Current Operative Treatment of Obstructive Hypertrophic Cardiomyopathy

Charles L. McIntosh, MD, PhD, and Barry J. Maron, MD

Operative intervention has been an important part of the therapeutic strategy for patients with hypertrophic cardiomyopathy (HCM) for the past 30 years.1-16 Cleland,1 in 1958, was the first to successfully perform a transaortic myectomy in a patient with the obstructive form of this disease by resecting a small amount of muscle from the thickened upper portion of the ventricular septum. Shortly thereafter, Morrow2 modified and refined the ventricular septal myotomy-myectomy operation,3-8 which he eventually performed on 350 patients. About the same time, Bigelow et al4 successfully pioneered the myotomy operation (ventricular myotomy) that was similar to the myotomy-myectomy except that no muscle was actually removed from the ventricular septum.

During the past 3 decades, operation on patients with obstructive HCM has continued to be performed frequently but primarily in a few selected referral centers.1-16 Operative intervention has improved symptoms in many patients with HCM, in whom medical therapy has failed, by virtue of relieving the subaortic pressure gradient and reducing left ventricular pressures.17,18 The experiences gained from the clinical appraisal of these patients during a long period of time, as well as from technical advances in echocardiographic techniques, have considerably enhanced our understanding of the role of operation in HCM and have altered our concepts regarding the intraoperative management of such patients. Because of this evolution in our knowledge, it would appear important at this time to appraise the current status of operation in the treatment strategy of patients with obstructive HCM.

Concept of Dynamic Subaortic Obstruction

Left ventricular outflow tract gradients and subaortic obstruction in HCM are dynamic in nature, that is, they can be reduced or augmented by certain interventions that alter arterial pressure, myocardial contractility, or ventricular volume.17-20 These gradients and the concomitant elevation in left ventricular systolic pressure ultimately have deleterious pathophysiological consequences for the left ventricle, probably by virtue of inappropriately increasing myocardial wall stress and enhancing oxygen demand and consumption, and by producing mitral regurgitation.17,20 Thus, in the approximately 25% of patients with HCM who have substantial subaortic gradients and obstruction to left ventricular outflow, the chronic elevation in intraventricular systolic pressure probably constitutes an important determinant of clinical course.17-20

Operative intervention in HCM is predicated on the premise that true mechanical impedance to left ventricular outflow is present in these patients. Indeed, there is substantial evidence available from a series of hemodynamic, contrast and radionuclide angiographic, Doppler, and echocardiographic studies that supports the principle that systolic anterior motion of the mitral valve and prolonged systolic contact between the mitral valve and ventricular septum creates true obstruction to left ventricular outflow.17-28 For example, the gradient develops at the same time in midsystole that the mitral valve initially makes direct contact with the ventricular septum; the earlier and more prolonged this mitral-septal apposition, the more severe the outflow gradient. Indeed, it has been the clinical experience of our institution and others18 that in individual patients with HCM a left ventricular outflow gradient under basal conditions is, with rare exception, associated with marked mitral systolic anterior motion and mitral-septal contact. Although the left ventricle in HCM is hyperdynamic and ejects a considerable proportion of its stroke volume rapidly and early in systole, a large proportion of left ventricular emptying (about 50%) nevertheless occurs in the presence of the subaortic gradient and systolic contact between mitral valve and ventricular septum. Hence, the left ventricle is not truly devoid of blood when the outflow gradient is present; the gradient and forward blood flow coexist in midsystole, flow persists throughout the prolonged period of left ventricular ejection in most patients.
and the left ventricle continues to shorten even after increased intraventricular pressures appear. Doppler investigations (including color-flow mapping) confirm that the site of jet formation is at or near the point of maximum mitral systolic anterior motion, that blood-flow velocity is increased in the left ventricular outflow tract, and that the magnitude of this velocity is a linear relation with the measured subaortic gradient.

**Indications for Operation and Hemodynamic Consequences of Myotomy-Myectomy**

About 1,700 operations (primarily ventricular septal myotomy-myectomy) have been performed on patients with obstructive HCM of all ages from about 10 to 75 years at 10 major institutions throughout the world that have active surgical experience with this disease. Operative mortality has been about 5%. The usual indications for operation are 1) marked symptoms unrelieved by adequate medical trial of β-blockers, calcium antagonists or other drugs and 2) obstruction to left ventricular outflow under basal conditions (subaortic gradient ≥50 mm Hg) or with provocative maneuvers alone (at some institutions). There are no conclusive studies demonstrating whether or not operation prolongs life, and therefore, surgery is only recommended and performed to improve severe symptoms and the quality of life. Because of the uncertainty regarding whether operation influences longevity and because of the risks of the procedure, asymptomatic or mildly symptomatic patients with large gradients are not usually selected as candidates for myotomy-myectomy even though they could theoretically benefit hemodynamically from this procedure.

