Current Concepts of Mitral Valve Reconstruction for Mitral Insufficiency

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In recent years, there has been a renewed interest in surgical reconstruction of the insufficient mitral valve because of reconfirmation of the limitations of existing prosthetic and bioprosthetic valves. A follow-up study, including late functional data, of 148 patients who underwent mitral valve reconstruction at our institution was combined with a review of the literature to assess the current status of mitral reconstruction. The results indicate that mitral reconstruction by Carpentier techniques is widely applicable, durable, and relatively free of complication. Freedom from late thromboembolic and anticoagulant complications is particularly notable. These factors could prove to justify earlier operative intervention in patients with mitral insufficiency before permanent myocardial damage evolves. As mitral valve reconstruction techniques become more familiar and widely used, mitral reconstruction may become the operative procedure of choice for mitral insufficiency, especially insufficiency due to degenerative disease. (Circulation 1988;78:1087–1098)

Open-heart surgery became a clinical reality in 1955, but a reliable prosthetic valve was not available until 1961. During this time Lillehei et al. and Merendino and Bruce separately reported mitral anuloplasty techniques that were successfully used in selected patients. Once prosthetic valves became available, however, interest in mitral reconstruction sharply decreased for more than 20 years.

At least three groups maintained some interest, however, in reconstruction in selected patients. In 1960, McGoon reported his technique of leaflet plication for isolated ruptured chordae tendineae, a method subsequently used at the Mayo Clinic, Rochester, Minnesota, in certain patients. Twenty-five years later, the total Mayo Clinic experience with the technique in 131 patients was reported by Orszulak et al.

In 1963, Kay and Egerton reported a technique of anuloplasty combined with repair of ruptured chordae tendineae. In 1986, Kay et al. reported that early repair was performed in 101 of 141 patients treated for mitral regurgitation secondary to coronary disease.

In 1965 Reed et al. at New York University (NYU) reported a technique of asymmetric mitral anuloplasty, particularly valuable for mitral insufficiency in children. By 1980, 196 patients had been operated upon with this technique.

Renewed interest in mitral reconstruction began in Europe in the 1970s, primarily with the work of Carpentier et al. and Carpentier in Paris, Duran et al. in Spain, and Shore et al. in England. The most extensive experience was reported by Carpentier who described results in more than 1,400 patients in 1983. After a visit with Carpentier in Paris in 1980 by Stephen B. Colvin, the Carpentier techniques were cautiously evaluated at NYU and have been used with increasing frequency since that time. Our experience with mitral reconstruction using the Carpentier techniques now totals over 300 patients. For reasons described in this report, we now consider the Carpentier techniques to be the preferred method of mitral valve reconstruction in most patients with mitral insufficiency from degenerative disease. Cosgrove et al. at the Cleveland Clinic, Cleveland, Ohio, have similarly adopted the Carpentier techniques with increasing frequency, describing experiences with 117 patients in 1986.

The renewed interest in reconstruction is primarily due to the limitations of all prosthetic valves. Principal among these limitations is thromboembolism, requiring permanent anticoagulation in all patients with metallic prostheses and in many with bioprostheses. Serious problems occur with a thromboembolic frequency ranging from 2% to 4% each year, which are well summarized in the review by

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Edmunds in 1987 that included 232 references. Bioprostheses, though less dependent on anticoagulants, are not reliably durable (80—85% at 10 years), with the strong probability that 15-year durability, not yet known, will be significantly worse.

In this review, the NYU experience is compared with reports of mitral valve reconstruction from other institutions, with the conclusion that sufficient data now exist to indicate that mitral reconstruction is both possible and durable and should be seriously considered in the majority of patients undergoing operation for mitral insufficiency.

**Anatomy of the Normal Mitral Valve**

A clear understanding of the anatomy of the normal mitral valve is crucial to mitral valve reconstruction because most decisions at operation are based upon visual evaluation of the abnormalities. The earlier widespread lack of interest in mitral valve reconstruction is well reflected by the near absence of significant anatomic publications covering mitral valve surgery from the United States during a 30-year period (1955—1985). However, the 1970 publications by Lam et al. and Ranganathan et al. in Canada are excellent.

The normal mitral valve is a complex, left-sided atrioventricular valve composed of the fibrous anulus, leaflets, chordae tendineae, and papillary muscles arising from the left ventricular myocardium. The fibrous anulus is in continuity anteriorly with the fibrous cardiac skeleton of the heart. The leaflet tissue is actually a continuous "curtain" extending around the entire circumference of the valve but is normally divided into two leaflets, the anterior (aortic) leaflet and the posterior (mural) leaflet. Two indentations in the leaflets are regularly present, constituting the anterolateral and the posteromedial commissures. These commissures do not extend to the anulus, however, and a few millimeters of normal leaflet tissue are present between the base of the commissure and the anulus.

