

Hypertrophic Cardiomyopathy

Outcome of Patients With Hypertrophic Obstructive Cardiomyopathy After Percutaneous Transluminal Septal Myocardial Ablation and Septal Myectomy Surgery

Jian Xin Qin, MD, Takahiro Shiota, MD, PhD, FACC, Harry M. Lever, MD, FACC, Samir R. Kapadia, MD, Marta Sitges, MD, David N. Rubin, MD, Fabrice Bauer, MD, Neil L. Greenberg, PhD, Deborah A. Agler, RDCS, Jeanne K. Drinko, RDCS, Maureen Martin, E. Murat Tuzcu, MD, FACC, Nicholas G. Smedira, MD, FACC, Bruce Lytle, MD, FACC, James D. Thomas, MD, FACC

Cleveland, Ohio

OBJECTIVES	This study was conducted to evaluate follow-up results in patients with hypertrophic obstructive cardiomyopathy (HOCM) who underwent either percutaneous transluminal septal myocardial ablation (PTSMA) or septal myectomy.
BACKGROUND METHODS	Controversy exists with regard to these two forms of treatment for patients with HOCM. Of 51 patients with HOCM treated, 25 were treated by PTSMA and 26 patients via myectomy. Two-dimensional echocardiograms were performed before both procedures, immediately afterwards and at a three-month follow-up. The New York Heart Association (NYHA) functional class was obtained before the procedures and at follow-up.
RESULTS	Interventricular septal thickness was significantly reduced at follow-up in both groups (2.3 ± 0.4 cm vs. 1.9 ± 0.4 cm for septal ablation and 2.4 ± 0.6 cm vs. 1.7 ± 0.2 cm for myectomy, both $p < 0.001$). Estimated by continuous-wave Doppler, the resting pressure gradient (PG) across the left ventricular outflow tract (LVOT) significantly decreased immediately after the procedures in both groups (64 ± 39 mm Hg vs. 28 ± 29 mm Hg for PTSMA, 62 ± 43 mm Hg vs. 7 ± 7 mm Hg for myectomy, both $p < 0.0001$). At three-month follow-up, the resting PG remained lower in the PTSMA and myectomy groups (24 ± 19 mm Hg and 11 ± 6 mm Hg, respectively, vs. those before procedures, both $p < 0.0001$). The NYHA functional class was also significantly improved in both groups (3.5 ± 0.5 vs. 1.9 ± 0.7 for PTSMA, 3.3 ± 0.5 vs. 1.5 ± 0.7 for myectomy, both $p < 0.0001$).
CONCLUSIONS	Both myectomy and PTSMA reduce LVOT obstruction and significantly improve NYHA functional class in patients with HOCM. However, there are benefits and drawbacks for each therapeutic method that must be counterbalanced when deciding on treatment for LVOT obstruction. (J Am Coll Cardiol 2001;38:1994-2000) © 2001 by the American College of Cardiology

Hypertrophic obstructive cardiomyopathy (HOCM) is a disease characterized by: hypertrophy of the interventricular septum (IVS), a narrowed left ventricular (LV) outflow tract (LVOT) and, frequently, systolic anterior motion (SAM) of the mitral valve resulting in LVOT obstruction (1-3). The aim of treatment is to reduce SAM to eliminate the pressure gradient (PG) and the mitral insufficiency in order to improve myocardial performance and symptoms. Several therapeutic options have been suggested for treating patients with HOCM. Medications, including beta-adrenergic blocking agents, verapamil and disopyramide, are

used for initial treatment. However, high doses of medication frequently required to treat the symptoms of HOCM may produce side effects (4). Surgical septal myectomy has been shown to be safe and effective for eliminating the LVOT obstruction and improving symptoms (5-9). However, experience with myectomy is limited at many centers. Several reports show that the role of dual pacemakers as an option in the treatment of LVOT obstruction is still controversial (10,11). Recently, a nonsurgical procedure, percutaneous transluminal septal myocardial ablation (PTSMA), has been introduced and might replace surgical myectomy in some patients (12-18). Ethanol is infused into one or more septal perforator branches of the left anterior descending coronary artery to cause necrosis and then shrinkage of the proximal hypertrophied septum, which results in an enlargement of the narrowed LVOT. This novel therapeutic modality might become standard treatment for HOCM, but its results in comparison with

From the Cardiovascular Imaging Center, Department of Cardiology, The Cleveland Clinic Foundation, Cleveland, Ohio. Supported, in part, by grant NCC9-60 from the National Aeronautics and Space Administration, Houston, Texas, Grant #9951522V from the America Heart Association Ohio Local Chapter, Columbus, Ohio, and Grant #R01 HL56688-01A1 from the National Institutes of Health, Bethesda, Maryland. Presented, in part, at the 49th Annual Scientific Sessions of American College of Cardiology, March 2000, Anaheim, California.

Manuscript received June 4, 2001; revised manuscript received August 21, 2001, accepted August 31, 2001.

