Echocardiographic Evaluation of Aortic Valve Stenosis: Problems and Pitfalls

Nikos T. Kouris1, Dimitris P. Tsiapras2, Christoforos D. Olympios1
1“Thriassio” General Hospital of Elefsina, 2Onassis Cardiac Surgery Centre, Athens, Greece

Aortic valve stenosis (AS) is the most common valvular heart disease and the third most common cardiac disease (after coronary artery disease and hypertension) in the developed countries.1 Doppler echocardiography is the method of choice for both the diagnosis and the estimation of the severity of AS, since it describes with precision the anatomy of the aortic valve (AV), evaluates its hydraulic behaviour, and determines its effect on left ventricular (LV) function.

According to the latest guidelines of the European Society of Cardiology and the American Heart Association, AS is considered severe when the aortic valve area is <1.0 cm² (or <0.6 cm²/m²), the mean transvalvular pressure gradient is >50 mmHg (or >40 mmHg according to the American guidelines) and the peak transvalvular velocity is >4 m/s (which corresponds to a peak transvalvular pressure gradient >64 mmHg), all measured echocardiographically.1,2 However, in daily clinical practice, the clinical cardiologist is required to deal with patients who show discrepancies between the various findings and the echocardiographic measurements. For example, in a given patient the AV may be found to be severely calcified, with limited opening, with preserved LV function, and a valve area of 0.8 cm², but in combination with a peak flow velocity of 3.5 m/s and a mean pressure gradient <40 mmHg. Similarly a small valve area may be detected (0.9 cm²) with a low transvalvular pressure gradient, in combination with a deterioration in LV function. Such situations raise uncertainty with regard to the real severity of AS, as well as concern about the indicated management of these patients. This issue of the Hellenic Journal of Cardiology includes a very interesting and extensive review article by Antoniou et al,3 which thoroughly analyses the new echocardiographic indices and their clinical use. Prompted by that article, this editorial will discuss methods for the correct evaluation of the severity of AS, as well as the cases of possibly conflicting findings.

Correct imaging of the anatomy and evaluation of the hydraulic behaviour of the AV

The appearance of the AV and the mobility of its leaflets on the two-dimensional echocardiogram provide the first information about the severity of AS (Figure 1). The anatomy and the mobility of the AV are best evaluated using the parasternal long-axis view, the parasternal short-axis view at the level of the great vessels, or the apical long-axis three-chamber view, rather than from the apical five-chamber view.4 In cases of suboptimal imaging from the parasternal or apical views, it is possible to obtain good images using the
If even one leaflet of the AV opens well, despite the presence of severe calcification on the other two, the existence of severe AS is not likely. In contrast, a severely calcified and immobile AV suggests severe AS, so that if the Doppler recording shows a moderate increase in velocity, it is better to re-evaluate this more carefully. However, it should be taken into account that the opening of the AV is affected by the stroke volume, so that in cases of systolic LV dysfunction the mobility of the aortic leaflets may appear to be reduced. Direct planimetry of the valve area, from transthoracic and especially from transoesophageal imaging, is considered reliable, but in clinical practice it does not always agree with the haemodynamically determined effective orifice area.

Apart from the cases where the AS is apparently mild (good mobility of aortic leaflets, Vmax <3 m/s, with good LV function), the peak AV transvalvular velocity should be recorded by continuous wave Doppler from the apical five-chamber view and from at least one other view (usually the right parasternal or subxiphoid). The use of a stand-alone probe may prove particularly useful for these recordings (Figure 2).

The recording of Vmax, the calculation of the mean pressure gradient and the aortic valve area are the minimum information required. The morphology of the wave is indicative of the severity; a signal of triangular form is suggestive of severe AS, whereas early peaking of the velocity indicates moderate AS (Figure 3). The mean transvalvular pressure gradient is derived from instantaneous velocity calculations through the whole spectrum of the signal, and not by calculating the mean transvalvular velocity and inserting it into Bernoulli’s simplified formula. It is, therefore, more representative of the severity of the stenosis than is the peak velocity, which describes only one part of the signal.

Figure 1. Short-axis view of a moderately (left) and severely (right) calcified aortic valve.