There are two major papillary muscles, the anterolateral and the posteromedial. From these muscles, the commissural chordae tendineae extend in a fanlike fashion to the leaflet tissue adjacent to the commissures. The attachments of these chordae precisely identify the commissures, as the extent of each commissure is defined by the insertion of the commissural chordal branches. This is a particularly important point because the chordae of the commissures are seldom elongated, providing an accurate reference point at operation to determine the proper closing plane for the valve leaflets.

The anterior mitral valve leaflet is composed of an inner "rough" thickened zone near its edge, which defines the line of closure, along with a larger "clear" zone that makes up the remainder of the anterior leaflet. Similarly, the posterior leaflet has an inner rough closure zone, a clear zone, and a basal zone. The posterior leaflet is usually a triscalloped structure, with the middle scallop being the largest in more than 90% of the hearts. Occasionally, however, either the anterolateral or the posteromedial scallop is larger, and rarely, accessory scallops are present.

Based on these definitions, the posterior leaflet of the mitral valve comprises all leaflet tissue posterior to the two main commissures, encompassing accessory scallops that are classified as separate leaflets in other systems. The posterior leaflet occupies about 65% of the anular circumference, and the anterior leaflet occupies about 35%, although it has significantly more functional leaflet tissue.

Lam et al. and Ranganathan et al. revised the classification of the chordae tendineae and accurately defined the various types of chordae and the structural support supplied by each type. The chordae tendineae insert into the rough zones of both the anterior and the posterior leaflets as well as into the basal zone of the posterior leaflet. Each rough zone chord splits into three chords shortly after its origin from the papillary muscle, one of which inserts into the free margin of the leaflet, one near the junction of the rough and clear zones, and one intermediate between the two. Classically, two of the anterior rough zone chordae are large and thick, named "strut" chordae because of their importance to leaflet function. One arises from the anterolateral papillary muscle and one from the posteromedial papillary muscle. The posterior leaflet rough chordae are somewhat shorter and thinner than those to the anterior leaflet. Basal chordae to the posterior leaflet are found in about 60% of the patients; these are less structurally important than the rough zone chordae.

Each mitral valve has on average 25 chordae tendineae, nine to the anterior leaflet (two strut and seven rough), 14 to the posterior leaflet (10 rough, two basal, and two cleft), and two commissural chordae. The various surgical techniques used in mitral valve reconstruction cannot be used effectively unless this normal anatomic structure is readily understood and recognized.

**Pathophysiological Consideration**

There are five principal causes of mitral insufficiency: rheumatic heart disease, degenerative heart disease, endocarditis, ischemic heart disease, and congenital defects. In the initial NYU series of 148 patients undergoing mitral valve reconstruction, 43% were degenerative, 30% rheumatic, and only 11% ischemic. The specific pathological changes caused by each process have been well described.

A "functional classification" was devised by Carpenter from an anatomic study of 100 cadaver hearts combined with measurements obtained during his first 400 mitral valve reconstructions. The "functional" classification has three basic types. Type I includes insufficient valves with normal leaflet motion, the insufficiency resulting either from anular dilation or leaflet perforation. Type II valves have increased leaflet motion (prolapse), produced by either elongation or rupture.
of chordae tendineae. Type III valves have restricted leaflet motion from fusion or shortening of commissures, leaflets, and chordae. This type is principally seen with rheumatic disease. The concept of functional classification is particularly useful at the time of operation in determining which components of the mitral valve apparatus are causing the insufficiency.

With rheumatic valve disease, there is an asymmetric dilatation of the anulus, developing primarily in the posteromedial portions, that changes the contour of the mitral valve from an ellipse with a long transverse axis between the commissures to an ellipse with the long axis in the anteroposterior direction. The commissures may be fused with fibrosis and calcification of the leaflets, producing either valvular stenosis or insufficiency. The chordae tendineae may be thickened, fused, and shortened or, rarely, elongated. A critical point in pathological anatomy is that the anulus of the aortic leaflet is not dilated, being fixed to the fibrous skeleton of the base of the heart. This anatomic fact provides the basic reference point in determining the reconstruction of the anulus.

The pathological abnormalities with valvular insufficiency from degenerative changes are quite different. All chordae are thinned and elongated. The leaflet tissue is thinned and increased in size (the “billowing” mitral valve). The posterior anular dilatation is symmetric, involving both commissures equally, unlike that found in the rheumatic valve.

