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) muscle mass was increased out of proportion to myocardial vascular insufficiency with ischemia resulting from myocardial oxygen supply/demand imbalance. Left ventricular (LV) muscle mass was increased out of proportion to myocardial capillary oxygen supply. As one might expect in medical science, this is not the end of the story. Examining the arterial pulsed wave dynamics that characterize features of both epicardial and microcirculatory blood flow, Davies et al detail additional mechanisms beyond coronary reserve impairment to better understand why angina may occur in the AS patient with normal coronary arteries. PAVR produced not only a fall in myocardial microvascular resistance, but also improved diastolic filling waves related to favorable changes in LV wall stress as another postulated mechanism for a reduction in anginal symptoms in patients relieved of their valvular LV outflow obstruction.

In this group’s most recent application of pulsed wave analysis, Davies et al report changes in the intracoronary diastolic suction wave (the principal accelerator of coronary blood flow to the myocardium) in 11 patients with AS and normal coronary arteries. Before PAVR, the diastolic suction wave intensity was related to the severity of AS; the higher the gradient was, the greater the diastolic suction wave intensity was. However, in contrast to normal wave patterns, pacing at 90 and 120 bpm decreased the diastolic suction wave intensity, a response that represented impaired coronary flow reserve. A reduced diastolic filling wave reduces diastolic myocardial flow. More important, after PAVR, repeat measurements demonstrated a fall in the diastolic suction wave intensity at rest, which no longer correlated to the transvalvular gradient or LV wall stress as it did before, and in contrast to the pre-PAVR state, pacing-induced myocardial stress produced a rise in the diastolic suction wave intensity and hence more coronary flow to the myocardium. The coronary physiological reserve measured in this way specifically incorporates the influence of the backpressure of coronary microvascular resistance, which is impaired in AS and improved immediately after PAVR with restored and nearly normalized intracoronary pressure wave patterns.

Understanding coronary pulsed wave analysis requires a reintroduction to the method. The pulse analysis begins with the acquisition of simultaneous high-fidelity intracoronary pressure and flow velocity signals obtained from sensor-tipped angioplasty guidewires positioned during coronary angiography. From the pressure and flow data, normal coronary perfusion is the net result of pressure waves (and flow) generated in the proximal aorta and those counterbalancing pressure waves moving back toward the aorta originating in the distal microcirculatory end of the coronary flow path. The cycle of cardiac contraction and relaxation produces 6 pressure waves with different magnitudes (intensity), directions, and velocities as a result of competing acceleration and decelerations (Figures 1 and 2). Coronary blood flow into the myocardium is determined largely by the prominent coronary 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.
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. 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 myocardiopathies 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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