

## Current Strategies of Mitral Valve Repair

Dan Spiegelstein MD, Probal Ghosh MD, Leonid Sternik MD, Salis Tager MD, Amihai Shinfeld MD and Ehud Raanani MD

Department of Cardiothoracic Surgery, Sheba Medical Center, Tel Hashomer, Israel  
Affiliated to Sackler Faculty of Medicine, Tel Aviv University, Ramat Aviv, Israel

**Key words:** mitral valve repair, valve surgery, surgical techniques, minimal invasive approach, degenerative mitral valve

### Abstract

**Background:** During the last decade new surgical techniques for mitral valve repair were developed. We have been using those techniques in order to widen the spectrum of patients eligible for MV repair.

**Objectives:** To assess the operative and mid-term results of using a wide variety of surgical techniques.

**Methods:** From January 2004 through December 2006, 213 patients underwent MV repair in our institution. Valve pathology was degenerative in 123 patients (58%), ischemic in 37 (17%), showed annular dilatation in 25 (12%), endocarditis in 16 (8%), was rheumatic in 13 (6%), and due to other causes in 14 (7%). Preoperative New York Heart Association class was  $2.35 \pm 0.85$  and ejection fraction  $53 \pm 12\%$ . Isolated MV repair was performed in 90 patients (42%) and 158 concomitant procedures were done in 123 patients (58%). A wide variety of surgical techniques was used in order to increase the number of repairs compared to valve replacement.

**Results:** There were 7 in-hospital deaths (3.3%). NYHA class improved from  $2.19 \pm 0.85$  to  $1.4 \pm 0.6$ , and freedom from reoperation was 100%. Echocardiography follow-up of patients with degenerative MV revealed that 93% of the patients (115/123) were free of mitral regurgitation greater than 2+ grade. In patients operated by a minimal invasive approach there were no conversions to sternotomy, no late deaths, none required reoperation, and 96% were free of MR greater than 2+ grade. The use of multiple surgical techniques enabled the repair of more than 80% of pure MR cases.

**Conclusions:** MV repair provides good perioperative and mid-term results, and supports the preference for MV repair over replacement, when feasible. Multiple valve repair techniques tailored to different pathologies increases the feasibility of mitral repair.

*IMAJ 2007;9:303-309*

Mitral valve repair is currently considered to be superior to MV replacement. It offers improved perioperative and long-term survival, greater regression of left ventricular dimensions and maintenance of left ventricular function. The durability of mitral repair is at least equivalent to replacement and offers freedom from long-term anticoagulation. [1,2] Greater understanding of the structure, function and pathology of the MV in recent decades improved the surgical results of MV repair, leading to excellent long-term survival. Surgical repair of the MV is the preferred goal of modern MV surgery, with the use of prosthetic

valves reserved for a select patient population. Techniques of MV repair have undergone many modifications and improvements in the last 50 years, which have made it a more predictable and reproducible method.

Valve repair techniques popularized in the last decade include the use of artificial chordae with polytetrafluoroethylene sutures for anterior and posterior mitral leaflet pathology, use of posterior and semi-rigid rings, novel approaches and devices for ischemic mitral regurgitation repair, a minimal invasive approach (mini-thoracotomy), and most recently new percutaneous approaches are starting to evolve for the repair of mitral regurgitation. These technical improvements have enabled the successful repair of more complex MV disease. This study describes our recent experience using various techniques and approaches for MV repair.

### Patients and Methods

This is a retrospective review of 213 consecutive MV repairs in our institution. All patients in whom MV repair was done as a primary or secondary procedure were included in the study.

Between 1 January 2004 and 31 December 2006, a total of 464 patients underwent cardiac surgery involving the MV at Sheba Medical Center, Tel Hashomer. MV replacement was performed in 251 patients, and 213 patients who underwent MV repair were included in the study. This number represents 80% of patients who underwent surgery due to pure MR. In 90 patients (42%) MV repair was the only intervention, and 123 patients (58%) underwent concomitant cardiac procedures (54 coronary artery bypass graft, 27 tricuspid valve repair, 28 aortic valve replacement/repair, 10 ascending aorta/aortic root replacement/repair, 6 atrial septal defect closure, 31 Maze, and 2 myectomy). Twelve patients (5.6%) had previous cardiac surgery. Most operations were performed electively, but 20 patients (9%) required urgent surgical intervention because of intractable congestive heart failure, active infective endocarditis, or both. A minimal invasive approach was used in 30 patients (14%), of whom 23 (77%) had degenerative disease. Preoperative characteristics are detailed in Table 1.

### Surgical technique

The surgical technique for repairing the valve was selected according to the etiology of the failing valve, and the abnormal segments of the valve.

