Does the intertrigonal distance dilate? Never say never

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In an era of dynamic 3-dimensional (3-D) reconstruction using advanced magnetic resonance, echocardiographic, or computed tomographic imaging, a fundamental assumption of mitral valve repair has been challenged by a study based on an old method, anatomic observation and measurement of pathologic specimens. Hueb and colleagues¹ from São Paulo have updated basic observational techniques by using computer analysis of digital photographs. The authors compared mitral valve and ventricular size in fixed cadaver hearts from normal patients (trauma death) with hearts from patients with advanced heart failure resulting from ischemic or idiopathic (non-Chagas) dilated cardiomyopathy.

Some of their findings were not surprising and confirmed prior studies and surgical observations. The left ventricle dilated to a globular shape and the mitral annulus dilated.²⁻⁴ However, there was no proportionality of left ventricular to mitral valve dilatation. Therefore, left ventricular remodeling and dilatation itself (increased sphericity) can lead to mitral regurgitation, not necessarily through a proportional amount of mitral valve annular dilatation.⁵ The majority of the mitral annular dilatation occurred in the muscular portion (primarily along the posterior leaflet) with a mean increase of 78 mm (ischemic) to 164 mm (idiopathic). This also was expected and accounts for the success of mitral valve repair techniques for functional mitral regurgitation that reduce the posterior annulus.

The unexpected finding was an increase in the fibrous portion of 72 mm (ischemic) and 84 mm (idiopathic). This corresponds to the intertrigone distance (ITD), commonly used as an aid in sizing mitral repair rings. Furthermore, the ITD change was proportional to the changes in the muscular portions, so that if the annulus became severely dilated, the ITD also changed a large amount. Is it possible that the fibrous skeleton of the heart can stretch?

We have been taught that the ITD is part of the fibrous skeleton of the heart and therefore is fixed and does not dilate.⁶⁻⁸ The data to support this conclusion are primarily based on 2-D echocardiographic measurements and surgical observation. However, 2-D reconstruction of the 3-D shape of the anterior annulus may not allow accurate measurement. Furthermore, we know the ITD expands and contracts in systole and diastole.⁹⁻¹¹ Scar and ligaments can stretch with enough repetitive pressure; maybe the fibrous portion of the heart can too.

The current study has limitations. Are measurements from the excised fixed pathologic specimens accurate representations of in vivo function of the ITD in normal and cardiomyopathic hearts? Kunzelman and colleagues¹² documented an increase in mitral annulus dimensions in the excised heart. Therefore, could the measurements in this study partly be explained by the use of pathologic specimens? Can the investigators accurately pinpoint the trigone from digital photographs? Sometimes a surgeon can "feel" the trigone when placing a suture better than one can "see" the trigone. If the fibrous skeleton dilated by 72 to 84 mm, as reported, would there not be associated changes in the adjacent aortic annulus? This is certainly not a hallmark of dilated cardiomyopathy. Since this is a pathologic study, there is no information on premortem mitral regurgitation.

When new information is presented, one has to question why it is at odds with the prevailing opinion and previous studies. The answer could be as simple as that the human ITD has not been investigated in a variety of normal and pathologic conditions. Conventional wisdom may have been substituted for hard facts and data. This certainly has been done before with the mitral valve, because it was incorrectly

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assumed that patients with severe left ventricular dysfunction and mitral regurgitation would not benefit from mitral valve repair or replacement because they had lost the “pop-off” mechanism that allowed the left ventricle to decompress into the left atrium.¹³

Assuming this study is accurate, the implications of increased ITD in cardiomyopathy relate to two aspects of mitral valve repair, sizing and the choice of a complete remodeling ring or a partial ring that modifies just the muscular portion of the anulus between the two trigones. One should not choose a ring size on the basis of the pathologic ITD. For functional mitral regurgitation, most of us already choose a ring that is at least two sizes smaller than what is measured, or the smallest ring available. This radical undersizing is needed to increase the zone of coaptation of the mitral leaflets. After all, the leaflets and subvalvular apparatus are (usually) normal, so we are trying to overcorrect the mitral anulus to make up for the ventricular dilatation that displaced the papillary muscles and restricted the leaflet motion.

A complete remodeling ring theoretically has advantages over a partial ring in functional mitral regurgitation.¹⁴ The entire anulus is returned to a more normal shape (important if the ITD is dilated), and the septal-lateral (or anterior-posterior) dimension is closed more completely, increasing the zone of coaptation.¹⁵ A recent study showed less late mitral regurgitation in cases of ischemic regurgitation repaired with a complete semirigid ring than with a partial flexible band.* The reason that late mitral regurgitation was lower in patients with the complete ring may have less to do with correction of the dilated ITD than with other factors such as use of a rigid ring and more effective closure of the septal-lateral dimension. This will be an area of careful study in the near future.

Although the present study casts doubt on the tenet of the fixed ITD, with important implications for valve repair and ring design, it is not yet a settled issue. Further confirmation of these observations would be welcome. The ideal study would serially monitor patients with 3-D left ventricular and annular imaging from early after myocardial infarction through the pathologic development of left ventricular remodeling with subsequent functional mitral regurgitation.

*Aklog L, Soltesz EG, Filsoufi F, Sepic J, Cohn LH, Nascimben L, et al. The impact of residual mitral regurgitation on survival after CABG and annuloplasty for ischemic mitral regurgitation. Unpublished data.

This study should confirm in vivo the findings of this article. In the meantime, surgeons should never assume that the ITD is fixed and will not dilate.

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