Abbreviations and Acronyms

HOCM	= hypertrophic obstructive cardiomyopathy
IVS	= interventricular septum
LV	= left ventricle or left ventricular
LVOT	= left ventricular outflow tract
NYHA	= New York Heart Association
PG	= pressure gradient
PTMSA	= percutaneous transluminal septal myocardial ablation
PW	= posterior wall
SAM	= systolic anterior motion of mitral leaflet
TEE	= transesophageal echocardiography

myectomy are not clear. While we feel PTMSA is a viable alternative to surgery, we have taken a cautious approach with it. We have done it, for the most part, in elderly patients or patients with other comorbid conditions because we feel that this is an investigational procedure that has not been definitively proven. Thus, the optimal treatment for patients with HOCM refractory to medical therapy is still unknown (19,20).

Several factors including age and risk of treatment modality might influence the clinical decision making when choosing the optimal treatment to relieve LVOT obstruction in patients with HOCM who have failed to respond to medical therapy. It is essential to understand the outcome of these treatments and which candidates are best suited for these two therapeutic modalities. We did not feel that alcohol ablation was ready for randomization at the time of this study or in its developmental stage. Therefore, the purpose of this nonrandomized cohort study is to assess the subjective and objective outcomes after PTMSA and surgical septal myectomy for the treatment of LVOT obstruction in a single center's population with HOCM.

METHODS

Patients. The study group consisted of 51 symptomatic patients with HOCM who underwent septal myectomy ($n = 26$, mean age 48 ± 13 years, men = 16) or PTMSA ($n = 25$, mean age 63 ± 14 years, men = 7) and had three months follow-up at the Cleveland Clinic Foundation from March 1997 to November 1999. During the same time, a total of 294 patients with hypertrophic cardiomyopathy were seen, and approximately half of the patients required an invasive therapeutic procedure (121 patients for myectomy and 37 patients for PTMSA). All patients had baseline echocardiography, immediately after the procedures and at three months. Most of our patients who underwent PTMSA did not require transesophageal echocardiography (TEE) to evaluate the mitral valve. Only if suitable images could not be obtained was TEE performed. One patient required a TEE before deciding about the suitability of PTMSA because of poor image quality, and this patient had a TEE during the PTMSA so that the septum could be properly imaged. The diagnosis of HOCM

was based on the presence of a hypertrophied, nondilated LV. Other diseases capable of producing significant hypertrophy and systolic anterior motion of the mitral valve were absent in these patients. In addition, each patient had clinical symptoms characteristic of severe HOCM, such as syncope in 12 patients (24%), angina in 22 patients (42%) or dyspnea in 40 patients (78%) of at least New York Heart Association (NYHA) functional class III. Patients with resting or provokable LVOT gradient ≥ 50 mm Hg and refractory to medical treatment were considered candidates for these therapies (18,21). Percutaneous transluminal septal myocardial ablation was usually recommended in patients who were elderly and had other comorbid conditions that could increase the risk of surgical therapy. When there was felt to be a need for concomitant valvular surgery or if a patient had a severe coronary artery disease, they were excluded for the PTMSA. All patients gave informed consent before these respective procedures.

Septal myectomy surgery. Standard cardiopulmonary bypass and myocardial preservation techniques were used (7-9). After aortic cross clamping and aortotomy, a portion of the septal muscle was excised. Then, another TEE was performed. If there was no LVOT PG, isoproterenol was infused, and the TEE was repeated to check for the elimination of LVOT PG and for the reduction or elimination of mitral regurgitation before closure of the chest.

PTMSA. In brief, two catheters were placed in the LV and ascending aorta to record simultaneous pressures and measure the PG across LVOT. Next, a balloon catheter was introduced into the first septal branch of the left anterior descending coronary artery. After the balloon was inflated, the distribution of the first septal branch was verified by contrast two-dimensional echocardiography after the injection of an echo contrast agent (18), Optison (Mallinckrodt Inc., San Diego, California) 0.5 to 1 ml diluted 1 to 20, through the catheter. If the first septal branch did not supply the area of SAM septal contact, the second branch was selected and retested. After confirming which territory of the basal septum contributed to the LVOT obstruction and no the other myocardial territory was involved, 1 to 3 ml (average 2.7 ± 1.0 ml) of alcohol was infused through the catheter with the balloon inflated and maintained for 10 min. The LVOT PGs were measured before and immediately after the procedures by catheter and by two-dimensional echocardiography.

