

Survival by stroke volume index in low-gradient normal EF severe aortic stenosis: insights into myocardial systolic dysfunction

To the Editor,

We read with interest the recent article on survival by stroke volume index (SVI) in patients with low-gradient normal EF severe aortic stenosis, which demonstrated lower SVI is incrementally associated with mortality.¹

The authors discuss a putative mechanism of low stroke volume (SV) secondary to concentric remodelling which results in reduced LV cavity size. This impedes LV diastolic filling culminating in diminished systolic function despite normal EF. The authors quote evidence of systolic impairment, for example reduced longitudinal strain, in similar cohorts with preserved EF.² In the current study, the subgroup with lowest SVI, and therefore presumed most severe systolic impairment despite EF >50%, demonstrated the thickest relative wall measurements. We believe this observation helps to explain the apparent paradox between significant myocardial dysfunction and preservation of EF in this cohort and in the wider 'heart failure with preserved ejection fraction' context. Recent mathematical modelling of LV contraction has shown that both myocardial shortening and end-diastolic wall thickness are determinants of EF.³ Essentially, absolute LV wall thickening, as defined by the absolute difference between wall thickness at end-systole and end-diastole, may be nearly normal in patients with concentric LV hypertrophy (LVH) because absolute systolic thickening will be augmented in response to increased end-diastolic LV wall thickness. As a result, the endocardial displacement and EF will also be normal, as the external LV volume remains fairly constant throughout the cardiac cycle⁴ and the absolute wall thickening may appear to compensate for any contractile strain abnormality. The development of concentric LVH may be viewed as a compensatory response that normalises contractile stress and total contractile force. However, if contractile stress remains reduced, the contractile force will be inadequate and result in a fall in stroke volume despite the preserved EF. In order to understand the apparent discrepancy in SV and EF, one must distinguish between contractile strain and stress and the relationship between end-diastolic wall thickness and EF.

The authors elected to investigate SV indexed to body surface area. However, it would be interesting to know whether correcting EF for the presence of concentric LVH (EF_c), as described in mathematical modelling studies of the LV,³ would be a useful prognostic marker in this cohort of patients. After all, EF_c is potentially an even more relevant allometric indexed value given the importance of end-diastolic wall thickness in patients with concentric LVH and systolic impairment but preserved EF.

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