Various techniques were used for degenerative MR. These included triangular or quadrangular resection of the posterior mitral

MV = mitral valve

NYHA = New York Heart Association

MR = mitral regurgitation

**Table 1.** Patients' characteristics

	All patients (n=213)	Degenerative MV (n=123)	All other etiologies (n=90)	P
Age (yrs)	60 ± 13	59.7 ± 12	60.4 ± 14.6	NS
Male/Female	142/71	87/36	55/35	NS
Hypertension	114 (54%)	65 (53%)	49 (54%)	NS
Hyperlipidemia	73 (34%)	38 (31%)	35 (39%)	NS
Diabetes mellitus	39 (18%)	15 (12%)	24 (27%)	0.011
Chronic obstructive pulmonary disease	7 (3.3%)	4 (3%)	3 (3.3%)	NS
Peripheral vascular disease	12 (5.6%)	4 (3%)	8 (9%)	NS
Smoking	49 (23%)	28 (23%)	21 (23%)	NS
Cerebrovascular	13 (6%)	6 (5%)	7 (8%)	NS
<b>Etiology*</b>				
Degenerative	123 (58%)	123 (100%)	0 (0%)	< 0.001
Ischemic	37 (17%)	0 (0%)	37 (43%)	< 0.001
Rheumatic	13 (6%)	1 (0.8%)	12 (13%)	< 0.001
Endocarditis	16 (8%)	6 (4.9%)	10 (11%)	NS
Annular dilatation	25 (12%)	5 (4.1%)	20 (22%)	< 0.001
Prosthetic dysfunction	6 (3%)	0 (0%)	6 (6.7%)	0.004
Other	8 (4%)	0 (0%)	6 (6.7%)	0.004
Previous cardiac surgery	12 (5.6%)	1 (0.8%)	11 (12.2%)	< 0.001
Standard Euroscore	5.2 ± 3.1	4.3 ± 2.8	6.7 ± 3.1	< 0.001
Logistic Euroscore	6.6 ± 9.0	4.9 ± 8.3	9.4 ± 9.6	0.002
Preoperative NYHA	2.35 ± 0.85	2.19 ± 0.85	2.57 ± 0.80	0.008
Preoperative EF (%)	53 ± 12	58 ± 8	46 ± 14	< 0.001
Preoperative creatinine (mg/dl)	1.16 ± 0.85	1.1 ± 0.3	1.25 ± 1.28	NS
Elective/Urgent	193/20	117/6	76/14	0.009

Values presented are (mean ± SD)

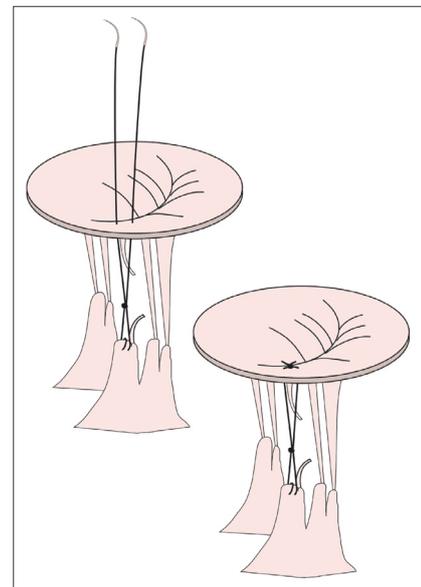
\* 8% had more than one etiology

leaflet, commissural closure with primary suturing, implantation of artificial chordae to anterior or posterior prolapsing leaflets, and Alfieri edge-to-edge repair between anterior and posterior leaflets. Most patients with PML pathology underwent triangular resection of the prolapsing segment with posterior band, whereas in most cases of anterior mitral leaflet pathology we used artificial chordae to repair the prolapsing segment [Figure 1]. In cases of chordal elongation of PML without leaflet prolapse, or extensive prolapse of the PML, we used artificial chordae to repair the posterior leaflet. Almost all patients with degenerative mitral regurgitation underwent annuloplasty.

Ischemic MR repair included annuloplasty with closed rings in 100% of cases, with or without myocardial revascularization. In one case we used artificial chordae for papillary muscle displacement toward the mitral annulus. MR caused by annular dilation was repaired by annuloplasty in 100% of cases.

Endocarditis MR repair techniques included excision of all infected and inflamed tissue, and repair of the missing tissue with fresh pericardial patch or primary suturing. Some patients needed implantation of artificial chordae and most underwent annuloplasty.

PML = posterior mitral leaflet



**Figure 1.** Technique of artificial chordae for repairing prolapsing mitral valve segment.

Rheumatic valve repair techniques included commissural fusion release, detachment of papillary muscle fusion, resection of prolapsing segments and annuloplasty.

