

Transaortic Chordal Cutting

Mitral Valve Repair for Obstructive Hypertrophic Cardiomyopathy With Mild Septal Hypertrophy



Paolo Ferrazzi, MD,* Paolo Spirito, MD,† Attilio Iacovoni, MD,‡ Alice Calabrese, MD,‡ Katrin Migliorati, PhD,* Caterina Simon, MD,§ Samuele Pentiricci, MD,§ Daniele Poggio, MD,* Massimiliano Grillo, MD,* Pietro Amigoni, MD,* Maria Iascone, PhD,|| Andrea Mortara, MD,* Barry J. Maron, MD,¶ Michele Senni, MD,‡ Paolo Bruzzi, PhD, MD#

ABSTRACT

BACKGROUND In severely symptomatic patients with obstructive hypertrophic cardiomyopathy (HCM) and mild septal hypertrophy, mitral valve (MV) abnormalities may play an important role in MV displacement into the left ventricular (LV) outflow tract. Therefore, isolated myectomy may not relieve outflow obstruction and symptoms, and MV replacement is often the surgical alternative.

OBJECTIVES This study sought to assess the clinical and hemodynamic results of cutting thickened secondary MV chordae combined with a shallow septal muscular resection in severely symptomatic patients with obstructive HCM and mild septal hypertrophy.

METHODS Clinical features were compared before surgery and at most recent clinical evaluation in 39 consecutive patients with obstructive HCM.

RESULTS Over a 23 ± 2 months follow-up, New York Heart Association functional class decreased from 2.9 ± 0.5 pre-operatively to 1.1 ± 1.1 post-operatively ($p < 0.001$), with no patient in class III at most recent evaluation. The resting outflow gradient decreased from 82 ± 43 mm Hg to 9 ± 5 mm Hg ($p < 0.001$) and septal thickness decreased from 17 ± 1 mm to 14 ± 2 mm ($p < 0.001$). No patient had MV prolapse or flail and 1 had residual moderate-to-severe MV regurgitation at most recent evaluation. MV geometry before and after surgery was compared with that of 25 consecutive patients with similar clinical profile and septal thickness that underwent isolated myectomy. After adjustment for differences in pre-operative values between the groups, the post-operative anterior MV leaflet-annulus ratio was 17% greater and tenting area 24% smaller in patients with chordal cutting, indicating that MV apparatus had moved to a more normal posterior position within the LV cavity, preventing MV systolic displacement into the outflow tract and outflow obstruction.

CONCLUSIONS This procedure relieves heart failure symptoms, abolishes LV outflow gradient, and avoids MV replacement in patients with obstructive HCM and mild septal thickness. (J Am Coll Cardiol 2015;66:1687-96)

© 2015 by the American College of Cardiology Foundation.

Surgical septal myectomy effectively relieves left ventricular (LV) outflow gradient and progressive heart failure symptoms, restores quality of life, and extends survival in patients with hypertrophic cardiomyopathy (HCM) (1-4). Because myectomy requires adequate resection of muscle from the basal ventricular septum to sufficiently enlarge the LV outflow tract and abolish mitral valve

From the *Centro per la Cardiomiopatia Ipertrofica e le Cardiopatie Valvolari, Policlinico di Monza, Monza, Italy; †Divisione di Cardiologia, Ente Ospedaliero Ospedali Galliera, Genoa, Italy; ‡USC Cardiologia, Ospedale Papa Giovanni XXIII, Bergamo, Italy; §USC Cardiocirurgia, Ospedale Papa Giovanni XXIII, Bergamo, Italy; ||Laboratorio Genetica Medica, Ospedale Papa Giovanni XXIII, Bergamo, Italy; ¶Hypertrophic Cardiomyopathy Center, Minneapolis Heart Institute Foundation, Minneapolis, Minnesota; and the #Istituto Nazionale per la Ricerca sul Cancro, Genoa, Italy. Dr. Senni has served as a consultant for Abbott Vascular and Novartis. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

Listen to this manuscript's audio summary by JACC Editor-in-Chief Dr. Valentin Fuster.

Manuscript received May 26, 2015; revised manuscript received July 23, 2015, accepted July 24, 2015.



ABBREVIATIONS AND ACRONYMS

AML = anterior mitral leaflet

HCM = hypertrophic
cardiomyopathy

LV = left ventricular

MV = mitral valve

(MV) systolic anterior motion, the most favorable results have been obtained in patients with marked septal hypertrophy (4-6).

However, a sizable minority of HCM patients with significant LV outflow gradients has relatively mild septal hypertrophy, which can make it technically difficult to achieve an adequate reduction in muscle thickness (4,7).

Such patients commonly have abnormalities of the MV apparatus, which contribute importantly to LV outflow obstruction (8-12), and they often undergo MV replacement as an alternative to septal myectomy (4,7,11,12). These technical difficulties in HCM patients with mild septal hypertrophy have stimulated the search for novel operative approaches, including transaortic extension or plication of the anterior mitral leaflet (AML) (13-16), for the purpose of avoiding MV replacement and the complications associated with prosthetic valves.

SEE PAGE 1697

In the present study, we report the results of a novel operative procedure using transaortic cutting of thickened MV secondary chordae. We performed this procedure in association with a shallow septal muscular resection (myectomy) to abolish the LV outflow gradient, relieve symptoms, and avoid MV replacement in a consecutive cohort of patients with obstructive HCM, mild septal thickness, and drug refractory symptoms.

METHODS

STUDY POPULATION. Between February 2000 and December 2013, a total of 268 consecutive patients with obstructive HCM underwent a surgical myectomy performed by the same surgeon (P.F.) at the participating institutions. In the present study, we report the clinical results in each of the 39 HCM patients who, between January 2011 and December 2013, underwent cutting of MV secondary chordae combined with a shallow septal myectomy because the magnitude and distribution of septal hypertrophy was judged inadequate for a classic extended myectomy (6). Each of these 39 patients had a LV outflow gradient ≥ 50 mm Hg at rest or with physiologic provocation, disabling symptoms unresponsive to medical therapy, and septal thickness ≤ 19 mm. These 39 patients include all patients with obstructive HCM and mild septal thickness (≤ 19 mm) who had surgery for relief of the LV outflow gradient performed between January 2011 and December 2013. Our institutional review committees approved this study. All study patients were fully informed of the nature

and risks of their condition, the standard therapies currently used to treat it, as well as the innovative nature of the surgical procedure they were offered, and gave written informed consent.

