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Commentary: Chain reaction: Incorporating the anterior mitral leaflet during mitral valve replacement distorts left ventricular mechanics

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As endovascular technology evolves, transcatheter mitral valve replacement (TMVR) has become a viable option for those patients at too high risk to undergo surgical MVR.¹ Although TMVR avoids the burden of cardiopulmonary bypass and open-heart surgery, a cardiovascular mortality rate up to 23.3% at 10 months post-TMVR has been observed, despite excellent valve hemodynamic parameters.¹ Brunel and colleagues² investigate the hemodynamic effects of anterior mitral leaflet (AML) incorporation into the annulus by mitral valve prostheses, as it occurs in TMVR, to aid in understanding the physiologic influence these devices have on left ventricle function.²

TMV prostheses are designed to fix the AML to the MV annulus to minimize the risk of an incompetent AML obstructing the left ventricular outflow tract (LVOT). However, the authors hypothesized that incorporating the AML into the annulus could potentially disrupt valvular–ventricular interactions with a subsequent deleterious effect on left ventricle function.³ To test their theory, they placed mechanical MV prostheses in 14 sheep with an exteriorized snare that allowed them to manipulate the AML between the incorporated and released position. Postoperative hemodynamic outcomes demonstrated incorporation of the AML, compared with release, was associated with significant decreases in the following critical indices: cardiac output,

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Disclosures: Dr Kilic is a member of the Medical Advisory Board for Medtronic, Inc. All other authors reported no conflicts of interest.

The *Journal* policy requires editors and reviewers to disclose conflicts of interest and to decline handling or reviewing manuscripts for which they may have a conflict of interest. The editors and reviewers of this article have no conflicts of interest.

Received for publication May 21, 2021; revisions received May 21, 2021; accepted for publication May 26, 2021; available ahead of print June 14, 2021.

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JTCVS Open 2021;7:123–4
2666-2736

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<https://doi.org/10.1016/j.jtc.2021.05.013>

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CENTRAL MESSAGE

The incorporation of the anterior mitral leaflet into the annulus during mitral valve replacement in an ovine cohort alters valvular–ventricular interactions resulting in decreased left ventricle function.

stroke volume, stroke work, and left ventricular end systolic pressure. Load-independent indices of left ventricular contractility (eg, end-systolic pressure-volume relationship, and preload recruitable stroke work) and left ventricle length were also significantly decreased during AML incorporation.

The authors attribute these findings to disruptions in valvular–ventricular interactions caused by immobilization of the AML. They suggest that an incorporated and immobile AML is unable to generate a closing force or to transfer that closing force to the left ventricle wall through the chordae tendinae and papillary muscles.

In the setting of severe mitral regurgitation, the principal goal of MV intervention is to improve forward flow. If incorporation of the AML during TMVR partially negates this improved forward flow, then, in the words of the authors, the procedure has merely “exchanged 1 hemodynamic derangement for another.”² However, LVOT obstruction is a serious and potentially fatal complication of TMVR. In a recent study of 194 patients undergoing TMVR, 13.4% of patients experienced LVOT obstruction, resulting in 34.6% of these patients having procedure-related death, 19.2% being converted to surgery, and 38.5% requiring emergency transcatheter reintervention.⁴ Thus, the contractile benefit of AML release must be weighed against the serious sequelae of LVOT obstruction.

The balance of avoiding LVOT obstruction with optimizing left ventricle function adds another layer of complexity to decision making for high-risk patients with MV disease. Brunel and colleagues² present compelling

preliminary evidence that release of the AML could provide improved left ventricle mechanics after TMVR and prompt future inquiry into this phenomenon in clinical settings.

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