Echocardiography. For both myectomy and PTMSA patient groups, conventional two-dimensional echocardiography and Doppler echocardiography was performed using a Vingmed System Five (GE/Vingmed, Milwaukee, Wisconsin) or a Sequoia 512 (Acuson, Mountain View, California) before, immediately after the procedures and at follow-up. All images were recorded on one-half inch VHS videotapes with selected images and loops also stored on magneto-optical disks in the DICOM format. Thickness of basal IVS and posterior wall (PW) and diameters of the LV and left atrium were measured using a standard M-mode echocar-

diagram, according to the recommendations of the American Society of Echocardiography. The ratio of IVS/PW was calculated, and LV ejection fraction was assessed (22). The degree of SAM of the mitral valve was divided into three grades as follows: 1) mild SAM was defined as the minimal mitral-septal distance ≥ 10 mm, 2) moderate SAM, when the distance was < 10 mm with only brief mitral-septal contact ($< 30\%$ of systole), and 3) severe SAM, when there was prolonged mitral-septal contact ($\geq 30\%$ of systole) (23). Mitral regurgitation was assessed by color Doppler and quantified by the flow convergence method (24). Continuous-wave Doppler technique was used to measure maximum velocity across LVOT with apical five- or three-chamber view. Then, peak PG through LVOT was estimated using the simplified Bernoulli equation (25). In patients with resting PG < 50 mm Hg, amyl nitrite inhalation was performed to check the presence of a provokable gradient.

Criteria for evaluation of success. For both myectomy and PTSMA patients, the procedures were considered successful when resting PG was less than 16 mm Hg or when the percentage of PG reduction was more than 50% immediately after procedures and during follow-up (13,16,26-28). The percentage (%) of PG reduction was calculated as:

$$\frac{(\text{PG before procedure} - \text{PG after procedure})}{\text{PG before procedure}} \times 100\%$$

Follow-up study. After discharge, patients underwent a comprehensive clinical evaluation, including assessment of NYHA functional class, by a cardiologist at a mean follow-up of 128 ± 84 days for myectomy and 117 ± 36 days for PTSMA ($p = \text{NS}$).

Statistical analysis. Data are presented as mean \pm SD. Analysis of variance with repeated measures was used to compare the values before and after the procedures and during follow-up. Due to the small sample size of the groups compared, paired Student *t* test was used (pre vs. post, pre vs. follow-up and post vs. follow-up). To adjust for the multiple comparisons being made, a *p* value < 0.01 was considered statistically significant. Chi-square tests were used to assess differences in success rates and complications between the two groups.

RESULTS

Characteristics of patients before procedures. There were 25 patients in the PTSMA group and 26 patients in the myectomy group. More women were included in the PTSMA group ($n = 18$) than in the myectomy group ($n = 10$) ($p = 0.02$). The patients in the PTSMA group were significantly older (63 ± 14 years, range: 39 to 85 years) than those in the myectomy group (48 ± 13 years, range: 30 to 70 years; $p < 0.001$). The patients receiving PTSMA were older and tended to have more comorbid conditions that might increase the risk for surgery than those found in

Table 1. The Comorbid Conditions in Two Groups

	PTSMA (n = 25)	Myectomy (n = 26)	p Value
Patients with comorbid conditions*	18	10	0.02
Congestive heart failure	8	3	NS
Coronary artery disease	3	5	NS
Peripheral vascular disease	2	0	NS
Azotemia	4	1	NS
Hypertension	10	3	0.02
Diabetes	5	2	NS
Chronic obstructive pulmonary disease	3	1	NS
History of stroke	1	0	NS

*Comorbid condition is defined as one or multiple conditions described below in the list.

PTSMA = percutaneous transluminal septal myocardial ablation.

patients receiving myectomy (18 [72%] vs. 10 [38%], $p = 0.02$) (Table 1). At the time of the procedures, 58% of patients took beta-blocking agents, 30% of patients took calcium channel blockers and 12% of patients took both medications.

Echocardiographic parameters. There was no significant difference in IVS thickness between the patients receiving PTSMA and the patients receiving myectomy before the procedures (Table 2). The IVS thickness was significantly decreased in both groups at follow-up (2.3 ± 0.4 cm vs. 1.9 ± 0.4 cm for PTSMA, 2.4 ± 0.6 cm vs. 1.7 ± 0.2 cm for myectomy, both $p < 0.001$). The end-diastolic diameter of the LV in the myectomy group was larger than that in the PTSMA group before procedure ($p = 0.03$). The end-diastolic diameter of the LV was significantly increased after PTSMA at follow-up ($p = 0.01$) and tended to increase after myectomy ($p = 0.6$) (Table 2). For mitral regurgitant volume and the degree of SAM, no significant difference was found between the two groups before procedures, and there was no difference at follow-up (Table 2). Mitral regurgitant volumes and the degree of SAM in both groups were significantly decreased during follow-up, as compared with those before procedures.

LVOT pressure gradients after procedures. PTSMA. The average resting PG of 25 patients decreased from $64 \pm$

Table 2. Echocardiographic Parameters Before and After Procedures

	PTSMA		Myectomy	
	Baseline	Follow-Up	Baseline	Follow-Up
LA (cm)	4.7 ± 0.8	4.6 ± 0.8	5.0 ± 0.7	4.7 ± 0.7
LVID (cm)	4.0 ± 0.8	$4.4 \pm 0.8^*$	$4.4 \pm 0.7^\ddagger$	4.5 ± 0.6
IVS (cm)	2.3 ± 0.4	$1.9 \pm 0.4^\ddagger$	2.4 ± 0.6	$1.7 \pm 0.2^\ddagger$
PW (cm)	1.5 ± 0.4	1.4 ± 0.3	1.4 ± 0.5	1.4 ± 0.2
EF (%)	62 ± 5	60 ± 6	63 ± 7	$58 \pm 5^*$
MR (ml)	23 ± 12	$9 \pm 3^\ddagger$	28 ± 12	$8 \pm 5^*$
SAM	1.4 ± 1.1	$0.6 \pm 0.8^\ddagger$	1.1 ± 0.6	$0.6 \pm 0.5^\ddagger$

* $p < 0.01$; $^\ddagger p < 0.001$, comparison with those before procedures; ‡ comparison with ablation, $p = 0.03$.