The standard ventricular septal myotomy-myectomy is performed through an aortotomy without the benefit of complete direct anatomic visualization. Two vertical and parallel incisions are made in the basal ventricular septum about 1 cm apart and 1.0–1.5 cm deep into muscle (Figure 1) and are extended toward the apex about 4 cm (Figure 2E). A third transverse incision connects the first two incisions at their distal extent, and the bar of septal muscle is excised. Generally, a rectangular channel is created, extending from a point near the aortic anulus distally to beyond the site of mitral-septal systolic contact (Figures 1 and 2E). This channel is made in the 12 o’clock position (on the echocardiographic short-axis orientation), avoiding portions of the conducting system and the membranous septum that are located more medially toward the 11 o’clock position.

In addition to the standard myotomy-myectomy, a number of other operations have been proposed through the years for patients with obstructive HCM, including myotomy alone, modifications of myotomy-myectomy with approaches other than an aortotomy, or mitral valve replacement. Although these operations differ in design, the immediate objective has been the same in each case, that is, to abolish or substantially relieve the subaortic gradient and systolic anterior motion of the mitral valve and, thereby, reduce the elevated left ventricular systolic pressures. With septal myotomy-myectomy, this goal is achieved in about 95% of patients without compromising global left ventricular function; in the vast majority of these patients, the gradient under basal conditions is abolished, and in the remainder, only trivial gradients (≤20 mm Hg) are evident postoperatively. When relieved by operation, the basal gradient does not reoccur later; however, significant provocable gradients can be elicited with maneuvers after an operation that has been successful in relieving the basal gradient. The pathophysiological significance of such inducible gradients is unknown.

Although the amount of muscle excised (usually 1.5–3.0 g) constitutes only a small fraction of overall left ventricular mass, it is removed from a localized and critical site in the left ventricular outflow tract where mitral-septal contact and increased outflow tract velocities occur. Consequently, operation has the effect of widening and enlarging the left ventricular outflow tract, increas-

![Figure 1. Illustration of standard ventricular septal myotomy-myectomy operation performed through an aortotomy. Panel A: Two vertical and parallel incisions are made in the most basal portion of ventricular septum about 1 cm apart. A third incision is made transversely, connecting the initial two parallel myotomies. Attachments of the muscle bar to the septum are divided. Panel B: This segment of muscle is isolated and excised. Panel C: At completion of the myotomy-myectomy operation, a rectangular channel is created, about 1 cm wide, 1 cm deep, and 4 cm long, extending from a point 5–10 mm below the aortic anulus to a point just distal to the systolic contact between the distal portion of mitral valve leaflets and ventricular septum. In some patients, additional tissue may be resected from the margins of the channel to achieve greater enlargement of the left ventricular outflow tract.](http://circ.ahajournals.org/)

Downloaded from http://circ.ahajournals.org/ by guest on April 29, 2017
ing the distance between septum and mitral valve, and thereby substantially reducing the high outflow tract velocities and Venturi forces that probably create mitral systolic anterior motion and mechanical impedance to outflow.9,30–33 Because mitral regurgitation is a consequence of systolic anterior motion of the valve, it is also relieved or reduced by operation.18

Ventricular septal myotomy-myectomy may improve left ventricular function in other ways as well. Postoperative decrease in left ventricular pressure is associated with a decrease in myocardial oxygen consumption,17,34 an improvement in lactate metabolism,17,34 and restoration of a normal forward blood-flow profile.23,24 Although unproven, it is possible that some beneficial effects of operation may be related to improved diastolic function, an important disease component of HCM.35–37 After the septal myotomy-myectomy, left ventricular end-diastolic pressure decreases in about two thirds of patients and is normalized in one fourth.8

Morphological Spectrum of Hypertrophic Cardiomyopathy and Its Significance to Operation

