SURGICAL MANAGEMENT OF HYPERTROPHIC OBSTRUCTIVE CARDIOMYOPATHY

Early and late results

From 1975 through 1993, 178 patients underwent surgical management of hypertrophic obstructive cardiomyopathy. Operations included isolated septal myectomy (n = 95), septal myectomy and coronary artery bypass grafting (n = 41), septal myectomy plus a valve procedure (n = 25), septal myectomy, valve procedure, and coronary artery bypass grafting (n = 14), and mitral valve replacement without septal myectomy (n = 3). Recent myectomy results were monitored with transesophageal echocardiography. After initial myectomy, 32 patients (20%) underwent a second pump run for more extensive myectomy only (n = 22), mitral valve replacement only (n = 5), or both (n = 2). In-hospital mortality was 6% (n = 11) and 4% (n = 6) for patients undergoing septal myectomy or septal myectomy plus coronary artery bypass grafting, respectively. Heart block occurred in 17 patients (10%). Left ventricular outflow tract systolic gradients decreased from a mean of 93 mm Hg to 21 mm Hg after myectomy. Late survival was 86% and 70% at 5 and 10 postoperative years, respectively, and 93% and 79% for patients undergoing septal myectomy alone or septal myectomy plus coronary artery bypass grafting, respectively. Only 3 of 131 in-hospital survivors of septal myectomy or septal myectomy plus coronary artery bypass grafting died late cardiac deaths, for a yearly mortality of 0.6%. However, the 5-year late survival of patients undergoing valve operation plus septal myectomy was 51%, and multivariate testing confirmed the adverse influence on late survival (p = 0.008), as well as adverse influences of increasing age (p = 0.016) and return to cardiopulmonary bypass for mitral valve replacement (p = 0.038). At follow-up 136 patients (94%) had New York Heart Association class I or II symptoms. For patients with hypertrophic obstructive cardiomyopathy, septal myectomy alone or in combination with coronary artery bypass grafting produces effective symptom relief, excellent long-term survival, and a low risk of late cardiac death. (J THORAC CARDIOVASC SURG 1995;110:195-208)

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Hypertrophic obstructive cardiomyopathy is a condition of unknown origin characterized by septal hypertrophy and associated dynamic, variable subaortic obstruction in the left ventricular (LV) outflow tract. This obstruction is associated with abnormal systolic motion of the anterior mitral leaflet. Treatment of patients with symptomatic conditions includes beta and calcium channel blockers,[1-4] antiarrhythmics,[5, 6] and, recently, dual chamber pacing.[7-8] Surgical myectomy to relieve outflow obstruction is reserved for patients who have significantly symptomatic conditions while receiving medical therapy, those with associated surgical cardiac disease, and younger patients with mildly symptomatic conditions and a history of syncope.

Transaortic septal myectomy[9] has been the primary surgical approach at our institution over the last 18 years. Our increasing experience with this technique and emerging controversy[10] over the role

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Table I. *Preoperative and postoperative gradient, MR, and SAM*

	Preop. (n)	*Postop. (n)*
Maximum gradient (mm Hg)		
0–25	4	97
26–50	7	30
51–75	39	2
76–100	74	3
101–200	46	1
Total	170	133
MR		
None	15	
1+	36	
2+	43	
3+	48	
4+	12	
Total	154	
SAM		
None	13	67
Mild	19	46
Moderate	75	6
Severe	24	2
Total	131	121

MR, Mitral regurgitation; *SAM,* systolic anterior motion; *Preop.,* preoperative; *Postop.,* postoperative.

of surgical myectomy and dual chamber pacing therapy prompted us to review our early and late results.

Patients and methods

A consecutive series of 178 patients undergoing surgical treatment of hypertrophic cardiomyopathy, primarily septal myectomy alone, or myectomy in combination with a valve or coronary procedure at the Cleveland Clinic Foundation from 1975 through 1993 was reviewed. Over half of the procedures were performed over the last 3 years of this period. Patients with congenital subaortic membrane were excluded. Clinical information was derived from a retrospective individual chart review and the Cleveland Clinic Cardiovascular Information Registry.

One half (n = 89) of our patient population was male, and 70% were in their sixth to ninth decade of life. Eight percent (n = 11) had a documented family history of hypertrophic cardiomyopathy. Ninety percent of the patient population (n = 165) had normal or mildly impaired left ventricular function, and 36% (n = 58) had significant surgical coronary artery disease. Ten patients (6%) underwent reoperation, and four (3%) required emergency operative intervention.

Preoperative diagnosis was based on clinical history, echocardiographic examination, and cardiac catheterization. Preoperative medications included calcium channel blockers in 77 (61%), beta blockers in 78 (62%), amiodarone in seven (6%), and norpace in seven (6%). The most common symptoms were dyspnea in 103 patients, followed by angina (n = 87), syncope (n = 55), congestive heart failure (n = 39), atrial arrhythmias (n = 49), and ventricular arrhythmias (n = 11). Individual patients typically had multiple symptoms. Syncope includes patients with near-syncopal symptoms.

Preoperative gradients were determined at cardiac catheterization or at echocardiography and were determined with and without provocation. The preoperative and postoperative gradients and the degree of mitral regurgitation (MR) and systolic anterior motion of the anterior mitral leaflet are summarized in Table I (The gradient listed in Table I is the larger of either the gradient at rest or with provocation. When only one value was available, that gradient is listed.) The degree of postoperative MR was not consistently available on retrospective review and is not included in Table I.

Surgical technique. Standard cardiopulmonary bypass with systemic moderate hypothermia, LV venting, and either blood or crystalloid antegrade and/or retrograde cardioplegic solution was used. Since 1984 intraoperative echocardiography has guided transaortic resection of the region of septal hypertrophy. The echocardiographic techniques have been previously reported by our group[11] and others.[12, 13] After an oblique transverse aortotomy, the aortic cusps were retracted to expose the subvalvar hypertrophied septum. The involved septal area is beneath the right coronary cusp and is characterized by a thickened and fibrotic endocardial plaque from contact with the anterior leaflet of the mitral valve.[12] Two parallel incisions in the direction of the left ventricular apex are made encompassing this area of septal hypertrophy, and a muscle bar 1 cm in depth and 1 to 2 cm in width that extends 3 to 5 cm toward the apex is resected. The resection is extended toward the LV apex until the bases of the papillary muscles are visualized. This intraoperative visual localization is corroborated by intraoperative echocardiography, which allows precise localization of this area of septal hypertrophy. In particular the relation of the obstructing septum to the aortic valve anulus, the apical extent of hypertrophy and mitral valve septal contact, and maximal septal thickness are useful echocardiographic parameters in guiding precise surgical resection. On completion of the septal myectomy and any associated procedures, patients were weaned from cardiopulmonary bypass, and echocardiographic determination of the LV outflow tract (LVOT) gradient, with and without progressively higher doses of isoproterenol (Isuprel), was performed. The presence of an LVOT gradient greater than 50 mm Hg, with or without provocation, or greater than moderate MR was generally an indication for a return to bypass and either a more extensive myectomy or mitral valve replacement (MVR).

