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INVITED COMMENTARY

In this issue of *The Annals*, Cho and colleagues [1] reported the association of thoracic aorta calcium score (TACS) to left ventricular (LV) hypertrophy and clinical outcomes in patients with severe aortic stenosis (AS). The main finding of the study was that high TACS was associated with LV hypertrophy, and it predicted less regression of LV mass and poor clinical outcomes following aortic valve replacement. This observation timely recalls that LV remodeling, a major determinant of outcome, is a complex pathophysiologic process. It interplays the inherited ability of the LV to adapt to increased afterload, the severity of valvular obstruction, and the reduced arterial compliance. The contribution of the vascular compliance to the LV remodeling through the so-called ventriculoarterial coupling is a well-known phenomenon. Increased arterial stiffness as estimated by an increase in pulse wave velocity has recently been shown to be associated with decreased quality of life and cognition after aortic valve replacement [2, 3]. The clinical relevance of these reports [2, 3], in line with the study by Cho and colleagues [1], is to place AS within a degenerative vascular disease continuum that involves the arterial tree from the aortic valve and the large arteries toward the arterioles, sharing common risk factors.

While carotid-femoral pulse wave velocity is the gold standard measure of arterial stiffness, with validated normal reference range values [4], TACS only remains an indirect and potentially late measure of the vascular compliance. The methodology for TACS has to be standardized and widely tested for interscan, scanrescan, and interobserver reproducibility. TACS normative values for age, sex, and ethnicity have to be compiled before it can enter the clinical armamentarium. Nevertheless, unenhanced computed tomography used for TACS has the advantage of comprehensively evaluate broadly the calcification load, including coronary and valve calcification scores that are inaccessible to other techniques, which respectively are risk markers for coronary artery stenosis and of postoperative outcomes in patients with AS [5]. Cho and colleagues [1] failed to replicate the former finding because the patients with notorious

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coronary vascular disease were excluded from the cohort. On a clinical view, the study by Cho and colleagues [1] provided no sufficient evidence to change how we care for patients with AS, because of limited study sample and inherent composite input limitations pertaining to its retrospective nature. However, they should be congratulated for indicating how it could be important to redefine the risk factors in patients with AS using vascular compliance assessment, both for disease severity and prognosis-based operative decision making. Their report thus paves the way for larger prospective and controlled trials to improve stratification of AS using vascular compliance and calcification scores.

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