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See Article page e353.

Commentary: Primum non nocere

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The surgical correction of functional tricuspid regurgitation (FTR) by reduction annuloplasty is an increasingly utilized procedure in the armamentarium of the cardiac surgeon. Malinowski and colleagues¹ report an ovine model in which chronic epicardial pacing induced biventricular tachycardia-mediated cardiomyopathy and moderate FTR. Ten sheep underwent placement of sonomicrometry crystals on the right ventricle epicardium, tricuspid annulus, leaflet body, and edges followed by a modified DeVega annuloplasty that was exteriorized to enable progressive cinching to produce tricuspid annular reduction (TAR). A total of 5 reductions (TAR 1-5) were carried out while evaluating biventricular function, tricuspid regurgitation (TR), tricuspid gradient, and various objective parameters by echocardiography. The main conclusion was that TAR of 55% perturbed anterior leaflet and posterior leaflet dynamics while maintaining normal septal leaflet motion. More profound TAR triggered severe changes in anterior leaflet and posterior leaflet excursion, closing angle, and strain, which suggested that aggressive undersizing impaired leaflet kinematics.

The ovine model is desirable over other nonprimate models due to its similarity to the human heart.² The current study¹ is elegant, and the authors are to be congratulated; however, there are limitations that should caution the direct application of its conclusions to clinical practice. The method of annuloplasty used was a modified DeVega suture annuloplasty.^{3,4} Remodeling annuloplasty applied for FTR

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CENTRAL MESSAGE

Overzealous tricuspid annular reduction severely impairs leaflet mobility and could be inimical to a satisfactory long-term result.

in the contemporary clinical setting utilizes rigid rings that often mimic the nonplanar geometry of the normal tricuspid annulus.⁵ It is quite conceivable or likely that leaflet mechanics by echo and sonomicrometry would be different with a rigid prosthesis to those produced by a simple, 2-dimensional, sutured annuloplasty. In addition, the influence of TAR on FTR was studied under acute conditions during the terminal procedure and did not allow animal recovery that might have produced different outcomes. Finally, sonomicrometry crystals by themselves may have influence on leaflet kinematics due to their weight and bending stiffness of associated wires.⁶

Nevertheless, the authors appear to effectively show that there may be a cost to leaflet excursion impairment from an undersized annuloplasty. Although progressive TAR abated TR, leaflet kinematics were impaired commensurate with degree of reduction. The average gradient for TAR-5 was only 3.6 mm Hg, a level that would not be expected to produce clinically relevant tricuspid stenosis. As the authors noted, leaflet tethering associated with restrictive mitral annuloplasty has been shown to induce biological changes in

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leaflets, including increase of collagen synthesis and elevation of transforming growth factor beta. These in turn promote fibrosis, thickening, and calcification that could produce suboptimal long-term clinical results. Certainly, immobile calcified posterior mitral leaflets associated with rings are commonly found.^{7,8} This study stimulates conjecture that overzealous reduction annuloplasty may adversely influence leaflet mechanics and durability. Finally, the article provides evidence there is an optimal TAR that reduces TR while minimizing the adverse effects on leaflet kinematics. For effective repair of FTR, we must first do no harm.

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