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Role of Echocardiography in Aortic Stenosis

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ARTICLE INFO

Keywords:
Echocardiography
Aortic stenosis
Aortic valve disease
Low gradient aortic stenosis
Valve disease

ABSTRACT

Aortic stenosis is a valve disorder that includes not only valve narrowing but also changes in the left ventricle and intracardiac hemodynamics. Older patients with aortic stenosis often have co-existing pathologic disorders, which influence the pathophysiology, symptom expression and prognosis. There is also increasing awareness that severe aortic stenosis could be associated with low transvalvular pressure gradient caused by a variety of mechanisms. Surgical and transcutaneous valve replacements are currently available interventions for patients with severe aortic stenosis. This article reviews the role of echocardiography in the comprehensive assessment of aortic stenosis, its severity and associated pathophysiologic abnormalities.

During the last decade, new insights on the clinical features, presentation, and prognosis of patients with aortic stenosis (AS), and the emerging option of transcutaneous aortic valve implantation (TAVI) in selected patients have placed emphasis on the need to approach aortic stenosis not just in terms of valve stenosis and gradients, but much more comprehensively. The etiology of AS include congenital lesions, commonly a bicuspid aortic valve, degenerative calcification of the valve cusps, rheumatic fever-related commissural fusion, and rare systemic or therapy-induced mechanisms. Severe AS, if uncompensated and uncorrected, leads to disabling symptoms and decreased survival.[1,2] For optimal clinical decision making, comprehensive diagnostic evaluation of patients with aortic valve disease requires assessment of the morphology of the valve, severity of the valve lesion, size of the aorta, systemic arterial afterload, impact on LV size and function, consequences of abnormal LV function and hemodynamics on pulmonary artery pressures, right ventricular size and function, and co-existent disorders. Echocardiography, with its two- and three-dimensional imaging capabilities, and various Doppler modalities, allows for examination of the valve pathology, as well as hemodynamics, and has therefore become the diagnostic method of choice, and the guide for optimal management of patients with AS. Echocardiographic data should be integrated with the clinical features of a patient, other imaging, and, when needed, catheterization findings in order to approach AS in a syndromatic fashion. This article will review the role of echocardiography in patients with AS.

Assessment of the severity of aortic stenosis

Normal aortic valve area (AVA) is considered to range from 2 to 4.5 cm^2. This observation is based on a few reports from a small series of subjects. As AVA progressively decreases, it results in an increasing pressure gradient between the LV and aorta, and an increasing flow velocity across the valve. In the past, the severity of AS was determined by invasively measuring cardiac output and pressure gradients across the valve, and employing the Gorlin equation, or other similar equations, to derive the AVA. Such methods of obtaining AVA, however, were poorly validated and their accuracy never appropriately tested in the setting of abnormal flow states, increased afterload, or co-existent disorders.

Statement of Conflict of Interest: none.

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http://dx.doi.org/10.1016/j.pcad.2014.05.006
0033-0620/

Abbreviations and Acronyms
AR = aortic regurgitation
AS = aortic stenosis
AVA = normal aortic valve area
AVAi = AVA index
EF = ejection fraction
ELI = energy loss index
SAVR = surgical aortic valve replacement
SV = stroke volume
TAVI = transcutaneous aortic valve implantation
VTI = velocity–time integral

Aortic valve pathology

Two-dimensional and three-dimensional imaging portray the pathology of a stenotic aortic valve. The number of valve cusps, valve thickening, commissural fusion, calcific burden on the valve, and restricted motion are visualized in the long-axis and short-axis imaging planes [3–5] (Fig 1). In most adult patients with AS, the valve has three thickened and calcified cusps. Valve thickening and calcification may involve all cusps or only part of the valve. A bicuspid valve has two cusps with complete or partial fusion in the conjoint cusp, and an oval appearance. The raphe is generally directed towards the pulmonic valve or tricuspid valve direction. If stenosis is not severe, the valve exhibits systolic bowing in the long-axis view that gives a clue to the bicuspid nature of the valve (Table 1).