The pathological changes from ischemic mitral insufficiency are less precise; basic concepts are still evolving. First, the ischemic changes may be reversible and may improve after coronary bypass surgery. However, at least three or four permanent discrete anatomic changes can occur. A ruptured papillary muscle or ruptured chordae may result from a myocardial infarction. With less severe injury, there may be a scarred contracted papillary muscle with associated elongation of the chordae. If an extensive myocardial scar results from an infarction or an aneurysm develops, the papillary muscle and chordae may be grossly deformed. Finally, ventricular hypertrophy and dilatation may result in symmetrical dilatation of the posterior anulus.

Endocarditis may produce a discrete perforation, rupture of a chord, or varying degrees of leaflet destruction. When destruction is not extensive, reconstruction has been quite effective. Congenital forms of mitral insufficiency are well described in the recent textbook by Kirklin and Barratt-Boyce.

Operative Indications

Because significant complications after successful mitral reconstruction appear to be much less common than those after mitral valve replacement, operation can now be considered earlier in the course of the disease. It is well known that with mitral insufficiency significant, irreversible ventricular injury (manifested by dilatation, hypertrophy, and a fall in ejection fraction) can insidiously evolve with few or no symptoms. The known serious complications of prosthetic valves, however, have created a problem with each such patient in deciding whether the course of the disease has progressed to a point where the hazards of a prosthetic valve were preferable. We currently recommend prompt operation for nearly all patients with significant symptoms due to severe mitral regurgitation. Based upon our experience with mitral reconstruction during the past 7 years, operation is now also recommended for asymptomatic patients when significant ventricular dilatation has developed or when there is a fall in ejection fraction with exercise. Earlier operation is an exciting concept, hopefully decreasing the well-known 20–40% mortality that occurs in the first 5 years after operation when mitral valve replacement is performed in New York Heart Association (NYHA) functional Class IV patients.

At the opposite end of the spectrum, NYHA Class IV patients with far-advanced disease are similarly recommended for reconstruction unless associated diseases mandate a shorter operative procedure. In our initial series, 60% of patients were NYHA Class III, and 19% were NYHA Class IV preoperatively.

In the NYU experience, a specific clinical contraindication to operation has simply not been found. Reconstruction can be seriously considered for any age group (the youngest patient in our series was 3; the oldest was 83 years). Unstable clinical conditions, advanced congestive heart failure, or associated diseases are not absolute contraindications. The final decision on all patients regarding the feasibility of valve reconstruction is made at operation where visual inspection and analysis of the valvular pathology allow selection of the appropriate reconstructive technique.

Operative Technique

A median sternotomy incision with standard cardiopulmonary bypass is used. Excellent myocardial protection is essential because periods of ischemia for 1, 2, or, rarely, 3 hours may be required for complex repairs, especially when additional cardiac procedures are performed. At NYU, the mean aortic cross-clamp time was 94 minutes in the overall experience with valve reconstruction, but it was usually less than 60 minutes in patients undergoing isolated mitral valve reconstruction. The standard myocardial protection routine includes cold potassium blood cardioplegia in conjunction with topical hypothermia, keeping myocardial temperatures well below 15°C.

Exposure is obtained with a long incision in the left atrium, posterior to the interatrial septum. The Carpentier mitral retractor is essential to provide optimal exposure of the valve. Good exposure with a still, dry operative field is critical to precise mitral valve reconstruction, for this permits a systematic unhurried evaluation of the abnormalities causing the mitral insufficiency.
Mitral Valve Evaluation and Diagnosis

The keystone of mitral reconstruction is to visually examine all components of the valve and to decide which abnormalities are causing the insufficiency. Then, techniques should be chosen to correct these problems, and specific maneuvers should be used to confirm that the insufficiency has been corrected.

Initially, the atrial endocardium above the anulus is inspected for the presence of a "jet lesion," represented by a roughening and thickening of the atrial endocardium from the regurgitation jet. This may provide a clue to the site of the dominant insufficiency. Dilatation of the anulus is principally an increase in the anteroposterior diameter of the valve from dilatation of the anulus of the mural leaflet. The anulus of the aortic leaflet is not dilated, forming part of the fibrous skeleton of the heart; this fundamental anatomic fact provides a valuable guideline for anuloplasty.

Changes in valve leaflets or chordae are detected by elevating different leaflets with a nerve hook. Commissural chordae are seldom elongated, providing a reference point to detect significant chordal elongation. If leaflet tissue can be elevated more than 1 cm above the normal plane of the anulus, the degree of prolapse is considered significant. Carpentier et al. and Carpentier10 found that patients with prolapse often had chordae elongation of more than 1 cm (3–15 mm). Significant chordal shortening and fusion is found predominantly in rheumatic valves.

