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Review Article

A Review of the 2017 American Society of Echocardiography Guidelines for Evaluation of Aortic Stenosis: Considerations for Perioperative Echocardiography

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AORTIC STENOSIS (AS) is characterized by progressive obstruction of left ventricular outflow—the end result being insufficient cardiac output and heart failure and possibly death.[1] Even though the prevalence of AS is only 0.2% among patients in their 50s, as many as 9.8% of patients in their 80s experience this disease.[1] Prompt diagnosis of AS is critical because symptomatic patients experience a 2-year mortality exceeding 50% in the absence of aortic valve replacement (AVR). In 1 study, for example, 5-year survival in patients who undergo AVR was 68% compared with 22% in those who did not undergo valve replacement.[2]

Echocardiography is an invaluable tool both to diagnose and assess the severity of AS. Given that clinical decisions are directed by echocardiographic data, echocardiographic standards to facilitate accuracy and consistency were deemed necessary.[3] In 2009, the American Society of Echocardiography (ASE) and the European Association of Echocardiography released recommendations based on scientific literature and expert consensus to assist in the evaluation of valvular stenosis, including AS.

Recently, the ASE released updated guidelines specifically focused on AS.[4] This update does not significantly alter the approach for structural assessment of the aortic valve (AV). It does, however, discuss means to more accurately assess the severity of AS. This update evolved in part from the need to address techniques for measuring the left ventricular outflow tract (LVOT)—a critical variable in grading AS. Calculation of the LVOT area can be problematic given assumptions of circular geometry as data demonstrate a high prevalence of oval-shaped LVOTs.[5] Addressing this discrepancy may improve proper classification of patients with AS. Furthermore, this update elaborates on the topic of low-flow, low-gradient AS, and it also provides a new classification scheme.

Although the ASE guidelines acknowledge some inconsistencies, several potential sources of error are not discussed. This review will begin by discussing these inconsistencies. Then standard evaluation methods for AS, as defined by the previous set of guidelines, are briefly summarized, followed by a focus on the recently updated recommendations. In addition, relevant topics for perioperative echocardiographers that are not addressed in the recent guidelines are discussed is this review.

Inconsistencies Within the Guidelines

Even before the updated guidelines were published, some of the diagnostic criteria in the previous guidelines were criticized for their inconsistencies.[6] One of the reasons for this

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perhaps relates to the origin of the criteria to diagnose AS. The initial criteria for defining severe AS were based on outcome studies using the Gorlin equation and cardiac catheterization.[7] Gorlin suggested that there is a critical aortic valve area (AVA) at which patients become symptomatic and suggested a value of ≤ 0.5 cm^2 for pure AS, and subsequent studies around the mid-1950s suggested a range of 0.5 to 1.0 cm^2.[8–10]

Cardiac Catheterization Versus Echocardiography

Over time, echocardiography was reported to have good correlation with cardiac catheterization measurements and was adopted to diagnose severe AS with the advantage of being a noninvasive option.[11–13] Severe AS still was defined by an AVA of < 1.0 cm^2, with no differentiation based on whether this measurement was obtained using catheterization or echocardiography. However, some studies have questioned whether there is a strong correlation between measurements using the 2 modalities.[6,14] For example, 1 study found that 20% of patients with severe AS as defined by an AVA < 1.0 cm^2 using echocardiography had an AVA > 1.0 cm^2 using catheterization data.[14] This raises the question of whether one set of criteria can be equally applied to multiple modalities.

Methods of Obtaining AVA

The method used to obtain the AVA also can have implications regarding its accuracy in diagnosing severe AS.[6] Doppler echocardiography using the continuity equation provides the effective orifice area (EOA), and cardiac catheterization provides the Gorlin area.[15] Even though originally it was believed that the EOA and Gorlin area always would be equal, inconsistencies have been found that can be caused by differences in valve inflow shape and aortic cross-sectional area. Accounting for pressure recovery and energy loss may resolve these differences.

The variation in valve shape also can contribute to differences between the EOA and the geometric orifice area (GOA). The GOA is anatomic orifice area and can be measured using planimetry. The EOA generally is smaller than the GOA because it represents the area at the vena contracta that occurs just downstream of the valve (Fig 1).[16] Variations in valve shape can lead to further discrepancies

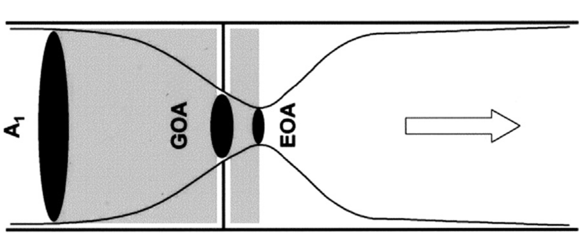

Fig 1. Geometric orifice area versus effective orifice area. Schematic representation of flow through rigid orifice plate. A_1, inlet cross-sectional area; EOA, effective orifice area; GOA, geometric orifice area. The contraction coefficient is the ratio of EOA to GOA (EOA/GOA). Reproduced from Garcia et al.[16]

between EOA and GOA due to alterations in flow dependence. Planimetry therefore has been discouraged by some groups despite being a level 2 recommendation in the ASE guidelines.[4] Overall, the potential for discrepancies is evident considering that there is one set of criteria for diagnosing severe AS with multiple means of data acquisition that have inherent differences.

Diagnostic Criteria

In addition to previously described reasons for inconsistencies when obtaining the AVA, multiple criteria also may be a factor. For example, in addition to using AVA, a mean pressure gradient ≥ 40 mmHg also can be used to diagnose severe AS.[4] It is interesting to note the evolution of this criterion. Historically, the 1998 guidelines released by the American College of Cardiology and American Heart Association required a mean gradient of 50 mmHg for severe AS.[17] The criteria appear to originate from "conservative general guidelines" from 1989.[18] In 2006, the 1998 guidelines were revised and the mean gradient to diagnose severe AS was decreased to 40 mmHg, the criterion seen in the current guidelines.[19] Criticism of this metric has arisen due to noted discrepancies. For example, it is possible for a patient to have an AVA of 1.0 cm^2 but only a mean pressure gradient of 26 mmHg using the Gorlin formula.[20] In fact, several patients with an AVA < 1.0 cm^2 have been found to have mean gradients < 40 mmHg, and this is explained only partially by low-flow states.[21] This discrepancy also is highlighted by theoretical models that show that a gradient of only 30 to 35 mmHg is expected with normal flow through the EOA of 1.0 cm^2.[22] Furthermore, Minners et al. demonstrated that this inconsistency is not just limited to echocardiography, but also extends to cardiac catheterization.[6] Some studies therefore have suggested that the AVA used to define severe AS should be decreased to 0.8 cm^2 to resolve the discordance. However, this change has not been incorporated into the guidelines, perhaps due to some studies suggesting that survival benefits of AVR correlate well with the current definition.[23] Nevertheless, these findings raise concern regarding the ability of a single cutoff for a mean gradient of 40 mmHg used across multiple modalities to capture all patients with severe AS.

Transthoracic Echocardiography Versus Transesophageal Echocardiography

The ASE guidelines also do not differentiate between the use of transthoracic echocardiography (TTE) versus transesophageal echocardiography (TEE) to diagnose AS. Furthermore, the guidelines clearly were written with a focus on TTE. Although TTE and TEE share the common modality of echocardiography, the techniques have their differences. For example, studies have shown TEE to have superior levels of sensitivity regarding detection of sources for cardiac emboli and diagnosing endocarditis.[24,25] The findings of Stoddard et al. showed that differences also extended to the diagnosis of

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