with MV disease based on basic physiological aspects of the left ventricle secondary to the diastolic overload. We do not have good data regarding the impact of failing MV repair and MV dysfunction on the right and left atria and on the role of the severity of pulmonary vascular disease. We also do not know whether the major publications discussed truly represent surgical outcomes, as centers with excellent reputations in MV repair do get referrals earlier and probably see less of the significant impact of long-standing MR in their patient populations.

We want to bring attention to another potential modifier on late outcomes. David and colleagues¹ demonstrated that late recurrence of MR and early TR were associated with late moderate TR. Therefore, it is important to look at the quality of the MV repair, both early and late, in addition to the TV repair, to understand the potential impact on long-term outcome.

In summary, the contribution of McCarthy and colleagues³ to the field is significant, as they have highlighted the importance of AF, even when treated effectively and consistently, as a risk factor for recurrent TR.

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See Article page 2030.

Commentary: Gate of success and corridor of failure

Tomasz A. Timek, MD, PhD

Functional tricuspid valve regurgitation is a poorly understood entity most frequently associated with left-sided valvular pathology. Although historically little heed was paid to the “forgotten” valve, current surgical guidelines chart a more aggressive course in addressing functional tricuspid insufficiency.¹ However, there is ample controversy in the literature whether less than moderate tricuspid

Tomasz A. Timek, MD, PhD

CENTRAL MESSAGE

Tricuspid annular dilatation of 45 mm or more and preoperative atrial fibrillation may predispose to progression of tricuspid insufficiency in patients with degenerative mitral regurgitation.

regurgitation (TR) should be treated at the time of left-sided valvular surgery.^{2,3} In the current issue of the *Journal*, McCarthy and colleagues⁴ from Northwestern University

From the Division of Cardiothoracic Surgery, Spectrum Health, Michigan State University College of Human Medicine, Grand Rapids, Mich.

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Address for reprints: Tomasz A. Timek, MD, PhD, Division of Cardiothoracic Surgery, Spectrum Health, Michigan State University College of Human Medicine, 100 Michigan Ave NE, Grand Rapids, MI 49503 (E-mail: tomasz.timek@spectrumhealth.org).

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present a series of more than 800 patients who underwent mitral valve repair for degenerative disease with less than moderate TR at the time of surgery. Although 97% underwent a surgical maze procedure, patients with preoperative atrial fibrillation were at higher risk for developing significant late TR, as were those with annular diameter of 45 mm or more on preoperative echocardiography. Regardless of the present preoperative risk factor, progression to distant functional tricuspid valve regurgitation was associated with increased long-term mortality.

The current report represents one of the largest series in the literature addressing the vexing problem of TR progression after mitral repair, and these results should be carefully considered. Atriogenic functional mitral regurgitation and TR^{5,6} have been described in the literature, and the physiology of normal atrial valve closure requires atrioventricular synchrony to achieve presystolic annular reduction.⁷ Current data corroborate these prior reports because patients with preoperative atrial fibrillation had a higher rate of annular diameter 45 mm or greater than those who did not (20% vs 3%). In the absence of atrial fibrillation, the authors discovered that annular diameter of greater than 45 mm was associated with progressive TR rather than an annular size of 40 mm recommended by current guidelines. As such, it is not surprising that TR progression was more pronounced in patients with preoperative atrial fibrillation, yet a majority of these patients remained in normal sinus rhythm in the long term because of concomitant surgical ablation. Unfortunately, the authors do not provide data on whether the ablated patients who progressed to TR developed recurrent atrial fibrillation because it would be helpful to elucidate if progression of TR is related to electrical synchrony or whether right-sided ultrastructural changes begotten by severe mitral regurgitation and atrial fibrillation are irreversible regardless of durable mitral repair and ablative therapy. Because annular diameter on follow-up echocardiography was not reported, the link

between progressive annular dilatation and distant TR, although plausible, also cannot be firmly established. Last, 43% of patients (22/51) with an annular size of 45 mm or greater underwent tricuspid annuloplasty at the time of mitral repair, but the outcomes of those patients is not reported or compared with those who were not treated for the same degree of annular dilation.

The study by McCarthy and colleagues⁴ provides further clarity in addressing less than moderate TR at the time of degenerative mitral valve repair. The data suggest prophylactic annular reduction at a diameter of 45 mm or more or in the presence of preoperative atrial fibrillation. However, these recommendations assume that the etiology of functional tricuspid valve regurgitation is predominantly related to annular pathology, but the exact mechanisms of TR progression remain to be defined.

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