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Commentary: Left ventricular outflow tract obstruction by mitral bioprostheses. Still a problem?

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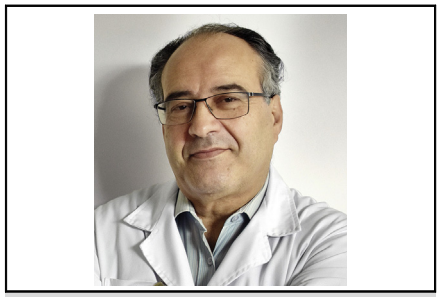

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Although repair is now the most preferred option for the treatment of mitral valve disease, especially regurgitation and in degenerative disease, mitral valve replacement (MVR) is often required. In this case, the choice of prosthesis is of paramount importance. Mechanical valves are low-profile and usually cause no problem with regard to flow in the left ventricular outflow tract (LVOT). By contrast, bioprostheses are significantly greater profile, and LVOT obstruction is frequent. The problem is more frequent when bioprosthetic implantation is associated with preservation of the native mitral valve apparatus, which is believed to be associated with better left ventricular (LV) function.

As replacement is most often performed for rheumatic disease and endocarditis, typically in patients with normal LV ejection fraction, preservation of the posterior leaflet only appears to be sufficient to maintain near-normal LV function. In contrast, in ischemic mitral regurgitation, preservation of the whole apparatus appears to be of greater importance. In these cases, because the most important appears to be preservation of the chordae tendineae, splitting the anterior mitral leaflet (AML) in the middle, with or without resection of part of the body of the leaflet, has been suggested to move it away from the LVOT and avoid the risk of obstruction.

In a paper published in this issue of the *Journal*, Brunel and colleagues[1] from Sidney, Australia, deal with this problem. In an animal study, the authors analyze methods of safely retaining the anterior leaflet at the time of MVR.

CENTRAL MESSAGE

LVOT obstruction during bioprosthetic implantation with preservation of the native mitral valve apparatus, which is believed to be associated with better left ventricular function, is frequent.

They studied 15 insertions of the Medtronic Mosaic porcine bioprosthesis, which has asymmetric interstrut sectors, to identify the best position to avoid left ventricular outflow tract obstruction (LVOTO), which was defined by a peak instantaneous pressure gradient >30 mm Hg. Thirteen of the 15 valve insertions resulted in LVOT, independently of the valve orientation. Transapical echocardiography confirmed systolic anterior motion of the AML as the cause of LVOTO in all episodes, but a wider interstrut distance subtending the anterior leaflet was associated to lesser obstruction that was, here, late systolic rather than holosystolic.

In their investigation, the authors departed from the concept that "resection of the AML chordae impairs the mitral valvular ventricular interaction resulting in reduced LV contractility" and that "it seems possible that the perioperative mortality rate from valve replacement could be reduced by retention of the AML in patients with substantially impaired LV function," especially in ischemic mitral regurgitation. This is still subject of some controversy, at least for some groups of patients.[2] In addition, this group of authors has recently published in this *Journal* a paper suggesting that incorporating the anterior mitral leaflet to the annulus, preserving the chordae tendineae, impairs LV function in an ovine model.[3]

In any case, as indicated, modification of the AML to prevent LVOTO can be achieved with minimal resection of chordae and no other native valve modification, and in transcatheter methods, splitting of the AML is often used.

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In contrast, the prosthesis studied is, to my knowledge, the only one with this asymmetry, and the use of porcine bioprostheses is in fast decline, anyway. Also, in their limitations section, the authors state that "in this study, a bioprosthetic mitral prosthesis was used which limits the extrapolation of our results to transcatheter mitral valve replacement as the design of the transcatheter valves is different."

As one of the reviewers argued during the editorial process, the Mosaic valve is a third-generation bioprosthesis with lower strut profile. In these cases, LVOTO depends on the size of the LV cavity, in terms of hypertrophy or dilatation (ischemic regurgitation is different form rheumatic or endocarditis), strut position and orientation, and mitroaortic angulation. However, none of this information was included in the manuscript. Another problem is the eventual anatomical differences between human and sheep hearts; annular size was >33 mm in all animals, which is much different from humans, and all sheep received 31-mm Mosaic prostheses.

The authors suggest that "modification or redesigning of the bioprosthesis should involve a wide inter-strut distance subtending the AML (a large anterior bioprosthetic leaflet)." However, as already mentioned in this commentary, this is the problem of only this particular prosthesis as, to my knowledge, all other bioprostheses, especially the currently preferred pericardial valves, are symmetrical and have similar interpost distances. Therefore, the clinical interest of this study is limited from the surgical point of view, and I do not understand the conclusion that "this model represents an effective method for research into prevention of left ventricular outflow tract obstruction following mitral valve replacement with preservation of the native valve."

However, as the authors stated, "the model may have several implications for transcatheter MVR," where the risk of LVOTO is apparently greater than in surgical MVR, but this was not the subject of this study and has to be confirmed by further experience and analysis. In contrast, in these procedures, the authors differentiate the LVOTO caused by systolic anterior motion from that obstruction due to the prosthesis impinging in the outflow tract. In the former case, splitting the anterior mitral leaflet would solve the problem, whereas we can't define a solution for the latter. But these are "beads from another rosary," which are not to be discussed here and now.

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