The operative procedures performed are listed in Table II. Seventy-six percent of patients underwent septal myectomy alone or in combination with coronary artery bypass grafting (CABG). Twenty-one patients underwent a mitral valve procedure during initial hospitalization, two of whom underwent MVR on reoperation. Of these 21 patients who underwent a mitral valve procedure during their initial hospitalization, 16 had an MR grade of 3 to 4+ before the operation. Ten of these patients underwent MVR at initial operation with and without associated septal myectomy. The origin of MR in this group was rheumatic in four patients, bacterial endocarditis in three,

Table II. *Operations performed (n = 178)*

Myectomy only	95 (54%)
Myectomy/CABG	41 (23%)
Myectomy/valve	25 (14%)
Mitral	9 (5%)
Aortic	12 (7%)
Mitral/Aortic	3 (2%)
Mitral Tricuspid	1 (1%)
Myectomy/CABG/valve	14 (8%)
Mitral	4 (2%)
Aortic	9 (5%)
Mitral/aortic	1 (1%)
Mitral valve replacement	3 (2%)
Mitral	2 (1%)
Mitral/aortic	1 (1%)

and cleft mitral valve, papillary fibroelastosis, and degenerative myxomatous mitral valve disease in the remaining three patients. Four patients underwent mitral valve repair. The origins of MR in these four patients included rheumatic, cleft posterior mitral leaflet, myxoma, and, in the fourth patient, the origin was unclear. The remaining seven patients underwent MVR because of the presence of a persistent postmyectomy gradient or significant MR. Six of these seven patients had normal mitral valves, and one had a myxomatous valve.

Clinical follow-up was obtained in 175 of 178 patients (98%) by direct telephone interview with individual patients and their referring physicians.

Statistical analysis. The mean period of follow-up was 44 months (standard deviation 3.9 months). Means and percentages were based on the set of nonmissing observations. Preoperative, intraoperative, and postoperative factors were considered as potential predictors of early and late death. Risk factors that were significant ($p < 0.05$) or nearly significant univariate predictors of death were also considered for inclusion in multivariate models of death. In-hospital mortality was analyzed for all patients, and late death was studied in the cohort of hospital survivors. Nonparametric survival curves were based on Kaplan-Meier estimates, and the p values for these curves were based on the log-rank test. Semi-parametric models of late death were based on a Cox regression analysis. Univariate and multivariate models of hospital death involved the logistic regression technique. Separate models including and excluding postoperative factors were used; p values based on Wald χ^2 statistics were used to evaluate significance in these models.

Results

Hospital mortality. Hospital mortality was 6% overall, 4% for patients undergoing myectomy alone, and 5% for those undergoing myectomy and CABG. Addition of a valve procedure to myectomy increased the mortality to 8%. Patients requiring a combined myectomy, CABG, and valve procedure had a mortality of 21%. Eight of the 11 hospital deaths occurred in female patients. The causes of in-hospital mortality and late deaths are outlined in Table III. Univariate logistic regression analysis of hospital deaths (Table IV) showed age and preoperative amiodarone usage to be significant predictors of in-hospital mortality. Hospital mortality was 10% in patients over 60 years of age compared with 1.3% in those under 60 years of age. Amiodarone usage increased mortality to 29% compared with

Table III. *Causes of hospital and late death*

		Late death (n = 18)	
	Hospital death (n = 11)	*Myectomy with or without CABG*	*Valve**
Cancer	0	4	1
Nondocumented (probable cardiac)	0	1	4
LV failure	3	1	1
CVA	3	0	1
Sudden cardiac death	1	1	1
Renal failure	0	0	2
Endocarditis	1	0	0
RV rupture	1	0	0
LV rupture	1	0	0
Bleeding	0	1	0
Sepsis	0	0	1
Multiorgan failure	1	0	0

CVA, Cerebrovascular accident; *RV,* right ventricular.

*Patients who underwent a valve procedure with or without associated myectomy or CABG.

Table IV. *Risk of hospital death*

Risk factor	*Odds ratio*	*p Value*	*Confidence interval*
Univariate logistic regression models of hospital death*			
Age†	1.9	0.029	1.1, 3.5
Amiodarone	7.2	0.029	1.2, 41.8
Functional class‡	2.8	0.054	1.0, 8.2
Valve operation	2.9	0.090	0.8, 10.1
Multivariate logistic regression model with only preoperative predictors			
Amiodarone	6.9	0.040	1.1, 43.2
Age†	1.9	0.043	1.0, 3.6
Valve operation	2.8	0.117	0.8, 10.5

*With preoperative and operative predictors of hospital death.

†Odds ratio for age represents increased odds of hospital death for every 10 years of age.

‡Functional class odds ratio represents the increased odds for each increased functional class level (I, II, III and IV).

Table V. *Postoperative complications**

Complete heart block		17
Return to operating room		22
Bleeding	13	
MVR	2	
Redo myectomy	1	
Redo myectomy/MVR	1	
LV rupture	2	
RV rupture	1	
Pericardial effusion	1	
Atrial arrhythmias		46
Ventricular arrhythmias		13
Respiratory		13
Renal		9
CVA		5
Wound		3

RV, Right ventricular; *CVA,* cerebrovascular accident.
*A total of 128 complications in 87 patients.

2% in untreated patients. New York Heart Association functional class ($p = 0.054$) and valve operation ($p = 0.090$) tended toward statistical significance. A multivariate logistic regression model of hospital deaths also showed age and amiodarone usage to be statistically significant preoperative predictors. Valve operation was less strongly associated with hospital death and not statistically significant.

Hospital morbidity. Postoperative complications are listed in Table V. The overall incidence of complete heart block was 10%. Of the 17 cases, 10 occurred in the isolated myectomy surgical group, and six developed in patients undergoing a concomitant valve procedure. One patient underwent deliberate surgical interruption of atrioventricular conduction to produce complete heart block and control malignant supraventricular arrhythmias. The incidence of complete heart block in the patient undergoing isolated myectomy requiring a second pump run was 30%, which is significantly higher ($p = 0.005$) than the 6% incidence in the myectomy group undergoing a single pump run. Four patients underwent five in-hospital reoperations which included MVR in two, redo myectomy and MVR in one, and isolated myectomy for a persistent residual gradient in the remaining patient.

Hemodynamics. The mean maximal preoperative LVOT gradient measured in 170 patients at rest or with provocation was 93 mm Hg and decreased to a mean of 21 mm Hg in the 133 patients in whom measurements were recorded. In the immediate postoperative period, six patients had persistent measured gradients greater than 50 mm Hg, eight had moderate to severe systolic anterior motion of the anterior mitral leaflet (SAM), and 17 had MR grade 2 to 3+. Statistical analysis failed to show any relationship between persistent gradient, MR, or SAM and hospital morbidity, mortality, late morbidity or mortality, or return to New York Heart Association functional class III or IV symptom status. Thirty-two patients returned to cardiopulmonary bypass at their initial operation because of either a persistent LVOT gradient or significant MR and SAM. Twenty-two patients underwent repeat myectomy, and five patients underwent MVR alone or in combination with myectomy. Of the 60 patients with MR grade 3 to 4+ on preoperative echocardiography, 16 underwent MVR. Five patients who required MVR did not have significant MR documented before the operation. Ten were performed as planned on the initial pump run. Five were performed on return to cardiopulmonary bypass because of persistent MR after myectomy, and two were performed at reoperation during the same hospitalization. No late MVRs were performed in patients with grade 3+ or 4+ MR noted before the operation.

Late deaths. Fig. 1 shows overall survival and event-free survival. Fig. 2 shows a late survival at 10 years of 79% for myectomy with or without CABG, compared with 26% when a valve procedure is added. The causes of late death are listed in Table III. Two patients undergoing myectomy, with or without concurrent CABG, experienced sudden death on long-term follow-up, and one died of progressive LV failure.

Predictors of late death with the use of univariate and multivariate Cox regression models are listed in Table VI. For age, the risk ratio rates represent the increased risk for every 10 years of age. For MR, the risk ratio represents the increased risk for each increased level of MR (1+, 2+, 3+, etc.). Strong predictors of late death included concomitant valve operation, increasing age, a history of syncope or near-syncope, complete heart block, and the performance of MVR when initial myectomy resulted in either a significant residual gradient or MR. Twelve of the 19 late deaths and two thirds of all deaths occurred in women.

