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Commentary: Is length (of the anterior mitral leaflet) important?

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Our understanding of left ventricular outflow tract obstruction (LVOTO) has evolved significantly over the past 25 years. It was long thought that hypertrophy was absolutely necessary to generate obstruction and that obstruction was uncommon. These myths have been dispelled. Although only 25% of patients have resting gradients, more than 70% of patients will develop obstruction with exercise.¹ We found, using detailed magnetic resonance imaging analysis of left ventricle and outflow tract angles, septal thickness, and mitral valve abnormalities, that the greater the acuteness of the angle between the aorta and the apex, the greater the gradient independent of basal septal thickness.² So, if hypertrophy is less important than the outflow angle, what is the mechanism of LVOTO? It was thought that Venturi forces related to high-flow velocity pulled the anterior mitral valve leaflet toward the septum. Careful echocardiographic analysis of flow vortices found that drag forces push the posterior leaflet and coaptation point into the LVOT before the generation of outflow tract flow acceleration^{3,4} (Figure 1). It was always perplexing to me how anterior mitral leaflet extension (with a very limited myectomy) eliminated obstruction.⁵ The patch likely produces a posterior force that resists this drag. Any surgical intervention must neutralize drag forces on the mitral leaflets.

Carvalho and the Mayo Clinic team⁶ attempt to address whether leaflet length is material to managing LVOTO. They concluded that mitral leaflets were longer in patients undergoing myectomy compared with those undergoing coronary artery bypass grafting or aortic valve replacement,

Obstruction: flow vectors, septal thickness, aortic angle, and valve/subvalvular anatomy.

CENTRAL MESSAGE

Flow dynamics and drag forces produce left ventricular outflow tract obstruction. All components of the left ventricular outflow tract must be evaluated to determine the cause(s) of obstruction.

FIGURE 1. Obstruction: flow vectors, septal thickness, aortic angle, and valve/subvalvular anatomy

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Disclosures: The author reported no conflicts of interest.

The *Journal* policy requires editors and reviewers to disclose conflicts of interest and to decline handling or reviewing manuscripts for which they may have a conflict of interest. The editors and reviewers of this article have no conflicts of interest.

Received for publication Jan 25, 2021; revisions received Jan 25, 2021; accepted for publication Jan 26, 2021; available ahead of print Feb 3, 2021.

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J Thorac Cardiovasc Surg 2023;165:90-1

0022-5223/\$36.00

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<https://doi.org/10.1016/j.jtcvs.2021.01.110>

that longer length was related to systolic anterior motion (SAM) in the univariate analysis but not in the multivariable model. What we cannot discern from the manuscript is whether the assessment of postoperative SAM is at rest or with provocation. (We routinely administer high-dose Isuprel [$>10 \mu\text{g}/\text{min}$] in the operating room and amyl nitrate before discharge to provoke obstruction.) If it is only at rest, then it is noteworthy and concerning that 66% of patients with anterior leaflet length greater than 30 mm had postoperative SAM and 11% of this cohort and 14% of the nonelongated cohort had moderate or greater residual mitral regurgitation.

Extending the myectomy toward the apex as advised by the authors is critically important to eliminate the drag force vectors under the posterior leaflet especially in patients with acute apex to LVOT angulation. This can eliminate obstruction in the vast majority of patients. There is no doubt with extensive experience, septa as thin as 12 to 13 mm can be safely reduced with an extended myectomy alone. The margin for an error, which can be catastrophic, is quite small.

It is very useful to consider hypertrophy as a frequent but not required component of LVOTO. This has allowed us to identify latent provokable obstruction in symptomatic

patients with completely normal-appearing resting echocardiograms. By understanding the fluid dynamics, flow vortices, and the anatomical substrate that predisposes to obstruction, surgeons are developing innovative techniques to tailor a safe operation to the variable anatomy of the outflow tract.

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