The application of two-dimensional echocardiographic imaging to a large number of patients within the broad clinical spectrum of HCM has emphasized the great morphological diversity present in this disease, particularly with respect to the distribution of left ventricular hypertrophy.17,18,38–40 Left ventricular hypertrophy may be particularly marked, including some patients with the greatest ventricular septal thickening observed in any cardiac disease. Alternatively, in other patients septal thickening is mild and localized to only a portion of the ventricular septum, usually the basal anterior septum. Most patients with localized septal hypertrophy have the nonobstructive form of HCM, although others may show typical systolic anterior motion of the mitral valve and obstruction to left ventricular outflow.17,40

Frequently, in patients with HCM, the pattern of wall thickening is strikingly heterogeneous. Wall thickness may change abruptly, often within only a few millimeters. Consequently, contiguous segments of the left ventricular wall may differ greatly in thickness; in fact, it is not uncommon for markedly thickened left ventricular segments to be adjacent to portions of the wall that are of normal thickness. Such heterogeneity may also occur within the ventricular septum and even within the relatively confined region of the potential myotomy-myectomy, that is, the basal portion of septum extending about 4 cm from the aortic anulus distally toward the left ventricular apex (Figures 2 and 3).

Our appreciation of the morphological and clinical spectrum of those patients with obstructive HCM referred for operative therapy has evolved through the years, and we are now cognizant of a more diverse expression of the disease than perhaps was the case in the 1960s and 1970s. Certainly, since 1980, we have been exposed to more varied

FIGURE 2. Illustrations of the morphological spectrum of obstructive hypertrophic cardiomyopathy and importance of the distribution of ventricular septal (VS) thickening to the myotomy-myectomy operation (shown in diastole). Panels A–D: Different distributions of ventricular septal hypertrophy in the longitudinal cross-sectional plane; thickened areas of septum are denoted by parallel slanted lines. Panel A: Septal hypertrophy is quite localized to the most proximal 2 cm of the anterior basal septum. Panel B: Hypertrophy involves the upper and midseptal areas, extending over the proximal 4 cm of anterior septum. Panel C: Basal portion of the anterior ventricular septum is relatively thin, whereas substantially increased septal thickness is evident at the point of systolic contact between mitral valve and septum as well as in the more distal portion of septum. Panel D: Hypertrophy is more diffuse and involves the entire septum homogeneously. Panel E: Completed myotomy-myectomy channel (arrows) created in the same left ventricle that is depicted in Panel D, extending from near the aortic anulus to just beyond the mitral valve tips. Panel F: Low-profile disc prosthesis implanted in the mitral position after the native mitral valve has been removed from a patient with relatively thin ventricular septum. AML, anterior mitral leaflet; Ao, aorta; LA, left atrium; LV, left ventricle; LVFW, left ventricular free wall; PML, posterior mitral leaflet; RV, right ventricle.
patterns of left ventricular hypertrophy by virtue of expanding and diversified referral patterns and our extensive use of diagnostic echocardiography. This experience has had an important impact on the operative treatment of patients with HCM, including the selection of patients for septal myotomy-myectomy and the precise techniques used to relieve the left ventricular outflow tract gradient.

**Application of Intraoperative Echocardiography to Ventricular Septal Myotomy-Myectomy**

Two major complications of the myotomy-myectomy operation that may contribute greatly to mortality and morbidity are complete heart block and ventricular septal defect. Complete heart block results when the integrity of the conducting system is interrupted by improper placement of the myotomy incisions (or even with appropriate myectomy in those rare patients with preexisting right bundle branch block). This complication can now usually be treated adequately with implantation of atioventricular sequential or rate-responsive pacemakers. Conduction defects (including left bundle branch block) are also common sequelae of septal myotomy-myectomy and are present in about 75% of patients after operation. These electrocardiographic abnormalities do not, however, appear to be of particular electrophysiological consequence.

Iatrogenic ventricular septal defects, produced by removal of an excessive amount of muscle from the septum (or by excessively deep myotomy incisions), are probably the most significant complication of septal myotomy-myectomy. Septal defects usually occur when myectomy is performed in areas of the septum that are of normal or only mildly increased thickness, either by direct perforation or by creating an area of necrosis that in turn is predisposed to perforation. Therefore, it is critical for the surgeon to be aware of any heterogeneity in septal thickness so that relatively thin areas of the septum can be excluded from the myectomy.

