Resection, Plication, Release - the RPR procedure for obstructive hypertrophic cardiomyopathy

Obstrüktif hipertrofik kardiyomiyopati için RPR prosedürü - Rezeksiyon, Plikasyon, Salınma

Daniel G. Swistel, Sandhya K. Balaram

Division of Cardiovascular and Thoracic Surgery, St. Luke's/Roosevelt Hospital Center
Columbia University, College of Physicians and Surgeons, New York, NY, USA

ABSTRACT

Objective: The surgical management of left ventricular outflow tract (LVOT) obstruction secondary to hypertrophic cardiomyopathy (HCM) has classically consisted of a septal myectomy. To address inconsistent results the extended myectomy or resection (R) and papillary muscle release (R) have been described. Our group introduced a novel addition to the surgical management consisting of an anterior mitral leaflet plication (P). We call the procedure resection - plication- release for repair of complex HCM pathology - the RPR operation. We investigated the mid-term results of all our patients undergoing surgical management for simple and complex HCM pathology.

Methods: Forty-two patients have undergone surgery for HCM at our hospital center since we began to look critically at the pathophysiology. Patients received either an extended myectomy alone, a myectomy plus either papillary muscle release or mitral leaflet plication, or the total RPR procedure. Pre and post-operative transesophageal echocardiograms were obtained in all patients to assess LVOT gradient, adequacy of resection and degree of mitral insufficiency. Subsequently, all patients had a trans-thoracic echocardiogram at a mean follow-up period of 3.4 ± 3.1 years (range, 0.5 to 7).

Results: Twenty-one patients underwent the full RPR procedure; thirteen received portions of the procedure and only seven underwent myectomy alone (including three with concomitant mitral valve replacement (MVR) for insufficiency unrelated to their obstructive pathology). One patient had an isolated MVR as primary therapy for HCM management. The average age was 56 ±14 years. The preoperative LVOT obstruction gradient was 137 ± 45 mm Hg and reduced to 10 ± 17 mm Hg post-operatively. All patients had mitral insufficiency pre-operatively, grade 3.1 on average (scale 0-4), and reduced post-operatively to trivial, grade 0.2. During the follow-up period, LVOT gradient remained low at 6 ± 14 mm Hg, and mitral insufficiency remained trivial, grade 0.4 (All p values < 0.0001). There were no hospital deaths and overall, no need for reoperations.

Conclusions: Hypertrophic cardiomyopathy patients often present with wide anatomic variation. When these variations are understood, the operative approach should be directed to correct or ameliorate those specific aspects, termed simple or complex pathophysiology. Durable long-term results can be achieved in all patients when the mitral valve pathology is appreciated and appropriately repaired, along with a properly located and adequately sized septal myectomy. *(Anadolu Kardiyol Derg 2006; 6 Suppl 2: 31-6)*

Key words: Hypertrophic obstructive cardiomyopathy, mitral valve plication

ÖZET

Amaç: Hipertrofik kardiyomiyopatiye(HKM) ikincil olarak gelişmiş sol ventrikül çıkış yolu (SVÇY) obstrüksiyonun cerrahi tedavisi klasik olarak septal miyektomiye dayanmaktadır. Bu tekniklerin tutarsız sonuçlar nedeni ile yaygın miyektomi veya rezeksiyon (R) ve papiller adalenin açığa çıkarma (release, R) gibi prosedürler tanımlanmıştı. Bizim grubumuz cerrahi tedavisine ilave olarak anteriyor mitral yaprakçığın plikasyonu (P) işlemi ortaya koymuştur. Kompleks HKM patolojisinin tamiri için ortaya koyduğumuz prosedüre rezeksiyon-plikasyon-salınma (release) - RPR operasyonu- adını verdik. Bu çalışmada, basit ve kompleks HKM patolojisi nedeni ile cerrahi tedavi uygulanan tüm hastalarımızda orta donem sonuçlarını inceledik.

