A RIGHT TRACK STEMS FROM THE RIGHT LEARNING

To the Editor:

Wan and Hu's commentary¹ on our work² is notable; however, some points need clarification.

The geometric disorder of the ischemic mitral valve (MV) is caused by the 3-dimensional modification of the anteroposterior annular diameter, area/volume of tenting, and interpapillary muscle distance (IPMD). In extreme conditions of altered geometry (left ventricular [LV] end systolic volume/body surface area/mL/m² ≥ 65 , CL $\geq 12^\circ$, α^2 angle $\geq 45^\circ$, IPMD > 20 mm), the correction of all measures is needed to normalize function.³⁻⁶ The clinical benefit of concomitant MV repair in patients with ischemic etiology is through the optimal recovery of all ischemic regions to ensure favorable LV reverse remodeling over time. Reverse remodeling is improved by reducing myocardial scar tissue burden, explaining the rationale for complete revascularization of viable myocardium.^{4,5} Long-term survival outcomes and recurrence of mitral regurgitation (MR) are closely related.^{4,5}

Persistent ischemic MR (IMR), especially with reduced LV ejection fraction, commonly results in LV dilatation, an independent risk factor for mortality. The exact biological and molecular events leading to adverse ventricular remodeling remain unclear. Adverse ventricular remodeling alters the geometrical configuration of left-sided chambers, resulting in MV distortion at the annular and subannular levels, promoting moderate-to-severe MR. As noted by Michler,⁶ the physiologic consequences of a severely dilated LV with depressed systolic function are severe

functional MR and heart failure, which cannot be solved by combined reparative surgery.³⁻⁶

In the CTSN trial, 26.2% of patients who underwent restrictive mitral annuloplasty (RMA) did not have concomitant coronary artery bypass grafting,⁷ which offers a biological mechanism to explain the observed advantage in clinical outcomes of subvalvular repair in addition to RMA, matching those who had revascularization.^{1,4} In our randomized subvalvular repair trial, all patients had myocardial revascularization.³ Although revascularization is critical, we believe that criticisms of subvalvular repair procedures may derive from the presence of 22.5% of patients who have mild to moderate MR at a 5-year follow-up.³ This can be partially explained by large areas of scar tissue formation, dyskinesia, or basal aneurysms. Current prospective trials have not sufficiently highlighted improvements in postoperative tethering among patients with LV lateral wall dysfunction, persistent LV dyskinesia, severely altered LV sphericity, and predominant apical displacement of both leaflets due to symmetric tethering.

Numerous studies have focused on the degree of annular restriction by mitral annuloplasty given the reported risk of stenosis, systolic anterior motion, and recurrent MR in over-correction or excessive undersizing.¹⁻⁹ The anterior leaflet enlargement patch as performed by the authors is progressive¹⁰ (Figure 1), provided that there is a marked reduction of tethering forces.^{11,12} The forces on the fibrous skeleton of the heart exerted by annular undersizing would counteract the displacement vectors relative to the LV dilation expected in these patients. The consequent imbalance of internal forces within the ventricle pushes the system toward a more unstable mechanical equilibrium, eventually leading to MR recurrence.¹¹

FIGURE 1. A, After detachment of the anterior mitral leaflet, the length of the incision is measured. The pericardium is sutured to the valve, and prosthetic ring annuloplasty is performed according to downsizing. B, Recipient of anterior leaflet enlargement patch.

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article published in the JTCVS will be considered if they are received within 6 weeks of the time the article was published. Authors of the article being commented on will be given an opportunity to offer a timely response (2 weeks) to the letter. Authors of letters will be notified that the letter has been received. Unpublished letters cannot be returned.

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The use of subvalvular repair is associated with a significantly lower risk of exacerbating leaflet tethering caused by the anterior displacement of the posterior leaflet because of IPMD correction. This was more evident in patients with a lower LV end diastolic diameter (<60 mm) asymmetric tethering and a well-circumscribed infarct of the inferior myocardium. The papillary muscle approximation was not a significant effect modifier in 17% of patients with anterior myocardial infarctions and symmetrical patterns with predominantly severe apical tethering.⁵ Because the attrition rate of combined valvular and subvalvular surgery on asymmetric tethering, but not on symmetric tethering, increases with time, whether the clinical benefits seen at 5 years could increase with a longer follow-up period remains unknown.

Our recent meta-analysis highlighted the evidence of conflicting results between mitral repair surgery and the MitraClip procedure that adds a veil of mystery regarding the future of IMR treatment. The seemingly neglected importance of the subvalvular apparatus in this context may partially account for the poor results.

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REPLY: CROSSING THE RUBICON— VENTRICULAR DIMENSION CONTROLS THE ULTIMATE FATE OF ISCHEMIC MITRAL REGURGITATION PROCEDURES

Reply to the Editor:

In 49 BC, Julius Caesar defied the Roman Senate's ultimatum. Rather than disbanding his army, he marched them south across the Rubicon in northern Italy, initiating the civil war that ended the republic. Likewise, there is a ventricular dimension, which when crossed, determines the fate of ischemic mitral regurgitation (MR) repairs. In their letter to the Editor, Nappi and Singh¹ address the complexity of this cardiac pathology.

There is a ventricular dimension after myocardial infarction (as yet not absolutely defined) in which the left ventricle (LV) geometry results in wall tensions where positive reverse remodeling after mitral repair is not possible. Rather, the unameliorated high wall strain in remote myocardium results in further injury to the LV. From the initial reports of Braun and colleagues² through outcomes of the Cardiothoracic Surgery Network severe ischemic MR trial,³ large ventricular volumes after infarct have been associated with recurrent MR and poor outcomes. LV reshaping strategies^{4,5} have shown promise by decreasing remote myocardial wall tension and perhaps inhibiting remote apoptosis.⁶ The fault is not in our stars, but in the dilated ventricle.

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