Myocardial Mechanical Remodeling after Septal Myectomy for Severe Obstructive Hypertrophic Cardiomyopathy

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Background: Septal myectomy for symptomatic patients with hypertrophic obstructive cardiomyopathy (HOCM) is a well-established procedure for symptomatic relief. Myocardial mechanics are abnormal in patients with HOCM, demonstrating low longitudinal strain, high circumferential strain, and high apical rotation compared with healthy subjects. The aim of this study was to determine whether functional improvement after myectomy is associated with improved myocardial mechanics.

Methods: Clinical data and paired echocardiographic studies before and after myectomy (6–18 months) were retrospectively analyzed and compared in 66 patients (mean age, 54 ± 13 years; 64% men) with HOCM. Myocardial mechanics including longitudinal and circumferential strain and rotation were assessed using two-dimensional strain software (Velocity Vector Imaging).

Results: Patients had significant symptomatic alleviation (mean New York Heart Association class, 2.8 ± 0.4 at baseline and 1.3 ± 0.5 after myectomy; $P < .05$). Left ventricular outflow gradient decreased dramatically (from 93 ± 26 to 17 ± 12 mm Hg; $P < .05$), and left atrial volume index decreased (from 48 ± 16 to 37 ± 13 cm^3/m^2; $P < .05$). Low longitudinal strain decreased at the myectomy site, increased in the lateral segments, and remained unchanged globally (−16 ± 4). High circumferential strain decreased (from −31 ± 5 to −25 ± 6, $P < .05$). High left ventricular twist normalized (from −15.5 ± 6.2° to 12.8 ± 4.2°, $P < .05$). Independent predictors of symptomatic response included younger age before myectomy, thinner posterior wall, and higher lateral early diastolic velocity (e′).

Conclusion: In patients with HOCM, surgical myectomy alleviated symptoms, relieved obstruction, and decreased left atrial volume index. Longitudinal strain remained unchanged, but circumferential strain and rotation decreased, demonstrating different mechanical adaptations to chronic elevated afterload seen in patients with severe aortic stenosis undergoing valve replacement. Disease extent (age, posterior wall involvement) and the presence of diastolic dysfunction seem to be related to partial symptomatic response to myectomy. (J Am Soc Echocardiogr 2013;■:■-■.)

Keywords: Hypertrophic cardiomyopathy, LV outflow obstruction, Septal myectomy, Myocardial strain

Hypertrophic obstructive cardiomyopathy (HOCM) may cause disabling symptoms, and dynamic left ventricular (LV) outflow tract obstruction is associated with a worse prognosis.[1] Invasive interventions are usually considered when pharmacotherapy either fails to control symptoms or is not tolerated.[2] These invasive options include dual-chamber permanent pacing, surgical myectomy, and septal ethanol ablation. Transaortic septal myectomy is currently considered the most appropriate treatment for the majority of patients with HOCM and severe symptoms unresponsive to medical therapy.[3] Surgical myectomy for the relief of LVOT obstruction is associated with excellent perioperative and long-term outcomes.[4,5]

The mechanics of acute unloading of a severely obstructed left ventricle have been described in patients with severe aortic stenosis (AS) with normal ejection fractions undergoing aortic valve replacement.[6] Low preoperative longitudinal and abnormally high circumferential strain normalized after aortic valve replacement. Because chronically increased pressure overload and its surgical alleviation are similar in severe AS and HOCM, we wished to define premyectomy and postmyectomy mechanics to determine whether they were altered by successful surgical myectomy.

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Abbreviations

AS = Aortic stenosis

CI = Confidence interval

FEARR = Fraction of early apical reverse rotation

HCM = Hypertrophic cardiomyopathy

HOCM = Hypertrophic obstructive cardiomyopathy

LV = Left ventricular

LVOT = Left ventricular outflow tract

2D = Two-dimensional

METHODS

Patients

Sixty-six patients undergoing septal myectomy without concomitant surgery (valve repair or replacement or maze procedure) done at Toronto General Hospital (Toronto, ON, Canada) for symptomatic HOCM were retrospectively studied. Patients required full baseline clinical records, echocardiograms obtained <6 months before myectomy and on follow-up 6 to 18 months after myectomy, and echocardiographic studies adequate for strain analysis in both baseline and follow-up studies. The control group consisted of 36 healthy subjects without family histories of hypertrophic cardiomyopathy (HCM) and normal echocardiographic findings with similar gender and age distributions. The study was approved by the Research Ethics Board of Toronto General Hospital, University Health Network.