EF = left ventricular ejection fraction; IVS = thickness of interventricular septum; LA = diameter of left atrium; LVID = end-diastolic diameter of left ventricle; MR = mitral regurgitant volume; PTSMA = percutaneous transluminal septal myocardial ablation; PW = thickness of posterior wall of left ventricle; SAM = degree of systolic anterior motion of mitral valve.

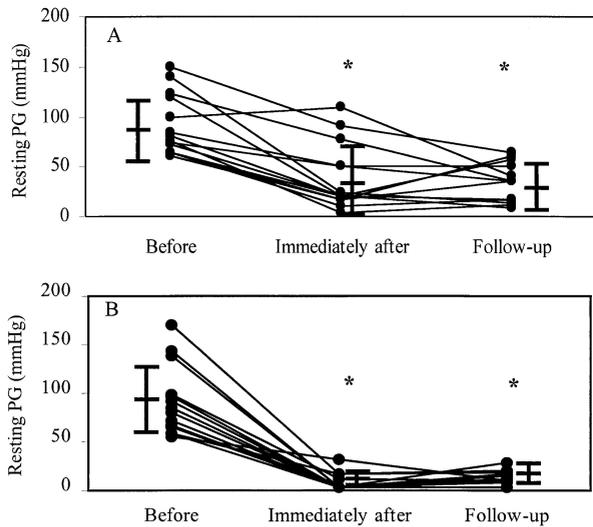


Figure 1. Resting pressure gradients (PG) before and immediately after percutaneous transluminal septal myocardial ablation (A) or myectomy (B) and at three-month follow-up in patients with resting PG ≥ 50 mm Hg. *in comparison with PGs before procedure, $p < 0.0001$.

39 mm Hg to 28 ± 29 mm Hg ($p < 0.0001$) after PTSMA and to 24 ± 19 mm Hg ($p < 0.0001$) at follow-up. Of these 25 patients, 12 had a post-PTSMA PG < 16 mm Hg, and five had > 16 mm Hg but $> 50\%$ reduction in PG. At follow-up, 13 patients had PG < 16 mm Hg, and nine patients had > 16 mm Hg but $> 50\%$ reduction in PG. Thus, between the post-procedure period and follow-up, an additional five patients had further reduction in their gradients. At three-month follow-up, three patients were considered a failure.

We also analyzed 14 patients with resting PG ≥ 50 mm Hg and 11 patients with resting PG < 50 mm Hg, separately. Left ventricular outflow tract PG decreased from 91 ± 30 mm Hg to 38 ± 33 mm Hg after PTSMA and to 33 ± 20 mm Hg at follow-up (vs. before PTSMA, both $p < 0.0001$) in the high PG group (Fig. 1A). In 11 patients with lower PG, the amyl nitrite induced provokable gradient decreased from 92 ± 30 mm Hg to 44 ± 22 mm Hg after PTSMA ($p < 0.001$) and to 55 ± 41 mm Hg at follow-up ($p = 0.03$) (Fig. 2A).

In the late follow-up, one patient had the PTSMA redone 15 months after initial PTSMA, and five others underwent myectomy for the elimination of the remaining obstruction because of continued symptoms. A permanent pacemaker was implanted in all five myectomy patients because complete atrioventricular block developed.

MYECTOMY. The average resting PG of 26 patients decreased from 62 ± 43 mm Hg to 7 ± 7 mm Hg ($p < 0.0001$) after myectomy and to 11 ± 6 mm Hg ($p < 0.0001$) at follow-up. Of these 26 patients, 25 had a postmyectomy PG < 16 mm Hg. One patient had a postmyectomy PG > 16 mm Hg and with $> 50\%$ reduction in PG. At follow-up, 21 patients had PG < 16 mm Hg, and

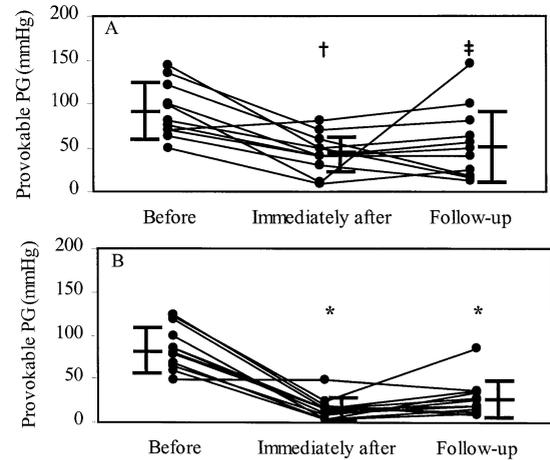


Figure 2. Provokable pressure gradients (PG) before and immediately after percutaneous transluminal septal myocardial ablation (A) or myectomy (B) and at three-month follow-up in patients with resting PG < 50 mm Hg. In comparison with PGs before procedure, * $p < 0.0001$; † $p < 0.001$; ‡ $p = 0.03$.

five patients had resting PG between 16 and 50 mm Hg. Thus, between the postprocedure period and follow-up, four patients had resting PG increase slightly, but it still remained significantly lower as compared with that before the procedure.