ECHOCARDIOGRAPHY. The criteria used in the present investigation for the diagnosis of HCM have been previously reported (17,18). Standard echocardiographic measurements were assessed before myectomy operation and at the most recent clinical evaluation, as previously described (17). The LV outflow tract gradient was measured under resting conditions and with physiological provocation. The degree of MV regurgitation was assessed using multiple Doppler echocardiographic criteria, including jet area, jet width, and spectral Doppler intensity, and was graded on a scale from 0 to 4 (0 = none, 1 = mild; 2 = moderate; 3 = moderate to severe; and 4 = severe) (4,19).

SURGICAL PROCEDURE. The papillary muscles were mobilized and a septal muscular resection (myectomy) was performed as previously described (17). Because the patients included in the present study had mild septal thickness, the septal resection was shallow. After papillary muscle mobilization and septal resection, the ventricular surface of the AML was examined to decide which secondary chordae to cut. This decision was made on the basis of intraoperative transesophageal echocardiographic assessment and direct surgical inspection of the anatomic and functional features of the MV apparatus. We have used the term secondary chordae (i.e., secondary order chordae) to describe all anterior leaflet chordae (including strut chordae) that insert beyond the free margin and rough zone of the leaflet (Figure 1A) (20,21). Those fibrotic secondary chordae judged to play an important role in tethering the AML toward the LV outflow tract and ventricular apex were cut selectively. The number of resected chordae varied in relation to the anatomical features of the chordal apparatus in the individual patient; resected chordae were particularly thickened, often agglutinated, and usually connected to a fibrotic area of the corresponding papillary muscle.

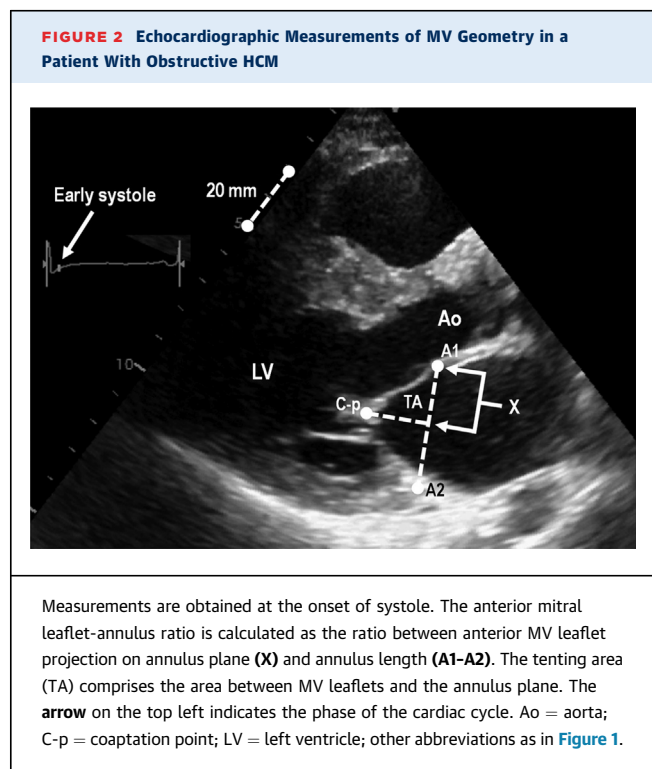
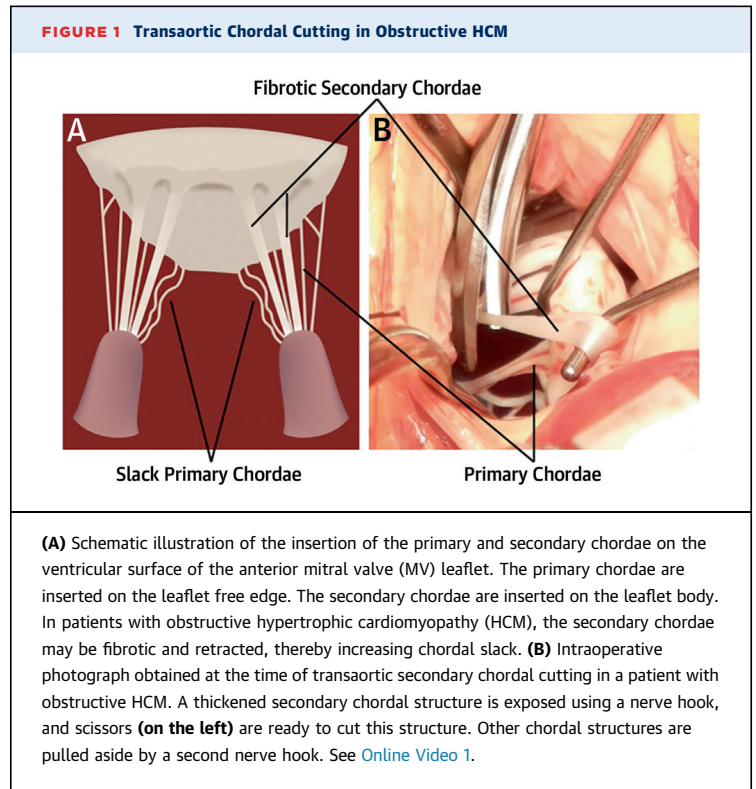
The leaflet was pushed toward the left atrium using 2 forceps to identify the retracted secondary chordae and the lines of traction exercised by the retracted chordae on the anterior leaflet. The identified secondary chordae were then selected with a nerve hook (Figure 1B). The corresponding primary chordae (located in front of the secondary chordae) were also identified to confirm that primary chordae were supporting the free margin of the anterior leaflet. In most of our study patients, the abnormal traction exerted

by the secondary chordae on the papillary muscle was confirmed by the presence of fibrosis at the site of attachment of the chordae to the papillary muscle. The distal extreme of the fibrotic secondary chordae were then cut from their connection to the papillary muscle and the proximal extreme was cut from the ventricular surface of the AML (Online Video 1). In some patients, overgrown fibrotic tissue was attached to the proximal extreme and ventricular surface of the severed secondary chordae. When present, this overgrown tissue was removed together with the chordae in order to increase leaflet mobility (Online Video 1). To prevent lesions of the leaflet body, great caution was used in disconnecting the secondary chordae from the leaflet.