A minimal invasive approach was used in selected patients with optimal conditions for valve repair. None of them required any other cardiac intervention, and all were elective cases. The most common pathology in patients operated by the minimal invasive approach was isolated PML P2 prolapse. The approach was through a 5 cm lateral incision in the fourth right intercostal space. Cardiopulmonary bypass cannulation was performed through the femoral vessels and right internal jugular vein. A specialized balloon-tipped endoaortic catheter was introduced through femoral arterial cannula (Endoclamp™, Heartport, Redwood City, CA, USA) and positioned in the aortic root. Aortic clamping was performed by inflating the Endoclamp balloon in the ascending aorta, and cardioplegia was given in the aortic root through the Endoclamp aortic catheter.

All patients with chronic atrial fibrillation had a concomitant Maze procedure. Patients were anticoagulated with warfarin sodium during the first 3 postoperative months if they were in sinus rhythm and permanently if they were in atrial fibrillation. Detailed operative data are displayed in Table 2.

#### Follow-up

Demographic, morphologic, echocardiographic and surgical data were obtained from our institute medical records. Patients (or family members) with degenerative etiology of the mitral disease were contacted for clinical follow-up. Echocardiography data were obtained from our medical institute database, patients, or ambulatory medical services.

#### Data analysis

All statistical analyses were performed with the SPSS software (SPSS 10.0 for Windows). Group statistics were expressed as

**Table 2.** Perioperative data

	All patients (n=213)	Degenerative MV (n=123)	All other etiologies (n=90)	P
Extracorporeal circulation time (min)	119 ± 44	114 ± 38	127 ± 53	0.063
X-clamp time (min)	88 ± 29	87 ± 29	89 ± 29	0.551
Minimal invasive approach	30 (14%)	23 (19%)	7 (8%)	0.02
<b>Concomitant procedure*</b>	123 (58%)			
Coronary artery bypass	54 (25%)	14 (11%)	40 (44%)	< 0.001
Tricuspid valve repair	27 (13%)	15 (12%)	12 (13%)	NS
Aortic valve replacement/repair	28 (13%)	6 (5%)	22 (24%)	< 0.001
Ascending aorta replacement/repair	10 (5%)	4 (3.3%)	6 (7%)	NS
Maze procedure	31 (15%)	20 (16%)	11 (12%)	NS
Closure of atrial septal defect	6 (3%)	3 (2.4%)	3 (3.3%)	NS
Myectomy	2 (1%)	1 (0.8%)	1 (1.1%)	NS
Mortality	7 (3%)	2 (1.6%)	5 (5.6%)	NS
Neurology event	5 (2%)	2 (1.6%)	3 (3.3%)	NS
Acute renal failure	12 (6%)	5 (4%)	7 (8%)	NS
ICU time (hr)	49 ± 82	31 ± 23	76 ± 123	0.003
Ventilation time (hrs)	21 ± 69	9 ± 5	39 ± 106	0.023

Values presented are (mean ± SD)

\* Some patients had more than one concomitant procedure

mean ± 1 standard deviation. Continuous variables were compared with Student's *t*-test. Categorical variables were compared with the chi-square test. *P* values less than 0.05 were considered significant.

## Results

### Patient characteristics

The patients' mean age was 60 ± 13 years (range 17–87), and 142 (66.6%) were males. Degenerative disease was the most prevalent cause of MR and was present in 123 patients (58%). Ischemic MR was present in 37 (17%), annular dilatation in 25 (12%), endocarditis in 16 (8%), rheumatic heart disease in 13 (6%) and other causes in 14 (7%). Twelve patients (5.6%) had previous cardiac surgery in which one patient was operated on by the minimal invasive approach. Preoperative New York Heart Association score was 2.35 ± 0.85 and ejection fraction 53 ± 12% for all patients. NYHA class was 2.19 ± 0.85, 2.76 ± 0.83 and 2.86 ± 0.36 in degenerative disease, ischemic and annular dilatation, respectively; and EF was 58 ± 8, 39 ± 14 and 45 ± 13 in degenerative disease, ischemic and annular dilatation, respectively. Twenty percent of patients were asymptomatic at surgery (NYHA class I). Standard and logistic Euroscore were 5.2 ± 3.1 and 6.6 ± 9.0% respectively.

### Hospital mortality

Seven patients (3.3%) died within 30 days of the operation. Three of them died perioperatively because of low cardiac output syndrome. Another three patients had sepsis-induced multiple

organ failure. One patient died 3 days postoperatively because of delayed tamponade and sudden cardiac arrest in the ward. Five of them had ischemic or annular dilatation MR, one had MV repair for degenerative MR and concomitant aortic root repair, and one had degenerative MR with remote endocarditis and underwent MV and tricuspid valve repair. There was no operative or in-hospital mortality in the subset of patients with isolated degenerative MV disease, or in patients operated on by the minimal invasive approach.

