

Transaortic Septal Myectomy for Obstructive Hypertrophic Cardiomyopathy



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Transaortic extended septal myectomy is the best treatment option for patients with obstructive hypertrophic cardiomyopathy whose symptoms are refractory to medical management. In this article, we describe our operative technique of transaortic septal myectomy, which relieves subaortic left ventricular outflow tract obstruction and systolic anterior motion of the mitral valve associated with hypertrophic cardiomyopathy. In experienced centers, surgical outcomes are excellent (operative mortality <1%) and long-term symptomatic relief is achieved in the majority of patients.

Operative Techniques in Thoracic and Cardiovascular Surgery 22:200–215 © 2018 Elsevier Inc. All rights reserved.

KEYWORDS hypertrophic cardiomyopathy, transaortic septal myectomy, transapical myectomy

Introduction

Although transaortic septal myectomy is the preferred method for septal reduction for obstructive hypertrophic cardiomyopathy (HCM),¹ many centers and surgeons have little experience with the procedure due to reluctance of some clinicians to refer patients with HCM for operation. Indeed, concern regarding residual left ventricular outflow tract (LVOT) gradients and risk of iatrogenic ventricular septal defects has led many cardiologists to use alcohol septal ablation as first line therapy for obstructive HCM refractory to medical therapy.² At institutions that evaluate and treat large numbers of patients with HCM, transaortic myectomy can be performed with very low operative risk and reliable relief of outflow tract gradients.^{3,4}

Patient selection is based on symptomatic status and preoperative imaging. In general, septal reduction therapy is reserved for patients whose symptoms persist despite medical therapy or those who have serious side effects of medications.⁵ Transthoracic Doppler echocardiography should be performed in all patients to document LVOT gradients, presence or absence of mitral valve regurgitation, and septal and left ventricular (LV) morphology. Patients with minimal resting LVOT gradients (<30 mmHg) should undergo provocative maneuvers, including Valsalva, inhalation of amyl nitrite, exercise, or infusion of isoproterenol.⁶ High LVOT gradients are associated with reduced late survival, and this may be an important consideration when selecting patients for surgery.⁷

On echocardiography, basal septal thickness measures approximately 20–22 mm in most patients, but in some cases, massive hypertrophy (30–50 mm) can be seen. Increased thickness of the basal septum is an important criterion in diagnosing obstructive HCM, but a relatively thin septum (<18 mm) is not a contraindication to septal myectomy.⁴ Structural mitral valve disease, including abnormal papillary muscles, should also be identified on preoperative imaging, as these pathologies can be addressed at the time of surgery. In patients with intrinsic mitral valve disease, repair of the native valve at the time of septal myectomy is preferred over valve replacement because valvuloplasty is associated with better late survival.⁸ Systolic anterior motion and mitral valve regurgitation are usually abolished following adequate transaortic septal myectomy. In our experience, interventions on a structurally normal mitral valve are rarely needed to relieve systolic anterior motion (SAM) or mitral valve regurgitation.⁸

Finally, it is important to identify other phenotypic HCM variants on preoperative echocardiography or cardiac magnetic resonance imaging. Hypertrophic cardiomyopathy with midventricular obstruction or apical HCM requires a transapical incision to relieve intraventricular gradients or to enlarge the LV cavity.^{9,10} Complex long-segment septal hypertrophy may necessitate a combined procedure involving transaortic and transapical incisions, and is discussed in this article.¹¹

Operative technique

After induction of general anesthesia, transesophageal echocardiography is performed to assess the cardiac anatomy and quantify preoperative LVOT gradient, systolic anterior motion, and mitral valve regurgitation. On prebypass echocardiography, additional information pertinent to the surgeon may include the maximum septal thickness, the

Disclosures: None.

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distance from the aortic annulus to the greatest septal bulge, as well as the distal extent of the septal hypertrophy towards the apex. The steps in transaortic septal myectomy are presented in [Figures 1-13](#).

Following completion of myectomy, the left ventricle is irrigated and any debris from the resection is removed. The aortic and mitral valves are inspected carefully for inadvertent injury. Then, the aortotomy is closed in 2 layers using 4-0 polypropylene suture.

Cardiopulmonary bypass is discontinued in the usual fashion, and atrial and ventricular pacing wires are placed. Postbypass, we again measure the LVOT gradient using transesophageal echocardiography and direct needle measurement (at rest and following PVC). If there is a residual gradient of more than 15-20 mmHg associated with significant systolic anterior motion, we reinstitute cardiopulmonary bypass in order to resect any remaining muscle causing residual obstruction.

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