SECONDARY CHORDAL CUTTING VERSUS CONVENTIONAL SURGICAL TECHNIQUES. Between February 2000 and December 2010, a total of 29 consecutive severely symptomatic patients with obstructive HCM and mild septal thickness (≤ 19 mm) underwent surgery for relief of LV outflow gradient without associated chordal cutting. Clinical and hemodynamic results in our 39 study patients with chordal cutting were compared with those in this group of 29 patients without chordal cutting to investigate the potential advantages of our novel operational technique over conventional surgery for relief of LV outflow gradient.

ASSESSMENT OF THE EFFECTS OF SECONDARY CHORDAL CUTTING ON MV GEOMETRY AND FUNCTION. To investigate the effects of secondary chordal cutting on MV geometry and function, echocardiographic indexes that measure the position of the MV coaptation point in the LV cavity were obtained in the 39 study patients with chordal cutting. These measurements were compared with measurements obtained in 25 of the 29 patients with obstructive HCM and mild septal thickness who had undergone a conventional septal myectomy without chordal cutting. Four patients in the group without chordal cutting were excluded from this analysis because they had undergone septal myectomy with associated MV repair and insertion of a prosthetic ring or MV replacement.

Two echocardiographic indexes of the displacement of the MV coaptation point in the LV cavity were obtained before and after surgery in the parasternal long-axis view at the onset of systole and were compared between the 39 study patients with chordal cutting and the 25 patients with isolated septal myectomy (Figure 2). Displacement of the coaptation point in the outflow tract was assessed as the ratio between the AML projection on the mitral annulus plane and annulus length, and expressed as AML-annulus ratio (22). Displacement of the coaptation point toward the LV apex was assessed as the area



enclosed between the valve leaflets and annulus plane, and expressed as MV tenting area (23).

STATISTICAL ANALYSES. Primary analyses were aimed at describing and comparing pre-operative and post-operative clinical characteristics of the 39 patients with secondary chordal cutting associated with myectomy. Variables were presented as means with standard deviations, or frequencies with proportions; differences between pre-operative and post-operative values were assessed for statistical significance using the paired Student *t* test, chi-square test for trend, Wilcoxon signed rank test for paired data, or Fisher exact test, as appropriate. The changes observed after surgery in the AML-annulus ratio and MV tenting area in the 39 patients with chordal cutting associated with myectomy were compared with those observed in the 25 patients with isolated myectomy using the standard analysis of covariance, with post-operative values as the dependent variable and pre-operative values as covariates. Relationships between variables were determined by Pearson or Spearman ρ correlation coefficients. Values of $p < 0.05$ were considered statistically significant. Computations were performed with IBM SPSS 19.0 statistical software (SPSS, Inc., Chicago, Illinois).

RESULTS

OUTCOME AFTER SURGERY IN PATIENTS WITH CHORDAL CUTTING.

The baseline clinical characteristics and the

hemodynamic and clinical outcomes in the 39 study patients with chordal cutting are summarized in **Table 1**. Duration of follow-up was 23 ± 2 months (median 24 months) and was ≥ 6 months in each patient. None of the study patients died during hospitalization or follow-up, and none required MV replacement.

Clinical and hemodynamic benefit. Each of the 39 patients with chordal cutting experienced substantial symptomatic and hemodynamic improvement after surgery. Pre-operatively, 32 (82%) patients were severely symptomatic in New York Heart Association functional class III or IV, and 7 (18%) were in class II. Post-operatively, 34 (87%) were asymptomatic in New York Heart Association functional class I, and 5 (13%) were in class II ($p < 0.001$) (**Figure 3A**).

Peak LV outflow gradient at rest was 82 ± 43 mm Hg (median 75 mm Hg) pre-operatively and 9 ± 5 mm Hg (median 10 mm Hg) post-operatively ($p < 0.001$), with an outflow gradient < 15 mm Hg in 34 (87%) patients at the most recent evaluation (**Figure 3B**). The left atrial diameter was 49 ± 6 mm before surgery and 45 ± 7 mm at the most recent evaluation ($p < 0.001$). Septal thickness was 17 ± 1 mm (median 17 mm) before surgery and 14 ± 2 mm (median 14 mm) after surgery ($p < 0.001$), indicative of a shallow myectomy.

A median of 3 chordae (up to a maximum of 8) was cut in each patient. None of the 39 study patients had MV prolapse or valve flail before surgery or at the most recent evaluation. Of the 9 study patients with grade 3 or 4 MV regurgitation before surgery, 8 had lower grades of MV regurgitation after surgery, whereas 1 patient had persistent moderate-severe MV regurgitation post-operatively.

Of the 39 study patients, 13 had atrial fibrillation before surgery (paroxysmal in 6, persistent in 6, and permanent in 1 patient). Of these 13 patients, 10 underwent pulmonary vein isolation using radio-frequency ablation at the time of the myectomy operation; 7 of these 10 patients were in sinus rhythm at most recent evaluation.

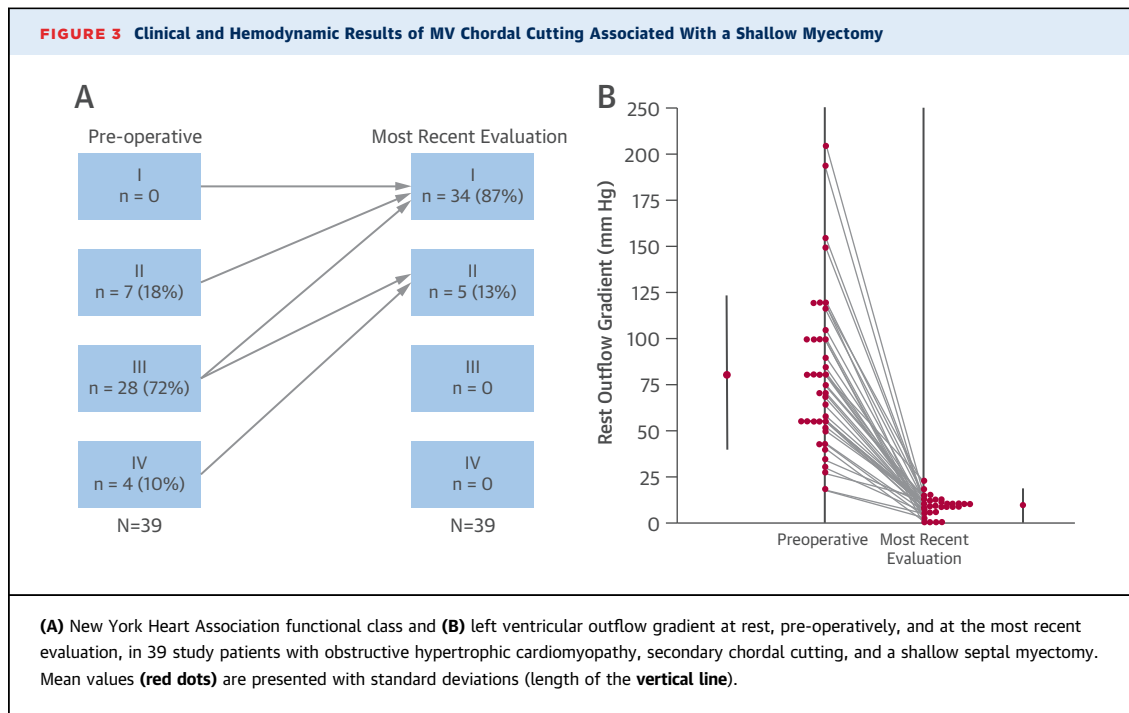
ADVANTAGES OF CHORDAL CUTTING OVER CONVENTIONAL SURGERY FOR OUTFLOW GRADIENT RELIEF.