Figure 2. Recording of the peak velocity through a stenotic aortic valve in the apical four-chamber view (left) and the right parasternal view (right), where a higher velocity is recorded.
Calculation of the aortic valve area (Figure 4) using the continuity equation (aortic valve area = \( \pi \times (d_{\text{LVOT}}/2)^2 \times \text{VTI}_{\text{LVOT}} / \text{VTI}_{\text{V-max}} \)) is very important and should be performed in every case of AS, \(^4\) since it is relatively flow-independent. The most common mistakes are in the measurement of the diameter of the LVOT, whose value is squared in the continuity equation. It is measured from the parasternal long-axis view, during systole, directly below the base of the aortic leaflets, and from the internal surfaces of the interventricular septum and the posterior aortic wall (inner to inner edge). Care is also needed in the placement of the sample volume in the LVOT, so that the signal recorded will have a “clean” interior without many sounds, with clear and thin boundaries. The use of velocity values in the continuity equation instead of their integrals (VTI) is appropriate only when the LVOT and peak velocity signals are of the same form and is best avoided. In addition, correction of the aortic valve area for body surface area (BSA) is very important; for example, an aortic valve area of 1.3 cm\(^2\) corresponds to obviously moderate AS in a small-bodied individual with BSA 1.5 m\(^2\) (corrected valve area 0.87 cm\(^2\)/m\(^2\)). However, in a large-bodied person with BSA 2.4 m\(^2\) the corrected valve area is 0.54 cm\(^2\)/m\(^2\) and the AS should be assessed as severe. In cases where the image quality does not allow a certain estimation of the aortic valve area, a ratio \( \text{VTI}_{\text{LVOT}} / \text{VTI}_{\text{V-max}} < 0.25 \) is an indication of severe AS.

Care is also needed in cases with coexisting aortic regurgitation or anaemia. In aortic regurgitation, because of the increased transaortic volume, the peak velocity and the mean transvalvular pressure gradient are also increased for a given aortic valve area. The combination of moderate AS and moderate AV regurgitation suggests severe mixed aortic disease. \(^4\) In the presence of anaemia, a high pressure gradient is recorded when there is mild or moderate AS.

Measurement of the patient’s blood pressure at examination time is also required, since velocity measurements are affected by this, and it must be taken into account in repeated studies. In particular, systolic blood pressure contributes to the already increased afterload, has a marked effect on its evaluation, and may cause the appearance of symptoms even in non-critical AS. \(^6\) Thus, in these cases it is necessary to estimate the double load that the LV faces during its
Ejection phase, the valvular and the arterial, by evaluating the valvulo-arterial impedance (Z_{va}). This new index is calculated by dividing the estimated LV systolic pressure (systolic blood pressure + mean transvalvular pressure gradient) by the stroke volume index: \( Z_{va} = \frac{(SBP + MnPG)}{SV} \). Values of the Z_{va} index >4.5 mmHg/ml/m² are considered to be indicative of an excessively increased afterload, while patients with values ≥ 4.9, or according to others >5.5 mmHg/ml/m², show increased mortality and morbidity if not treated surgically.⁸

### Evaluation of AS in patients with a low ejection fraction

Patients with depressed LV function and concomitant AS of whatever severity have a poor prognosis. However, the subgroup of patients with true severe AS and a low ejection fraction (EF), who make up no more than 5-10%, are those who have the highest mortality and the worst prognosis.⁹¹⁰ In these patients the primary problem is the valvular disease and the LV dysfunction is secondary and is due to the increased afterload (inadequate compensatory LV hypertrophy: afterload mismatch¹¹), so that replacement of the AV will lead to an improvement in LV performance. In the remaining patients the LV dysfunction is due to other causes and the moderately stenotic AV does not open fully because of the low stroke volume (pseudostenosis). This new index appears to improve the diagnostic accuracy in discriminating between real AS and pseudostenosis.¹⁴