Aortic valve area can be measured by planimetry, except in the presence of a markedly calcified or deformed valve.[6–8] Three-dimensional imaging is helpful in correctly representing the valve in an optimal orientation.[5] In addition to the valve area, other measurements of importance include diameters of the aortic ring and aortic root at the level of the sinus of Valsalva, sino-tubular junction, and proximal ascending aorta. If surgical replacement or transcatheter implantation is considered, these parameters have implications on the choice of the valve prosthesis. In patients with bicuspid aortic valve, which is often associated with a morphologically weak aortic wall that may dilate into an aneurysm and risk rupture, the dimensions of the aortic root and ascending aorta should be noted and monitored.

Severity of aortic stenosis

Patients with severe AS can be categorized as mild (AVA > 1.5), moderate (AVA 1.0–1.5), or severe (AVA ≤ 1.0 or AVAi ≤ 0.6 cm^2/m^2) based on AVA and various other empirical criteria.[9,10,10a] Severe AS can be further divided into those with normal flow and high gradient (mean gradient above 40 mm Hg), and those with low flow and low gradient (mean gradient 40 mm Hg or less). Reflection on the mechanism of low gradient is needed in gauging the prognosis and deciding on appropriate therapy.

Flow velocity and pressure gradients in aortic stenosis

As determined by the Doppler echocardiographic approach, the severity of aortic stenosis is discerned by deriving flow velocity, stroke volume, pressure gradient, and quantitation of aortic valve area (Fig 2). Maximum aortic flow velocities of < 3, 3.0–3.9, and > 4 m/s are thought to indicate mild, moderate, and severe AS, respectively. The shape of the maximum aortic flow velocity profile gives a clue to the stenosis severity. In mild to moderate aortic stenosis, the maximum flow velocity is commonly noted during the early phase of ejection time, whereas the maximum flow velocity in severe AS occurs at mid-ejection phase (Fig 2). However, it should be noted that an early peaking profile might be seen despite severe AS. If there is significant co-existent mitral regurgitation, the forward flow may decrease during the systolic phase if a sizable amount leaks into the left atrium, thereby distorting velocity since it is determined by flow rate. The ratio of proximal and distal velocity-time integral (VTI) is an index of AS severity, with an index of 0.25 pointing to severe AS. Using the maximum velocity profile of flow recorded distal to the valve, both maximum instantaneous and mean gradients are calculated using a modified Bernoulli equation, $4 \times (V_2^2 - V_1^1)$, or simply $4 \times V_2^2$, where V^1 is the velocity proximal to the valve and V^2 is the distal velocity.[11] The Doppler method's accuracy of calculating pressure gradients has been validated by simultaneous catheter-derived valve gradients.[12] Notice should be made to the differences in maximum instantaneous gradient as deduced from Doppler versus the peak-to-peak gradient deduced from the catheterization method. While mean gradients by Doppler and catheterization are similar, the maximum gradients are not. Doppler method yields the true maximum gradient, while the peak-to-peak gradient measured between LV and aortic pressure via catheterization is lower. Mean gradients of < 20 mm Hg, 20–39 m/s and > 40 mm Hg are considered to indicate mild, moderate, and severe AS, respectively. Although the AHA/ACC/ASE guidelines state that severe AS is associated with flow velocity of > 4 m/s, peak gradient of > 65 mm Hg, and mean gradient of > 40 mm Hg,[9] it would be a folly to rely on velocities and gradients alone, unless the aortic flow velocity is more than 5 m/s in the setting of a normal stroke volume (SV). Efforts should be made to obtain aortic valve area in patients with AS because a low forward SV due to a variety of disorders could result in lower velocities and lower pressure gradients despite the presence of severe AS.

Aortic valve area and AVA index

The aortic valve area indexed to the body size is the best indicator of the severity of AS. The AVA can be derived from Doppler hemodynamic data (effective or functional AVA) or by planimetry (anatomic AVA). In order to calculate the aortic valve area by Doppler hemodynamics, stroke volume must first be determined by multiplying the LV outflow area (from its diameter, use radius [r] to find area via πr^2) by the VTI of proximal flow profile; then, employing the concept of the continuity equation, AVA is calculated by dividing the stroke volume by VTI of the maximum distal flow velocity profile (Fig 3). Potential technical errors that need to be avoided in the Doppler method include wrong measurements of LV outflow diameter and incorrect recordings of flow velocities. The assumption in computing the stroke volume in the LV outflow is that the outflow area is circular. This may not be valid if