While none of the 39 study patients with chordal cutting associated with a shallow septal myectomy died during hospitalization or follow-up, and none required MV repair with insertion of a prosthetic ring or MV replacement, 1 of the 29 patients with a conventional septal myectomy and without chordal cutting died during hospitalization due to respiratory complications and renal failure, 3 required MV repair with insertion of a prosthetic ring, and 1

TABLE 1 Clinical Characteristics Before Surgery and at Most Recent Evaluation in 39 HCM Patients With Secondary Chordal Cutting and a Shallow Septal Myectomy

	Before Surgery	Most Recent Evaluation	p Value
NYHA functional class			$< 0.001^*$
I	0	34 (87)	
II	7 (18)	5 (13)	
III-IV	32 (82)	0	
Atrial fibrillation	13 (33)	6 (15)	
Echocardiographic data			
LVOT gradient at rest, mm Hg	82 ± 43	9 ± 5	< 0.001
Basal septal thickness, mm	17 ± 1	14 ± 2	< 0.001
LVEDD, mm	40 ± 7	42 ± 5	0.085
LVEF, %	68 ± 6	63 ± 5	< 0.001
Left atrial diameter, mm	49 ± 6	45 ± 7	< 0.001
Mitral valve regurgitation grade			$< 0.001^*$
1	17 (44)	33 (85)	
2	13 (33)	5 (13)	
3	7 (18)	1 (3)	
4	2 (5)	0	

Values are n (%) or mean \pm SD. *Wilcoxon signed rank test for paired data.
LVEDD = left ventricular end-diastolic dimension; LVEF = left ventricular ejection fraction; LVOT = left ventricular outflow tract; NYHA = New York Heart Association.



required MV replacement. Clinical and hemodynamic results in the patients with or without chordal cutting are summarized in **Table 2**.

MECHANISM BY WHICH CHORDAL CUTTING REDUCES LV OUTFLOW GRADIENT. The differences in the geometry of the MV apparatus before and after surgery in the 39 study patients with chordal cutting and a shallow myectomy, and the 25 patients with isolated myectomy and without chordal cutting, are summarized in **Table 3** and illustrated in **Figures 4** and **5**, and **Online Videos 2** and **3**.

AML-annulus ratio. In the patients with chordal cutting, the AML-annulus ratio increased significantly after surgery compared to pre-operative values, from 0.45 ± 0.08 to 0.57 ± 0.08 ($p < 0.001$), a 27% increase. This indicates that the leaflet coaptation point was repositioned more normally; closer to the posterior LV wall and farther from the outflow tract, with a consequent increase in the LV outflow tract size (**Table 3**, **Figures 4A**, **4B**, and **5A**, **Online Videos 2** and **3**). This increase in the post-operative AML-annulus ratio showed a moderate variability and, in absolute terms, appeared to be relatively constant (**Figure 5A**).

In the patients without chordal cutting, the AML-annulus ratio changed slightly, but significantly, after surgery, from 0.49 ± 0.08 to 0.51 ± 0.08 ($p = 0.003$), a 3% increase (**Figures 4C**, **4D**, and **5A**). After adjustment for the differences in pre-operative values between patients with and without chordal

cutting, the difference between the mean post-operative values was $+0.09$ (SE: 0.01; $p < 0.001$), representing a 17% greater post-operative AML-annulus ratio in patients with chordal cutting compared with controls.

MV tenting area. In patients with chordal cutting, the MV tenting area decreased significantly after surgery compared with pre-operative values, from 2.14 ± 0.62 cm² to 1.51 ± 0.54 cm² ($p < 0.001$), a 29% decrease. This indicates a reduction in the systolic displacement of the mitral leaflets toward the apex

TABLE 2 Comparison of Intraoperative Results and Clinical Outcome in HCM Patients With and Without Chordal Cutting

	Patients With Chordal Cutting (n = 39)	Patients Without Chordal Cutting (n = 29)	p Value
Age, yrs	58 ± 13	55 ± 15	0.535
Number of in-hospital/follow-up deaths	None	1 (3)	0.426*
Number of MV repair/replacement	None	4 (14)	0.029*
Number of cut chordae	1-8 (3)	—	
Clinical features at most recent evaluation			
NYHA functional class III-IV	None	3 (11)	0.068*
Residual LVOT gradient ≥30 mm Hg	None	2 (7)	0.171*
Post-operative LVOT gradient, mm Hg	9 ± 5	13 ± 10	0.041
Post-operative LVEF, %	63 ± 5	65 ± 7	0.193
Mitral valve regurgitation grade ≥3	1 (3)	3 (11)	0.301*

Values are n, mean ± SD, n (%), or range (median). *Fisher exact test.
HCM = hypertrophic cardiomyopathy; MV = mitral valve; other abbreviations as in **Table 1**.

