Commentary: One cannot fix what one cannot see

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Surgeons have a simplistic way of classifying heart disease purely based on 2-dimensional imaging assessment of cardiac anatomy. As such, we embraced the definition of heart failure with preserved ejection fraction (HFP EF) as the presence of heart failure symptoms with a left ventricular ejection fraction (LVEF) ≥50%. However, a preserved LVEF can only exclude heart failure with reduced ejection fraction; it has no diagnostic role to prognosticate patients in the group of HFP EF. We are missing measurements of abnormal physiology and impaired ventricular relaxation to further understand the mechanisms in HFP EF. In this context, the Heart Failure Association Pretest Assessment, Echocardiography and Natriuretic Peptide, Functional Testing, Final Etiology (HFA-PEFF) score was developed as a novel diagnostic tool for HFP EF. This novel heart failure assessment tool incorporates not only anatomical criteria but also biochemical and functional variables.

In this issue of the Journal, Lee and colleagues used the HFA-PEFF score to stratify patients in a retrospective cohort study of 3593 patients with a preserved LVEF who underwent coronary artery bypass grafting. Thirty-day mortality was similar among those with and without HFP EF. However, the 5-year survival was significantly lower in the HFP EF group compared with the non-HFP EF group (91.9% vs 97.0%; hazard ratio, 2.41; 95% confidence interval, 1.29-4.50; \( P = .006 \)).

Another important finding from this study is that there was no significant improvement in early diastolic mitral annular velocity (\( e' \)) or LV filling pressures (\( E/e' \)) at echocardiographic follow-up in the HFP EF group and indeterminate group. In other words, diastolic dysfunction in patients with coronary disease may not be reversible despite complete revascularization, even in the early stages of HFP EF before becoming detectable on diagnostic testing. This finding elicits the following question: is ventricular reverse remodeling possible in HFP EF?

Myocardial revascularization may not promote the same degree of ventricular reverse remodeling in HFP EF as it does in heart failure with reduced ejection fraction. This is possibly due to ongoing microvascular disease and inflammation in HFP EF, which cannot be addressed by macroscopic revascularization. It is therefore not surprising that Lee and associates report a greater long-term mortality and complication rate in patients with HFP EF after coronary artery bypass grafting compared with patients without HFP EF. These patients are also at greater risk of poor quality of life after surgical revascularization.

In light of these mortality and quality of life outcomes, is it possible that surgical revascularization may not be as useful patients with HFP EF and coronary artery disease? Do these patients need some future alternative or concomitant therapy for microvascular disease to optimize their results? Perhaps our understanding of how to innovate to address this is limited by the inability of surgeons to see this therapy with our bare eyes.
References