We also analyzed the 15 patients with resting PG ≥ 50 mm Hg and 11 patients with resting PG < 50 mm Hg. Left ventricular outflow tract PG decreased from 91 ± 35 mm Hg to 8 ± 8 mm Hg immediately after myectomy and to 13 ± 7 mm Hg at follow-up (both $p < 0.0001$) in the high PG group (Fig. 1B). In 11 patients with lower resting PG, the provokable gradient decreased from 86 ± 26 mm Hg (induced by amyl nitrite) to 16 ± 13 mm Hg (induced by infusion of isoproterenol) in the operating room after myectomy ($p < 0.0001$) and to 28 ± 22 mm Hg (induced by amyl nitrite) at follow-up ($p < 0.0001$) (Fig. 2B).

Comparison of both therapies for reduction of PG. Before the procedure, there was no significant difference for resting PG between the two groups (Fig. 3). However, the resting PGs were significantly lower in the myectomy group than they were in the PTSMA group immediately after the procedure and during follow-up. In 21 (81%) patients

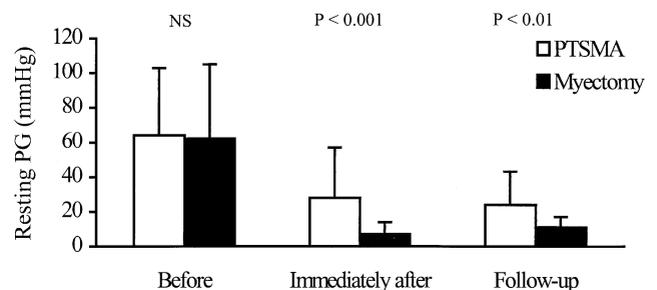


Figure 3. Comparison of average pressure gradients (PG) between percutaneous transluminal septal myocardial ablation (PTSMA) and myectomy group.

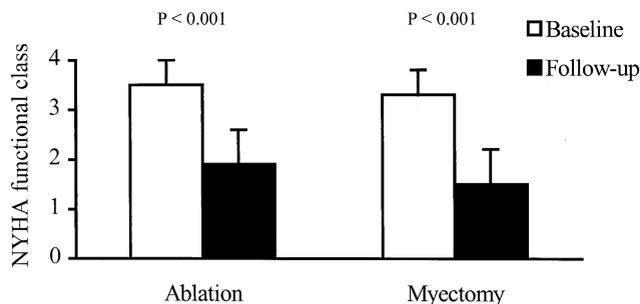


Figure 4. Improvements in New York Heart Association (NYHA) functional class after percutaneous transluminal septal myocardial ablation and myectomy in patients with hypertrophic obstructive cardiomyopathy.

receiving myectomy, the PG was completely eliminated, while only 13 (52%) PTSMA patients showed a resting PG <16 mm Hg at follow-up time ($p = 0.03$). Twenty-six (100%) patients in the myectomy group and 19 (76%) patients in the PTSMA group also showed a reduction of PG >50% ($p < 0.01$) at that time.

NYHA functional class. Before the procedures, each patient's NYHA functional class was \geq III (Fig. 4). Average NYHA functional class was 3.5 ± 0.5 for ablation patients and 3.3 ± 0.5 for myectomy patients ($p = \text{NS}$). All patients felt better after the procedure in both groups except one PTSMA patient. Average NYHA functional class was significantly improved in both PTSMA patients (1.9 ± 0.7 , $p < 0.001$) and myectomy patients (1.5 ± 0.7 , $p < 0.001$) after the procedure. There was no significant difference for NYHA functional class between the two groups during follow-up ($p = \text{NS}$).

Untoward effects of both procedures. There were no deaths in either myectomy or PTSMA patients within three months after the procedures. Eleven patients had conduction system block after PTSMA (complete right bundle branch block in nine and complete left bundle branch block in two), while 16 patients had complete left bundle branch block after myectomy, $p > 0.05$. Permanent pacemakers were required in six patients (24%) in the PTSMA group and in two patients (7.7%) in the myectomy group, respectively ($p > 0.05$).

Hospital stay. The recovery after the procedure was much quicker in the PTSMA patients than it was in the myectomy patients (mean hospital stay: 5.6 ± 2.3 days vs. 8.1 ± 3.5 days, $p = 0.01$).

