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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 (HFpEF) as the presence of heart failure symptoms with a left ventricular ejection fraction (LVEF) $\geq 50\%$. 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 HFpEF.¹ We are missing measurements of abnormal physiology and impaired ventricular relaxation to further understand the mechanisms in HFpEF. 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 HFpEF.¹ 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 colleagues² 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 HFpEF. However, the 5-year survival was significantly lower in the HFpEF group compared with the non-HFpEF 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

Microvascular dysfunction can be difficult to visualize with the naked eye.

CENTRAL MESSAGE

Diastolic dysfunction in heart failure with preserved ejection fraction and coronary disease may not be reversible with surgical revascularization due to ongoing microvascular disease and inflammation.

annular velocity (e') or LV filling pressures (E/e') at echocardiographic follow-up in the HFpEF 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 HFpEF before becoming detectable on diagnostic testing. This finding elicits the following question: is ventricular reverse remodeling possible in HFpEF?

Myocardial revascularization may not promote the same degree of ventricular reverse remodeling in HFpEF as it does in heart failure with reduced ejection fraction. This is possibly due to ongoing microvascular disease and myocardial inflammation in HFpEF, which cannot be addressed by macroscopic revascularization.^{2,3} It is therefore not surprising that Lee and associates report a greater long-term mortality and complication rate in patients with HFpEF after coronary artery bypass grafting compared with patients without HFpEF. These patients are also at greater risk of poor quality of life after surgical revascularization.⁴

In light of these mortality and quality of life outcomes, is it possible that surgical revascularization may not be as useful patients with HFpEF 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.

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