### Perioperative data

The mean operating, cardiopulmonary bypass, and aortic cross-clamp times averaged 295 ± 89, 119 ± 44, and 88 ± 29 minutes respectively. The mean intensive care unit stay and ventilation time was 49 ± 82 and 21 ± 69 hours, respectively (median 23 and 9 hours), with 57% being discharged from the ICU within 24 hours and an additional 22% within 48 hours. Prolonged inotropic support was needed in 30% of the patients, and intraaortic balloon pump was inserted in five patients. Other major post-operative complications were cerebrovascular accident/transient ischemic attacks in 5 patients (2.3%), and acute renal failure in 12 (5.6%) of whom 4 required dialysis. Maximal mean postoperative creatinine level was 1.3 ± 1.6 mg/dl. In the patients operated with the minimal invasive approach there were no conversions from mini-thoracotomy to median sternotomy.

### Patient survival and clinical follow-up

Clinical and echocardiographic follow-up were complete in the subset of patients with degenerative MV disease (123 patients). Mean follow-up was 13 ± 6.8 months. During follow-up there were no reoperations for valve repair failures. NYHA functional class improved from 2.19 ± 0.85 to 1.4 ± 0.6 in this group. Late echocardiography follow-up revealed that 93% of patients (115/123) were free of MR greater than 2+ grade. Comparison of patients with PML pathology to those with AML or bi-leaflet pathology revealed similar perioperative and mid-term results. Mean NYHA functional class at follow up was 1.2 ± 0.6 and 1.48 ± 0.6 in PML repair and AML/bi-leaflet repair, respectively. Echocardiography follow-up demonstrated that 2 of 38 patients (5%) had MR greater than 2+ grade in AML/bi-leaflet repair compared to 6 of 85 (7%) in PML repair (not significant). Detailed operative and follow-up data for degenerative MV disease are provided in Table 3.

In the subset of patients with degenerative MV disease operated by the minimal invasive approach (n=23), there was no late mortality during follow-up and none of them required reoperation for valve repair failure. NYHA functional class improved in this subset of patients from 1.9 ± 0.9 to 1.4 ± 0.6. Echocardiography follow-up revealed that 96% of patients (22/23) were free of MR greater than 2+ grade.

In the 16 patients with MV infective endocarditis (remote, active or abscess), there was one perioperative death. Among the

NYHA = New York Heart Association  
EF = ejection fraction

ICU = intensive care unit  
AML = anterior mitral leaflet

**Table 3.** Degenerative MV disease – patients' characteristics, operative data and follow-up

	All degenerative MV (n=123)	Posterior leaflet (n=85)	Anterior or bi-leaflet (n=38)	P
Age (yrs)	59.7 ± 12	59 ± 12	61 ± 13	0.41
Male/Female	87/36	58/27	29/9	0.36
Previous cardiac surgery	1 (0.8%)	0 (0%)	1 (2.6%)	NS
Standard Euroscore	4.3 ± 2.8	4.2 ± 2.9	4.4 ± 2.7	0.76
Logistic Euroscore	4.9 ± 8.3	5.0 ± 9.2	4.5 ± 5.6	0.76
Operative time (min)	285 ± 86	301 ± 87	250 ± 76	0.003
Extracorporeal circulation time (min)	114 ± 38	114 ± 38	114 ± 38	0.92
Aortic clamp time (min)	87 ± 29	85 ± 28	90 ± 31	0.44
Minimal invasive approach	23 (19%)	23 (27%)	0 (0%)	< 0.001
Annuloplasty	117 (95%)	81 (95%)	36 (95%)	NS
Posterior leaflet resection	91 (74%)	79 (93%)	12 (32%)	< 0.001
Alfieri edge-to-edge repair	5 (4%)	0 (0%)	5 (13%)	< 0.001
Artificial chorda/PTFE	47 (38%)	16 (19%)	31 (82%)	< 0.001
MR grade 2+ at follow-up	8 (6.5%)	6 (7.1%)	2 (5.3%)	NS
Follow-up NYHA	1.4 ± 0.6	1.48 ± 0.6	1.2 ± 0.6	NS

Values presented are (mean ± SD)

PTFE = polytetrafluoroethylene.

survivors, none had recurrence of infective endocarditis or any need for reoperation for valve repair failure.

## Discussion

Mitral valve surgery gradually evolved over the period of the last half century. Initially, most surgeons replaced the mitral valve in patients with MR. It is now accepted that the long-term outcome for repair is superior to that for replacement.

The first major advance in mitral surgery came with functional classification of MV pathologies as proposed by Carpentier [3], who divided the MV disease process into three types: type I – normal leaflet motion with annular dilation or leaflet perforation; type II – leaflet prolapse, chordal rupture/elongation, or papillary muscle rupture/elongation; type III – restricted leaflet motion with commissural fusion, leaflet thickening, or chordal fusion or thickening. The abnormal leaflet motion or abnormal valvular apparatus results in MV incompetence. Subsequent structural delineation (A1, A2, A3, P1, P2, P3, ALC, PMC) further enhanced allocation of valve architectural lesions.