DISCUSSION

In this study, we found that PTSMA and myectomy could reduce the hypertrophied septum, decrease LVOT obstruction and improve the NYHA functional class and maintain these effects for three months. The reduction of LVOT PG was more complete in patients receiving myectomy, while the hospital stay was shorter in patients receiving PTSMA. In our opinion, PTSMA may be more desirable for older patients with HOCM with comorbid conditions.

Advantages and disadvantages of myectomy surgery. The results of myectomy for patients with HOCM have significantly improved in the recent four decades (5-9). About 70% of patients with HOCM have reported substantial and persistent symptomatic improvement for five or more years after myectomy surgery (8,9,21). In more than 90% of patients with HOCM, resting LVOT gradients were completely eliminated or greatly reduced (8,9,21). However, myectomies are currently performed with experienced hands only at selected centers in the world (9,29). Previously, the surgical mortality appeared to be higher (10% to 17%) among elderly patients and those with concomitant diseases (30,31). Review of our myectomy experience reveals the hospital mortality is 1.0% (2 deaths of 194 patients) for all HOCM surgeries between 1994 and 1999, and the mortality reduces to zero for pure myectomy between 1997 and 1999. A group in Germany reported their operated mortalities were <2% (6).

Benefits and drawbacks of PTSMA. The short-term results of PTSMA are excellent for the reduction of LVOT obstruction (13,17,32). Previous studies report that mean resting PGs significantly reduced from 60 mm Hg to 14 mm Hg after PTSMA (13,17,32), and other studies with longer follow-up (16,18,33) do not show any recurrence of obstruction over time. One- to two-year follow-up shows continued improvement in symptoms with a mean increase of over one NYHA functional class (16,18,33). More importantly, objective tests show increases of exercise time around 40% over follow-up (18). Recently, Lakkis et al. (18) reported results of a one-year follow-up study in 50 patients. In his study, resting PGs dropped significantly from average 74 ± 23 mm Hg to 6 ± 18 mm Hg, and dobutamine-induced gradient decreased from 84 ± 28 mm Hg to 30 ± 33 mm Hg. The exercise duration increased by 2 min at one year. Similar results were also found in our study, with both resting and provokable PGs significantly reduced immediately after the PTSMA and remaining lower after three months.

Although PTSMA completely eliminated LVOT PGs in most patients with HOCM, significant resting or provokable obstruction may remain in some others. There were seven patients (7/50, 14%) in Lakkis' et al. (18) study who needed to have the procedure redone within one year after the initial ablation. There was also some significant scatter from the mean, as shown in our figures. This scatter was wider for the PTSMA group at follow-up. Left ventricular outflow tract obstruction is not only caused by hypertrophy of IVS but also by anterior movement of mitral valve leaflets. Percutaneous transluminal septal myocardial ablation will only cause reduction of the septum. If there is elongation of the anterior or posterior leaflet of the mitral valve, PTSMA will not be sufficient to relieve the outflow tract obstruction. In addition, recent data in abstract form from Faber et al. (34) suggests that patients with "thicker septums" obtain less success in reducing the gradient. One patient in our study who underwent PTSMA initially had

no gradient reduction because of an elongated anterior leaflet. When she underwent surgery, mitral valve repair was needed in addition to myectomy to completely abolish her gradient. Five PTSMA patients in our study subsequently underwent myectomy to eliminate the obstruction (one due to dissection of ascending aorta late after PTSMA, one due to elongation of mitral valve leaflets and cause for remaining three patients is unclear).

The overall requirement for pacing is 21%, varying from 0% to 40% among different reports, and the mortality is 2% for PTSMA (13,15,17,26,33). A total of 24% of PTSMA patients in our study needed permanent pacemakers after the procedure, while 7.7% patients needed them after myectomy. However, if myectomy is required after a failed PTSMA, there is a high likelihood that the patient will require a permanent pacemaker. The right bundle is usually supplied by the proximal septal perforators, and, thus, PTSMA frequently leads to complete right bundle branch block, and septal myectomy causes complete left bundle branch block in a majority of patients. So if a patient has both procedures, it is quite likely that complete heart block will develop. Despite the high incidence of permanent pacemaker insertion when both procedures are required, subsequent myectomy is not difficult.

In comparison with myectomy, however, PTSMA obviously has distinct advantages. Percutaneous transluminal septal myocardial ablation is a less invasive technique and has shorter hospital stay. Therefore, both physicians and patients may prefer PTSMA rather than myectomy in the treatment of HOCM as an initial nonpharmacologic intervention. Percutaneous transluminal septal myocardial ablation is an exciting alternative therapeutic strategy for older patients with HOCM with concomitant disease or higher-risk surgery. However, some patients do not derive enough relief immediately from the PTSMA, and months are required for reduction in the gradient, a potential disadvantage of the procedure for some patients. In the last five years, more than 1,000 patients with HOCM have undergone PTSMA worldwide (35). In addition, if the PTSMA fails, myectomy can still be performed, but a permanent pacemaker is almost always needed. In our experience, all five patients who underwent both procedures had permanent pacemakers implanted when both procedures were required. **Study limitations.** Our follow-up is relatively short in this nonrandomized study, and the longer-term results are unknown at this point. The groups are dissimilar in several respects. There were no criteria for dividing patients into PTSMA and myectomy groups. The general, accepted patient selection criteria for PTSMA and myectomy is based on marked LVOT PG under basal conditions (LVOT PG \geq 50 mm Hg) or a provokable PG $>$ 50 mm Hg and severe symptoms (NYHA functional class III or IV) that fail to cease with medication (21). In this study, the procedures were chosen based on patient and physician preference. However, if the patient was elderly or had significant comorbid conditions, PTSMA was strongly

