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Commentary: Picking up the slack—The case for conservative management of postrepair systolic anterior motion

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Small ventricular volume causing excessive leaflet slack and SAM.

CENTRAL MESSAGE

SAM postrepair results from an anterior shift of leaflet coaptation. Surgical intervention is warranted in $\leq 1\%$ of the total population, and outcome with conservative management is favorable.

Primary abnormalities of the mitral valve apparatus constitute the root cause of systolic anterior motion (SAM) of the mitral valve, resulting in outflow tract obstruction and mitral regurgitation.¹⁻³ In hypertrophic obstructive cardiomyopathy, the straightforward connection between altered geometry of the mitral valve and its dysfunction was long obscured by an overwhelming focus on asymmetric hypertrophy, particularly because septal myectomy abolishes SAM so efficiently. However, already early on, it was recognized that the surgical procedure corrects, to no small extent, the abnormal anterior displacement of the mitral valve within the ventricular cavity by relative posterior realignment with respect to the streamlines of flow.⁴ The subsequent observation of SAM after mitral valve repair in the absence of any left ventricular hypertrophy was a turning point in our mechanistic understanding of SAM.^{5,6} Once its root cause was recognized, surgical strategies could be developed to prevent this complication.⁷

In a large, contemporary cohort of patients, Ashikhmina and colleagues⁸ identified 3 geometric predictors favoring postrepair SAM: excessive posterior leaflet height (shifting the coaptation point anteriorly), small left ventricular systolic volume, and bileaflet prolapse. The first predictor is well recognized⁹ and has been addressed by resective measures; others favor neochordal restraint.¹⁰⁻¹³ The second most probably acts by moving the papillary muscles closer together and closer to the outflow tract,

producing excessive leaflet slack, while exposing the insufficiently tethered leaflets to the outflow stream (Figure 1). The third predictor may be related to the first: an unbalanced bileaflet prolapse with posterior dominance may cause an anterior shift of the valve. Not surprisingly, therefore, the surgical remedy in all cases included posterior leaflet intervention. In our series, with a more liberal use of artificial chordae (60%), bileaflet prolapse was not a predictor of SAM.¹⁴ In fact, use of artificial chordae appeared to have a protective effect, indicating it may facilitate leaflet balance more predictably than by relying exclusively on resective techniques.¹⁴ Of note, the authors did not find an association between basal septum thickness and SAM. Concentric left ventricular hypertrophy may act only indirectly, by reducing systolic cavity size. Inappropriate small cavity size may be treated effectively by volume loading, beta-blockers, and/or vasoconstrictors.^{8,14}

The initially observed SAM incidence of 13%, about 60% greater than previously reported,^{14,15} included chordal, nonobstructive (and nonregurgitant) SAM, which, unlike true leaflet SAM, does not represent a clinical problem.

As in our series, the vast majority of patients were managed conservatively with favorable outcomes. Only about 3.6% of the total study population showed persistent SAM at discharge, and only 1% was discharged with SAM and moderate mitral regurgitation (using stress echocardiography, we could even demonstrate lack of a provokable

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FIGURE 1. Mitral tethering in a normal (left) and small left ventricular cavity (right). *Top:* long-axis view; *bottom:* short-axis view. *Left,* with normal mitral tethering the streamlines of flow keep the valve in a posterior position. *Right,* a decrease in ventricular volume moves the papillary muscles closer together and closer to the outflow tract (OT) (double arrows), producing excessive leaflet slack while exposing the insufficiently tethered leaflets to the outflow stream. The valve is pushed toward the septum. LV, Left ventricle; Ao, aorta; LA, left atrium.

outflow tract obstruction at follow-up in patients with an initial tendency to SAM¹⁴).

We suspect these favorable numbers do not imply that there is rarely an a priori potential but rather that awareness of the problem by experienced surgical teams has led to its effective prevention. If, nonetheless, SAM occurs, it is in many cases a functional rather than structural problem, with volume loading and beta-blockers capable of picking up the slack.

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