Yöntemler: Hastane merkezimizde, kritik olarak patofizyolojiyi değerlendirmeye başladığımız zamandan beri, 42 hastaya cerrahi tedavi uygulandı. Hastalara sadece yaygın miyektomi, miyektomi ile birlikte papiller adalenin salınması; mitral yaprakçık plikasyonu, veya total RPR prosedürü yapıldı. Tüm hastalara SVÇY gradiyentini, rezeksiyonun yeterliliğini ve mitral yetersizliğini değerlendirmek amacı ile pre- ve postoperatif olarak transözofajiyal ekokardiyografi yapıldı. Takiben tüm hastalara ortalama 3.4 ± 3.1 yıl (yayılma aralığı 0.5-7) sonra transtorasik ekokardiyografi yapıldı.

Bulgular: Toplam 21 hastaya RPR prosedürü uygulandı; 13 hastaya prosedürün farklı kısımları yapıldı ve sadece 7'sine miyektomi yapıldı (obstrüksiyon patolojisine bağlı olmayan yetersizliği nedeni ile 3 mitral kapak replasmanı (MVR) dahil). Bir hastada HKM'nin primer tedavisi

Address for Correspondence: Daniel G. Swistel MD, Chief, Division of Cardiovascular and Thoracic Surgery,
St. Luke's/Roosevelt Hospital Center, 1111 Amsterdam Avenue, New York, New York 10025, USA
Telephone: 212-523-2798 Fax: 212-523-5344 E-mail: dswistel@chpnet.org

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olarak izole MVR uygulandı. Ortalama yaş 56 ±14 yıl idi. Preoperatif SVÇY gradiyenti 137 ± 45 mm Hg olup postoperatif dönemde 10 ± 17 mm Hg ya kadar düşüş gösterdi. Tüm hastalarda preoperatif olarak ortalama 3.1 şiddetinde (yayılma aralığı 0-4) mitral yetersizliği mevcuttu, ve postoperatif dönemde şiddeti 0.2 önemsiz dereceye kadar azalmıştır. Takip dönemimde, SVÇY gradiyenti düşük (6 ± 14 mm Hg) seviyelerde seyretti ve mitral yetersizliği önemsiz 0.4 derecede kaldı (tüm p değerleri <0.0001). Hastanede ölümler olmadı ve genel olarak reoperasyonlara ihtiyaç duyulmadı.

Sonuçlar: Çoğu zaman HKM'li hastalarda geniş anatomik varyasyonlar görülür. Bu varyasyonlar anlaşıldığı zaman, operatif yaklaşım düzeltme ve, basit ve kompleks patofizyolojisi olarak adlandırılan, spesifik yönlerin iyileşmesine yönelik olmalıdır. Tüm hastalarda, gerekli yerde ve yeterli büyüklüğünde yapılan septal miyektomi ile beraber mitral patolojisi belirlenip uygun tamiri yapıldığı zaman sağlam uzun süreli sonuçlar elde edilebilir. *(Anadolu Kardiyol Derg 2006; 6 Özel Sayı 2: 31-6)*

Anahtar kelimeler: Hipertrofik kardiyomiyopati, mitral kapak plikasyonu

Introduction

Early reports of individuals with signs and symptoms similar to those well known today associated with hypertrophic cardiomyopathy (HCM) date back to the nineteenth century (1). Autopsy reports from the same time recognized the pathology of severe left ventricular (LV) hypertrophy with outlet obstruction, often with near total cavity obliteration (2). This is not surprising since prevalence is 1 in 500 individuals (3).

Operative management of HCM required use of the heart lung machine and consequently, first reports first date from the mid nineteen-fifties (4). The classic septal myectomy by Morrow (5) remained the gold standard of surgical management, but inconsistent surgical technique and other pathophysiologic factors led to a significant failure rate for this procedure. Since the mitral valve was noted to play a dominant role in obstruction, mitral valve replacement had been advocated to relieve obstruction by some authors in all cases (6) and by others when the septum is thin < 18 mm or when myectomy fails to relieve obstruction. However, there is consensus that adequate myectomy and mitral repair is always preferable to mitral valve replacement.

The true pathophysiology of HCM has now been more clearly elucidated. Obstruction occurs because of mitral-septal contact during systole, and three primary causes promote this systolic anterior motion (SAM). Flow is directed around the hypertrophied septum towards the mitral valve anterior leaflet and pushes it into the outflow tract causing not only obstruction, but loss of systolic coaptation. This results in mitral insufficiency. The papillary muscles are often positioned more anteriorly and frequently bound to the asymmetrically hypertrophied septum. And lastly, the mitral anterior leaflet is often structurally abnormal. It is too long and redundant and protrudes in the outflow tract allowing early trapping by the flow stream during systole (7-10).

