Commentary: Opening the left ventricular outflow tract in hypertrophic cardiomyopathy requires a nuanced approach

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Surgical transaortic extended septal myectomy is the gold standard operative technique for treatment of hypertrophic cardiomyopathy (HCM) in patients experiencing symptoms refractory to medical therapy and accompanied by a resting or provokable left ventricular outflow tract (LVOT) gradient (ie, >50 mm Hg).¹⁻³ Over the past 2 decades, septal myectomy has undergone important evolution in surgical technique and approach.¹⁻³

In this issue of the *Journal*, Hodges and colleagues⁴ report on the outcomes of 1559 operations for treatment of HCM with obstruction. Of these, 586 represented isolated septal myectomies, 522 involved myectomies performed with mitral valve (MV) or subvalvular apparatus interventions, 422 included myectomies with another concomitant procedure, and 29 involved isolated MV interventions without myectomy. Of those procedures involving MV interventions, the most common techniques included anterior leaflet shortening (16%), chordae tendineae resection (9.8%), papillary muscle resection (7.2%), papillary muscle reorientation (7.5%), and MV replacement (42 for LVOT obstruction and 50 for intrinsic MV pathology). Patients undergoing MV interventions had thinner septums (18 ± 0.4 mm vs 22 ± 0.5 mm) and had less myocardium removed (6.2 ± 3.5 g vs 8.8 ± 3.8 g) than patients without an MV intervention. Overall operative mortality was 0.38%, with only 2 (0.13%) postoperative ventricular septal defects. Permanent pacemaker was required in 4.2% ($n = 1334$) in those with pre-existing conduction defects and 1.1% ($n = 464$) for isolated septal myectomy with normal preoperative conduction.

This is an important clinical report written by a highly experienced group in the surgical treatment of HCM. The strengths of this study include its large patient cohort, excellent detail and clarity provided on the surgical technique and approach to the different pathophysiologies contributing to LVOT obstruction, uniform approach to the disease process by a single surgeon, and the outstanding surgical results that set benchmarks for other experienced or emerging

Mitral valve abnormalities contribute to obstruction in hypertrophic cardiomyopathy.

Central Message

Mitral valve abnormalities, as well as septal hypertrophy, contribute to obstruction in hypertrophic cardiomyopathy. The mitral valve becomes more relevant as the basal septal thickness diminishes.

See Article page 2289.

programs. The highlight of this experience is the recognition that MV abnormalities, as well as septal anatomy, contribute to the pathophysiology of the obstruction in an important number of patients and that the contribution of the MV abnormalities to the obstructive physiology becomes more relevant as the basal septal thickness diminishes. Importantly, this report emphasizes the need for surgeons to be sufficiently experienced to recognize the various MV pathologies that contribute to obstruction and use multiple techniques to resolve the obstruction in addition to those techniques that address the hypertrophied septum.

The major limitation of this report is the obvious lack of intermediate and long-term follow-up necessary to understand the efficacy and durability of the various surgical techniques that address the MV. Further, it is not known whether the criteria used to define a successful operation by this group, which include a provokable gradient with dobutamine less than 36 mm Hg and moderate or less MV regurgitation, represent durable metrics. Despite these important limitations, the report by Hodges and colleagues is informative and contributes significant information to the surgical approaches to treatment of HCM. Surgical septal myectomy has been performed for more than 50 years and has represented an enduring and effective treatment for HCM with obstruction. An improved understanding of the phenotypic

patterns of interventricular anomalies continues to refine the surgical approach and improve the outcomes of this procedure.

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