With the new techniques that were developed in the 1990s for MV repair, cardiac surgeons have a wide arsenal of repair techniques for various valve pathologies, and nowadays over 90% of pure non-rheumatic MR requiring surgical intervention are considered to be reparable by experienced surgeons.

Understanding the underlying pathological features is important for determining whether mitral valve repair is feasible, how the valve should be repaired, and the prospect for long-term durability of the repair.

LV = left ventricular

## Degenerative MR

Degenerative MV disease is currently the most common cause of MR in developed countries. MV repair is associated with very good short and long-term outcomes in these cases. Gillinov and Cosgrove [2] reported 0.3% surgical mortality and 93% freedom from reoperation. There is a longstanding debate regarding the timing of surgery in degenerative MV disease, and the surgical treatment for asymptomatic patients with severe degenerative MR. It has been shown that early surgical treatment compared to conservative management can improve long-term survival and reduce cardiac mortality and morbidity [4]. Even in patients with coexisting coronary heart disease, repair of severe MR is superior to MV replacement [5].

Asymptomatic patients with severe degenerative MR should be recommended for early surgical intervention, to prevent LV dysfunction [6]. Long-term results of surgical MV repair in asymptomatic patients is better compared to those in symptomatic patients, and 15 year survival for those asymptomatic patients was identical to the general population matched for age and gender [7].

In the early era of mitral repair, only the PML pathologies were considered for durable satisfactory repair. Until the early 1990s, AML pathology involving more than one-sixth of the AML surface was considered beyond repair. As surgeons gained more experience, newer techniques for repairing the AML became common and the durability of those newer techniques was proven. Now almost all degenerative mitral valves can be repaired. With transesophageal echocardiography evaluation of the MV by an experienced team, 95% of degenerative MV requiring surgical repair can be repaired.

## Posterior leaflet repair

The most common posterior leaflet problem is ruptured chordae or prolapse of the middle scallop P2. The traditional technique for this repair is excision of the prolapsing tissue of the PML by quadrangular or triangular resection. Quadrangular resection usually requires leaflet detachment from the annulus and a sliding plasty to repair the resulting defect. Furthermore, a quadrangular resection/sliding plasty requires additional steps, creates a longer suture line, and is more prone to technical failure at the annular level. The triangular defect is repaired with interrupted or continuous sutures [8]. In cases where the PML is very wide and redundant, systolic anterior motion can result, causing left ventricular outflow obstruction. In such cases a sliding plasty of the posterior leaflet should be performed, which involves detaching the PML from the annulus on both sides of the quadrangular resection, lowering the height of the posterior leaflet and resuturing it closer to the opposite segment.

In our series the most common repair for posterior prolapse was triangular resection and primary closure of the resected portion of the posterior leaflet with prolene 4-0. The repair was completed with annuloplasty and a flexible posterior band. The posterior band was first described in 1964 by Belcher, but came into common practice only in the 1990s after the introduction of the Cosgrove-Edwards annuloplasty system [9]. In almost

all cases we used flexible rings, which have been shown to preserve LV function, in MR secondary to degenerative MV [10].

We also used artificial chordae to repair complex PML pathologies (16/85 patients) when there was not enough tissue to resect in the posterior leaflet (fibro-elastic deficiency syndrome), or in cases in which there was a very wide segment that was prolapsing.

### Anterior leaflet repair

The anterior leaflet is larger than the posterior but has a shorter attachment to the mitral annulus. It is in direct continuity with the fibrous skeleton of the heart, and the annulus portion attached to the anterior leaflet is considered relatively non-distensible. Those anatomic characteristics make the repair of the anterior leaflet more complicated than posterior leaflet repair [11]. The surgical techniques for AML repair evolved over the years. Resection of more than one-sixth of the AML is usually not feasible because there is not enough tissue for reconstruction of the prolapsing segment. Earlier techniques included chordal shortening and chordal transfer [3]. Chordal shortening is technically difficult, and there are reports of late failure of this repair, which was the reason cardiac surgeons abandoned this technique [12].

Chordal transfer is also a challenging technique, especially in cases where there is bi-leaflet prolapse. Chordal transfer involves moving normal chordae from the posterior leaflet (flip-over) or secondary chordae on the anterior leaflet to correct the prolapsed segment of the anterior leaflet. Since the degenerative disease is not limited only to the elongated or ruptured chordae, but extends to whole valve and subvalvular tissue, the transferred chordae are prone to rupture, resulting in late failure of the repair.

The concept of replacing the elongated or ruptured chordae by artificial chordae (fashioned out of silk, pericardium, etc.) was introduced many years ago. This technique did not gain popularity for some time, until a suitable material like polytetrafluoroethylene was found to be used for the neo-chordae. PTFE sutures are highly durable. They are rapidly covered by endocardium and fibroblasts and become non-thrombogenic. Postoperative transesophageal echocardiography can hardly distinguish between the native chordae and artificial chordae. The long-term durability of anterior leaflet repair with PTFE sutures has been demonstrated by several authors [13,14].