It is for this last reason that we have proposed adding an anterior leaflet horizontal plication, when necessary, to the operative management of HCM, along with an extended myectomy and papillary muscle release (11). We coined the phrase R-P-R for resection, plication and release, to be used in the management of HCM patients with complex pathophysiology (12). The horizontal anterior leaflet plication is easy to perform and does not require any additional incisions. It seems to not only shorten the anterior leaflet, but also stiffens it somewhat, further limiting its motion into the outflow tract (13).

Patients and Methods

Between 1997 and 2006, forty-two patients underwent surgery for obstructive HCM at St. Luke's/Roosevelt Hospital Center. The Divisions of Cardiology and Cardiac Surgery, jointly run an HCM Center, with the intent to not only deliver specific and individualized patient care, but also continue in depth study of the pathophysiology of the disease and improve management options, both medical and surgical. The Center follows approximately 400 patients; the vast majority are treated medically. Initial management of patients with symptomatic obstruction is β- blockade. If the patient is still symptomatic and still obstructed disopyramide is added (3, 16). If the patient cannot tolerate disopyramide a trial of verapamil is used. Individuals who fail pharmacologic therapy (defined as still obstructed with gradient at rest or after provocation >50 mm Hg and NYHA 3) are referred to surgery unless there are significant co-morbidities that might increase the risk of surgery. The majority of patients referred for surgery are those who do not tolerate the pharmacologic therapy because of severe side-effects or because it is ineffective. Elderly patients or those with co-morbidities may be referred for alcohol septal ablation or dual-chamber pacing.

Pre-operative evaluation

The mainstay of the pre-operative evaluation is transthoracic echocardiography (TTE). It is critical to elucidate the nature of the outflow tract obstruction. Important measurements include the thickness of the septum at the point of mitral-septal contact and beyond; the distance from the aortic annulus to beyond the point of mitral-septal contact (11,14). Though septal hypertrophy is usually asymmetric and dominant near the base of the heart, in some instances, the entire left ventricle can be uniformly hypertrophied and exhibit not only outflow obstruction but mid-ventricular obstruction as well. Additionally, the nature of the mitral leaflets is examined, including their length and thickness and the degree of mitral insufficiency. Severe structural abnormalities of the mitral valve may necessitate mitral valve replacement. The pre-operative transthoracic echocardiography (TTE) is also used to measure LVOT gradients, whether resting supine, standing, at exercise or after Valsalva maneuver.

A small number of patients who need further elucidation of anatomy have elective outpatient preoperative transesophageal echocardiography (TEE), but all patients have TEE in the operating room for both repeat pre-operative evaluation and post operatively to confirm adequacy of the procedure. Mitral insufficiency is graded from 1(trivial) to 4 (severe). These repeated examinations are used to assess the pathology under varying conditions. The decision to proceed with valvular plication and papillary muscle release is based on a combination of these echo findings and direct open visualization of redundancy of the leaflets, how slack they are, and the presence of abnormal papillary muscle attachments.