Because ventricular septal morphology is not identical in all patients with obstructive HCM who are candidates for myotomy-myectomy (Figures 2 and 3), the precise mode of muscular resection should not necessarily be identical in all patients. Consequently, we have found detailed echocardiographic mapping of septal hypertrophy by echocardiography to be crucial for the safe and efficacious performance of this operation. However, in an important minority of patients, conventional trans-thoracic echocardiography cannot provide the necessary precise definition of septal thickness or location of hypertrophy; this may be due to a number of variables including unfavorable body habitus or chest configuration or the presence of pulmonary disease. Ultimately, such considerations led us to routinely use intraoperative echocardiography to define septal morphology in patients with obstructive HCM. During the past 5 years, each patient undergoing operation for HCM has had the distri-
bution and magnitude of septal hypertrophy mapped in the operating room with an integrated two-dimensional and M-mode echocardiographic examination, using a sterile transducer placed directly on the surface of the right ventricle of the beating heart before the institution of cardiopulmonary bypass.

The information obtained from this intraoperative assessment is used to determine whether myotomy-myectomy should be performed and, if so, whether the standard resection needs to be tailored to the individual patient's septal anatomy (Figures 2 and 3). At our institution, it is relatively uncommon to encounter a patient with obstructive HCM at operation in whom septal hypertrophy is both particularly marked and homogeneously distributed so that the standard myotomy-myectomy can be undertaken with no preoperative deliberation regarding the pattern and magnitude of septal thickness. Instead, a variety of patterns of septal hypertrophy are usually encountered. Atypical forms of septal hypertrophy include those in which the thickening of the septum is displaced laterally, medially, or distally, or the anterior septum is indented toward the right coronary leaflet at the 12 o'clock position (in the short axis). Hence, the most crucial information provided by the intraoperative echocardiographic examination is whether the most basal portion of ventricular septum at or near the 12 o'clock position on the short-axis orientation is thick enough to permit myotomy-myectomy and resection of septal muscle to be performed without incurring undue risk for iatrogenic ventricular septal defect.

Based on the information derived from intraoperative echocardiographic studies, the routine at our institution is to avoid beginning the proximal portion of the myectomy resection directly beneath the aortic anulus (Figures 1 and 2E). Outflow obstruction does not occur at this point; the most proximal 5-10 mm of ventricular septum is frequently thin; and support for the aortic valve anulus (and avoidance of iatrogenic aortic regurgitation) may well depend upon leaving this area of the septum intact. If echocardiography reveals that the area of most marked septal hypertrophy is located at or near the point of systolic mitral-septal contact (about 2-3 cm from the aortic anulus) but that the most basal portion of septum is relatively thin, then the resection of muscle must begin even farther from the aortic anulus than usual, bypassing the proximal thin area of septum (Figure 2C). In another variation of septal morphology, the most lateral portion of the anterior basal septum is relatively thin, whereas the more medial portion is markedly thickened (Figure 3D). The precise localization of the muscular resection can sometimes be shifted slightly medially in this circumstance; however, should the surgeon judge this revision to be impractical because of the limited exposure permitted through the aortotomy (i.e., between the 11 and 1 o'clock positions in the echocardiographic short-axis orientation), the myotomy-myectomy is not performed, and mitral valve replacement is considered a more appropriate alternative. Similarly, if all portions of the basal septum are relatively thin (<18 mm) and the risk of incurring a ventricular septal defect with a myotomy-myectomy is judged to be unacceptably high, mitral valve replacement is undertaken instead (Figure 2F). At our institution, we have found that intraoperative echocardiography plays a major role in determining the precise operative approach in about one third of those operations performed on patients with HCM.

In addition, intraoperative echocardiography provides the surgeon with the capability of assessing the site and adequacy of the myotomy-myectomy and its impact on mitral systolic anterior motion (and hence, subaortic obstruction) immediately after septal resection as well as visualizing iatrogenically produced ventricular septal defect or damage to the mitral valve apparatus. Consequently, in the event that operative relief of obstruction is judged to be incomplete, the myotomy-myectomy can be revised at that time in the operating room, and additional septal muscle can be removed, or mitral valve replacement can be performed.

**Clinical Consequences of Myotomy-Myectomy Operation**

The hemodynamic changes that result from operation have been consistently associated with an improved quality of life for the patient as manifested by a reduction in congestive symptoms (exer-tional dyspnea, fatigue, orthopnea, and paroxysmal nocturnal dyspnea), chest pain, and symptoms of impaired consciousness (near-syncope and syncope). Of the 240 patients operated upon at the National Institutes of Health by Dr. Andrew G. Morrow (between 1960 and 1982), about 70% reported symptomatic improvement during an average follow-up period of about 5 years.14 This improvement occurred in elderly42 as well as in young patients, and the subjective relief of symptoms has been associated with an objective increase in exercise capacity43,44 and threshold for pacing-induced angina.17,34 However, almost 10% of patients had persistent or recurrent, marked functional limitation, and 7% died of causes related to their underlying cardiomyopathy (either suddenly or due to congestive heart failure); postoperative atrial fibrillation appeared to be an important contributing factor to poor clinical outcome.

