Aortic Valve Area Index
A Partial Answer to an Enigma

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It is generally believed that, (aortic valve) organic disease being once established ... is progressive ... must end in death, either by rupture of the valves, organic disease of the remaining portion of the heart, or obstruction to the current of blood. ... but ... this disorganizing process may be occasionally of singular slowness, so that the patient may live for many years in the enjoyment of good, or at least tolerable health, or that the diseased action is really arrested and the lesion becomes stationary.

Thus, did William Stokes categorize the uncertain natural history of aortic stenosis (AS) more than a century and half ago.1 Since his elegant description, AS has remained an enigmatic problem, particularly in the absence of symptoms. On one hand, the presence of typical symptoms and a peak transaortic velocity of >4 m/s and a valve area of <1 cm² results in a poor prognosis if untreated, as further elegantly described >50 years ago.2 In the absence of symptoms, however, the relation between aortic valve area (AVA) and prognosis becomes much less certain; every cardiologist struggles with the question of when (if at all) to intervene upon such patients. Will their fate “end in death” as Stokes lamented or will they be among the fortunate who “live for many years... (as) the lesion becomes stationary.”

See Article by Tribouilloy et al

In these patients, the cutoff of 1 cm² may mislead because, by itself, it provides no information about actual forward flow. Multiple studies have shown that this value (1 cm²) corresponds unevenly to a transvalvular velocity of 4 m/s, generating an inherent inconsistency in assessment of AS severity.3 If, for instance, an AVA <1 cm² is measured in the setting of a low transvalvular gradient, determination of transvalvular flow becomes a vital factor in the decision-making process.4 As a partial solution, current guidelines recommend indexing AVA to body surface area (BSA); a value of 0.6 cm²/m² has been used as a cutoff for defining AS as severe.5 The value of 0.6 cm² has a long history. As early as 1967, the indexed value provided greater specificity in the setting of increased ventricular stroke work loss, a variable that identifies severe left ventricular outflow tract obstruction.6 Some investigators have suggested a value of 0.7 cm²/m², whereas still others support the value 0.4 cm²/m² as an optimal prognostic dividing line.7

In light of such challenges, the continued emergence of novel methods for evaluation of extent of AS is both laudable and unsurprising. The ultimate objective is to be able to offer a surgical or transcatheter intervention for AS such that the benefit of treatment at that moment will outweigh the risks of further watchful waiting. In this context, the article by Tribouilloy et al8 in this issue of Circulation: Cardiovascular Imaging is a welcome addition to the conversation surrounding identification of those at highest risk for progression of disease. In their study, the authors queried a prospectively assembled comprehensive database to select asymptomatic patients with an AVA of ≤1.3 cm², preserved left ventricular systolic function, and no other significant valvular disease. Subsequently, they observed and recorded their progression to death or aortic valve replacement, outcomes consonant with the development of severe AS. They determined that an AVA indexed to BSA of <0.6 cm²/m² did not distinguish those who enjoyed favorable outcomes from those who suffered adverse ones; indexing to height proved to be superior for this purpose. Their findings held true after adjustment for multiple relevant variables including age, sex, comorbidities, and ejection fraction. Among their patients, the clearest inflection point between a benign and adverse outcome occurred at an indexed AVA of 0.4 cm²/m². Thus, their findings call into the question the applicability of 0.6 cm²/m² as a universal cutoff for severe AS, and whether indexing to AVA to BSA is the best possible approach.

Across all studies, the ongoing variability of findings adds strength to the authors’ assertion that BSA by itself will not distinguish those patients with a rapidly progressive course from those who progress more slowly. As the authors suggest, BSA does not precisely track with AVA. AS might not produce significant limitation in a patient with increased fat, yet that increased weight from fat translated into increased BSA will reclassify that patient from moderate to severe AS without any change in valve area. As an indexation tool, BSA increases sensitivity for the diagnosis of severe AS, but its positive predictive value is limited by its changeability within a given individual. The rate of progression in a patient with asymptomatic AS is not likely to be affected much by weight gain or loss alone. An indexation approach that uses a variable that does not fluctuate to the same degree as BSA might well be a more powerful predictive tool, and the authors argue convincingly for indexing to height as such a superior instrument. In contrast to weight, height changes slowly once adulthood is reached and then in only one
direction. Although AS correlates with competing indexation methods, in the author’s model, at least, height proved to be better than those variables at predicting prognosis.

Height alone is not a perfect tool; it provided the best fit to their statistical model, yet many patients with severe AS remained above the index’s cutoff. Such limitations provide a note of caution, reminding us that any anthropometric measure used as the sole arbiter of risk stratification for progression to severe AS will tell a story that is incomplete at best and misleading at worst. As we know, stroke volume has a significant incremental role to play. In the setting of a low transaortic velocity, a low flow state produces a condition that has a natural history indistinguishable from severe symptomatic AS; when it’s normal, the low-gradient AS produces a natural history that is far more benign.9 In totality, although indexing to height is a method that seems to increase sensitivity, it is not a substitute for careful, comprehensive, clinical assessment. Perhaps, the ideal approach to identifying those who at greatest risk will combine indexation to a basket of other variables, such as contractility, left ventricular mass, stroke volume, and stroke work. The current effort by Tribouilloy et al advances our understanding of the complexity of predicting the fate of patients with AS. For now, AS remains an enigma that will test the clinician’s skill and judgment on into the foreseeable future.

Disclosures
None.

References

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