TABLE 3 Geometry of the MV Apparatus Before and After Surgical Myectomy in 39 HCM Patients With and 25 Without Chordal Cutting

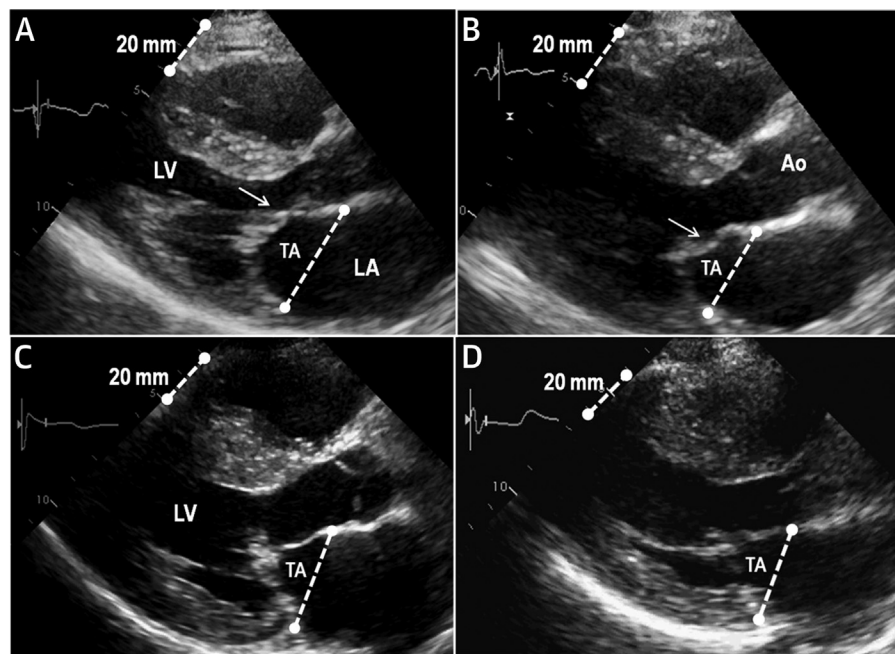
	Patients With Chordal Cutting (Mean ± SD)	Patients Without Chordal Cutting (Mean ± SD)	Crude Post-Operative Difference Between Groups (mean ± SE)	Adjusted Post-Operative Difference Between Groups* (mean ± SE)	p Value
AML-annulus ratio					
Pre-operative value	0.45 ± 0.08	0.49 ± 0.08			
Post-operative value	0.57 ± 0.08	0.51 ± 0.08	+0.06 ± 0.02	+0.09 ± 0.01	<0.001
Absolute change	+0.11 ± 0.07	-0.01 ± 0.02			
p value	<0.001	0.003			
Proportional change	+27%	+3%		+17%	
MV tenting area, cm²					
Pre-operative value	2.14 ± 0.62	1.78 ± 0.48			
Post-operative value	1.51 ± 0.54	1.70 ± 0.41	-0.19 ± 0.13	-0.42 ± 0.10	<0.001
Absolute change, cm ²	-0.63 ± 0.42	-0.08 ± 0.27			
p value	<0.001	0.172			
Proportional change	-29%	-4%		-24%	

*Difference between post-operative mean values, after adjustment for differences in pre-operative values between the groups using the analysis of covariance.
AML = anterior mitral leaflet.

(Table 3, Figures 4A and 4B). This decrease in post-operative MV tenting area showed an important variability and was more marked in patients with a larger pre-operative tenting area (Figure 5B).

In the patients without chordal cutting, the MV tenting area did not change significantly after surgery, from $1.78 \pm 0.48 \text{ cm}^2$ to $1.70 \pm 0.41 \text{ cm}^2$ ($p = 0.172$) (Figures 4C and 4D). After adjustment

FIGURE 4 Echocardiographic Images Showing the Effects of Secondary Chordal Cutting on the MV Apparatus in Obstructive HCM



Pre-operative (A) and post-operative (B) echocardiographic images obtained in early systole in a patient with obstructive HCM and mild septal thickness. After secondary chordal cutting (5 chordae) and a shallow septal myectomy, the abnormal tethering of the anterior leaflet by thickened secondary chordae is abolished (arrows), the MV apparatus has moved away from the LV outflow tract to a more posterior and normal position in the LV cavity, and the MV tenting area is substantially reduced, from 3.5 to 2.2 cm². See [Online Videos 2 and 3](#). Pre-operative (C) and post-operative (D) echocardiographic images obtained in early systole in a patient with obstructive HCM, relatively mild septal thickness, and isolated septal myectomy without secondary chordal cutting. After surgery, the MV TA remained substantially unchanged, from 2.70 to 2.65 cm². Abbreviations as in [Figures 1 and 2](#).

for the differences in pre-operative values between the 2 patient groups, the difference between the means was -0.42 cm^2 (SE: 0.10; $p < 0.001$), representing a 24% smaller post-operative MV tenting area in patients with chordal cutting compared with controls.