Operative Technique

Our operative technique has been described previously (12). Briefly reviewed, an extended myectomy is performed similar to that described by Messmer (15). A trefoil hook is introduced within the ventricular chamber as deeply as possible but must pass beyond the measured point of mitral-septal contact (Fig. 1). In rare instances the outflow tract is so small, that a preliminary resection is carried out closer to the aortic annulus just so enough room is achieved for deeper visualization. Unless the outflow tract is very narrow a #10 knife blade is used for the best chance at adequate resection with the first pass. Once the endomyocardium is violated it becomes relatively more difficult to resect any subsequent large amounts of muscle. That notwithstanding, additional portions of muscle are resected until it is felt by palpation that adequate amounts of muscle have been removed without creating an iatrogenic ventricular septal defect. Once the myectomy is complete, visualization within the ventricular cavity is remarkably improved and the abnormal papillary muscle attachments can be identified, if present and resected, along with portions of the papillary muscle itself (Fig 2). Care is used not to divide any chordal structures. Lastly, the anterior leaflet of the mitral valve is examined. It is best gauged for redundancy on the echocardiogram, but further confirmed by direct inspection. A truly slack leaflet will be easy to plicate. Anywhere from four to seven fine sutures of 5-0 polypropylene are placed in a horizontal orientation to shorten and stiffen the leaflet (Fig. 3). If the anterior leaflet is too thick or not sufficiently redundant, the sutures will not function to shorten the leaflet and just tear out. Care must be used when the sutures are tied down to forestall this eventuality. If the leaflet is very thin and floppy but not necessarily too long, then only 1 or 2 mm of tissue is used for the plication. The function of the plication then is more to stiffen the leaflet than to shorten it. On the other hand, a very long leaflet can be shortened as much as 4 or 5 mm without fear of post-operative insufficiency. In such a patient, shortening will often reduce the protruding portion of the leaflet that had extended past the coaptation point.

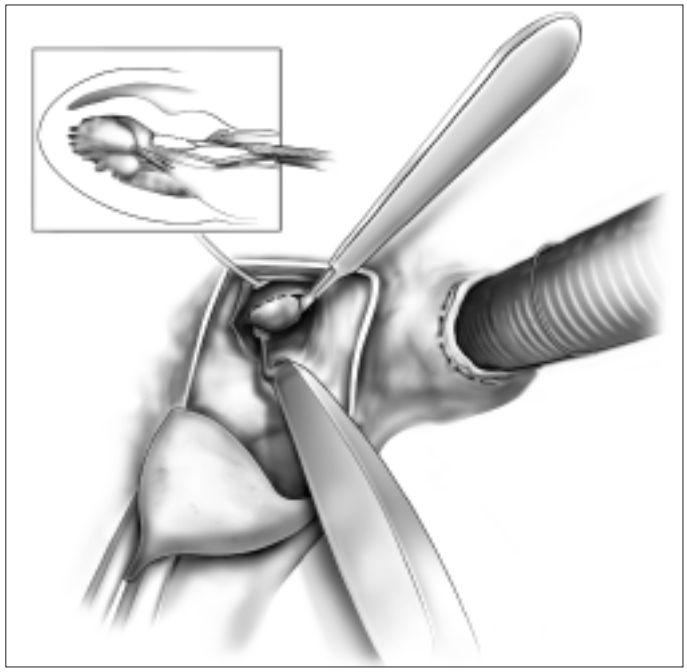

Figure 1. Resection of hypertrophied septum is carried out with a number 10 Blade knife, starting at the level of the right coronary ostia and moving anteriorly (leftwards). Traction is supplied by a trefoil hook placed deeply within the ventricular cavity

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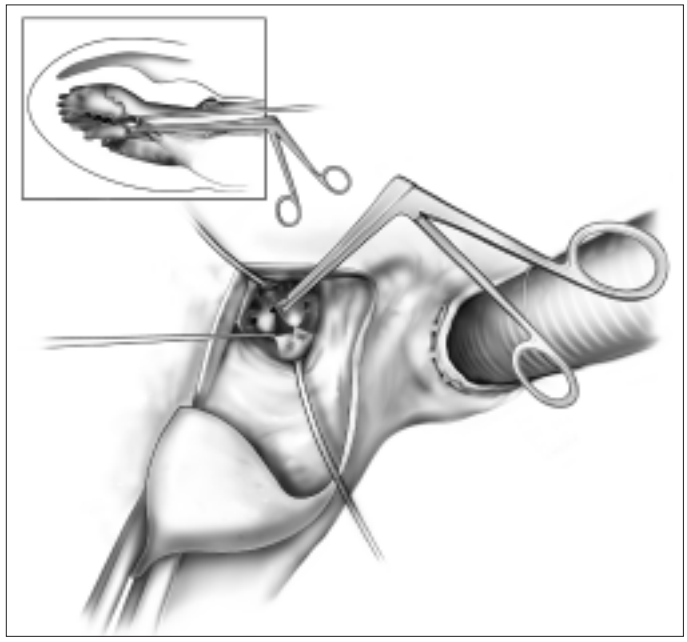