We have recently analyzed the most current clinical outcome for the 123 patients operated upon at our institution between 1960 and 1975 to assess the consequences of septal myotomy-myectomy in that subgroup of patients in whom the longest longitudinal evaluation after operation is available (Figure 4). At the time of myotomy-myectomy, the mean age of these patients was 42 years; the follow-up period ranged to 25 years (mean, 11.5 years). Forty percent of the patients have survived to date,
and two thirds of those continue to be improved from their preoperative state. The mean age of the survivors is now 57 years, and more than one half have reached the age of 60. On the other hand, 29 patients (24%) are known to have ultimately died of causes related largely to HCM; the average age of these patients at death was 54 years, nine had attained 60 years of age, and the mean period of survival after operation was greater than 10 years. Although continuing attrition and morbidity was observed during the long follow-up period of 11.5 years, the annual mortality rate due to HCM of 2.2% was practically identical to that observed for the shorter 5-year period of postoperative observation (i.e., 1.8%). In addition, there is little evidence to suggest that patients commonly evolve to the dilated phase of HCM many years after operation, and none ultimately required cardiac transplantation for “end-stage” progression of their disease.17

These data emphasize that it is most appropriate to view ventricular septal myotomy-myectomy as a palliative rather than a curative procedure for obstructive HCM. Outflow obstruction is an important disease component in many patients with HCM, and the symptomatic benefit achieved by operative relief of outflow obstruction and experienced by patients for considerable periods of time reflects this fact. However, it is important to emphasize that symptoms in HCM are often caused by a complex interaction of several pathophysiological mechanisms (e.g., diastolic dysfunction, small vessel disease, and arrhythmias) that undoubtedly influence the long-term outcome of patients after operation.17 Hence, even after complete operative relief of subaortic obstruction, the underlying disease process in patients with HCM can progress, leading to recurrent symptoms and even premature death.8,14 Consequently, patients with a successful myotomy-myectomy often require further pharmacological therapy for symptoms after operation, and in some patients, aggressive diagnostic strategies and treatment may be required for clinically important arrhythmias.

Potential Role of Mitral Valve Replacement

Prosthetic mitral valve replacement was first proposed and used as a mode of operative therapy for patients with obstructive HCM by Cooley et al.45 The rationale for mitral valve replacement in patients with obstructive HCM is that removal of the native mitral valve with prosthetic replacement abolishes both the subaortic obstruction gradient and mitral regurgitation (which are due to anterior motion of the mitral leaflets during systole),15 and removal also eliminates the risk of ventricular septal perforation that may result from septal muscle resection. Valve replacement has been a controversial treatment because of the inherent risks for thromboembolic or anticoagulant complications and prosthetic valve dysfunction.46 The initial concerns associated with inserting a mechanical valve into patients with a small left ventricular cavity have been largely absolved by the demonstration that low-profile prosthetic valves do not obstruct left ventricular outflow and will function normally as long as the size of the mitral orifice is sufficient.

Currently, practically all investigators agree that ventricular septal myotomy-myectomy is the preferred operation for most patients with obstructive HCM. However, as suggested above, valve replacement with a low-profile disc or bileaflet prosthesis may have an application in those selected patients with obstructive HCM in whom the risk of resection of septal tissue is judged to be unacceptably high, that is, when the basal anterior septum is relatively thin (<18 mm, usually 15–17 mm) or when the regions of greatest septal thickness are inaccessible to a myotomy-myectomy performed through an aortotomy. Additional (but rare) indications for mitral valve replacement include inadequate relief of subaortic obstruction after myotomy-myectomy or severe mitral regurgitation secondary to an intrinsic abnormality of the valve. However, severe pulmonary arterial hypertension associated with significant mitral regurgitation in a patient with a structurally normal mitral valve is not, as such, an indication for mitral valve replacement.

