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EDITORIAL

Is the stress aortic valve index a fractional flow reserve for the stenotic aortic valve?

Morton J. Kern[1,2]*, MD; Derek Antoku[1,2], MD

Corresponding author: Long Beach Veterans Administration Medical Center, 5901 East 7th Street, 111C, Long Beach, CA, 90822, USA. E-mail: mortonkern2007@gmail.com

In patients with severe aortic stenosis (AS), aortic valve replacement (AVR), performed percutaneously or by surgery, is truly life-changing. However, in patients with moderate or mild AS, decision-making can be vexing as the optimal treatment remains unclear. Uncertainty around the need for valve replacement in such patients is especially keen in those who have dyspnoea, chest pain, or presyncope that cannot be completely attributed to the aortic valve. Moreover, evidence suggesting that moderate AS is associated with increased morbidity and mortality[1,2] makes the decision for AVR even more poignant.

In this issue of EuroIntervention, Eerdekens et al examine the haemodynamic and clinical outcomes of a blinded, prospective, observational cohort of 52 patients with symptomatic moderate or mild aortic stenosis and preserved left ventricular (LV) function[3]. They tested the hypothesis that the stress aortic valve index (SAVI), defined as the aortic/left ventricular pressure ratio during dobutamine stress, would distinguish physiological responses beyond traditional valve metrics, better identifying patients for AVR who otherwise might not be treated appropriately.

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The study group underwent echocardiography and transvalvular haemodynamic testing at rest, during bicycle exercise, and during dobutamine stress, as well as calcium scoring, brain natriuretic peptide (BNP) testing, etc. The novel SAVI was calculated from invasive haemodynamics but can also be derived from echocardiographic data, using aortic (Ao) pressure and estimated LV pressure (defined as aortic pressure plus left ventricular outflow tract velocity gradient pressure) during dobutamine stress[4,5]. The study was blinded insofar as the SAVI results were not provided to the treating physicians who made decisions for therapy according to existing standards over the 1-year follow-up period. Calcium scoring, BNP, and other adjunct measures used in clinical decision-making for AS were also analysed.

Dobutamine infusion increased the transvalvular aortic gradient from 25 mmHg to 42 mmHg, with the resting Ao/LV pressure ratio declining from 0.82 to 0.70 (the SAVI). The aortic valve area (AVA) at rest, as calculated by the Gorlin formula, did not predict the stress haemodynamic response. Furthermore, 25 of 52 patients demonstrated a SAVI of <0.70, a value which was comparable to that found in patients with severe aortic stenosis who were studied on a separate occasion[5]. A low SAVI was associated with the need for AVR as well as death (3 patients with a SAVI <0.70 vs 0 patients with a SAVI >0.70) but not with stroke, heart failure, atrial fibrillation, or the need for a pacemaker. The SAVI independently predicted clinical aortic valve intervention (hazard ratio 5.7; p=0.007), whereas AVA, BNP, and sex-specific calcium thresholds did not. The authors concluded that the SAVI unmasked patients not identified by conventional metrics who were at higher risk for valve intervention and who would otherwise not be treated for their moderate/mild aortic stenosis.

By identifying patients with marginally severe AS needing AVR, the SAVI seems to be a step forward in that it was better than AVA and transvalvular gradient alone. For patients with clearly haemodynamically severe AS, the SAVI was comparable to AVA/transvalvular gradient and therefore added little clinical value in this group. While the SAVI provides information gleaned from stress responses,