Figure 2. The hypertrophied papillary muscles are released and thinned with a pituitary rongeur. Often the papillary muscle has grown into the anterior septum or there are abnormal attachments, which can be divided without fear of causing mitral insufficiency

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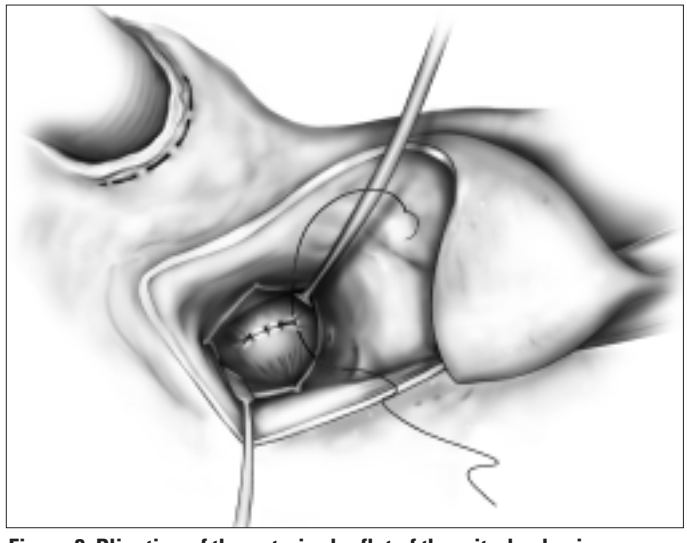

Figure 3. Plication of the anterior leaflet of the mitral valve is accomplished with the placement of four to six horizontal mattress sutures of 5-0 proline

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Anadolu Kardiyol Derg 2006: 6 Özel Sayı 2; 31-6
Anatol J Cardiol 2006: 6 Suppl 2; 31-6

The adequacy of the procedure is tested after the cessation of cardio-pulmonary bypass, but before the cannulas are removed. Once it is ascertained that loading conditions are appropriate, Doppler measurements are used to ascertain the presence or absence of an outflow tract gradient. The degree of mitral insufficiency is also examined. If these measurements show the appropriate resolution of a gradient and mitral leakage, the patient is provoked with high-dose dobutamine and the measurements are repeated. Cardiopulmonary bypass is reinstituted for persistent gradient greater than 30 mmHg, persistent mitral-septal contact, or mitral insufficiency of moderate or greater degree.

Follow-up

All patients are followed within our HCM program. All post-operative patients receive a TTE at their 3 month visit to assess LVOT gradient, mitral insufficiency and LV function. Subsequent TTE's are performed yearly, unless symptomatology dictates otherwise.

Statistical Analysis

Data is collected prospectively, presented as mean ± standard deviation. The range of data is also presented. Continuous variables were compared using paired Student's t test with significance accepted for p values of less than 0.05.

Results

Forty-two patients underwent surgery for HCM over a nine year period. The majority, twenty-one, underwent the full RPR procedure, seven had myectomy and papillary muscle release, seven had myectomy alone (three of these patients had mitral valve replacement for insufficiency unrelated to their HCM), six had myectomy and anterior mitral leaflet plication alone, and one patient required a mitral valve replacement as primary therapy for obstruction (Table 1). There were fourteen women (33%) and 28 men (67%) with a mean age of 54.5 ± 18 years of age. Other characteristics included coronary artery disease with prior myocardial infarction (34%), hypertension (41%), diabetes mellitus (24%) and nonsustained ventricular tachycardia (20%) (Table 2). Resting peak LVOT gradient measured by Doppler echocardiography was 135 ± 43 mm Hg (range 30 to 230 mm Hg). The mean degree of mitral insufficiency was 3.1 (range 1 to 4). All patients had SAM present on preoperative TTE or TEE. The average ejection fraction was 59% ± 12% (range 46% to 75%) with a preoperative New York Heart Association symptom class of 3.2 ± 0.8 (range 2 to 4) (Table 3).

