Changing Reflections of the Coronary Microcirculation After Percutaneous Aortic Valve Replacement
Novel Observations With Arterial Pulsed Wave Dynamics

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In this issue of Circulation, Davies et al assess the changes in coronary blood flow and reserve after the relief of aortic stenosis (AS) by percutaneous aortic valve replacement (PAVR) and implicate these findings as additional mechanisms bearing on the critical role of the coronary microcirculation in this population.

One cannot help but be intrigued by the novel physiological insights that pulsed wave analysis brings to one of the oldest cardiovascular diseases, AS, and one of the newest procedures in interventional cardiology, namely PAVR. For decades, the diagnosis and treatment of AS have rightly focused on identifying and then ameliorating the adverse consequences of abnormal transvalvular pressure and flow. In the absence of coronary artery disease, the mechanisms of angina in AS were debated and attributed mostly to microvascular insufficiency with ischemia resulting from myocardial oxygen supply/demand imbalance. Left ventricular (LV) angina in AS were debated and attributed mostly to microvascular insufficiency with ischemia resulting from myocardial oxygen supply/demand imbalance. Left ventricular (LV) muscle mass was increased out of proportion to myocardial blood flow to the myocardium) in 11 patients with AS and their valvular LV outflow obstruction. Before PAVR, the diastolic suction wave of LV relaxation at the beginning of diastole (wave 5), increasing epicardial flow from the aorta into the epicardial coronary artery and into the myocardium. During systolic ejection with a normal aortic valve, LV and aortic pressures are coupled, with LV pressure being the major determinant of intramyocardial stress. Because pressures at each end of the coronary artery (aortic and LV myocardial) are similar during systole, the net change in coronary flow during systole is normally minimal. In diastole, the aortic valve closure uncouples aortic from LV pressure, producing an aortic–LV myocardial pressure gradient accelerating coronary blood flow (via the diastolic suction wave) into the epicardial artery and myocardium.

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However, in patients with aortic stenosis during systole, intramyocardial compression, resistance, and myocardial wall tension are already markedly increased relative to patients without AS. The normal physiological reserve is impaired with reduced diastolic suction during stress. The pathophysiological states of increased LV afterload (valve stenosis) and myocardial stress (wall tension, thickness/chamber volume) and the suboptimal uncoupling of aortic and LV pressures, critical for optimal coronary perfusion, are improved after PAVR.

Beyond renewing an appreciation of the complexities of pulsed wave analysis, this study has relatively few limitations. The study population is small, involving mostly elderly women with normal arteries. Applicability to other patient groups, such as elderly men or younger patients, may not hold completely, but this is probably unlikely. Other confounding factors include the influence of anesthesia, volume status before and after PAVR, and preexisting hypertension and LV hypertrophy. Pacing rather than a pharmacological stimulation was used as the myocardial stressor to test coronary reserve. Although the authors noted that it is uncertain whether pharmacological stress with dobutamine would have produced different findings, one can speculate that adding more inotropic stimulation (increasing contractility and myocardial compression) to the tachycardia would likely make the abnormalities more pronounced. None of these issues significantly negates the interpretation of the unique pulsed wave dynamics measured in the experimental setting used.

This study supports the notion that anginal symptoms are attributable to more than the impaired microcirculation produced by excessive LV hypertrophy. AS produces increased myocardial wall stress with increased microvascular compression, reduced time-varying elastance, high LV end-diastolic pressures, and attenuated diastolic filling time and filling wave magnitude into the microvasculature.5,6 Davies et al show us that elimination of aortic valve resistance after PAVR impacts these mechanisms positively and can improve coronary flow, all findings that may explain, in part, the highly favorable clinical response to both surgical AVR and PAVR. Outcome studies characterizing patients by pulsed wave analysis will likely make this method a relevant tool in the future for understanding the complex relationships among peripheral vascular disease, coronary artery disease, and LV mechanics in our patients with a variety of valvular and nonvalvular myocardopathies and potentially compromised microcirculatory reserve.

**Disclosures**

Dr Kern serves on the speakers’ bureau for Volcano Therapeutics and St. Jude Medical Inc.
References


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