Recent Operative Results

Since 1982, after the death of Dr. Andrew G. Morrow, 156 patients with obstructive HCM (unassociated with other cardiac diseases) have been operated upon at the National Institutes of Health; 108 underwent myotomy-myectomy, but the other 48 patients received mitral valve replacement in accordance with the previously mentioned selection criteria. Indeed, in 70% of these 48 patients, valve
replacement was performed as the initial operation because the basal anterior ventricular septum was estimated by intraoperative echocardiography to be less than 18 mm thick or to demonstrate an atypical pattern of septal hypertrophy. Operative mortality in the myotomy-myectomy group was reduced to 2.7% (6.2% with valve replacement), presumably due largely to minimizing the risk for iatrogenic ventricular septal defect but also by virtue of greater understanding and improved methods of myocardial preservation.

Hemodynamic benefit from mitral valve replacement (assessed 6–12 months after operation) compares favorably with that achieved with myotomy-myectomy (Figure 5). Both operations produce similar reduction in the outflow tract gradient under basal conditions. Although the two groups of patients are not strictly comparable in all morphological, hemodynamic, or clinical variables, it is of note that mitral valve replacement resulted in somewhat greater reduction in postoperative provokable gradient and left ventricular end-diastolic pressure. Implantation of the mitral prostheses in patients with HCM was not associated with an important increase in pulmonary arterial wedge pressure.

The postoperative clinical course of patients with HCM receiving mitral valve replacement has been encouraging over a relatively short follow-up period of 6 months to 5 years (mean, 2.2 years). Of the 48 patients, there have been five late deaths, including three patients with a clinical course complicated by earlier unsuccessful myotomy-myectomy; two of the late deaths were caused directly by the prosthetic valve (i.e., cerebral hemorrhage as a complication of anticoagulant therapy and congestive heart failure secondary to perivalvular leak). Complications related to the prosthetic mitral valve also occurred in three other patients (i.e., valvular thrombosis or leak). About 70% of these 48 patients have experienced a substantial improvement in symptoms and functional capacity; most of those patients who did not improve postoperatively were those in whom mitral valve replacement was not their first operation, and a previous septal myotomy-myectomy had been unsuccessful. However, at this time, there is little available information regarding the long-term clinical outcome of patients with HCM who have undergone mitral valve replacement. It is uncertain whether these patients, who realize substantial hemodynamic benefit, will ultimately achieve the same symptomatic relief and longevity as have patients with myotomy-myectomy during similar time periods, and it is uncertain whether the potential complications of prosthetic valves will prove to be critical during a substantial period of follow-up.

Conclusions

Operation remains an important and rational therapeutic alternative when drug therapy is unsuccessful in relieving or controlling the severe symptoms experienced by many patients with obstructive HCM. After operative relief of outflow obstruction and normalization of left ventricular systolic pressure, the vast majority of patients experience an important benefit in symptoms, functional limitation, and quality of life that are often long lasting. However, even though operation results in permanent relief of outflow obstruction, it cannot be regarded as curative because patients may ultimately develop progressive cardiac symptoms or die from their cardiomyopathy because of impaired left ventricular filling, myocardial ischemia, atrial fibrillation, ventricular arrhythmias, or other undefined components of the disease. Long-term annual mortality rate related to HCM is about 2% during an average follow-up period of 11.5 years (ranging to 25 years).
The greater awareness and understanding of the morphological spectrum of HCM afforded by the application of two-dimensional echocardiography (including intraoperative imaging) has had an important impact on the operative management of patients with this disease. In particular, preoperative characterization of the distribution of ventricular septal hypertrophy before operation permits the myotomy-myectomy operation to be planned and performed so that muscle is resected only from sufficiently thickened regions of the septum, thereby minimizing the risk of iatrogenic ventricular septal defect. With these considerations in mind, those patients with particularly modest degrees of septal hypertrophy (<18 mm wall thickness) or heterogeneous patterns of septal thickening may be judged to be more appropriate candidates for mitral valve replacement than for myotomy-myectomy. Prosthetic mitral valve replacement provides hemodynamic benefits similar to myotomy-myectomy, but the long-term clinical outcome of those patients undergoing valve replacement is not yet known.

Acknowledgment
We acknowledge the superb efforts of our research assistant, Mrs. Gail Greenberg, in assembling the clinical data used in this report.

References


(Circulation 1988;78:487–495)
Current operative treatment of obstructive hypertrophic cardiomyopathy.
C L McIntosh and B J Maron

Circulation. 1988;78:487-495
doi: 10.1161/01.CIR.78.3.487
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1988 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/78/3/487.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/