Table 1. Operative Procedures

Complete RPR procedure	21
Myectomy and papillary muscle release	7
Myectomy alone*	7
Myectomy and anterior mitral leaflet plication	6
Mitral valve replacement	1

* Three patients had mitral valve replacement in this group as a concomitant procedure, unrelated to HCM pathology.
RPR - Resection-Plication-Release

Table 2. Patient Characteristics

Age, years	54.5 ± 18
Sex	
Male, n(%)	28 (67)
Female, n(%)	14 (33)
Coronary artery disease, % (with prior myocardial infarction)	34
Hypertension, %	41
Diabetes mellitus, %	24
Malignant ventricular arrhythmias, %	20

Patients often required concomitant procedures, including coronary artery bypass grafting (n = 10), radiofrequency ablation for atrial fibrillation (n = 5), aortic valve replacement (n = 4), mitral ring annuloplasty (n = 1), closure of atrial septal defect (n = 1), and resection of ascending aortic aneurysm (n = 1). Average aortic cross-clamp time was 104 ± 34 minutes with a cardiopulmonary bypass time of 152 ± 49 minutes. Two patients required placement back on cardiopulmonary bypass because of an unacceptable residual gradient and mitral insufficiency. In one patient, after additional resection of more muscle mass both from the septum and the base of the anterior papillary muscle, the gradient and mitral leakage resolved. In the other patient who underwent a full RPR procedure, there appeared to be persistent structural abnormality to the mitral anterior leaflet, causing significant insufficiency. After reinstituting cardio-pulmonary bypass, the left atrium was opened and an additional complex valvuloplasty was performed with the addition of an anterior-posterior leaflet coaptation suture (after Alfieri). The mitral insufficiency completely resolved.

One patient required mitral valve replacement as primary therapy for HCM. In that instance, the septum was only 1.5 cm in thickness, and structural abnormalities of the valve precluded any type of valvuloplasty. Three additional patients with obstructive HCM in our program underwent surgery and required concomitant mitral valve replacement. Severe structural abnormalities of the mitral leaflets and/or apparatus were the cause of insufficiency and could not be repaired with a valvuloplasty. Obstructive pathology was related to septal thickness and primary therapy for HCM was myectomy; though, replacement of the mitral valve also contributed to LVOT obstruction relief.

Follow up was 100%. There were no hospital deaths. Thirty-day mortality was also 0%. One patient, age 72, with multiple comorbidities including severe chronic obstructive pulmonary disease, died of respiratory failure at a rehabilitation institute. Patients were followed for a mean of 3.1 ± 2.1 years (range, 0.5 to 9). There were no other deaths at follow-up. Total mortality of the series to date was 1 of 42 patients, 2.4%.

NYHA class decreased from 3.2 ± 0.9 to 1.52 ± 0.6 (range 1 to 4; $p < 0.0001$). Most patients remained on a regimen of β-blockers. None required disopyramide and four patients were discharged on amiodarone. None of the patients have developed heart failure or required reoperation. Two patients required pacemaker for complete heart block. There were no other morbidities either during the post-operative period or subsequently during the follow-up period, including stroke or wound infection.

Length of stay (LOS) was 6.7 ± 3.4 days. This was lengthened in 4 cases by the need for implantation of an internal defibrillator. Indications include syncope and preoperative ventricular arrhythmias, one with pre-operative ventricular fibrillation and successful resuscitation. The devices were never implanted before post-operative day five.

Table 3. Summary of Results

	Pre-operative	Post-operative	p
New York Heart Association class	3.2 ± 0.8	1.52 ± 0.6	< 0.0001
LVOT obstruction gradient, mm Hg	135 ± 43	10 ± 18*	< 0.0001
Mitral insufficiency **	3.1	0.2	< 0.0001
SAM, %	100	0	< 0.0001
Ejection fraction, %	59 ± 12	56 ± 14	NS

* no significant change in those patients receiving dobutamine for provocation post-bypass
** scale 1-4
LVOT- left ventricular outflow tract, SAM- systolic anterior motion

Initial postoperative TEE in the operating room confirmed marked reduction in LVOT gradient to 10 ± 18 mm Hg (range 0 to 40; $p < 0.0001$). All patients were pharmacologically stressed to elicit a provocable gradient. Fifteen of the patients had intraoperative provocation with high dose dobutamine. In that group, the initial LVOT gradient was 9 ± 17 mmHg (range 0 to 40; $p < 0.0001$), and after provocation the mean gradient was 12 ± 16 mm Hg (range 0 to 40; $p < 0.0001$). Mitral valve insufficiency also markedly decreased from 3.1 to 0.2 (range 0 to 1; $p < 0.0001$). The results were the same in the group who were provoked with dobutamine.