In our series we used PTFE sutures to repair the prolapsing AML in 31 of 38 patients with AML pathology. During follow-up, none of them required reoperation, and 36 of the 38 (95%) were free of MR greater than 2+ grade. In one MV repair failure, annuloplasty was not feasible due to severe mitral annulus calcification, and in the second failure the Alfieri edge-to-edge repair was used. Our follow-up demonstrates similar mid-term echocardiography results in patients with isolated PML prolapse and patients with involvement of the anterior leaflet. Those results are promising, although long-term follow-up is still needed

to assess the quality and durability of the repair for anterior leaflet.

### Alfieri edge-to-edge repair

In 1995 Alfieri introduced a novel way for repairing mitral leaflet prolapse using the "edge to edge technique." In this technique the free edges of the leaflets at the site of regurgitation, which can be near the commissure or the central part, are approximated with a single suture. Despite a reduction in MV effective orifice area, in Alfieri's experience no patient required reoperation for MV stenosis [15]. In patients with ischemic MR or annular dilation MR (dilated cardiomyopathy), addition of the edge-to-edge technique to the undersized annuloplasty can significantly improve the durability of the repair [16]. Mid-term results of this technique in various MV pathologies are promising considering the high prevalence of complex anatomic lesions [17], but there is a burning debate on the role of edge-to-edge technique in MV repair. We rarely used the edge-to-edge repair in our series. It was mainly used when technical difficulties in performing our usual techniques arose during surgery, or as a bail-out procedure to minimize operating time when intraoperative transesophageal echocardiography result was not satisfactory.

### Chronic ischemic MR

Ischemic mitral regurgitation is also referred to as functional MR, as opposed to organic MR, because of the normal appearance of the MV leaflets. In chronic IMR, the valve, the leaflets and the subvalvular apparatus appear normal. Chronic IMR is therefore not a disease of the valve *per se*, but rather a component of ischemic disease of the left ventricle. The indications for surgery in chronic IMR are not yet well defined. It is generally agreed that patients undergoing revascularization cardiac surgery with moderate-to-severe MR should also undergo concomitant MV surgery (repair or replacement). Though some authors have proposed the concept of using biodegradable annuloplasty in even mild IMR to prevent future worsening of MR grade, it is still controversial whether CABG patients with MR up to grade 2 should undergo concomitant MV surgery. It is well known that concomitant MV surgery increases the perioperative risk twofold compared to the risk without it [18]. The results for MV replacement led cardiac surgeons to start performing MV repair for IMR. Currently, there is general consensus in the cardiac surgery community that MV repair results in lower perioperative mortality than MV replacement and should therefore be performed whenever possible.

The most common surgical procedure currently performed for repairing chronic IMR is undersized mitral annuloplasty. Undersizing the mitral annulus will result in increased leaflet coaptation and decreased regurgitation. Isolated annuloplasty solves the problem only at the annular level by forcefully bringing the leaflets together. The issue of leaflet tethering, which results from apical and lateral displacement of the papillary muscles secondary to progressive left ventricle dilation, is not solved

PTFE = polytetrafluoroethylene

IMR = Ischemic mitral regurgitation  
CABG = coronary artery bypass graft

by isolated annuloplasty and can cause late recurrence of MR. Surgical approximation of the papillary muscles may be necessary in addition to annuloplasty to reduce late recurrence of MR. The type of ring that should be used for undersized annuloplasty is also unclear, with several authors arguing the merits of rigid versus flexible and complete versus incomplete [19]. We used the annuloplasty technique in all our patients undergoing repair for IMR or annular dilation MR, and in one case we did an approximation of the papillary muscles towards the mitral annulus. Our perioperative mortality in this subset of patients is 5/62, which is similar to other published data [20].

### Annuloplasty

The annuloplasty ring is an important additional component in each kind of repair for almost all MV pathologies. Its overall role is to reduce the size of the annulus and reduce the tension on the sutures while providing flexibility and mobility at the same time [21]. Almost any mitral repair is completed with annuloplasty, which increases leaflet coaptation and reinforces the annulus to prevent future dilatation. It is known that annuloplasty improves the durability of the valve repair.

Carpentier et al. [22] introduced the concept of using a complete rigid ring to reduce the tension along the entire mitral annulus. In patients with chronic MR secondary to degenerative MV, the flexible rings have been shown to improve left ventricular function, compared to rigid rings [10]. Patients with IMR and heart failure ( $EF \leq 30\%$  and no primary mitral pathology) have lower risk of recurrent MR and reduced need for reoperation when a non-flexible ring is used compared to a flexible ring [23]. The choice of annuloplasty ring is largely a matter of surgeon preference, and the valve pathology. In degenerative MR, we chose to use a flexible posterior band, while in IMR we used rigid closed rings. In cases where an annuloplasty ring/band is implanted, anticoagulation for 3 months is recommended, unless the patient is at high risk for hemorrhagic complication.