Periodic distention of the valve leaflets by injecting saline through a bulb syringe into the ventricle is a valuable technique to assess the valve during different stages of reconstruction. In the relaxed heart, the two leaflets normally coapt in a line 3–4 mm above and parallel to the anulus. The reliability of this visual evaluation in predicting valvular competence has been routinely confirmed by digital palpation of the valve in the beating heart shortly after discontinuing cardiopulmonary bypass, with the left ventricle generating a systolic blood pressure above 80 mm Hg. During the past 7 years at NYU, this palpation method of evaluating valvular competence has been found more useful and reliable than any other method in predicting postoperative valvular competence.

Techniques of Mitral Valve Reconstruction

After the specific anatomic abnormalities have been identified, each is corrected with one of the techniques developed by Carpentier et al. and Carpentier,10,12 In our experience,19 two or more specific abnormalities were corrected in more than 90% of the patients (Table 1). This basic fact explains the value of the Carpentier concept, as compared with other methods of reconstruction that apply the same repair for all types of insufficiency. These other methods include the posteromedial anuloplasty of Kay et al.;8 the leaflet plication, combined with anuloplasty, reported by Orszulak et al. and originally developed by McGoon; and the tailored commissural anuloplasty of Reed et al. The specific Carpentier techniques of valve repair are discussed in the following paragraphs.

Dilatation of the Mitral Anulus

The anatomic fact that merits repeated emphasis is that anular dilatation only involves the posterior mitral anulus, never the anulus of the aortic leaflet that is part of the fibrous skeleton of the heart. With rheumatic disease, the dilatation is often greatest toward the posteromedial commissure; with ischemic and degenerative disease, the posterior anulus is symmetrically dilated.9,10

The Carpentier anuloplasty ring has been used in nearly all adult reconstructions and in the majority of older children (Figure 1). The ring has several advantages. It permits a selective anuloplasty, depending upon the site of dilatation; tension is removed from suture lines and leaflets; and the mitral anulus is stabilized. Stabilizing the anulus is somewhat theoretical but may be most important. It is well known that insufficiency tends to become progressively more severe from anular dilatation. Stabilization of the anulus with the ring may keep mild residual insufficiency from progressing to a more severe form. Several reports have described repairs without an anuloplasty, but the data are limited and unconvincing. The studies by Yacoub et al.22 and by Nunley and Starr23 described operations upon diseases primarily associated with focal defects in the mural leaflet without significant anular dilatation. The study by Shore et al.14 in which a selective suture technique was used, was restricted primarily to diseases of the mural leaflet, with prosthetic replacement for more severe problems.

In children and in young adults with rheumatic mitral insufficiency, the Reed et al. commissural asymmetric anuloplasty has had excellent results for more than 15 years.8,14 However, the majority of Carpentier's extensive experiences of more than 1,400 patients has also been with rheu
matic patients. In 1987, Antunes et al\textsuperscript{25} reported experiences with 241 patients in South Africa, 107 of whom were 15 years or younger. A Carpentier ring was used in more than 90% of the group because the initial experience with a few patients in whom the ring was not used was not satisfactory. The usual size of the anuloplasty ring is between 28 and 34 mm. The size is determined by measuring the intercommissural distance at the base of the anterior anulus.

**Segmental Resection of Mural Leaflet**

An important basic point is that 60–70% of the mural leaflet can be safely resected as a quadrangular resection (Figure 2), leaving some intact leaflet tissue near each commissure. The resulting defect may be as much as 4–5 cm in width. This somewhat astonishing fact has been learned empirically with experience. Hence, usually all diseased portions of the posterior leaflet are excised, with little effort to minimize the extent of resection. Resection is used for multiple ruptured or elongated chordae or for intrinsic disease of the mural leaflet. After excision, the anulus of the excised segment is plicated with a figure-of-eight 2-0 Tevdek suture, carefully placed in the anulus to approximate the margins of the residual leaflet tissue without tension. These leaflet margins are then reapproximated with 2–4 figure-of-eight 4-0 or 5-0 Prolene sutures. This type of segmental resection is surprisingly safe, as evidenced by the reports of Carpentier,\textsuperscript{10,12} Carpentier

\textbf{FIGURE 1.} Illustration of insertion of anuloplasty ring.

\textbf{FIGURE 2.} Illustration of posterior leaflet resection and leaflet repair followed by ring anuloplasty.
et al., Kirklin, Antunes et al., Nunley and Starr, Oliveira et al., and Yacoub et al.