suggested. Thus, in our study elderly patients more often had PTSMA while younger patients had myectomy. Similar reduction in LVOT obstruction by PTSMA occurred in younger patients, as compared with myectomy, in the latest study by Nagueh et al. (36) when patient's age and LVOT PG were matched. However, for elderly patients, especially with concomitant disease, the less aggressive and less invasive approach of PTSMA may be a better, or the only, choice, even with incomplete elimination of the LVOT obstruction.

CONCLUSIONS

Both myectomy and PTSMA reduce LVOT obstruction and improve NYHA functional class significantly in patients with HOCM. However, there are benefits and drawbacks for each therapeutic method that must be counterbalanced when deciding on treatment for LVOT obstruction.

Acknowledgments

The authors thank Mr. David Tollon, BS, for his careful editorial assistance and Ms. Penny L. Houghtaling, MS, for her statistical assistance.

Reprint requests and correspondence: Dr. Harry M. Lever, Department of Cardiology-Desk F15, Cleveland Clinic Foundation, 9500 Euclid Avenue, Cleveland, Ohio 44195. E-mail: leverh@ccf.org.

REFERENCES

1. Wigle ED, Rakowski H, Kimball BP, Williams WG. Hypertrophic cardiomyopathy: clinical spectrum and treatment. *Circulation* 1995; 92:1680-92.
2. Wigle ED, Sasson Z, Henderson MA, et al. Hypertrophic cardiomyopathy: a review. *Prog Cardiovasc Dis* 1985;28:1-83.
3. Wigle ED. Novel insights into the clinical manifestations and treatment of hypertrophic cardiomyopathy. *Curr Opin Cardiol* 1995;10: 299-305.
4. Maron BJ. Hypertrophic cardiomyopathy. *Lancet* 1997;350:127-33.
5. Merrill WH, Friesinger GC, Graham TP, Jr, et al. Long-lasting improvement after septal myectomy for hypertrophic obstructive cardiomyopathy. *Ann Thorac Surg* 2000;69:1732-5.
6. Schulte HD, Borisov K, Gams E, Gramsch-Zabel H, Losse B, Schwartzkopff B. Management of symptomatic hypertrophic obstructive cardiomyopathy—long-term results after surgical therapy. *Thorac Cardiovasc Surg* 1999;47:213-8.
7. McCully RB, Nishimura RA, Bailey KR, Schaff HV, Danielson GK, Tajik AJ. Hypertrophic obstructive cardiomyopathy: preoperative echocardiographic predictors of outcome after septal myectomy. *J Am Coll Cardiol* 1996;27:1491-6.
8. Spirito P, Seidman CE, McKenna WJ, Maron BJ. The management of hypertrophic cardiomyopathy. *N Engl J Med* 1997;336:775-85.
9. Heric BLB, Miller DP, Rosenkranz ER, Lever HM, Cosgrove DM. Surgical management of hypertrophic obstructive cardiomyopathy: early and late results. *J Thorac Cardiovasc Surg* 1995;110:195-206.
10. Ommen SR, Nishimura RA, Squires RW, Schaff HV, Danielson GK, Tajik AJ. Comparison of dual-chamber pacing versus septal myectomy for the treatment of patients with hypertrophic obstructive cardiomyopathy: a comparison of objective hemodynamic and exercise end points. *J Am Coll Cardiol* 1999;34:191-6.
11. Erwin JP, III, Nishimura RA, Lloyd MA, Tajik AJ. Dual chamber pacing for patients with hypertrophic obstructive cardiomyopathy: a clinical perspective in 2000. *Mayo Clin Proc* 2000;75:173-80.

