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Commentary: Short-term pain, long-term gain with left ventricular function after mitral valve repair

Clancy W. Mullan, MD, and Arnar Geirsson, MD

Mitral regurgitation is a common disorder with serious consequences for patient morbidity, including arrhythmias, heart failure, and stroke, as well as mortality.¹ For acceptable-risk patients with severe, symptomatic disease or evidence of left ventricular (LV) dysfunction, surgical repair at a center of excellence is widely accepted as the gold standard therapy²; however, no formal recommendations for optimal repair strategies exist, likely due to the multiple and complex phenotyping of primary mitral valve disease. The Canadian Mitral Research Alliance CardioLink-2 (CAMRA-2) trial was designed to evaluate differences in mitral valve function after leaflet-preserving versus leaflet-resecting techniques, with no observed differences between strategies.³ Hibino and colleagues⁴ report a subanalysis of the CAMRA-2 trial to investigate potential phased recovery of the LV and timeline of LV ejection fraction (EF) improvement after mitral valve repair, aiming to inform the conflicting evidence that had previously existed.⁵⁻⁷

The authors evaluated LV echocardiographic parameters and describe 2 phases of LV recovery after mitral repair. Independent of leaflet resection or preservation in their cohort, the LV demonstrated a decreased EF with improved forward stroke volume and end diastolic volumes. Over the subsequent year, the EF improved, albeit not to baseline. Although not improved at discharge, systolic parameters recovered over the following year.⁴

From the Division of Cardiac Surgery, Department of Surgery, Yale School of Medicine, New Haven, Conn.

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Address for reprints: Arnar Geirsson, MD, Division of Cardiac Surgery, Yale School of Medicine, New Haven, CT 06510 (E-mail: arnar.geirsson@yale.edu).

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Clancy W. Mullan, MD (left), and Arnar Geirsson, MD (right)

CENTRAL MESSAGE

Hibino and colleagues present a subanalysis of the CAMRA CardioLink-2 trial, observing a 2-phased recovery of the left ventricle: Early diastolic recovery followed by late systolic recovery.

The authors should be applauded for the limitations they acknowledge, which are insightful, thorough, and exemplary in their honest self-evaluation. Two should be highlighted. First, the authors acknowledge the well-compensated nature of their population, which could limit generalizability. Although the observed EF of their population is similar to the real-world population of patients undergoing mitral repair,⁸ an interesting observation is a widening of the distribution of predischARGE EF. Given the relevance of postrepair EF to late survival,⁹ detailed phenotyping, including biomarker profiling,¹⁰ of patients to describe those demonstrating a significant reduction in EF would provide valuable clinical insight, although this analysis is, of course, limited by its nature as a subanalysis of a completed clinical trial. Second, the choice of 2-dimensional echocardiography is a meaningful limitation compared with the more reproducible cardiac magnetic resonance imaging or 3-dimensional echocardiography.¹¹ We are further limited by lack of information on whether or not imaging was contrasted.

Two additional observations are important for interpretation of the data. First, in the CAMRA-2 trial, twice as many patients in the resection group underwent concomitant coronary artery bypass.³ Second, the cardioprotection strategies employed by the trial are not described. Both may have influenced the observations described here.

Hibino and colleagues⁴ provide valuable, hypothesis-generating observations in their study, and they should be applauded for their efforts to inform a challenging and elusive question. Future studies to define patients likely to or to not demonstrate LV recovery after mitral repair should be conducted with more reliable imaging techniques, comprehensive phenotyping, and homogeneity of myocardial preservation.

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