Anterior Leaflet Prolapse

Repair of anterior leaflet prolapse from chordal rupture or elongation requires a different approach because significant amounts of the anterior leaflet cannot be safely resected, possibly because the underlying anulus cannot be plicated, resulting in significant stress on the suture line. Ruptured chordae are treated by attachment of the ruptured chord to an adjacent large rough chord (Figure 3). If this is not feasible, chordal transposition from a segment of the mural leaflet to the anterior leaflet, as described by Carpentier et al. and Carpentier, is used (Figure 5). Anterior leaflet abnormalities were successfully corrected with procedures on the anterior leaflet in 33 of the first 148 patients in the NYU experience. This point merits emphasis because many surgeons have previously treated ruptured chordae to the aortic leaflet with valve replacement.

Restricted Leaflet Motion

Mitral insufficiency from restricted leaflet motion is principally found in rheumatic hearts. Insufficiency results from a combination of disease in both the leaflets and the chordae. If segmental mural leaflet disease is present, a standard quadrangular resection can be done. Fused or shortened basal or intermediate chordae may be divided. Fused commissural chordae may be separated, also dividing the underlying papillary muscle.

Often commissural fusion is present as well, producing a combination of stenosis and insufficiency. Thirty-one of the first 148 patients in the NYU series had combined mitral insufficiency and stenosis.

Neither segmental calcification of the leaflet nor extensive calcification of the mural leaflet anulus has been found a contraindication to repair as long as the anterior leaflet is mobile. Most published studies have implied that extensive calcification was a contraindication to reconstruction because the calcified mural leaflet anulus could not be plicated. In the NYU series, calcium was cautiously debrided in 26 patients until enough mobility of the anulus was obtained to permit plication, after which reconstruction was satisfactorily accomplished.

Endocarditis

The techniques of reconstruction have been particularly applicable to different types of injury resulting from endocarditis (12% of the NYU series). The abnormalities range from simple leaflet perforation to chordal rupture or extensive segmental destruction of leaflet tissue. Repair of leaflet perforations by direct suture or application of a pericardial patch has been successful. Knowing the reliability of the Carpentier techniques has permitted a bold approach to reconstructing valves with more extensive abnormalities. This is particularly gratifying because such patients do not have any inherent valvular disease, such as rheumatic fever or degenerative disease; hence, their long-term prognosis after reconstruction should be excellent.

Contraindications

It should be emphasized that the only known contraindication to reconstruction has been severe disease in the anterior leaflet or unrepairable fusion of the leaflets and chordae. Valves with extensive mural leaflet disease, multiple elongated or ruptured chordae, or severe calcification of the anulus of the mural leaflet have all been successfully repaired. The reliability of the techniques is indicated by the fact that in the vast majority of patients repair has been accomplished once decided upon, almost never
Valve reconstruction is thought possible in well over 90% of the nonrheumatic valves operated upon for mitral insufficiency. Considering the overall experience with operative procedures on mitral valves at NYU in 1986 and 1987 (including reoperative mitral valve operation, operation for failure of previous valve prosthesis, and operation for severe mitral stenosis), 164 patients had mitral valve reconstruction, whereas 292 patients had mitral valve replacement. Thus, despite an intense interest in mitral valve reconstruction within our own institution, only 36% of the patients undergoing procedures on the mitral valve received mitral valve reconstruction. Certainly, many of the patients receiving mitral valve replacement were operated on because of severe mitral stenosis, previous prosthetic valve failure, or significant disease in the anterior leaflet that precluded valve reconstruction. However, there was a wide variation in the frequency with which the procedure was used between different surgeons, ranging from minimal use by some to use in 66.4% of the mitral valve patients operated on by the most experienced (S.B.C.).

Discussion and Recommendations

Significance of Current Data With Mitral Reconstruction

Current data from all sources, including 148 patients previously reported from NYU, as well as
TABLE 2. Cumulative Results of Mitral Valve Reconstruction Since 1980