12. Seggewiss H. Percutaneous transluminal septal myocardial ablation: a new treatment for hypertrophic obstructive cardiomyopathy. *Eur Heart J* 2000;21:704-7.
13. Seggewiss H, Gleichmann U, Faber L, Fassbender D, Schmidt HK, Strick S. Percutaneous transluminal septal myocardial ablation in hypertrophic obstructive cardiomyopathy: acute results and 3-month follow-up in 25 patients. *J Am Coll Cardiol* 1998;31:252-8.
14. Sigwart U. Non-surgical myocardial reduction for hypertrophic obstructive cardiomyopathy. *Lancet* 1995;346:211-4.
15. Knight C, Kurbaan AS, Seggewiss H, et al. Nonsurgical septal reduction for hypertrophic obstructive cardiomyopathy: outcome in the first series of patients. *Circulation* 1997;95:2075-81.
16. Faber L, Meissner A, Ziemssen P, Seggewiss H. Percutaneous transluminal septal myocardial ablation for hypertrophic obstructive cardiomyopathy: long-term follow up of the first series of 25 patients. *Heart* 2000;83:326-31.
17. Faber L, Seggewiss H, Gleichmann U. Percutaneous transluminal septal myocardial ablation in hypertrophic obstructive cardiomyopathy: results with respect to intraprocedural myocardial contrast echocardiography. *Circulation* 1998;98:2415-21.
18. Lakkis NM, Nagueh SF, Dunn JK, Killip D, Spencer WH. Nonsurgical septal reduction therapy for hypertrophic obstructive cardiomyopathy: one-year follow-up. *J Am Coll Cardiol* 2000;36:852-5.
19. Rubin DN, Tuzcu EM, Lever HM. Percutaneous transluminal septal myocardial ablation. *Curr Cardiol Rep* 2000;2:160-5.
20. Fananapazir L, McAreavey D. Therapeutic options in patients with obstructive hypertrophic cardiomyopathy and severe drug-refractory symptoms. *J Am Coll Cardiol* 1998;31:259-64.
21. Spirito P, Maron BJ. Perspectives on the role of new treatment strategies in hypertrophic obstructive cardiomyopathy. *J Am Coll Cardiol* 1999;33:1071-5.
22. Sahn DJ, DeMaria A, Kisslo J, Weyman A. Recommendations regarding quantitation in M-mode echocardiography: results of a survey of echocardiographic measurements. *Circulation* 1978;58:1072-83.
23. Gilbert BW, Pollick C, Adelman AG, Wigle ED. Hypertrophic cardiomyopathy: subclassification by M-mode echocardiography. *Am J Cardiol* 1980;45:861-72.
24. Shiota T, Jones M, Teien DE, et al. Evaluation of mitral regurgitation using a digitally determined color Doppler flow convergence "centerline" acceleration method: studies in an animal model with quantified mitral regurgitation. *Circulation* 1994;89:2879-87.
25. Sasson Z, Yock PG, Hatle LK, Alderman EL, Popp RL. Doppler echocardiographic determination of the pressure gradient in hypertrophic cardiomyopathy. *J Am Coll Cardiol* 1988;11:752-6.
26. Lakkis NM, Nagueh SF, Kleiman NS, et al. Echocardiography-guided ethanol septal reduction for hypertrophic obstructive cardiomyopathy. *Circulation* 1998;98:1750-5.
27. Nagueh SF, Lakkis NM, Middleton KJ, Spencer WH, III, Zoghbi WA, Quinones MA. Doppler estimation of left ventricular filling pressures in patients with hypertrophic cardiomyopathy. *Circulation* 1999;99:254-61.
28. Seggewiss H, Faber L, Gleichmann U. Percutaneous transluminal septal ablation in hypertrophic obstructive cardiomyopathy. *Thorac Cardiovasc Surg* 1999;47:94-100.
29. ten Berg JM, van Tooren R, Jaarsma W, Suttrop MJ, Plokker HW. Therapeutic options in patients with obstructive hypertrophic cardiomyopathy and severe drug-refractory symptoms. *J Am Coll Cardiol* 1998;32:1485.
30. Robbins RC, Stinson EB. Long-term results of left ventricular myotomy and myectomy for obstructive hypertrophic cardiomyopathy. *J Thorac Cardiovasc Surg* 1996;111:586-94.
31. Cooper MM, McIntosh CL, Tucker E, Clark RE. Operation for hypertrophic subaortic stenosis in the aged. *Ann Thorac Surg* 1987;44:370-8.
32. Knight C, Sigwart U. Non-surgical ablation of the ventricular septum for the treatment of hypertrophic cardiomyopathy. *Heart* 1996;76:92.
33. Gietzen FH, Leuner CJ, Raute-Kreinsen U, et al. Acute and long-term results after Transcoronary Ablation of Septal Hypertrophy (TASH). Catheter interventional treatment for hypertrophic obstructive cardiomyopathy. *Eur Heart J* 1999;20:1342-54.
34. Faber L, Werlemann B, Krater L, Seggewiss H, Horstkotte D. Septal ablation for hypertrophic obstructive cardiomyopathy: an analysis of the patients with dissatisfactory reduction of the outflow gradient (abstr). *J Am Coll Cardiol* 2001;37:200A.
35. Kuhn H, Gietzen FH, Leuner C, et al. Transcoronary Ablation of Septal Hypertrophy (TASH): a new treatment option for hypertrophic obstructive cardiomyopathy. *Z Kardiol* 2000;89Suppl 4:41-54.
36. Nagueh SF, Ommen SR, Lakkis NM, et al. Comparison of ethanol septal reduction therapy with surgical myectomy for the treatment of hypertrophic obstructive cardiomyopathy (abstr). *J Am Coll Cardiol* 2001;37:200A.