Functional status/late events. Preoperative and follow-up functional class is shown in Table VII. Of the two patients who were in functional class 1 before the operation, one underwent "prophylactic" septal myectomy before a planned neurosurgical procedure, and the other was a young child with a large gradient and history of syncope. Nine patients

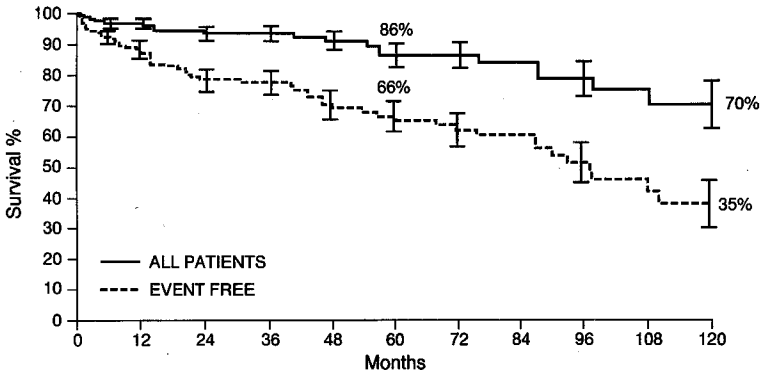

Fig. 1. Overall late survival and event-free survival for 167 in-hospital survivors. Late survival at 5 and 10 postoperative years was 86% and 70%. Event-free survival at 5 and 10 years was 66% and 35%. The numbers of patients remaining in the survival sample at 2, 4, 6, 8, and 10 years was 83, 53, 34, 18, and 6, respectively. The numbers of patients remaining in the event-free sample at 2, 4, 6, 8, and 10 years was 95, 63, 39, 24 and, 8, respectively. *Error bars* are ± standard deviation.

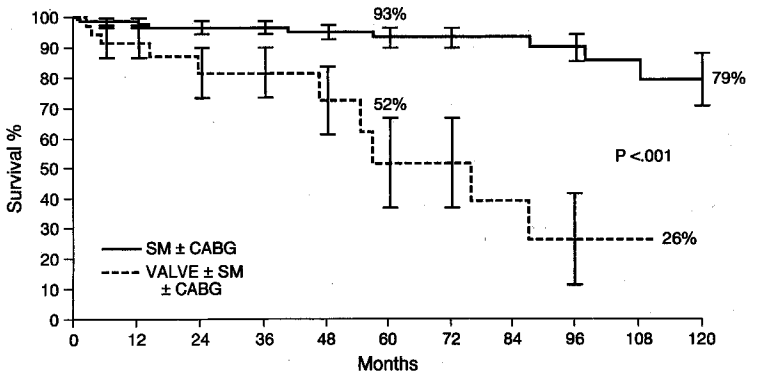

Fig. 2. Late survival by procedure of 167 in-hospital survivors. Survival of the septal myectomy *(SM)* with or without coronary artery bypass grafting *(CABG)* group at 5 and 10 years was 93% and 79%, respectively, compared with 51% and 26% in the group undergoing a valve procedure. The respective numbers of patients remaining in the sample for the myectomy with or without CABG group at 2, 4, 6, 8, and 10 years were 80, 15, 35, 22, and 8. The respective numbers remaining for the valve group at 2, 4, 6, 8, and 10 years were 15, 8, 4, 21, and 0. *Error bars* are ± standard deviation.

were in functional class III or IV on long-term follow-up. Univariate and multivariate analyses with preoperative, operative, and postoperative variables could not show any statistically significant predictors of this treatment failure group. The 108 late events in 75 patients are listed in Appendix A. Six patients underwent seven late reoperations that included CABG in two, and MVR/aortic valve replacement, tricuspid valve replacement, MVR, and ventricular septal defect repair in the remaining four patients.

Fig. 3 shows the statistically significant difference in event-free survival between those patients undergoing myectomy with or without CABG and those undergoing a concomitant valve procedure.

Discussion

Widespread use of two-dimensional echocardiography has led to increasing clinical recognition of the obstructive form of hypertrophic cardiomyopathy. This recognition has been reflected in our progressive experience with the surgical management of this morphologically diverse disease during the last 5 years. The variable clinical expression and natural history of hypertrophic obstructive cardiomyopathy and institution-specific referral patterns has complicated comparison of surgically treated patients with those treated medically. As a result, controversy remains over (1) the role of surgical myectomy compared with drug and pace-

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Table VI. *Risk of late death*

Risk factor	*Odds ratio*	*p Value*	*Confidence interval*
Univariate Cox regression models of late death			
Valve operation	6.6	<0.001	2.6, 16.6
RTB/MVR*	22.6	<0.001	6.5, 78.2
Age	2.3	0.001	1.4, 3.8
CHB	4.8	0.008	1.5, 15.6
Blood used	6.2	0.015	1.4, 26.9
Syncope	3.2	0.015	1.3, 13.5
MR	1.6	0.056	1.0, 2.4
CVA	7.3	0.057	0.9, 56.0
Multivariate model with preop., op., and postop. predictors			
CHB	9.5	0.001	2.6, 35.0
Valve operation	4.7	0.008	1.4, 15.3
Age	2.0	0.010	1.2, 3.1
Syncope	4.0	0.020	1.2, 12.6
RTB/MVR*	5.4	0.038	1.1, 26.8

RTB, Return to bypass; *CHB,* complete heart block; *CVA,* cerebrovascular accident; *preop.,* preoperative; *postop.,* postoperative.

*Patients who after initial myectomy required return to cardiopulmonary bypass and MVR with and without septal myectomy.

Table VII. *New York Heart Association functional class (n)*

	Preoperative	*At follow-up*
I	2	103
II	29	33
III	113	8
IV	4	1

maker therapy in the management of hypertrophic obstructive cardiomyopathy, (2) identification of patient subgroups at higher operative risk, (3) long-term functional results and the influence, if any, of surgical procedures on the incidence of sudden death, (4) the role of intraoperative echocardiography and the long-term significance of postoperative echocardiographic findings, and (5) the role of MVR.

Hospital mortality. Our overall 6% hospital mortality and 4% to 5% mortality for patients undergoing septal myectomy with or without CABG is comparable with that reported in other surgical series.[1, 14-20] Only increasing age and preoperative amiodarone usage were significant multivariate preoperative predictors of hospital death. Although concomitant valve operation did not predict hospital mortality, the addition of valve operation to septal myectomy with CABG increased perioperative mortality to 21%. The variation in reported mortality rates is primarily related to patient age and the performance of concomitant surgical procedures. Mohr and associates[21] reported no operative mortality in 47 consecutive patients under 40 years of age, 43 of whom underwent isolated myectomy. Cohn, Trehan, and Collins,[16] likewise, reported zero mortality in 32 older patients (mean age 55 years) undergoing isolated myectomy. The adverse influence of age and concomitant procedure was emphasized by the Mayo Clinic group[14] who reported 15.6% mortality in patients older than 65 years of age and 11.4% for patients undergoing an additional revascularization or valve procedure. Similarly, a recent large series from Dusseldorf[15] reported 2.9% hospital mortality in 272 patients undergoing isolated septal myectomy versus 10.9% in 92 patients undergoing simultaneous procedures. However, the adverse influence of concomitant CABG reported by the National Institutes of Health group, who describe a 2.7% operative mortality for isolated myectomy compared with 18% in those undergoing simultaneous CABG, was not apparent in our study.[22] The National Institutes of Health group concluded that their operative mortality was higher because of an increased incidence of ventricular septal defect (21%) potentially because of septal ischemia in the presence of coronary artery disease. Our mortality of 5% in patients undergoing simultaneous myectomy and CABG was nearly equivalent to that for myectomy alone. We did not encounter a higher incidence of ventricular septal defect.