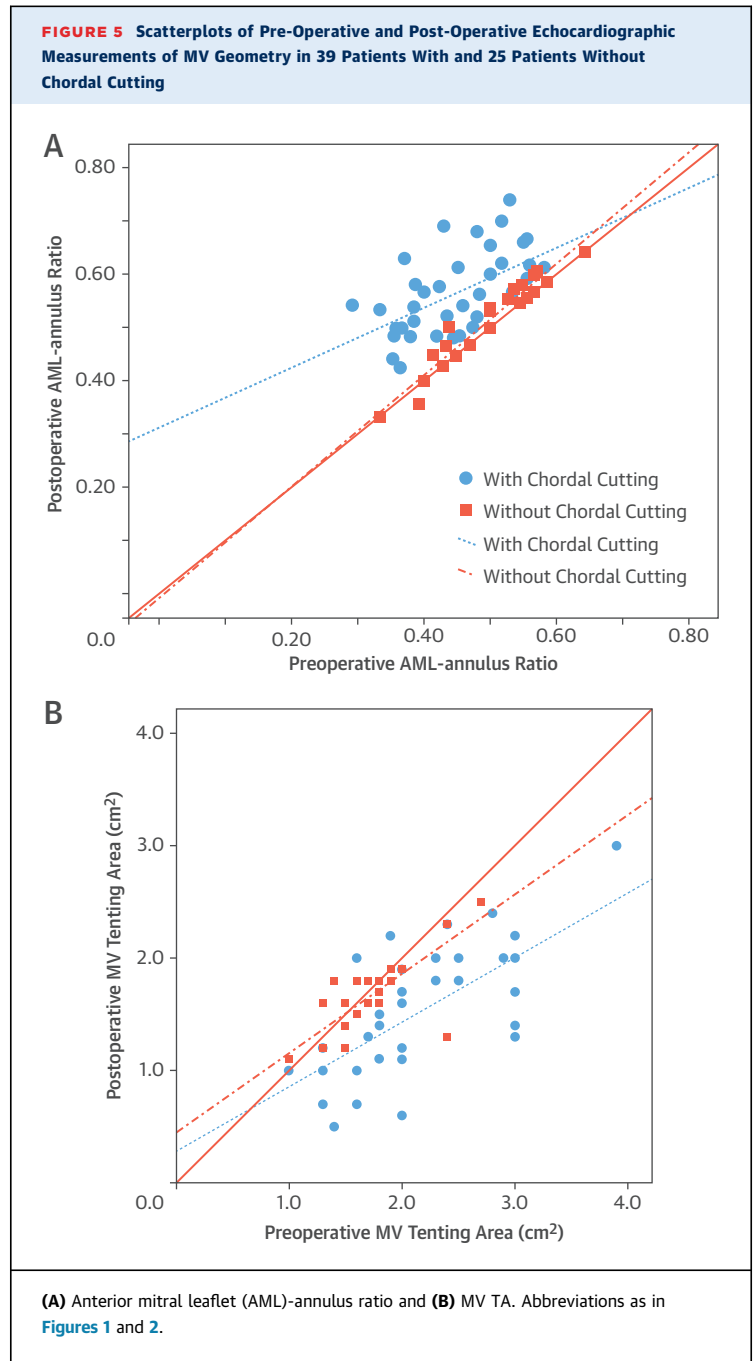
DISCUSSION

The results of the present study show that our novel transaortic surgical procedure of cutting fibrotic and retracted MV secondary chordae (in combination with a shallow septal myectomy) can abolish or substantially reduce the LV outflow gradient, relieve heart failure symptoms, and improve quality of life in patients with obstructive HCM and mild septal thickness. This procedure may also reduce the risk of iatrogenic septal defect and avoid MV replacement (4,7,24).

In each of our study patients with transaortic chordal cutting associated with a shallow myectomy, functional limitation decreased to class I or II and LV outflow gradient was abolished or substantially reduced after surgery. In the great majority of the study patients, moderate or severe MV regurgitation either decreased by at least 1 grade (to grade 1 to 2), or was abolished after surgery. None of the study patients required MV repair through the left atrium or MV replacement, and none had MV flail or prolapse at the most recent evaluation.

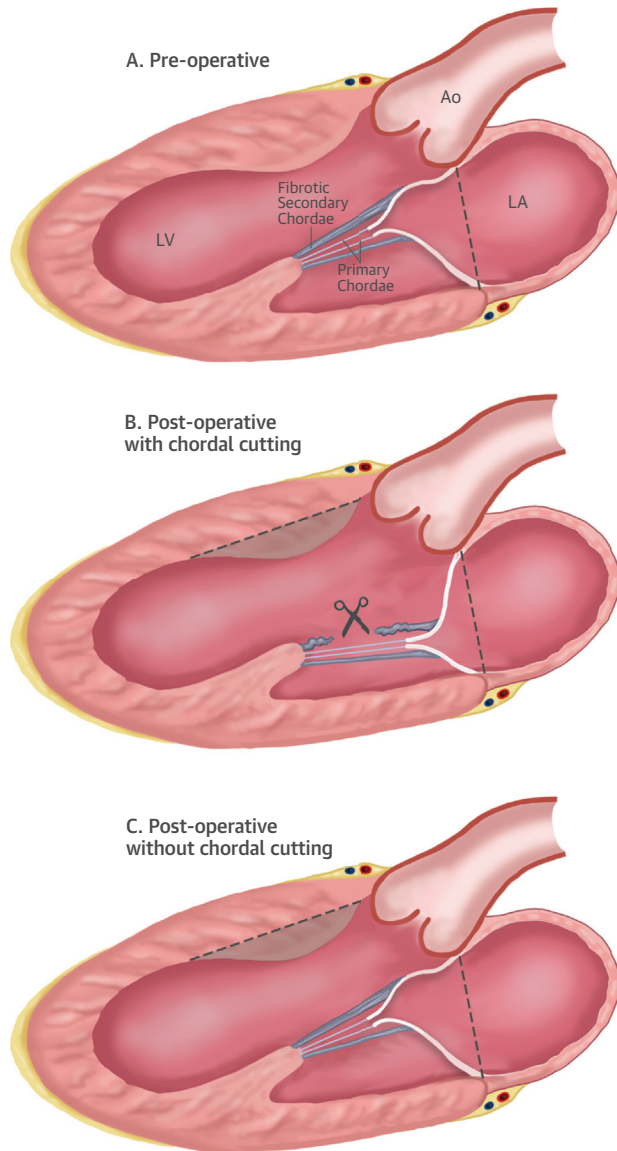
Our findings also clarify the mechanism by which this transaortic procedure led to substantial clinical and hemodynamic improvement in our study patients. In normal subjects, the secondary chordae (inserted on the body of the MV leaflet ventricular surface) help to preserve ventricular shape and function during ejection, while the primary chordae (inserted on the leaflet free edge) prevent valve flail and regurgitation by opposing the valve closing forces in systole (25,26). However, in patients with obstructive HCM, the secondary chordae of the anterior MV leaflet often appear thickened and retracted at surgery or necropsy (8,11). The effects of these chordal abnormalities on the MV apparatus have not been previously investigated. However, earlier studies have shown that in patients with obstructive HCM, remodeling of the left ventricle and MV apparatus increases leaflet and chordal slack, thereby exposing the tip of the anterior leaflet to the risk of being displaced into the outflow tract by blood flow drag forces during ejection (8-11,27-31).

The rationale for our novel operative approach relied on the hypothesis that, in many patients with



obstructive HCM and mild septal thickness, fibrotic and retracted secondary chordae may cause abnormal tethering of the AML and favor the displacement of slack portions of the leaflet (and attached primary chordae) into the LV outflow tract and the ejection flow. The results of our study show that in this patient subset, secondary chordal cutting in association with a shallow myectomy moves the MV coaptation point away from the LV outflow tract to a more posterior

CENTRAL ILLUSTRATION Secondary Chordal Cutting in Obstructive HCM: Effects of Secondary Chordal Cutting on the Geometry and Function of the MV Apparatus



Ferrazzi, P. et al. J Am Coll Cardiol. 2015; 66(15):1687-96.