The diagnosis of HOCM was based on echocardiographic findings of septal hypertrophy (≥15 mm) and septal/posterior wall thickness ratio > 1.3, in the absence of any other cause that could account for the degree of hypertrophy detected.[3,7] Myectomy was offered to patients with unacceptable symptoms despite maximally tolerated pharmacotherapy with LVOT gradients ≥ 50 mm Hg at rest or after provocation (Valsalva maneuver, inhalation of amyl nitrate, or ventricular premature beat), as measured during Doppler echocardiography or cardiac catheterization.[3,7] Patients were selected for surgical septal myectomy rather than septal ethanol ablation on the basis of their ages, comorbidities, and preferences.[8–10]

Myectomy Procedure

Myectomy was performed during cardiopulmonary bypass. Septal myectomy was performed through an oblique aortotomy [1]. The length of the septal myectomy ranged from 35 to 50 mm, the width ranged from 20 to 35 mm (wider toward the apex than in the subaortic region), and the depth of the resection was aimed at leaving 8 to 10 mm of residual thickness at the site of the myectomy. Concomitant surgical procedures were also performed if required.

Echocardiography

Baseline (preprocedural) and follow-up (6–18 months after the procedure) transthoracic echocardiographic images were obtained. Echocardiographic studies were interpreted at the time of acquisition. Good-quality echocardiograms were required for speckle-tracing analysis with minimal two-dimensional (2D) frame rate of 40 frames/sec with good LV endocardial border demarcation. Two-dimensional Doppler parameters were measured according to guidelines of the American Society of Echocardiography.[11,12]

Analysis of Myocardial Mechanics. Baseline (preprocedural) and follow-up strain measurements were performed using 2D tissue-tracking software (Velocity Vector Imaging version 3.0.0; Siemens Medical Solutions USA, Inc., Mountain View, CA) from archived 2D echocardiographic studies.[13] Longitudinal wall strain and strain rate were averaged from 18-segment measurements from the apical two-chamber, three-chamber, and four-chamber views. Circumferential strain, strain rate, and rotation velocities and angles were measured in six segments per short-axis plane (at the basal and midventricular papillary muscle LV level) and in four segments at the apical level. Measurements were averaged for each short-axis level. Averaged myocardial rotation angles were used to calculate basal-apical LV twist, defined as the maximal instantaneous mid-to-apical rotation angle difference.

Table 1 Patients' clinical characteristics (n = 66)

Variable	Value
Demographics	
Men	42 (64%)
Age (y)	54 ± 13
Age range (y)	30–80
Age at diagnosis of HCM (y)	46 ± 14
Body surface area (m^2)	1.95 ± 0.22
Symptoms	
Shortness of breath	62 (94%)
Chest Pain	43 (65%)
Syncope	16 (24%)
NYHA class II (before myectomy)	11 (17%)
NYHA class III (before myectomy)	55 (83%)
Comorbidities	
Coronary artery disease	7 (11%)
Arterial hypertension	26 (36%)
Diabetes mellitus	4 (6%)
Hyperlipidemia	26 (39%)
Atrial fibrillation	17 (26%)
Medications	
β-blockers	56 (85%)
Disopyramide	47 (71%)
Calcium channel blockers	7 (11%)

NYHA, New York Heart Association.
Data are expressed as mean ± SD or as number (percentage).

Fraction of Early Apical Reverse Rotation (FEARR)

We used early apical reverse rotation to assess early LV relaxation[14,15] and measured the fractional decrease in rotation angle from its peak value to its value at 10% of the cycle length later, using the equation $\text{FEARR} = [\theta_{\text{peak}} - \theta_{t(\text{peak})+10\%\text{CL}}]/\theta_{\text{peak}}$, where θ is the rotation angle, and CL is the cycle length. Ten percent into diastole time was selected because of previous studies demonstrating the largest decrease in fractional reverse rotation for moderate compared with mild LV hypertrophy.[16]

Statistical Analysis

Data were analyzed using MedCalc version 11.6.1 (MedCalc Software, Mariakerke, Belgium). Continuous data are presented as mean ± SD. Patients were compared with control subjects using unpaired t tests. Premyectomy and postmyectomy comparisons were done using paired t tests. Logistic regression analysis was used to identify independent predictors of clinical response to myectomy. Statistical significance was defined as a P value <.05. For test performance, intraobserver and interobserver variability, intraclass correlation coefficients with 95% confidence intervals (CIs) were calculated.