The antiarrhythmic efficacy and negative inotropic effects of amiodarone have led to its use in the management of hypertrophic obstructive cardiomyopathy with varying results.[6, 23] One group has found an increased incidence of sudden death in patients treated with amiodarone.[23] The adverse effects of preoperative amiodarone in the surgical management of hypertrophic obstructive cardiomyopathy has been reported by the National Institutes of Health group[24] who described an increased incidence of postoperative pulmonary, hepatic, and low cardiac output complications in treated patients. Seven patients were treated with amiodarone, and two deaths occurred among this seven. Of our two patients treated with amiodarone who died, one clearly had a sudden cardiac death 13 hours after the operation. The other patient had markedly elevated transaminase levels after the operation and diffuse hepatic necrosis on autopsy. These two patient deaths may have been directly related to amiodarone usage.

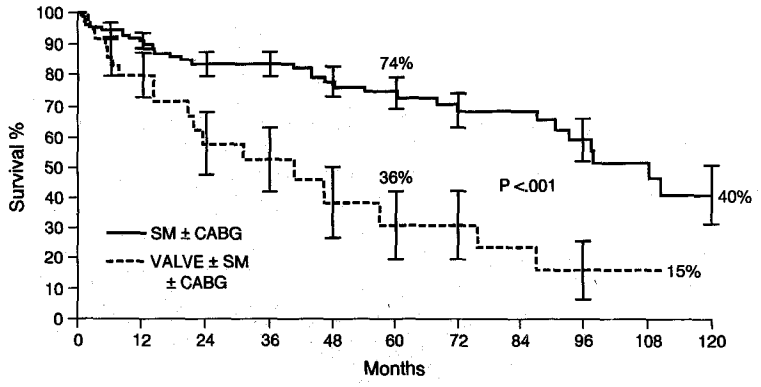

Fig. 3. Event-free survival by procedure (events listed in Appendix A) of 167 in-hospital survivors. Event-free survival of the septal myectomy *(SM)* with or without coronary artery bypass grafting *(CABG)* group at 5 and 8 years was 74% and 52%, respectively, compared with 36% and 15% in the group undergoing a valve procedure. The respective numbers of patients remaining in the sample in the myectomy with or without CABG group at 2, 4, 6, 8, and 10 years were 71, 48, 30, 16, and 6. The respective numbers of patients in the valve group at 2, 4, 6, 8, and 10 years were 12, 5, 4, 2, and 0. *Error bars* are ± standard deviation.

Complications. The 10% incidence of complete heart block in our series is higher than the 5% reported by Maron and others.[1, 24, 25] In the isolated myectomy group its occurrence is associated with return to cardiopulmonary bypass and redo myectomy. In addition, the presence of complete heart block was a strong predictor of reduced late survival on multivariate analysis. This association of complete heart block and need for repeat myectomy may reflect the presence of more severe septal hypertrophy in this group and subsequent need for a more aggressive myectomy leading to atrioventricular node injury. Although our higher incidence of complete heart block may reflect the degree of septal hypertrophy in those patients affected, it may also be a marker for the thoroughness of echocardiographically guided septal resection, durable relief of LV outflow obstruction, and excellent long-term symptom relief we have observed.

Postoperative iatrogenic ventricular septal defect has been reported in 3% of patients by Kirklin and Barratt-Boyes,[25] this complication occurred in only one of our patients in the early part of our experience before the routine use of echocardiography. Intraoperative two-dimensional echocardiography is instrumental in guiding the appropriate amount and location of septal resection and may be partly responsible for the low incidence of this complication observed in our population.

In-hospital reoperation was necessary in four patients: one underwent extended myectomy, two underwent MVR, and one underwent both MVR and myectomy. Of six patients undergoing late reoperation, none required repeat myectomy for a persistent gradient, although two required MVR. This 1.2% incidence of late MVR and absence of late repeat myectomy compares favorably with the late reoperation experience from the National Institutes of Health.[26] On long-term follow-up of 535 patients undergoing septal myectomy, they report 23 patients (4.3%) who required late reoperations that included MVR in 11 and repeat myectomy in 12.

Significant aortic insufficiency requiring late aortic valve replacement occurred in one patient. We did not review all incidences of mild-to-moderate aortic insufficiency which occurred in 54% of 67 patients followed up by Sasson and associates.[27] The National Institutes of Health group[28] reported a 4% incidence of significant late aortic insufficiency in 525 patients after myectomy. Six of their 12 patients required aortic valve replacement.

Late mortality. Late survival for our entire group was 86% and 70% at 5- and 10-year follow-up, respectively, and 93% and 79% for patients undergoing septal myectomy with or without CABG. Only 3 of 131 in-hospital survivors of septal myectomy with or without CABG died late cardiac deaths, for a yearly mortality of 0.6%. This late mortality compares favorably with natural history studies which report mortality rates of 3% to 6%/year.[1, 29] Fifty-six percent of our patient group was older than 60 years of age and may have had a distinct form of hyper-

trophic obstructive cardiomyopathy[30] with a more favorable natural history than that in younger patients. The Mayo Clinic has followed up a group of 95 patients over the age of 65 for a median duration of 4.2 years and found survival not significantly different from that in an age- and gender-matched control group.[31] A literature review by Seiler and associates[17] of 12 medical series from 1974 through 1990 including 869 medically treated patients, with a follow-up ranging from 1.2 to 9.1 years, reported yearly mortality rates ranging from 0.5% to 6%. They review five surgical series with reported mortality rates of 2.2% to 4.4%/year. Their long-term 9-year follow-up of patients undergoing myectomy and patients receiving verapamil treatment showed a mortality rate of 0.6%/year, although only 17 patients were treated in this manner. In a larger group of 127 patients undergoing septal myectomy, Krajcer and associates[32] reported late mortality of 0.7%/year.

Comparison of diverse patient groups from multiple institutions in a retrospective manner does not allow definitive conclusions regarding the superiority of one form of treatment over another. Furthermore, the reported mortality rates with medical and surgical therapy variably refer to both cardiac and noncardiac deaths. In general the younger the patient population the more often late premature deaths are due to cardiac causes in contrast to an older group such as ours in which late deaths may result from a variety of noncardiac causes. Four of the eight late deaths in our cases of septal myectomy with or without CABG were due to malignancy. Given the limitations of a retrospective review we can conclude that our overall operative mortality in a symptomatic, diverse group of patients undergoing myectomy is acceptably low and that the rate of late cardiac deaths is very low.

Our multivariate analysis identified late valve operation, increasing age, syncope, postoperative complete heart block, and return to bypass with MVR as factors significantly associated with late death.

Syncope is the symptom that has been shown to be most strongly associated with the development of sudden death in multiple studies that have investigated relevant risk factors.[1, 33] Other identified proposed risk factors include young age, increased left ventricular wall thickness, a family history of sudden death, and nonsustained ventricular tachycardia on ambulatory electrocardiographic monitoring.[1] The mechanism by which a history of syncope may predispose a patient to sudden death is uncertain.[34] Syncope is associated with inducible sustained ventricular arrhythmias.[35] These arrhythmias may be manifestations of the underlying pathologic myocardial cell disarray and fibrosis seen in hypertrophic obstructive cardiomyopathy which subsequently is not improved by surgical myectomy.

The association of complete heart block with late death may be a marker for those patients with more extensive septal hypertrophy[36, 37] who, accordingly, require a more aggressive subaortic resection to relieve outflow obstruction. The recognized association of this increased septal hypertrophy with sudden death[35] may provide an explanation for our findings. Confirmation would require echocardiographic evidence of increased septal hypertrophy in those patients in whom complete heart block developed compared with those unaffected (information not available from our study).