much like fractional flow reserve (FFR) does for coronary stenosis, the similarity ends there. FFR involves a small-tube mechanical model, translating hyperaemic translesional pressure into a percentage of normal flow across the coronary artery stenosis. The normal FFR value in the absence of coronary disease is 1. On the other hand, the SAVI is employed in a large-tube model with more complex mechanics than the simple tubular narrowing of a coronary artery. The SAVI normalises stress-induced left ventricular-to-aortic pressure, creating a new pressure index of valve severity. The threshold of <0.70 for the SAVI was selected based on its association with severe aortic stenosis[4,5]. The SAVI presumably eliminates some of the errors in the Gorlin valve area calculation (such as correction factors accounting for dysfunctional valve leaflets, shape of the valve orifice, aortic impedance, LV function, LV outflow transmission, etc.). The SAVI may be particularly useful for the low-flow, low-gradient, severe AS patients[4]. For the clinician, the SAVI could augment the interpretation of the resting transstenotic pressure gradient and AVA for decision-making about AVR. However, the SAVI responses cannot be predicted from resting transvalvular haemodynamics because of the complex nature of the valve system and its associated rheology. The SAVI is differentiated from AVA because it depends, in part, on whether the flow dynamics of the valve act like a variable resistor or a fixed orifice model[4]. The unique anatomical and physiological variables of the aortic valve are likely exaggerated during exercise and appear to contribute to inaccuracies in the Gorlin formula. Although the SAVI concept has been known for almost a decade, bringing a new index into routine clinical use is a monumental task. Like FFR users before them, SAVI proponents will have to address the same challenges in validation, and demonstrate a strong link to outcomes, before the SAVI can be used routinely.

There are several study limitations that should be noted. The small study population makes this clinical work underpowered, though it remains a highly suitable pilot study. The selection of the endpoint is also challenging. An obvious comparison to existing haemodynamic metrics is not a substitute for clinical outcomes. For severe AS, the SAVI is comparable to AVA and/or transstenotic gradient. For moderate/mild AS, the SAVI is at odds with AVA in some patients as it can produce highly variable responses. Eerdekens et al[3] used 2 standards (AVA/transstenotic gradient and the need for AVR) to validate the SAVI. A clinical standard could be questioned, since patient decisions are not based solely on a single measurement or index. We often use (or misuse) AVA as a decision point for AVR, but for those patients with equivocal findings, clinicians must incorporate several ancillary parameters (e.g., symptoms, calcium scores, BNP, etc.) in making the final decision.

Lastly, we do not know if the patients with moderate/mild AS with a low SAVI progressed to severe AS, or whether there was another cause for AVR such as coronary artery disease progression or new or refractory symptoms despite mild valve areas and a high SAVI that remained in the moderate/mild range. If the SAVI can accurately predict AS progression or identify those who may develop persistent symptoms, despite not meeting traditional criteria for AVR, the association of the clinical decisions with a SAVI <0.70 would further validate its usefulness[6].

As the authors acknowledge, theirs is a pilot study for larger trials such the PROGRESS trial and the EXPAND TAVR II trial[7,8] currently enrolling patients with moderate AS for transcatheter aortic valve implantation.

Just as FFR is useful to appropriately select patients for percutaneous coronary intervention, the SAVI may help us better identify those with borderline valve lesions who will benefit from AVR. Eerdekens et al[3] are to be applauded for providing a strong pilot study which suggests that the SAVI "…(has) proved its ability to predict clinical outcomes". Although "proved" may be premature, we believe that, with larger studies, the SAVI will likely become a valuable tool in the assessment of aortic stenosis.

In researching more about aortic stenosis, we found that Dr Richard Gorlin and his father, W.B. Gorlin[9], may have been prescient in their thinking about the aortic valve area formulae, stating "… the cardiologist should estimate valve obstruction by both anatomic and physiologic methods. The two data sets may not always match, because they should not necessarily match! The use of correlation coefficients that force the data to fit or not fit may no longer be the proper exercise. Unusual dissipation of pressure that yields a discrepancy in derived valve area may be telling us more about the valve than we knew before. The discrepancy may reveal that there are additional sources of energy loss…". These thoughts invite deeper and continuing consideration of the use of transvalvular haemodynamics such as the SAVI.

Authors' affiliations

1. Department of Medicine, VA Long Beach Health Care System, Long Beach, CA, USA; 2. Department of Medicine, University of California, Irvine, CA, USA

Conflict of interest statement

M.J. Kern is a speaker for Abbott, Acist Medical, Boston Scientific, CathWorks/Medtronic, Haemonetics/Opsens, and Philips; and a consultant to Merit Medical and SummaCor. D. Antoku has no conflicts of interest to declare.

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