(A) In patients with obstructive hypertrophic cardiomyopathy (HCM), fibrotic and retracted mitral valve (MV) secondary chordae contribute to displace the body of the anterior leaflet into the left ventricular outflow tract. **(B)** Cutting selected abnormal chordae (in combination with a shallow septal myectomy) moves the MV apparatus and leaflet coaptation point away from the outflow tract to a more posterior and normal position in the left ventricular cavity, substantially increasing outflow tract size and decreasing MV tenting area. **(C)** Isolated septal myectomy (i.e., without associated chordal cutting) does not alter the anterior displacement of the MV apparatus. Ao = aorta; LA = left atrium; LV = left ventricle.

and normal position within the LV cavity by reducing anterior leaflet tethering and restoring slack primary chordae to their function. These changes in MV geometry and function enlarge the outflow tract, prevent displacement of the MV apparatus into the path of the ejection flow, and contribute to abolition of LV outflow obstruction (**Central Illustration, Online Videos 2 and 3**).

The extent of MV repositioning in the LV cavity after chordal cutting varied among our study patients. In particular, the post-operative decrease in MV tenting area tended to be more marked in patients with a larger pre-operative tenting area. It is likely that several morphologic features that affect the MV position in the LV cavity of patients with HCM contributed to this variability, including the extent of anterior and apical papillary muscle displacement in the cavity, the severity of MV and chordal abnormalities, and variability in the impact of early systolic flow on the posterior leaflet (8-11,27-32).

Although secondary chordal cutting has previously been used to reduce systolic tethering of the AML and valve regurgitation in patients with ischemic dilated cardiomyopathy and severe MV insufficiency (33), this operative strategy remains controversial because the secondary chordal function of maintaining ventricular geometry and enhancing wall thickening may be helpful in the presence of LV dilation and systolic dysfunction (26). However, in patients with obstructive HCM, these potentially unfavorable effects of secondary chordal cutting are prevented by the preserved or hyperdynamic systolic function and small LV cavity.

We must underline that although chordal cutting may improve operative results in patients with obstructive HCM and mild LV hypertrophy undergoing a shallow septal myectomy, this operation requires surgeons with particular experience with the anatomy of obstructive HCM. Therefore, our observations support the recommendations of the American College of Cardiology Foundation/American Heart Association HCM guidelines that operator and institutional experience with surgical management of patients with HCM are key determinants of a successful outcome (34).

STUDY LIMITATIONS. In the present study, the only criteria used to select patients for secondary chordal cutting were presence of LV outflow gradient ≥ 50 mm Hg at rest or with provocation, relatively mild septal thickness (≤ 19 mm), and disabling symptoms unresponsive to medical therapy. We selected this subgroup because it is in patients with obstructive HCM and only mild septal hypertrophy that abnormalities of the mitral valve apparatus may play a particularly

important role in generating the LV outflow gradient. However, the extent of mitral valve repositioning in the LV cavity after surgery in patients with chordal cutting showed some variability. Therefore, a larger and morphologically more diverse study population, including patients with more marked LV hypertrophy, will be required to identify additional criteria that could help to clarify which patients with obstructive HCM may benefit most from combining secondary chordal cutting with septal myectomy.

CONCLUSIONS

We report the results of a novel operative procedure using transaortic cutting of thickened MV secondary chordae that, in association with a shallow myectomy, moves the leaflet coaptation point away from the septum to a more posterior position within the LV cavity, increases LV outflow tract size, abolishes or substantially decreases the LV outflow gradient, relieves heart failure symptoms, and may reduce the need for MV replacement in this important patient subset with obstructive HCM, drug refractory symptoms, and relatively mild septal thickness.

ACKNOWLEDGMENTS The authors gratefully acknowledge the assistance of Barbara Ottonello and Domenico Carratta in preparing the figures.

REPRINT REQUESTS AND CORRESPONDENCE: Dr. Paolo Ferrazzi, Centro per la Cardiomiopatia Ipertrofica e le Cardiopatie Valvolari, Policlinico di Monza, Via Amati, 111, 20090 Monza, Italy. E-mail: paolo.ferrazzi@policlinicodimonza.it.

PERSPECTIVES

COMPETENCY IN MEDICAL KNOWLEDGE: Fibrotic and retracted secondary MV chordae play an important role in systolic displacement of the MV apparatus into the LV outflow tract in patients with obstructive HCM, mild septal hypertrophy, and drug-refractory symptoms. Transaortic cutting of fibrotic secondary chordae, associated with a shallow myectomy, moves the leaflet coaptation point away from the septum to a more posterior position within the LV cavity, thereby enlarging the outflow tract, preventing displacement of the MV leaflets into the path of ejection flow, abolishing outflow obstruction, and reducing the need for deep muscular resection in this difficult patient subset.

TRANSLATIONAL OUTLOOK: Further studies are needed to enhance the pre-operative identification of patients who may benefit from this transaortic approach and assess the long-term durability of benefit in severely symptomatic patients with obstructive HCM and mild septal thickening.