The influence of concomitant valve operation on late survival was profound. The yearly mortality rate in hospital survivors undergoing valve operation was 6.6%/year—more than 10 times greater than that in the septal myectomy with or without CABG group. Those patients undergoing valve operation were particularly diverse with a variety of cardiac surgical diseases that included mitral or aortic valvular disease, hypertrophic obstructive cardiomyopathy, and, in 14 patients, concomitant coronary artery disease. The small number of patients in each valve category does not allow statistical analysis of associated risk factors for late death in comparison with other surgical subgroups. The adverse impact of valve operation on late survival may reflect diastolic dysfunction seen in hypertrophic obstructive cardiomyopathy in combination with valvular disease. This diastolic dysfunction may be even more severe in older patients[38] and potentially limits the normal pathophysiologic increases in myocardial wall thickness and chamber dimension that are usual adaptive responses to pressure and volume loading valvular disease.

Role of MVR. Those patients with significant MR who do not respond to initial myectomy and require a return to cardiopulmonary bypass and MVR were at significantly higher risk for late death on our multivariate analysis. Of the seven patients in this category, six had MR grade 3 to 4+ before the operation. Of the 60 patients with preoperative MR grade 3+ to 4+, 16 required MV replacement, and, of those 16, six procedures were performed after an attempted myectomy without MVR was inadequate to correct the underlying MR. Why this group should be at higher risk for late death is unclear.

The reason for this increased risk requires an understanding of the variable response of existing preoperative MR to attempted septal myectomy and may be related to our current understanding of the pathophysiologic condition of MR in hypertrophic obstructive cardiomyopathy.

Doppler echocardiographic study of dynamic LVOT obstruction during systole has provided our understanding of the relationship between the mitral valve and the hypertrophic septum.[1] The Venturi effect of a high velocity jet through an area of subaortic septal obstruction appears to direct the anterior mitral leaflet toward the septum during mid-systole, exacerbating the obstruction, interfering with normal mitral valve closure, and producing a variable degree of MR. The role of this septal hypertrophy and the precise site of SAM-induced septal contact provides the morphologic basis for our echocardiographic demonstration of the appropriate site for surgical resection.

The validity of this approach allowed us to successfully reverse MR grade 3+ to 4+ in 44 of 60 patients with septal myectomy alone. In 10 of the remaining 16 patients the underlying preoperative mitral valve disease was recognized (subacute bacterial endocarditis $n = 3$, rheumatic $n = 2$, cleft mitral valve $n = 2$, myxomatous $n = 2$, papillary fibroelastosis $n = 1$), and the operating surgeon performed mitral valve repair or replacement at the initial operation. Five patients required MVR because of either a persistent postmyectomy gradient or MR. Four of these five had normal mitral valves, and one had a redundant posterior mitral leaflet. Two patients required reoperation and MVR during initial hospitalization for persistent postoperative MR, and both of these patients were found to have apparently normal mitral valves. This experience suggests that a small subgroup of patients, despite myectomy, have persistent MR with no clearly identified mitral valve disease. In addition, this subgroup after MVR appears to be at higher risk for late death.

The concept of systolic anterior motion of the anterior mitral leaflet as a cause of MR emphasizes the role of septal disease rather than potential underlying mitral valve disease. A small number of patients have intrinsic mitral valve disease, usually recognized before the operation, in combination with hypertrophic obstructive cardiomyopathy. In addition, a separate group of patients may have more subtle mitral valve abnormalities combined with septal hypertrophy. Recent pathoanatomic studies have shown that, in addition to the characteristic asymmetric obstructing septal hypertrophy, intramural coronary artery anomalies, and myocardial fibrosis characteristic of hypertrophic obstructive cardiomyopathy, many patients have associated complex mitral valve abnormalities.[1] These include increased mitral valve surface area, asymmetry in scallop configuration, chordal rupture,[39] abnormal papillary muscle insertion,[40] and anterior displacement of the mitral valve. Klues and associates[41] have shown this spectrum of structural malformations in 66 of 94 explanted mitral valves from patients with hypertrophic cardiomyopathy. These studies suggest that the morphologic definition of hypertrophic obstructive cardiomyopathy may need to be expanded to include the mitral valve. They also emphasize the potential complex role of the mitral valve in combination with septal hypertrophy in producing MR. Understanding this subgroup of patients with apparently "normal" mitral valves before the operation and who subsequently require MVR after initial myectomy may help to elucidate why this group is at higher risk for late death.

Our study does not resolve the ongoing controversy over the role of MVR in hypertrophic obstructive cardiomyopathy.[43-48] Currently the National Institutes of Health group[47] recommends MVR (1) in the presence of a thin septum <18 mm, (2) in the presence of atypical septal structure, (3) after previous septal myectomy, and (4) when intrinsic mitral valve disease is present. This approach has led to 25% of their patients requiring MVR. Krajcer and associates[46] have long emphasized the excellent hemodynamic results of primary MVR in patients with severely symptomatic hypertrophic obstructive cardiomyopathy and have performed MVR in 29% of hypertrophic obstructive cardiomyopathy cases in the presence of either severe MR or after failure of septal myectomy. Walker and associates[43] have recommended primary MVR for hypertrophic obstructive cardiomyopathy by less experienced units because it provides a more consistent reduction in the LVOT gradient. However, the recognized bleeding, infectious and thromboembolic risks of prosthetic MVR, our excellent long-term results of myectomy alone, and the finding that valve operation itself is a risk factor for late death lead us to continue to recommend myectomy as the primary treatment for hypertrophic obstructive cardiomyopathy. Patients with intrinsic mitral valve disease are best treated with MVR. The small group of patients who have persistent MR after septal myectomy and who ap-

pear to have structurally normal valves on preoperative echocardiography are a difficult group. A better understanding of the exact mechanism of regurgitation in these valves may allow more precise future treatment recommendations.

The role of mitral valve repair is not clearly defined, but our anecdotal experience suggests that the results of repair are less predictable in the presence of significant hypertrophic cardiomyopathy.

Functional improvement. Symptomatic improvement in our diverse patient population was excellent. No risk factors could be identified that allowed us to predict treatment failure in this group. Seven percent of patients who underwent operation were in functional class III or IV on long-term follow-up. Comparable objective functional improvement after myectomy has been reported in other surgical series.[1, 25] Our findings further establish the efficacy of surgical myectomy in reducing the symptoms of hypertrophic obstructive cardiomyopathy. Objective improvement in exercise tolerance and maximal oxygen consumption,[49] improvements in thallium perfusion,[50] improvements in myocardial metabolism,[51] and reduction in LV hypertrophy[52] provide objective correlates of this observed clinical improvement.

Conclusion

Our retrospective review of this diverse group of patients shows that (1) transaortic septal myectomy provides effective symptom relief with low operative mortality and low risk of late cardiac death, (2) increasing age and preoperative amiodarone usage are strong predictors of perioperative mortality, (3) increasing age, postoperative complete heart block, valve operation, a history of syncope, and the need for MVR after return to cardiopulmonary bypass at initial operation adversely influences long-term survival, and (4) the majority of patients with significant MR before operation are effectively treated with septal myectomy alone, although a difficult minority of cases not regularly identified before surgery will require prosthetic MVR to control the MR and LVOT gradient in hypertrophic obstructive cardiomyopathy.