<table>
<thead>
<tr>
<th>Reference</th>
<th>Year</th>
<th>Patients (n)</th>
<th>Etiology</th>
<th>Operative technique</th>
<th>Operative mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Orszulak et al⁴</td>
<td>1985</td>
<td>131</td>
<td>Ruptured chordae</td>
<td>Leaflet plication and suture anuloplasty (McGoon)</td>
<td>6%</td>
</tr>
<tr>
<td>Kay et al⁵</td>
<td>1986</td>
<td>101</td>
<td>Ischemic</td>
<td>Suture plication anuloplasty</td>
<td>8%</td>
</tr>
<tr>
<td>Reed et al⁶</td>
<td>1980</td>
<td>196</td>
<td>70% rheumatic</td>
<td>Asymmetric anuloplasty</td>
<td>4.5%</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>12% degenerative</td>
<td></td>
<td>1.3% since 1965</td>
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<td></td>
<td></td>
<td></td>
<td>9% congenital</td>
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<td></td>
<td></td>
<td></td>
<td>3% ischemic</td>
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<tr>
<td>Carpentier⁷</td>
<td>1983</td>
<td>1,421</td>
<td>60% rheumatic</td>
<td>Carpentier</td>
<td>3.6%</td>
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<td></td>
<td></td>
<td></td>
<td>21% degenerative</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td>13% congenital</td>
<td></td>
<td></td>
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<tr>
<td>Cosgrove et al¹⁵</td>
<td>1986</td>
<td>117</td>
<td>80% degenerative</td>
<td>Carpentier</td>
<td>1.8% isolated</td>
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<td></td>
<td></td>
<td></td>
<td>11% rheumatic</td>
<td></td>
<td>4.3% overall</td>
</tr>
<tr>
<td>Galloway et al¹⁹</td>
<td>1988</td>
<td>148</td>
<td>43% degenerative</td>
<td>Carpentier</td>
<td>1.2% isolated</td>
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<td></td>
<td>30% rheumatic</td>
<td></td>
<td>5.4% overall</td>
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<td></td>
<td></td>
<td></td>
<td>12% ischemic</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>12% infectious</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kirklin and Barratt-Boyes²¹</td>
<td>1987</td>
<td>201</td>
<td>13–30% degenerative</td>
<td>Varied</td>
<td>3% isolated</td>
</tr>
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<td></td>
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<td>10–28% ischemic</td>
<td></td>
<td>7% overall</td>
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<td>12% rheumatic</td>
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<td></td>
<td></td>
<td>10% cardiomyopathy</td>
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<td></td>
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<tr>
<td>Yacoub et al²²</td>
<td>1983</td>
<td>86</td>
<td>Degenerative</td>
<td>Leaflet resection and suture anuloplasty</td>
<td>3%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>No ring anuloplasty</td>
<td></td>
</tr>
<tr>
<td>Antunes et al²⁵</td>
<td>1987</td>
<td>241</td>
<td>Rheumatic</td>
<td>Carpentier</td>
<td>3.3%</td>
</tr>
<tr>
<td>Oliveira et al²⁶</td>
<td>1983</td>
<td>82</td>
<td>Ruptured chordae</td>
<td>Carpentier (no ring anuloplasty)</td>
<td>4.9%</td>
</tr>
<tr>
<td>Angell et al²⁹</td>
<td>1987</td>
<td>112</td>
<td>NA</td>
<td>Carpentier</td>
<td>5.4%</td>
</tr>
<tr>
<td>Lessana et al³²</td>
<td>1983</td>
<td>130</td>
<td>Rheumatic</td>
<td>Carpentier</td>
<td>2.3%</td>
</tr>
<tr>
<td>Penkoske et al³⁹</td>
<td>1984</td>
<td>31</td>
<td>Degenerative and ruptured chordae</td>
<td>Leaflet plication and suture anuloplasty (McGoon)</td>
<td>3%</td>
</tr>
</tbody>
</table>

Echo, echocardiography; MR, mitral regurgitation; MV, mitral valve; NA, not available.

those reported from other institutions, are tabulated in Table 2. Experiences with a total of 2,997 patients from 13 different institutions are listed. These data tend to support the following generalizations:

1) The Carpentier techniques of mitral reconstruction seem potentially applicable to all types of mitral insufficiency, regardless of cause. Only limited data, however, are yet available with ischemic insufficiency.

2) The Carpentier techniques seem to us clearly superior to all other forms of mitral reconstruction. This view is somewhat controversial because each surgeon’s experience is limited to the technique used at his institution. However, the detailed review of all published reports that was done in the preparation of this report, considering both the size of the data base and the length of follow-up, is supportive of the above conclusion. Obviously, different, techniques can be used for different specific problems (i.e., asymmetric anuloplasty of Reed et al⁷,⁸ sutured plication of Kay and Egerton⁵ and Kay et al⁶ leaflet plication of McGoon⁴ and Orszulak et al⁴ and segmental resection of Yacoub et al²²), but the Carpentier methods are the most versatile and the most applicable for all types of problems.

3) There are no known fixed contraindications to reconstruction as long as a functioning anterior leaflet is present. This includes patients of all ages with all degrees of heart failure.

4) The frequency with which reconstruction as opposed to replacement will be undertaken will vary with the surgeon.