### Infective endocarditis

Surgical treatment of infective endocarditis has also undergone major developments in the last two decades. In the early era of surgical treatment for infective endocarditis, MV replacement was considered to be the gold standard. The growing experience and modern techniques in valve repairs, mainly in degenerative MV disease, enabled cardiac surgeons to repair complex MV pathologies, including infected MV, with lower hospital mortality and improved long-term outcome compared with replacement [24]. In the last decade, repair of infected MV has become more and more popular, with excellent long-term results and the same, or better rate of infection recurrence. In the 16 patients (8%) in whom we performed valve repair, 5 had involvement of the anterior leaflet, and we used artificial chordae in 3 cases.

### Minimal invasive approach

Our data show in the subset of isolated PML prolapse that the durability of MV repair with the minimal invasive approach is as

good as that with the standard median sternotomy approach. The clinical and echocardiography follow-up data demonstrated that the quality of MV repair was not affected and valve repair durability not impaired. We observed that only 1 of 23 patients (4.3%) in the minimal invasive subset had MR of grade 2+ in the late follow-up. Improvements in technology and surgical instruments and the increase in operative experience have reduced the complications rate of minimal invasive cardiac surgery. This approach is now considered as safe as standard median sternotomy in the hands of experienced surgeons, but MV repair by the minimal invasive approach with all its known advantages must always be weighed against the technical difficulties in repairing the valve with limited exposure. It is anticipated that as more experience is gained, more complex cases (bi-leaflet pathology, endocarditis, etc.) will be performed using the minimal invasive approach. Even complex MV repair with the minimal invasive approach has been shown to be safe and feasible [25], although the quality and long-term durability of complex valve repair has not yet been clearly demonstrated. Patients with complex MV pathology who are suitable for valve repair should be carefully selected for the minimal invasive approach.

### Conclusions

We have introduced several contemporary techniques and approaches for repairing the mitral valve in the most prevalent etiologies. Our MV repair results compared to the results of MV replacement support previous data on the superiority of outcome in patients undergoing repair over replacement. We use a wide arsenal of MV repair techniques for almost all valve pathologies, tailoring the best approach and technique for the valve pathology and for the patient.

### References

1. Suri RM, Schaff HV, Dearani JA, et al. Survival advantage and improved durability of mitral repair for leaflet prolapse subsets in the current era. *Ann Thorac Surg* 2006;82:819–26.
2. Gillinov AM, Cosgrove DM 3rd. Current status of mitral valve repair. *Am Heart Hosp J* 2003;1:47–54.
3. Carpentier A. Cardiac valve surgery – the “French correction.” *J Thorac Cardiovasc Surg* 1983;86:323–37.
4. Ling LH, Enriquez-Sarano M, Seward JB, et al. Early surgery in patients with mitral regurgitation due to flail leaflets: a long-term outcome study. *Circulation* 1997;96:1819–25.
5. Gillinov AM, Faber C, Houghtaling PL, et al. Repair versus replacement for degenerative mitral valve disease with coexisting ischemic heart disease. *J Thorac Cardiovasc Surg* 2003;125:1350–62.
6. Smolens IS, Pagani FD, Deeb GM, Prager RL, Nossad SS, Bolling SF. Prophylactic mitral reconstruction for mitral regurgitation. *Ann Thorac Surg* 2001;72:1210–15.
7. David TE, Ivanov J, Armstrong S, Rakowski H. Late outcomes of mitral valve repair for floppy valves: implications for asymptomatic patients. *J Thorac Cardiovasc Surg* 2003;125:1143–52.
8. Suri RM, Orszulak TA. Triangular resection for repair of mitral regurgitation due to degenerative disease. *Op Tech Thorac Cardiovasc Surg* 2005;10:194–9.
9. Cosgrove DM 3rd, Arcidi JM, Rodriguez L, Stewart WJ, Powell K, Thomas JD. Initial experience with the Cosgrove-Edwards annuloplasty system. *Ann Thorac Surg* 1995;60:499–503.
10. David TE, Komeda M, Pollick C, Burns RJ. Mitral valve annulo-