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REFERENCES

1. Schlant R, Alexander RW, O'Rourke RA, Roberts R, Sonnenblick EH. Hypertrophic cardiomyopathy. In: Maron BJ, Roberts W, ed. Hurst's the heart. 8th ed. New York: McGraw-Hill, 1994:1621-36.
2. Rosing DR, Kent KM, Borer JS, Seides SF, Maron BJ, Epstein SE. Verapamil therapy: a new approach to the pharmacologic treatment of hypertrophic cardiomyopathy. II. Effects on exercise capacity and symptomatic status. Circulation 1979;60:1201-7.
3. Betocchi S, Cannon RO, Watson RM, et al. Effects of sublingual nifedipine on hemodynamics and systolic and diastolic function in patients with hypertrophic cardiomyopathy. Circulation 1985;72:1001-7.
4. Gilligan DM, Chan WL, Joshi J, et al. A double-blind, placebo-controlled crossover trial of Nadolol and Verapamil in mild and moderately symptomatic hypertrophic cardiomyopathy. J Am Coll Cardiol 1993;21:1672-9.
5. Frenneaux MP, Counihan PJ, Porter A, Lipkin DP, McKenna WJ. Effects of amiodarone on erect and supine exercise haemodynamics and exercise capacity in patients with hypertrophic cardiomyopathy. Eur Heart J 1992;13:687-96.
6. McKenna WJ, Oakley CM, Krikler DM, Goodwin JF. Improved survival with amiodarone in patients with hypertrophic cardiomyopathy and ventricular tachycardia. Br Heart J 1985;53:412-6.
7. McAreavey D, Fananapazir L. Altered cardiac hemodynamic and electrical state in normal sinus rhythm after chronic dual-chamber pacing for relief of left ventricular outflow obstruction in hypertrophic cardiomyopathy. Am J Cardiol 1992;70:651-6.
8. Fananapazir L, Cannon RO, Tripodi D, Panza J. Impact of dual-chamber permanent pacing in patients with obstructive hypertrophic cardiomyopathy with symptoms refractory to verapamil and beta-adrenergic blocker therapy. Circulation 1992;85:2149-61.
9. Morrow AG. Hypertrophic subaortic stenosis. Operative methods utilized to relieve left ventricular outflow obstruction. J THORAC CARDIOVASC SURG 1978; 76:423-30.
10. Nishimura RA, Danielson GK. Dual Chamber pacing for hypertrophic obstructive cardiomyopathy:has its time come? Br Heart J 1993;70:301-3.
11. Marwick TH, Stewart WJ, Lever HM, et al. Benefits of intraoperative echocardiography in the surgical management of hypertrophic cardiomyopathy. J Am Coll Cardiol 1992;20:1066-72.
12. Goldman ME. Hypertrophic cardiomyopathy and intraoperative echocardiography: too much of a good thing may be dangerous. J Am Coll Cardiol 1992;20: 53-4.
13. Grigg LE, Wigle ED, Williams WG, Daniel LB, Rakowski H. Transesophageal Doppler echocardiography in obstructive hypertrophic cardiomyopathy: clarification of pathophysiology and importance in intraoperative decision making. J Am Coll Cardiol 1992;20:42-52.

14. Mohr R, Schaff HV, Danielson GK, et al. The outcome of surgical treatment of hypertrophic obstructive cardiomyopathy. J THORAC CARDIOVASC SURG 1989;97:666-74.
15. Schulte HD, Bircks WH, Loesse B, Godehardt EAJ, Schwartzkopff B. Prognosis of patients with hypertrophic obstructive cardiomyopathy after transaortic myectomy. J THORAC CARDIOVASC SURG 1993;106:709-17.
16. Cohn LH, Trehan H, Collins, JJ. Long-term follow-up of patients undergoing myotomy/myectomy for obstructive hypertrophic cardiomyopathy. Am J Cardiol 1992;70:657-60.
17. Seiler C, Hess OM, Schoenbeck M, et al. Long-term follow-up of medical versus surgical therapy for hypertrophic cardiomyopathy: a retrospective study. J Am Coll Cardiol 1991;17:634-42.
18. McIntosh CL, Maron BJ. Current operative treatment of obstructive hypertrophic cardiomyopathy. Circulation 1988;78:487-95.
19. Blanchard DG, Ross J Jr. Hypertrophic cardiomyopathy: prognosis with medical or surgical therapy. Clin Cardiol 1991;14:11-9.
20. Delahaye F, Jegaden O, de Gevigney G, et al. Postoperative and long-term prognosis of myotomy-myomectomy for obstructive hypertrophic cardiomyopathy: influence of associated mitral valve replacement. Eur Heart J 1993;14:1229-37.
21. Mohr R, Schaff HV, Puga FJ, Danielson GK. Results of operation for hypertrophic obstructive cardiomyopathy in children and adults less than 40 years of age. Circulation 1989;80(Suppl):I191-6.
22. Siegman IL, Maron BJ, Permut LC, McIntosh CL, Clark RE. Results of operation for coexistent obstructive hypertrophic cardiomyopathy and coronary artery disease. J Am Coll Cardiol 1989;13:1527-33.
23. Leon M, Tracy C, Winkler J, Bergum C, Bonow R, Epstein S. Amiodarone does not prevent and may increase sudden death in patients with hypertrophic cardiomyopathy [Abstract]. Circulation 1987;76(Suppl): IV248.
24. Kupferschmid JP, Rosengart TK, McIntosh CL, Leon MB, Clark RE. Amiodarone-induced complications after cardiac operation for obstructive hypertrophic cardiomyopathy. Ann Thorac Surg 1989;48:359-64.
25. Kirklin J, Barratt-Boyes B. Hypertrophic obstructive cardiomyopathy. In: Cardiac surgery. 2nd ed. New York: Churchill-Livingston, 1992:1239-62.
26. Roberts CS, McIntosh CL, Brown PS, Cannon RO, Gertz SD, Clark RE. Reoperation for persistent outflow obstruction in hypertrophic cardiomyopathy. Ann Thorac Surg 1991;51:455-60.
27. Sasson Z, Prieur T, Skrobik Y, et al. Aortic regurgitation: a common complication after surgery for hypertrophic obstructive cardiomyopathy. J Am Coll Cardiol 1989;13:63-7.
28. Brown PS, Roberts CS, McIntosh CL, Clark RE. Aortic regurgitation after left ventricular myotomy and myectomy. Ann Thorac Surg 1991;51:585-92.
29. Shah PM, Adelman AG, Wigle D, et al. The natural (and unnatural) history of hypertrophic obstructive cardiomyopathy. Circ Res 1974;35(Suppl):II179-95.
30. Lever HM, Karam RF, Currie PJ, Healy BP. Hypertrophic cardiomyopathy in the elderly: distinctions from the young based on cardiac shape. Circulation 1989;79:580-9.
31. Fay WP, Taliercio CP, Ilstrup DM, et al. Natural history of hypertrophic cardiomyopathy in the elderly. J Am Coll Cardiol 1990;16:821-6.
32. Krajcer Z, Leachman RD, Cooley DA, Coronado R. Septal myotomy-myomectomy versus mitral valve replacement in hypertrophic cardiomyopathy: ten-year follow-up in 185 patients. Circulation 1989;80(Suppl): I57-64.
33. McKenna WJ, Deanfield JE, Faroqui A, England D, Oakley C, Goodwin JF. Prognosis in hypertrophic cardiomyopathy: role of age and clinical, electrocardiographic and hemodynamic features. Am J Cardiol 1981;47:532-8.
34. Fananapazir L, Tracy CM, Leon MB, et al. Electrophysiologic abnormalities in patients with hypertrophic cardiomyopathy: a consecutive analysis in 155 patients. Circulation 1989;80:1259-68.
35. Maron BJ, Fananapazir L. Sudden cardiac death in hypertrophic cardiomyopathy. Circulation 1992; 85(Suppl):I57-63.
36. Spirito P, Maron BJ. Relation between extent of left ventricular hypertrophy and occurrence of sudden cardiac death in hypertrophic cardiomyopathy. J Am Coll Cardiol 1990;15:1521-6.
37. McKenna WJ, Camm AJ. Sudden death in hypertrophic cardiomyopathy. Assessment of patients at high risk. Circulation 1989;80:1489-92.
38. Chikamori T, Doi YL, Yonezawa Y, Dickie S, Ozawa T, McKenna WJ. Comparison of clinical features in patients ≥60 years of age to those ≤40 years of age with hypertrophic cardiomyopathy. Am J Cardiol 1990;66:875-8.
39. Zhu W, Oh JK, Kopecky SL, Schaff HV, Tajik AJ. Mitral regurgitation due to ruptured chordae tendineae in patients with hypertrophic obstructive cardiomyopathy. J Am Coll Cardiol 1992;20:242-7.
40. Klues HG, Roberts WC, Maron BJ. Anomalous insertion of papillary muscle directly into anterior mitral leaflet in hypertrophic cardiomyopathy: significance in producing left ventricular outflow obstruction. Circulation 1991;84:1188-97.
41. Klues HG, Maron BJ, Dollar AL, Roberts WC. Diversity of structural mitral valve alterations in hypertrophic cardiomyopathy. Circulation 1992;85: 1651-60.