### Evaluation of AS in patients with a low stroke volume, low pressure gradients, and normal EF

A quite significant proportion of patients with severe AS, when defined in terms of aortic valve area, show a paradoxically low pressure gradient despite the presence of a preserved EF (paradoxical low flow, low-gradient aortic stenosis).¹⁵ These patients (e.g. AV valve area 0.8 cm², mean pressure gradient 29 mmHg, EF 60%) fulfil one severity criterion for AS (valve area) but not the others (pressure gradient, peak flow velocity) (Figure 7) and this discordance may (erroneously) lead to postponement of surgical intervention. The error arises because these patients are often in a more advanced stage of their disease, which is characterised by a greater degree of concentric hypertrophy and a smaller LV cavity, a normal (but lower than expected) EF with an elevated total LV afterload, and hence a reduced stroke volume. The reduced stroke volume (<35 ml/m²), irrespectively of the EF, results in the creation of a pressure gradient that is lower than expected.¹⁵ In addition, in these patients it is particularly valuable to calculate the valvulo-arterial impedance via the Z_{va} index, in order to evaluate the afterload.
The problem of pressure recovery

The phenomenon of pressure recovery (PR) has been investigated in order to explain (in part) the difference between the Doppler and catheterisation measurements. The pressure gradient recorded during catheter withdrawal corresponds to the difference between the maximum pressure value in the LV and the aorta (peak-to-peak gradient) and does not exist physiologically, since the two pressure curves do not occur simultaneously. In contrast, Doppler measures the maximum instantaneous pressure difference at the neck of the stenotic flow (vena contracta) immediately below the orifice, which is usually 20 mmHg higher than the catheterisation value, though it may reach as high as 50 mmHg. As the blood passes through the stenotic AV, it loses momentum peripherally to the orifice, which results in an increase in the pressure within the aorta. According to Bernoulli's theorem, in a closed system the sum of the kinetic and potential energy remains constant; thus a reduction in kinetic energy leads to an increase in static pressure. This increase is the PR. The PR is negligible in cases with mild AS or with a dilated ascending aorta. However, it has clinical significance in cases with a small aortic diameter (<3 cm), although this is not very common (Figure 8). In practice, correction of the pressure gradient taking the PR into account uses the formula:

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PR = 4AV_{\text{max}}^2 \times 2 \frac{AV_{\text{Ac}}}{AoA} \times \left(1 - \frac{AV_{\text{Ac}}}{AoA}\right)
\]

where PR is pressure recovery, AVmax is the peak aortic velocity by continuous wave Doppler, AVAc is the aortic valve area from the continuity equation, and AoA is the cross-sectional area of the ascending aorta. It is recommended that PR be evaluated at the level of the sinotubular junction.

In general, it could be noted that in marginal cases between moderate and severe AS, the severity of the stenosis is considered greater if the aortic diameter is small.

Finally, in recent years, the development and use of percutaneous repair of the AV (TAVI) has resulted in an increase in the number of patients who undergo non-surgical intervention. This has created the need for a fuller evaluation and more precise selection of patients with AS, where an accurate estimation of AS severity is mandatory.

**Figure 5.** Clinical algorithm for patients with aortic stenosis and a low ejection fraction. AS – aortic stenosis; DSE – dobutamine stress echo; SV – stroke volume; MPG – mean pressure gradient; TSAS – true severe AS; PSAS – pseudo-severe AS; AVA – aortic valve area.
Figure 6. A. Low-dose dobutamine stress echo in a patient with true severe aortic stenosis and a low ejection fraction. At rest the mean pressure gradient is 30 mmHg and the calculated aortic orifice is 0.76 cm². At peak dobutamine infusion the mean pressure gradient increases to 55 mmHg and the orifice remains at 0.78 cm². B. Low-dose dobutamine stress echo in a patient with pseudo-severe aortic stenosis and a low ejection fraction. At rest the mean pressure gradient is 10 mmHg and the calculated aortic orifice is 0.81 cm², while at peak dobutamine infusion the mean pressure gradient increases to 21 mmHg and the orifice to 1.02 cm².
To conclude, AS is a common echocardiographic finding; it should, however, be considered as a dynamic manifestation of a systemic process, rather than as a disease restricted exclusively to the AV. The evaluation of its severity and the decision to perform surgical intervention are often also affected by the remainder of the heart and the aorta. In particular, the discrimination between moderate and severe AS is often difficult, and account should be taken of the interactions between valvular, vascular, and haemodynamic measurements of the LV (rule of three Vs: valve, ventricle, vascular).

References