At three month follow-up, the gradient continued to fall, measured under normal physiologic condition. Left ventricular outflow gradient decreased further to 6 ± 14 mm Hg for the whole group (range 0 - 36; $p < 0.0001$). Likewise, mitral insufficiency remained low at 0.4 (range 0 - 1, $p < 0.0001$) (Table 3).

Discussion

Hypertrophic cardiomyopathy patients with obstruction can be severely debilitated, often unable to exercise, walk uphill or upstairs, and occasionally are short of breath when eating. The fear of sudden death can be ever-present.

Improvements in medical management have relieved most patients' symptoms without surgery. Two-thirds of the patients are successfully treated without any invasive intervention (16). Negative inotropes work by decreasing ejection acceleration and decreasing drag (pushing) forces on the protruding mitral valve leaflet (17). Other studies suggest this decrease in dP/dt is associated with ventricular dilatation, a drop in ejection fraction though with a concomitant increase in cardiac index (18). But, residual gradients persist and increase in cardiac index is inconsistent and many patients are intolerant of this physiologic depression in cardiac function. Even those individuals who tolerate pharmacologic therapy, may have their physiologic state somewhat decreased, especially after exercise.

Surgical management, on the other hand, relieves the mechanical obstruction, allows normal outflow dynamics and eradicates the mitral insufficiency. Patients, though kept on a low dose of β-blockers, are functionally normal and encouraged to have normal physical activity. Surgery remains the gold-standard of management for HCM (19).

The risk of sudden death is not, however, as clearly understood. After successful surgery the risk of sudden death is low, about 1%/year (20). Although muscle mass in the asymmetrically hypertrophied septum is resected, there remains large amounts of muscle with disorganized fibers and fibrosis thought to contribute to the risk of fibrillation and sudden death. Relieving the obstruction appears to decrease this risk, (21) though one would need randomized trials to prove this, which are not likely to be done for ethical reasons. The known risks of sudden death include a confirmed prior episode of ventricular fibrillation, a family history of sudden death or a septum greater than 3.0 cm. Patients in our center receive defibrillators postoperatively if they have two or more risk factors, regardless of their post-operative gradient.

The biggest problem that occurs with surgical management is the incomplete operation. Too many surgeons misinterpret or are unaware of the procedure as described by Morrow in 1961 (22), and a completely inadequate myectomy or even just a myotomy is performed. Even if the Morrow procedure is properly performed a significant number of failures will occur. As detailed above, the pathophysiology can be varied which is why alternative or additional procedures which address the mitral valve are necessary to assure successful resolution of gradient, SAM and mitral insufficiency (23). The extended myectomy assures that adequate muscle is resected in the appropriate location (15). In many patients the 1 cm trough as described by many surgeons in the 1950's and 1960's will be insufficient. In other individuals the septum may not be overly hypertrophied but the papillary muscles may be too anteriorly displaced. In others, the mitral valve anterior leaflet may be so elongated that no amount of muscle resection may be enough to relieve the obstructive phenomenon. Hence the R-P-R procedure as described here-in.

We do not believe that obstruction recurs. There is no data in the literature suggesting such regrowth and there has been no recurrence of obstruction in any of our patients during this follow-up period. "Regrowth" of the septum may actually reflect an initially inadequate operation. After the proper myectomy procedure the raw myocardium heals with flat scar tissue, limiting any ability for regrowth. In our series, no patients required reoperation, but several patients operated on elsewhere have persistent gradients and are followed with medical therapy. We have operated on two patients who had unsuccessful alcohol ablation. In these instances, areas of fibrotic, scarred endomyocardium were visualized, although not in the right location.

We have documented excellent results in mid-term follow-up for surgery in HCM. Thirty day operative mortality was 0% and long-term total mortality to date in this series was 2.4%, with no cardiac mortality caused by HCM. Septal myectomy and the R-P-R procedure specifically, are safe, reproducible procedures. The results are long-lasting, with complete symptomatic relief.

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