42. Klues HG, Proschan MA, Dollar AL, Spirito P, Roberts WC, Maron BJ. Echocardiographic assessment of mitral valve size in obstructive hypertrophic cardiomyopathy: anatomic validation from mitral valve specimen. Circulation 1993;88:548-55.
43. Walker WS, Reid KG, Cameron EW, Walbaum PR, Kitchin AH. Comparison of ventricular septal surgery and mitral valve replacement for hypertrophic obstructive cardiomyopathy. Ann Thorac Surg 1989;48:528-35.
44. McIntosh CL, Greenberg GJ, Maron BJ, Leon MB, Cannon RO, Clark RE. Clinical and hemodynamic results after mitral valve replacement in patients with obstructive hypertrophic cardiomyopathy. Ann Thorac Surg 1989;47:236-46.
45. Roberts CS, Gertz SD, Klues HG, et al. Appearance of or persistence of severe mitral regurgitation without left ventricular outflow obstruction after partial ventricular septal myotomy-myectomy in hypertrophic cardiomyopathy. Am J Cardiol 1991;68:1726-8.
46. Krajcer Z, Leachman RD, Cooley DA, Ostojic M, Coronado R. Mitral valve replacement and septal myomectomy in hypertrophic cardiomyopathy: ten-year follow-up in 80 patients. Circulation 1988; 78(Suppl)I35-43.
47. Cooper MM, Tucker E, McIntosh CL, Cannon RO, Clark RE. Effect of left ventricular septal myectomy on concurrent mitral regurgitation. Ann Thorac Surg 1989;48:251-6.
48. Fritzsche D, Krakor R, Goos H, Lindenau KF, Will-Shahab L. Comparison of myectomy alone or in combination with mitral valve repair for hypertrophic obstructive cardiomyopathy. Thorac Cardiovasc Surg 1992;40:65-9.
49. Diodati JG, Schenke WH, Waclawiw MA, McIntosh CL, Cannon RO. Predictors of exercise benefit after operative relief of left ventricular outflow obstruction by the myotomy-myectomy procedure in hypertrophic cardiomyopathy. Am J Cardiol 1992;69:1617-22.
50. Cannon RO, Dilsizian V, O'Gara PT, et al. Impact of surgical relief of outflow obstruction on thallium perfusion abnormalities in hypertrophic cardiomyopathy. Circulation 1992;85:1039-45.
51. Cannon RO, McIntosh CL, Schenke WH, Maron BJ, Bonow RO, Epstein SE. Effect of surgical reduction of left ventricular outflow obstruction on hemodynamics, coronary flow and myocardial metabolism in hypertrophic cardiomyopathy. Circulation 1989;79: 766-75.
52. Curtius JM, Stoecker J, Loesse B, Welslau R, Scholz D. Changes of the degree of hypertrophy in hypertrophic obstructive cardiomyopathy under medical and surgical treatment. Cardiology 1989;76:255-63.

Discussion

Dr. William G. Williams *(Toronto, Ontario, Canada).* I congratulate Dr. Heric and his colleagues for focusing our attention on the mitral valve in hypertrophic obstructive cardiomyopathy. And I rise to add substantive evidence to his data which illustrate the success of septal myectomy alone in dealing with the mitral incompetence in this disease.

Intraoperative transesophageal echocardiography shows clearly the mechanism of the mitral incompetence. The basis of the disease, as has been outlined, is the thickening of the intraventricular septum which narrows the subaortic channel. As shown with color Doppler, the narrowed channel results in flow acceleration and turbulence in the subaortic area, and that flow acceleration drags the mitral valve toward the intraventricular septum, the so-called systolic anterior motion. As a result of the displacement of the valve, the leaflets fail to coapt and there is MR, which is typically posteriorly directed and occurs late in systole. It is important that the degree of outlet obstruction correlates with the degree of mitral incompetence. And when it does not, it indicates that there is independent mitral valve disease which must be addressed.

After myectomy, the intraoperative echocardiography shows a widening of the outflow tract and a loss of turbulence in the subaortic area, and consequently the valve closes in a normal plane, coapts properly, and eliminates or certainly reduces the mitral incompetence.

Our experience with the surgical treatment of hypertrophic cardiomyopathy consists of 189 patients. In 78% the lesion was isolated, although I hasten to add that all of these patients had some degree of mitral incompetence, usually mild or moderate, but in a few, severe. 22% of the patients had other concomitant lesions, but note that independent MR was present in only four patients, and in all it was dealt with by valve repair. In no patient was MVR required.

There are four operative deaths in this series, including one in the 174 patients who had an isolated myectomy. To date there are nine late deaths. Ten-year survival of 84% is similar to survival in your series. Importantly, no patient has required reoperation for mitral valve disease.

Our experience concurs with Dr. Heric's data in recommending that the mitral valve repair or replacement is seldom necessary in patients with hypertrophic obstructive cardiomyopathy. Subaortic myectomy alone restores mitral valve function.

I have a question for Dr. Heric regarding the amount of muscle excised. I think it is important to excise enough myocardium to eliminate the LVOT gradient both at rest and with provocation. We try to excise an area equivalent to the area of the aortic root. Do you have any data regarding the amount of muscle excised, particularly in that group of patients who you return to bypass to resect additional muscle?

I enjoyed your paper and presentation and thank you for the interesting data.

Dr. Heric. First, let me make a comment on your statements about the utility of performing myectomy in relieving significant MR. We looked at 60 of our patients who came to the operating room with documented 2+ to 3+ MR, and indeed, as you have shown, 44 of those patients who underwent isolated myectomy had complete

resolution of their MR. This concurs with our understanding of the pathophysiologic aspects of MR in this disease.

However, 8 of the remaining 16 patients who, despite having what appeared to be adequate myectomy as documented by echocardiography, still had persistent MR. Six of them underwent MVR at initial operation because of persistent MR, and two patients underwent MVR in the postoperative period. The other eight patients had intrinsic mitral valve disease that was apparent before the operation and actually underwent MVR initially, as planned.

I would agree that myectomy is effective for the vast majority of patients who have MR in association with hypertrophic obstructive cardiomyopathy and would also agree that patients who have what appears to be intrinsic mitral valve disease will require some type of mitral valve procedure. However, I believe there is a group of patients with hypertrophic obstructive cardiomyopathy who appear to have normal mitral valves before the operation who after myectomy do not improve and experience persistent MR.

I think this is a subgroup of patients that we do not understand completely. There is an increasing amount of literature documenting that, in fact, the mitral valve in hypertrophic cardiomyopathy that appears to be normal before the operation may be significantly pathologic. Pathoanatomic studies show that these mitral valves are indeed abnormal. The mitral valve may have a role in MR that is separate and more complex than the mechanistic role of systolic anterior motion alone.