- plasty: the effect of the type on left ventricular function. *Ann Thorac Surg* 1989;47:524-7.
11. Oliveira JM, Antunes MJ. Mitral valve repair: better than replacement. *Heart* 2006;92:275-81.
  12. Phillips MR, Daly RC, Schaff HV, Dearani JA, Mullany CJ, Orszulak TA. Repair of anterior leaflet mitral valve prolapse: chordal replacement versus chordal shortening. *Ann Thorac Surg* 2000;69:25-9.
  13. David TE, Omran A, Armstrong S, et al. Long-term results of mitral valve repair for myxomatous disease with and without chordal replacement with expanded polytetrafluoroethylene sutures. *J Thorac Cardiovasc Surg* 1998;115:1279-85.
  14. Suri RM, Schaff HV, Dearani JA, et al. Survival advantage and improved durability of mitral repair for leaflet prolapse subsets in the current era. *Ann Thorac Surg* 2006;82:819-26.
  15. Alfieri O, Maisano F, De Bonis M, et al. The double-orifice technique in mitral valve repair: a simple solution for complex problems. *J Thorac Cardiovasc Surg* 2001;122:674-81.
  16. De Bonis M, Lapenna E, La Canna G, et al. Mitral valve repair for functional mitral regurgitation in end-stage dilated cardiomyopathy: role of the "edge-to-edge" technique. *Circulation* 2005;112(9 Suppl):I402-8.
  17. Maisano F, Torracca L, Oppizzi M, et al. The edge-to-edge technique: a simplified method to correct mitral insufficiency. *Eur J Cardiothorac Surg* 1998;13:240-5.
  18. Borger MA, Alam A, Murphy PM, Doenst T, David TE. Chronic ischemic mitral regurgitation: repair, replace or rethink? *Ann Thorac Surg* 2006;81:1153-61.
  19. McCarthy PM. Does the intertrigonal distance dilate? Never say never. *J Thorac Cardiovasc Surg* 2002;124:1078-9.
  20. Kang DH, Kim MJ, Kang SJ, et al. Mitral valve repair versus revascularization alone in the treatment of ischemic mitral regurgitation. *Circulation* 2006;114(1 Suppl):I499-503.
  21. Tuladhar SM, Punjabi PP. Surgical reconstruction of the mitral valve. *Heart* 2006;92:1373-7.
  22. Carpentier A, Deloche A, Dauptain J, et al. A new reconstructive operation for correction of mitral and tricuspid insufficiency. *J Thorac Cardiovasc Surg* 1971;61:1-13.
  23. Spoor MT, Geltz A, Bolling SF. Flexible versus nonflexible mitral valve rings for congestive heart failure: differential durability of repair. *Circulation* 2006;114(1 Suppl):I67-71.
  24. Muehrcke DD, Cosgrove DM 3rd, Lytle BW, et al. Is there an advantage to repairing infected mitral valves? *Ann Thorac Surg* 1997;63:1718-24.
  25. Walther T, Falk V, Mohr FW. Minimally invasive mitral valve surgery. *J Cardiovasc Surg (Torino)* 2004;45:487-95.

**Correspondence:** Dr. D. Spiegelstein, Dept. of Cardiothoracic Surgery, Sheba Medical Center, Tel Hashomer 52621, Israel.  
Phone: (972-3) 530-2710  
Fax: (972-3) 530-2410  
email: danny.spiegelstein@gmail.com

## Capsule

### A receptor for vitamin A uptake

Vitamin A has a large number of biological functions, including roles in vision, reproduction, immunity, tissue regeneration, and neuronal signaling. The existence of a cell surface receptor for the vitamin A carrier retinol binding protein (RBP) was proposed more than 30 years ago. Kawaguchi et al. report the identification and characterization of the long-sought RBP

receptor. The RBP receptor is a multipass-transmembrane domain protein with robust RBP binding and vitamin A uptake activity and is localized to the expected cellular locations for vitamin A uptake.

*Science* 2007;315:820  
Eitan Israeli

## Capsule

### Viruses and prostate cancer

The recognition that a virus plays an etiological role in a specific type cancer can profoundly change the ways in which that particular disease is diagnosed, treated, or prevented. One recent guidepost is cervical cancer, where the identification of human papillomavirus as a causal agent has led to the development of a promising prophylactic vaccine. Previous studies have provided evidence in support of a possible viral origin for human prostate cancer. A retrovirus called XMRV (for xenotropic murine leukemia virus-related virus) was detected in 40% of prostate tumors from men who were homozygous for an allelic variant of the *RNASEL* gene and in only 2% of tumors from men of other genotypes. The *RNASEL* gene encodes RNase L, a ribonuclease whose activity is required for an innate antiviral response mediated by interferon (IFN); and the allelic variant

associated with XMRV-containing tumors encodes an enzyme with impaired activity. Dong and team show that a molecular viral clone of XMRV is infectious in human prostate cancer cell lines, that replication of the virus *in vitro* is sensitive to inhibition by IFN, and that suppression of RNase L enhances viral replication. In addition, they localized putative integration sites for the XMRV provirus to several host genes that encode functions with biologically plausible roles in prostate cancer, including a suppressor of androgen receptor trans-activation. Still unanswered is the critical question of whether XMRV plays a causal role in prostate cancer, but these provocative observations should stimulate further experiments to sort this out.

*Proc Natl Acad Sci USA* 2007;104:1655  
Eitan Israeli