Letter by Lumley et al Regarding Article, “Arterial Pulse Wave Dynamics After Percutaneous Aortic Valve Replacement: Fall in Coronary Diastolic Suction With Increasing Heart Rate as a Basis for Angina Symptoms in Aortic Stenosis”

To the Editor:

We read the recent article “Arterial Pulse Wave Dynamics After Percutaneous Aortic Valve Replacement: Fall in Coronary Diastolic Suction With Increasing Heart Rate as a Basis for Angina Symptoms in Aortic Stenosis”1 with great interest and would like to commend the authors for producing novel coronary wave intensity data in the aortic stenosis population. The microcirculatory decompression (suction) wave was reported to increase in proportion to the severity of aortic stenosis (r=0.59, \( P<0.05 \)), a change opposite to that which was reported previously by the same authors in patients with left ventricular hypertrophy.2 The severity of aortic stenosis in determining this relationship was judged by peak aortic gradient.

The 11 patients included in the study had an aortic valve area of 0.6±0.1 cm², suggesting a relatively homogeneous population with respect to aortic valve area. In contrast, a broad range of peak valve gradients was observed (81±24 mm Hg), which would be consistent with a range of left ventricular (LV) systolic function. It is therefore possible that the peak aortic gradient is a surrogate measure of LV function in this cohort, most of whom had an aortic valve area <0.8 cm² (2 SD above the mean). In this context, the magnitude of the microcirculatory decompression wave may, in fact, be a reflection of LV systolic and diastolic function rather than aortic stenosis per se. The authors state in their article that the extent of microcirculatory compression during systole is related to the rate and magnitude of decompression (recoil) in diastole. Hence, compared to those with impaired LV function, patients with preserved function would be expected to have greater systolic microcirculatory compression and consequently more pronounced recoil, leading to an increased microcirculatory decompression–associated wave energy.

Given that simultaneous pressure and Doppler velocity were measured in the left main coronary artery rather than the left anterior descending or circumflex vessel, it would be surprising if septal and posterior wall thickening (individually or collectively) were found to correlate with the amplitude of the microcirculatory decompression wave. On the other hand, it would be interesting to assess the relationship between the left main coronary artery backward decompression wave and global LV function (as assessed by LV ejection fraction or LV outflow tract–velocity-time integral) in this study cohort. Similarly, it may be instructive to assess the relationship between this wave and indices of aortic stenosis, which are least dependent on LV function, such as aortic valve area.

Percutaneous aortic valve replacement is an innovative model for assessing the impact of aortic stenosis and of relieving the valve stenosis. However, the use of rapid ventricular pacing (during balloon valvuloplasty and during positioning and implantation of the valve prosthesis) often has a detrimental effect on LV function, particularly when baseline function is poor. Any hypotensive effects of such changes are usually countered by the administration of vasoactive agents, which increases afterload. Both diminishing LV contractility and increasing afterload would be expected to decrease the amplitude of the backward decompression wave, which may in turn confound the effects of relieving the valve stenosis. Thus, interpretation of the data after percutaneous aortic valve replacement is complicated, and the precise contribution of valve stenosis to coronary wave intensity profiles is somewhat unclear.

Disclosures

None.

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