A unique feature of the NYU mitral reconstruction series is that in 1980 a decision was made to initially include one surgeon (S.B.C.) in almost all operations, permitting one person to gain a visual familiarity with the different types of valvular abnormalities encountered. This plan is probably the main reason that an increasingly higher percentage of mitral valves have been reconstructed at our institution in recent years. Our experience indicates that more than 90% of patients with mitral insufficiency from degenerative disease, the most common cause of mitral insufficiency in the United States, can be treated by reconstruction.

With the realization that a reliable, durable reconstruction technique is available, undoubtedly many
surgeons will quickly learn to perform valve reconstruction, and reconstruction will be used more frequently in future years.

The basic facts supporting the previous general statements are detailed in the following paragraphs.

**Hospital Mortality**

In the NYU series, the 30-day hospital mortality for mitral valve reconstruction was 1.2% for isolated mitral reconstruction, 5.8% when reconstruction was combined with other cardiac procedures. Similar mortality rates have been published from other institutions (Table 2). These data clearly indicate that reconstruction is not associated with an increased operative risk. Several studies suggest that the operative risk may actually be less than with replacement.\(^{29,30}\) This is possible but not proven by available data.\(^{31}\) In the NYU series, the statistically significant factors predicting increased operative risk \((p<0.001)\) included ischemic etiology, previous myocardial infarction, age over 70 years, preoperative NYHA Class IV cardiac status, or an emergency operation.\(^{19}\)

**Late Survival**

In the NYU study, the 5-year actuarial survival for late cardiac death was 90%, whereas the 5-year freedom from all deaths was 80%.\(^{19}\) Sand et al\(^{31}\) in 1987 reported a 5-year survival, including hospital deaths, of 76%. Although their analysis of 389 patients receiving mitral valve replacements during a similar period of time found the 5-year survival to be 56%, multivariant analysis indicated that replacement rather than repair was only possibly a risk factor \((p = 0.14)\).\(^{31}\) This is an important point because reports by others suggested that reconstruction was associated with a lower mortality than replacement.\(^{29,30}\) However, current data confirm that late survival after mitral valve reconstruction is at least equivalent to that obtained with mitral valve replacement. Performance of mitral reconstruction at an earlier time, before irreversible ventricular injury has developed, conceivably could produce an even better 5-year survival.

**Durability of Repair**

The persistent concern limiting wide-spread acceptance of mitral valve reconstruction has been

---

**Table 2. Continued**

<table>
<thead>
<tr>
<th>Mean follow-up</th>
<th>Late survival</th>
<th>Freedom from late valve replacement</th>
<th>Late follow-up echo</th>
<th>Freedom from thromboembolism</th>
</tr>
</thead>
<tbody>
<tr>
<td>NA</td>
<td>92% at 5 yr</td>
<td>91% at 5 yr</td>
<td>NA</td>
<td>1.8%/patient yr</td>
</tr>
<tr>
<td>NA</td>
<td>73% at 10 yr</td>
<td>81% at 10 yr</td>
<td>NA</td>
<td>Actuarial rate NA</td>
</tr>
<tr>
<td>NA</td>
<td>60% at 5 yr</td>
<td>94% at 7–10 yr</td>
<td>NA</td>
<td>94% at 5 yr</td>
</tr>
<tr>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>96% at 15 yr</td>
</tr>
<tr>
<td>4.3 yr</td>
<td>82% at 9 yr</td>
<td>1.6%/yr rheumatic</td>
<td>NA</td>
<td>0.6%/patient yr</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.6%/patient yr degenerative</td>
<td>NA</td>
<td>Actuarial rate NA</td>
</tr>
<tr>
<td>13 mo</td>
<td>90% at 2 yr</td>
<td>96% at 2 yr</td>
<td>NA</td>
<td>99% at 2 yr</td>
</tr>
<tr>
<td>26 mo</td>
<td>90% at 5 yr</td>
<td>90% at 7 yr</td>
<td>Late in 83%</td>
<td>95% freedom at 5 yr</td>
</tr>
<tr>
<td></td>
<td>(cardiac)</td>
<td></td>
<td>92% remained free from severe MR at 5 yr</td>
<td></td>
</tr>
<tr>
<td></td>
<td>80% at 5 yr (all deaths)</td>
<td></td>
<td>Mean MV gradient = 4.0 ± 2.7 mm</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Mean MV area = 2.3 ± 0.7 cm²</td>
<td></td>
</tr>
<tr>
<td>NA</td>
<td>76% at 5 yr,</td>
<td>91% at 5 yr</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td></td>
<td>59% at 10 yr (all deaths)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>38 mo</td>
<td>90% at 5 yr</td>
<td>97% at 5 yr</td>
<td>NA</td>
<td>97% at 5 yr</td>
</tr>
<tr>
<td>30 mo</td>
<td>90% at 4 yr</td>
<td>78% at 4 yr</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>3.6 yr</td>
<td>88% at 6 yr</td>
<td>91% at 5 yr</td>
<td>NA</td>
<td>0.2%/patient yr</td>
</tr>
<tr>
<td>NA</td>
<td>85% at 5 yr</td>
<td>90% at 5 yr</td>
<td>90% free from severe MR at 5 yr</td>
<td>94% at 5 yr</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Mean MV gradient = 4.1 mm</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Mean MV area = 2.5 cm²</td>
<td></td>
</tr>
<tr>
<td>38 mo</td>
<td>92% at 7 yr</td>
<td>1.7%/patient yr</td>
<td>Actuarial rate NA</td>
<td>91% at 7 yr</td>
</tr>
<tr>
<td>44 mo</td>
<td>78% at 5 yr</td>
<td>84% at 5 yr</td>
<td>NA</td>
<td>97% at 5 yr</td>
</tr>
</tbody>
</table>
the uncertain durability. Carpentier reported valve failure rates after reconstruction of 0.7% for each patient year for degenerative disease and 1.6% for each patient year for rheumatic disease but did not have late echocardiographic data regarding valve function. Many patients from other countries were not available for late evaluation in Carpentier’s series. Others have reported valve durability rates from 78% at 4 years with rheumatic valves to as high as 90–97% at 5 years in nonrheumatic valves (Table 2). Most of these studies also have little data regarding valve function. Significantly, in all of the reported experiences, the failure rates have been higher after repair of rheumatic valves than after repair of valves with other types of mitral insufficiency.

