most patients with severe AS and a low transvalvular pressure gradient manifested a substantial improvement in symptomatic status and LVEF after valve replacement surgery, and these improvements occurred with similar frequency in subjects with and without LV contractile reserve.

Normal Left Ventricular Contractility and Low-gradient Severe Aortic Stenosis

The above term is reserved for patients with normal left ventricular ejection fraction but with reduced stroke volume and reduced systolic function seen secondary to left ventricular hypertrophy secondary to aortic stenosis known as increased concentric hypertrophy (ICR). This ICR in turn leads to

decreased LV filling and reduced stroke volume (SV). Lower SV results in lower gradients across aortic valve.[18]

Inaccurate measurements and underestimation can occur due to LVOT shape which can alter the calculation of SV and AVA. This is in turn can lead to inconsistent measurement of Severity of AS.[19] It is here that role of TEE becomes important for planimetry of aortic valve.

Role of tissue Doppler imaging is important as it there would be impaired long axis shortening with reduced contractility. It was concluded in SEAS substudy[20] that LV myocardial systolic dysfunction is common in asymptomatic AS in particular in patients with low-flow AS and increased valvuloarterial afterload, whereas EF is generally preserved. A condition similar to LF-LGAS was seen in these patients with low SV and transaortic gradients. The major concern in these cases is that underestimation of AVA can lead to underutilization of valve replacement.[11]

Hachicha[11] et al described ventriculoarterial impedance (Zva), an index of global hemodynamic load and related this to the onset of symptoms and adverse events.

$$\text{Ventriculoarterial impedance (Zva)} = \frac{\text{Systolic arterial pressure + mean net transaortic gradient}}{\text{Stroke volume/m}^2}$$

Height can be substituted instead of BSA if SV is indexed to height in this formula. A value of Zva ≥4.5 mm Hg/mL m^2 may be useful to identify patients who are at risk of deterioration of myocardial function as per previously reported studies.[20,21]

Normal LVEF does not mean normal SV. Hachicha et al[11] showed that one-third of patients with severe AS had reduced SV Index (SV/BSA <35 mL/m^2) despite preserved LVEF. This will lead to low flow situation and which in turn leads to low transvalvular gradients. In their study of 512 consecutive patients with echocardiographically determined low gradient severe AS (AVA ≤ 0.6 cm^2/m^2) and LVEF ≥50%, Hachicha et al[11] concluded that normal flow (NF) having SV index ≥35 mL was seen in 65% of cases and paradoxically low flow (PLF) having SV index ≤35 mL in 35% of cases. During 5 year follow-up, patients with PLF had a reduced survival compared to those with NF.

Guidelines[22] regarding diagnostic and therapeutic recommendations for LF-LGAS. Further prospective studies are needed to determine the prognosis and most appropriate timing of AVR in these asymptomatic paradoxically LF-LGAS patients with preserved LV function.

In clinical practice we are not infrequently challenged by the reality of patients with severe AS by both valve appearance and calculated valve area (AVA <1 cm^2) and who present with a mean gradient in the mild-to-moderate range (<40 mm Hg) despite normal LV contractility. Obviously, technical errors have to be excluded, but this possible presentation of severe AS has recently been increasingly recognized[8-11] and has been described in up to 42% of patients with severe AS and normal LV contractility. Importantly, these patients do not seem to have a better prognosis than their 'high-gradient' counterparts.[9-11] Possible explanations for this hemodynamic pattern include:[9-11]

- Relatively low stroke volume, which is not suggested by an apparently normal EF—this could be related to small LV cavity (small-sized patients, severely hypertrophic ventricles) or occult L systolic dysfunction (elderly patients, LV hypertrophy); and
- Higher systemic vascular resistance and LV afterload. The importance of recognizing this not uncommon hemodynamic pattern cannot be overemphasised, since AVR, when appropriate, should not be denied to these patients due to a possibly misleading 'not severe enough' gradient.

CONCLUSION

Echocardiography is the first-line diagnostic tool in the assessment of patients with AS. Cut-off values define severity criteria used to decide appropriateness of intervention. A large number of patients do not fulfill all accepted criteria and may present with perplexing hemodynamic patterns and echocardiographic results. Awareness of the sources of possible errors and of less typical echocardiographic results is essential for the correct management of AS patients whose echocardiographic studies are, apparently, confounding.

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Cardiac Imaging Update 2017

Salient Features

- It is a unique blend of cardiovascular diagnostic and prognostic imaging and the appropriateness of clinical and procedural management
- Covers the full range of clinical cardiac imaging and its applications, focusing on how the information derived can be used in clinical decision-making and patient management as well as current topics in clinical cardiology
- Focuses on its major areas of strength with chapters on endocarditis, aortic stenosis, heart failure, and cardiomyopathy; covers the recent developments in speckle tracking; the applications of cardiovascular magnetic resonance (CMR)
- Discusses the relative roles of CMR and cardiac CT
- Explores invasive approaches to assessment of coronary plaque and flow physiology
- Provides a comprehensive update of the status of the clinical applications of the imaging approaches as well as an update of important issues of clinical cardiology
- Serves as a soapbox to encourage the development of an integrated cardiovascular imaging subspecialty

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