REFERENCES

1. Ommen SR, Maron BJ, Olivetto I, et al. Long-term effects of surgical septal myectomy on survival in patients with obstructive hypertrophic cardiomyopathy. *J Am Coll Cardiol* 2005;46:470-6.
2. Woo A, Williams WG, Choi R, et al. Clinical and echocardiographic determinants of long-term survival after surgical myectomy in obstructive hypertrophic cardiomyopathy. *Circulation* 2005;111:2033-41.
3. Ball W, Ivanov J, Rakowski H, et al. Long-term survival in patients with resting obstructive hypertrophic cardiomyopathy: comparison of conservative versus invasive treatment. *J Am Coll Cardiol* 2011;58:2313-21.
4. Desai MY, Bhonsale A, Smedira NG, et al. Predictors of long-term outcomes in symptomatic hypertrophic obstructive cardiomyopathy patients undergoing surgical relief of left ventricular outflow tract obstruction. *Circulation* 2013;128:209-16.
5. Maron BJ, Dearani JA, Ommen SR, et al. The case for surgery in obstructive hypertrophic cardiomyopathy. *J Am Coll Cardiol* 2004;44:2044-53.
6. Dearani JA, Ommen SR, Gersh BJ, et al. Surgery insight: septal myectomy for obstructive hypertrophic cardiomyopathy—the Mayo Clinic experience. *Nat Clin Pract Cardiovasc Med* 2007;4:503-12.
7. McIntosh CL, Maron BJ. Current operative treatment of obstructive hypertrophic cardiomyopathy. *Circulation* 1988;78:487-95.
8. Klues HG, Maron BJ, Dollar AL, et al. Diversity of structural mitral valve alterations in hypertrophic cardiomyopathy. *Circulation* 1992;85:1651-60.
9. Levine RA, Vlahakes GJ, Lefebvre X, et al. Papillary muscle displacement causes systolic anterior motion of the mitral valve: experimental validation and insights into the mechanism of subaortic obstruction. *Circulation* 1995;91:1189-95.
10. Sherrid MV, Gunsberg DZ, Moldenhauer S, et al. Systolic anterior motion begins at low left ventricular outflow tract velocity in obstructive hypertrophic cardiomyopathy. *J Am Coll Cardiol* 2000;36:1344-54.
11. Kaple RK, Murphy RT, DiPaola LM, et al. Mitral valve abnormalities in hypertrophic cardiomyopathy: echocardiographic features and surgical outcomes. *Ann Thorac Surg* 2008;85:1527-35, 1535.e1-2.
12. Patel P, Dhillon A, Popovic ZB, et al. Left ventricular outflow obstruction in hypertrophic cardiomyopathy patients without severe septal hypertrophy: implications of mitral valve and papillary muscle abnormalities assessed using cardiac magnetic resonance and echocardiography. *Circ Cardiovasc Imaging* 2015;8:e003132.
13. van der Lee C, Kofflard MJ, van Herwerden LA, et al. Sustained improvement after combined anterior mitral leaflet extension and myectomy in hypertrophic obstructive cardiomyopathy. *Circulation* 2003;108:2088-92.
14. Balam SK, Tyrie L, Sherrid MV, et al. Resection-plication-release for hypertrophic cardiomyopathy: clinical and echocardiographic follow-up. *Ann Thorac Surg* 2008;86:1539-44, discussion 1544-5.
15. Balam SK, Ross RE, Sherrid MV, et al. Role of mitral valve plication in the management of hypertrophic cardiomyopathy. *Ann Thorac Surg* 2012;94:1990-7.
16. Vriesendorp PA, Schinkel AFL, Soliman OII, et al. Long-term benefit of myectomy and anterior mitral leaflet extension in obstructive hypertrophic cardiomyopathy. *Am J Cardiol* 2015;115:670-5.
17. Iacovoni A, Spirito P, Simon C, et al. A contemporary European experience with surgical septal myectomy in hypertrophic cardiomyopathy. *Eur Heart J* 2012;33:2080-7.

18. Spirito P, Bellone P, Harris KM, et al. Magnitude of left ventricular hypertrophy and risk of sudden death in hypertrophic cardiomyopathy. *N Engl J Med* 2000;342:1778-85.
19. Zoghbi WA, Enriquez-Sarano M, Foster E, et al., for the American Society of Echocardiography. Recommendations for evaluation of the severity of native valvular regurgitation with two-dimensional and Doppler echocardiography. *J Am Soc Echocardiogr* 2003;16:777-802.
20. Lam JH, Ranganathan N, Wigle ED, et al. Morphology of the human mitral valve. I. Chordae tendineae: a new classification. *Circulation* 1970;41:449-58.
21. Kumar N, Kumar M, Duran CM. A revised terminology for recording surgical findings of the mitral valve. *J Heart Valve Dis* 1995;4:70-5, discussion 76-7.
22. Dellling FN, Sanborn DY, Levine RA, et al. Frequency and mechanism of persistent systolic anterior motion and mitral regurgitation after septal ablation in obstructive hypertrophic cardiomyopathy. *Am J Cardiol* 2007;100:1691-5.
23. Yiu SF, Enriquez-Sarano M, Tribouilloy C, et al. Determinants of the degree of functional mitral regurgitation in patients with systolic left ventricular dysfunction: A quantitative clinical study. *Circulation* 2000;102:1400-6.
24. Cooley DA, Wukasch DC, Leachman RD. Mitral valve replacement for idiopathic hypertrophic subaortic stenosis. Results in 27 patients. *J Cardiovasc Surg (Torino)* 1976;17:380-7.
25. Obadia JF, Casali C, Chassignolle JF, et al. Mitral subvalvular apparatus: different functions of primary and secondary chordae. *Circulation* 1997;96:3124-8.
26. Rodriguez F, Langer F, Harrington KB, et al. Importance of mitral valve second-order chordae for left ventricular geometry, wall thickening mechanics, and global systolic function. *Circulation* 2004;110:1115-22.
27. Spirito P, Maron BJ. Significance of left ventricular outflow tract obstruction cross-sectional area in hypertrophic cardiomyopathy: a two-dimensional echocardiographic assessment. *Circulation* 1983;67:1100-8.
28. Spirito P, Maron BJ, Rosing DR. Morphologic determinants of hemodynamic state following ventricular septal myotomy-myectomy in patients with obstructive hypertrophic cardiomyopathy: M-mode and two-dimensional echocardiographic assessment. *Circulation* 1984;70:984-95.
29. Klues HG, Proschan MA, Dollar AL, et al. Echocardiographic assessment of mitral valve size in obstructive hypertrophic cardiomyopathy. Anatomic validation from mitral valve specimen. *Circulation* 1993;88:548-55.
30. Sherrid MV, Chu CK, Della E, et al. An echocardiographic study of the fluids mechanics of obstruction in hypertrophic cardiomyopathy. *J Am Coll Cardiol* 1993;22:816-25.
31. Kim DH, Handschumacher MD, Levine RA, et al. In vivo measurement of mitral valve leaflet surface area and subvalvular geometry in patients with asymmetrical septal hypertrophy: insights into the mechanism of outflow tract obstruction. *Circulation* 2010;122:1298-307.
32. Ro R, Halpern D, Sahn DJ, et al. Vector flow mapping in obstructive hypertrophic cardiomyopathy to assess the relationship of early systolic left ventricular flow and the mitral valve. *J Am Coll Cardiol* 2014;64:1984-95.
33. Messas E, Guerrero JL, Handschumacher MD, et al. Chordal cutting: a new therapeutic approach for ischemic mitral regurgitation. *Circulation* 2001;104:1958-63.
34. Gersh BJ, Maron BJ, Bonow RO, et al. 2011 ACCF/AHA guideline for the diagnosis and treatment of hypertrophic cardiomyopathy: executive summary: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines Developed in Collaboration With the American Association for Thoracic Surgery, American Society of Echocardiography, American Society of Nuclear Cardiology, Heart Failure Society of America, Heart Rhythm Society, Society for Cardiovascular Angiography and Interventions, and Society of Thoracic Surgeons. *J Am Coll Cardiol* 2011;58:2703-38.

KEY WORDS heart failure, septal myectomy, ventricular septum

APPENDIX For supplemental videos and their legends, please see the online version of this article.