Commentary: A thorough understanding of the mitral apparatus will improve the results of mitral valve repair: Part 2

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Central Message

A wider comprehension of the whole mitral valve apparatus will drive to a more durable and efficacious treatment of mitral regurgitation.

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Plenty of studies have aimed to clarify the geometric changes of the left ventricle (LV) after the process of remodeling, causing secondary mitral regurgitation (MR).[1-5] Results are controversial. Some previous experiences found that apical displacement of the papillary muscles is the main reason of the apical tethering of the mitral leaflets; conversely, others showed no displacement of papillary muscles, especially for the posterior papillary muscle (PPM). More recently, it has been shown this muscle has a posterolateral displacement and moves toward the mitral annulus.[1,2,6]

In this issue of the *Journal*, Bothe and colleagues[7] add evidence that no apical papillary muscle displacement is observed and that the displacement vector of the PPM is directed posterolaterally. From their study of an in vivo ovine 3-dimensional vector model, Bothe and colleagues[7] conclude that this posterolateral displacement is the predominant mechanism leading to apical leaflet tethering during chronic secondary MR. A similar mechanism was reported by Timek and colleagues in an acute ovine model.

MR was the expression of a lateral and posterior displacement of the PPM away from the midseptal annulus with its heads, which moved toward the mitral annulus.[1]

Secondary MR is a disease of the ventricle and papillary muscles that leads to valve dysfunction. In this regard, Bothe and colleagues[7] have focused mainly on PPM posterolateral displacement, which represents an important modification in LV geometry, leading to leaflet tethering. It would also have been interesting, however, to analyze the dynamic changes in the interpapillary muscle distance.[4] During systolic closure of mitral valve, papillary muscles approximate, leading to a shortening of interpapillary muscle distance, with consequent slackness in the marginal and secondary chordae. This movement allows basal leaflet motion toward coaptation. In the setting of chronic IMR, Kalra and colleagues[4] demonstrated that the loss of systolic shortening of interpapillary muscle distance can alter mitral valve force balance, resulting in asymmetric MR.

In addition, no information has been reported regarding the impact of LV remodeling, a well-known predictor of recurrence of MR after mitral valve annuloplasty.[6]

Jensen and colleagues[6] reported an increased distance between the anterior papillary muscle and the PPM and their asymmetric displacement after LV remodeling. Moreover, they described an apical displacement of anterior papillary muscle at end-systole, which could be explained by global LV remodeling after regional myocardial infarction.[6]

The most common treatment of secondary MR is mitral valve annuloplasty. Solely reductive mitral annuloplasty is destined to failure over time,[8] however, because it has been associated with an increased rate of recurrent MR at 2-year follow-up.[9] Nevertheless, a standard technique for the correction of the subvalvular apparatus to prevent recurrence of MR has not yet been developed.

Bothe and colleagues[7] advise relocating PPMs in the presence of posterolateral displacement. Other techniques have been proposed[10]; however, few data are available on long-term efficacy, and no information has been reported how LV remodeling may affect the repair durability.[11,12]

The mitral valve is a complex structure that requires precisely timed and harmonic coordination of the LV, papillary muscles, chordae tendineae, leaflets, mitral annulus, and left atrium to achieve a competent valve closure.[13] We

know that failure of any one of these anatomic components causes MR, but we are still far from a real understanding, because different studies have drawn different conclusions. As reported in a previous editorial commentary,[13] we maintain that a thorough understanding of the mitral apparatus will improve the results of mitral valve repair.

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