Commentary: Repair for ischemic mitral valve regurgitation: Does the type of ring matter?

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Secondary mitral valve regurgitation subsequent to myocardial ischemia, acute or chronic, remains a dilemma to the cardiac surgeon. For most types of mitral valve pathology, valve repair has emerged as the first choice and the results are continuously improving, but repair of the ischemic mitral valve has consistently produced less than good results. Thus, valve replacement is increasingly being suggested in these circumstances, especially in patients with echocardiographic risk factors for residual or recurrent mitral regurgitation.¹

Remodeling of the left ventricle after acute myocardial infarction is recognized as the direct cause of mitral valve regurgitation. With the dilatation of the ventricle, the papillary muscles are displaced apically and posteriorly, and the chordae pull the free edge of the leaflets downward and laterally. In the most frequent inferior infarction, this leaflet tethering is asymmetric, involving the medial half of both leaflets, and on echocardiography, the regurgitation jet is observed in the area of the posteromedial commissure. Finally, the annulus dilates secondarily to both the dilatation of the ventricle and the mitral regurgitation, which enters a vicious cycle.

Consequently, regurgitation is a disease of the ventricle and not of the valve, which no procedure on the valve alone (annulus, leaflets, or chordae tendineae) can correct. Because misalignment of the papillary muscles appears to be the main problem, several techniques directed at papillary muscle realignment have been suggested, yet with limited acceptance by the surgical fraternity, probably because of difficult reproducibility.

On the other hand, extreme reduction of annular diameter with markedly undersized rings has been advocated and is the most used procedure aimed at compensating the effects of annular dilatation and chordal tethering. But the long-term results have been unsatisfactory, even in the cases with an apparently good initial leaflet apposition and competence of the valve. Subsequently, different modifications of the original annuloplasty ring designs were made to

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Central Message

Ischemic mitral regurgitation remains a major surgical challenge. All efforts must be pursued to clarify the mechanisms of valve and ventricular dysfunction that might be corrected surgically.

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specifically tackle this problem and have been widely used, mostly with contradictory results.

It is in this context that in an animal study described in this issue of the *Journal*, Dr Bothe and investigators² of Dr Craig Miller's group in Stanford set out to "quantify the effects of annuloplasty rings designed to treat ischemic/functional mitral regurgitation, caused by acute left circumflex artery occlusion, on left ventricular septal-lateral and commissure-commissure dimensions." To this aim, they used radiopaque markers in both the mitral annulus and the left ventricle in 30 sheep. True-sized Carpentier-Edwards (Irvine, Calif) Physio 1 (conventional), Edwards IMR ETlogix, and GeoForm (Edwards) (etiology-specific) annuloplasty rings were implanted, and the changes in the diameters of the mitral annulus and morphology of the left ventricle were measured in different settings with and without the ring, so that each animal was used as its own comparison.

Besides the clear modifications of the annular size caused by the rings, Bothe and colleagues² observed virtually no consequences in the geometry of the left ventricle. Thus, they concluded that "disease-specific IMR/FMR annuloplasty rings do not induce relevant changes of left-ventricular dimensions in the acutely ischemic ovine heart." That is, it appears that the specifically designed annuloplasty rings do not have a significant impact in

reversing the ventricular remodeling, and some observed improvements are either due to reversion of the ischemia (after concomitant coronary artery bypass surgery) or to valvular competence. The unresolved question is why the simple placement of a ring, any ring, should significantly alter the geometry of the left ventricle with ongoing ischemia, apart from the expected small reduction on the basal ventricular level in the septal-lateral dimension seen with the GeoForm ring. Otherwise, in my opinion, acute restoration of valve competence is unlikely to make the difference.

Other previously published works have questioned the benefits of these etiology-specific annuloplasty rings,³ but this interesting study by Bothe and colleagues² may shed more light on understanding this phenomenon. However, it remains to be proven that the findings in the acute ischemic phase in the animals can be translated into the clinical, usually more chronic situation, also because the geometry of the left ventricle and the ischemic mitral regurgitation are part of a dynamic process dependent on many variables. On the other hand, “true-sized” rings were used in this experiment, whereas markedly undersized rings usually are used in patients under these circumstances. Would these have made any difference? The authors discuss these and other aspects at length and recognize that their study does have some limitations.

On the other hand, some surgeons still use flexible rings and even suture annuloplasty, which would have the advantage of preserving the physiologic motion of the annulus

during the cardiac cycle. Adding rigidity to the base of the ventricle, in these circumstances, may contribute to worsen the dysfunction. Unfortunately, Bothe and colleagues² did not consider this alternative. In any case, it remains obvious that approaches focused exclusively on the mitral annuloplasty are largely inefficient and should be complemented by procedures directed at ventricular reshaping and alignment of the papillary muscles and chordae.⁴

Surgical treatment of secondary mitral regurgitation of ischemic origin remains a major challenge. Mitral valve replacement may be a better alternative in these cases, especially in the presence of severe and complex regurgitation. Still, all efforts must be pursued to completely clarify the mechanisms of the valve and ventricular dysfunction that might be corrected surgically. In this respect, Bothe and colleagues² are to be congratulated for their continuing endeavors in the study of this subject.

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