To answer your question about the amount of muscle that was removed, we did not document the amount of muscle removed at myectomy. Every surgeon had a slightly different approach to this operation and did not routinely weigh the specimen. We were dependent on echocardiography to basically document the necessary extent of resection and the precise volume of resection that would relieve the source of outflow obstruction.

Dr. Hagen D. Schulte *(Dusseldorf, Germany).* You showed 32 patients who had to undergo remyectomy. We had the same experience in our earlier series and therefore we extended our incision from the original Morrow procedure to a Bigelow incision. And also the excision was continued down to the baseline of the papillary muscles. And so I support the remarks of Dr. Williams that you have to extend the myectomy.

My first question is, did you only rely on echocardiography or did you also do pressure measurements simultaneously during the operation?

My second question is related to the amount of patients with total atrioventricular block after operation. Do you have any idea what the reason for this is, except in those patients who had previous right bundle branch block?

Dr. Heric. If I understand your second question, you were wondering why we had such a high incidence of complete heart block?

Dr. Schulte. What is the reason?

Dr. Heric. To answer your second question first, we were also impressed with the frequency of complete heart block. It developed in 10% of our entire patient group, which is twice the generally reported 5% incidence.

Six of the cases of complete heart block actually occurred in that more complex group who had concomitant valve operations. Looking at the myectomy group alone, there were only 10 patients in whom complete heart block developed, an incidence of 6%. We found a significant association between a return to cardiopulmonary bypass, a more extensive myectomy and the development of complete heart block.

I believe the incidence of complete heart block in our series was a consequence of being fairly rigorous about requiring an adequate result after initial myectomy as determined by the echocardiographers, who are insistent that we do so.

And as a consequence of doing what we think is a more thorough myectomy, we have a slightly increased incidence of complete heart block. This is somewhat problematic. We would like to think that the thorough degree of myectomy is associated with our good long-term results, but, in fact, we have shown that complete heart block is a risk factor for late mortality. I cannot resolve whether this aggressive approach is correct, but I think the return to bypass for increasing myectomy was associated with our higher incidence of complete heart block.

We generally, in the early portion of the experience, did direct measurements in the ventricle and in the aorta to document the LVOT gradient. As we became more comfortable and familiar with intraoperative echocardiography and because our cardiologists have an interest in this disease, we have relied on echocardiography to establish the LVOT gradients.

Dr. Bruno J. Messmer *(Aachen, Germany).* Dr. Heric, I congratulate you on an honest paper, demonstrating also all the complications that can appear.

Some 15 years ago we modified the original Morrow technique. First we insert a sharp triple hook into the left ventricle to pull at the deepest point of the septal muscle bulge. By this maneuver and cutting in front of the prongs, the muscle bulge can be excised over its whole long axis. With the traditional Morrow technique the deepest point is often not reached, which may result in residual obstruction.

The second major modification consists in liberal mobilization of both papillary muscles from lateral attachments to the left ventricular wall and from hypertrophic interpapillary trabeculae. If necessary we trim the papillary muscles to slim sugar hats. It is important to reach full mobilization of the subvalvular apparatus of the mitral valve.

Since 1979 this technique has been used in 59 patients with one early death caused by multiorgan failure. Survival at 10 years is 85%. Postoperative echocardiographic studies show either a fully competent valve or only trivial insufficiency in spite of the fact that one third of the patients had 2+ and 3+ mitral insufficiency before operation and nothing had been done to the mitral valve itself with the exception of generous mobilization of the papillary muscles. In your presented paper, you have 16 patients with persistent mitral insufficiency, and I suspect that this is due to noncorrected malattachment and hypertrophy of the papillary muscles. Did you have a chance to inspect the papillary muscles during the operation?

Furthermore can you provide us some late noninvasive or invasive dates on your patients? In our series we have

done postoperative invasive studies in 22 unbiased survivors (39%). The average gradient at rest fell from 79 mm Hg before the operation to 5 mm Hg after the operation with the gradient under stress decreasing from 147 to 16 mm Hg. Finally, I wonder if your group has any experience with simple insertion of a DDD pacemaker system without myectomy in patients with hypertrophic obstructive cardiomyopathy?

Dr. Heric. To answer your first question, generally, when performing the myectomy, the incision is extended toward the LV apex until the papillary muscles are visualized. That is part of the operative procedure that all of the involved surgeons perform. I cannot make any comment about the appearance of the papillary muscles or their potential involvement in persistent MR by variable operating surgeons who were doing the procedures at the time.

I would also comment that our understanding of the disease of hypertrophic obstructive cardiomyopathy should include more than just the interventricular septum. The pathologic studies I previously mentioned have shown abnormal attachments between the papillary muscles and the mitral apparatus that are potentially important in the development of MR. Your procedure might be directed against this pathology, I do not know. We did not do this procedure, and I do not have any other specific comments.

Regarding late echocardiographic follow-up, our patients were variably followed up. On late follow-up, only one patient underwent MVR and no patients underwent late redo myectomy. All the patients who were in our geographic area were followed up with echocardiography, and none of them had significant postoperative gradients that were brought to our attention. However, because echocardiographic follow-up of these patients was inconsistent, we have not reported on this aspect of follow-up. We will attempt more complete echocardiographic follow-up as a future project.

There is a recent experience with DDD pacemakers for hypertrophic obstructive cardiomyopathy at our institution that actually has occurred since I have left the institution. Our study only included patients to early 1993, so I cannot comment on our recent experience with DDD pacemaker therapy.

Dr. Khalid Rasheed *(Riyadh, Saudi Arabia).* Our experience with similar cases has been that, after coming off cardiopulmonary bypass, there is often a significant gradient across the outflow tract. Most of this gradient is due to dynamic obstruction secondary to hypertrophy of the left ventricle. If the gradient is again checked after 15 to 20 minutes there is already a sharp fall in it and in some patients in whom we have repeated these measurements once again just before closing the chest; to our surprise, the gradient had almost completely disappeared.

My question is whether you also do repeated measurement of the gradient because, in my opinion, this would probably have saved some patients from reoperation.

Also, transesophageal echocardiography alone would probably have a higher chance of error because of the dynamic element of obstruction.

Dr. Heric. Every surgeon's approach to this disease was somewhat different. I think every surgeon is naturally reluctant to go back on bypass and do another surgical procedure. I would say, in general, although I do not have data to show you, surgeons were reluctant and wanted to see persistent real gradients, real systolic anterior motion, and real MR after initially coming off bypass with administration of isoproterenol (Isuprel) to be convinced of the need for extended remyectomy.

I was interested in whether those patients who, despite having a persistent gradient, MR, or systolic anterior motion that we would generally find unacceptable in the operating room had any long- or short-term morbidity. Because, obviously, this issue is of importance to surgeons, your approach suggesting that by ignoring these echocardiographic abnormalities patients my do well after initial myectomy. I found on follow-up of this group of patients, as I pointed out in my talk, that none of them had any adverse events either in hospital or later. So I was not able to show that having moderate MR or systolic anterior motion and having a provokable gradient had any long-term adverse effect, so perhaps your approach is reasonable.

Appendix A. *Late events*

Event	*No. of patients*	*Total events*
Ventricular arrhythmias	22	39
Catheterization	13	16
Congestive heart failure	9	13
Return of New York Heart Association functional class III or IV	9	12
Reoperation	6	7
Cerebrovascular accident	4	4
Percutaneous transluminal coronary angioplasty	3	7
Myocardial infarction	3	4
Permanent pacemaker	2	2
Thromboembolic	1	1
Endocarditis	1	1
Wound complication	1	1
Respiratory	1	1
None	74	0