At NYU, the 5-year actuarial freedom from late valve replacement was 90%. Perhaps the most significant point is that among the late valve failures, only one occurred in a patient with degenerative disease. Long-term two-dimensional and color Doppler echocardiographic evaluation of 83% of the first 148 patients operated upon at NYU found excellent valve function: 56% had zero insufficiency; 27% had 1 + insufficiency; and 10% had 2 + insufficiency. Only 7% had moderate-to-severe mitral insufficiency at follow-up echocardiographic examination. When we defined valve durability after mitral valve reconstruction as the combined freedom from late valve replacement and freedom from echocardiographic evidence of severe late valvular insufficiency, repair durability was significantly better in degenerative than in rheumatic patients (Figure 6). Galler et al initially reported systolic anterior motion of the mitral valve with mild-to-moderate left ventricular outflow tract obstruction in six of the first 65 mitral valve reconstructions performed at NYU, but subsequently, the frequency of this finding has decreased.

Follow-up two-dimensional and color Doppler echocardiography was also used to estimate the postreconstructive mitral valve gradient in our patients. The overall mean postreconstructive mitral valve gradient was 4.04 ± 2.75 mmHg, and the mean ± SD postreconstructive mitral area was 2.34 ± 0.7 cm². The rheumatic valves had a mean gradient of 6.00 ± 3.22 mmHg with an area of 1.94 ± 0.60 cm², whereas the nonrheumatic valves had a significantly smaller mean gradient of 3.27 ± 2.30 mmHg and a significantly larger mean valve area of 2.58 ± 0.71 cm².

It is somewhat surprising that both function and durability in the degenerative group are much better than in the rheumatic group because degenerative tissue has much less tensile strength. The applicability of the Cardinier techniques of reconstruction to degenerative valves was the original question investigated and reported from NYU in 1985. Perhaps the excellent late results after valve reconstruction for degenerative disease (only one patient requiring late replacement) may be due to the stabilizing effect of the anuloplasty ring.

In the 1987 study of the Alabama experience by Sand et al, the important observation was made with precise statistical analyses that there was “no rising late hazard phase” for reoperation in the repair group. This contrasts markedly with the late valve failures known to occur at a predictable frequency after mitral valve replacement due to late prosthetic valve dysfunction, thrombosis, and endocarditis.

Thus, the functional late durability of mitral valve reconstruction is extremely encouraging, especially in nonrheumatic populations. Valve durability and freedom from late reoperation after mitral valve reconstruction appear to be equal or superior to the durability and reoperation rates seen after mitral valve replacement.

Late Functional Status

Ninety-five percent of the patients in the NYU series improved to NYHA functional class I or II after mitral valve reconstruction. Studies by David et al suggest that the chordae tendineae contribute significantly to left ventricular myocardial function. Thus, it seems reasonable theoretically that left ventricular cardiac function may be better after mitral valve reconstruction than after mitral valve replacement, although data are not yet available.

Thromboembolic and Anticoagulant Complications

The clearest advantage of mitral valve reconstruction is the striking freedom from late thromboembolic problems despite the absence of routine anticoagulation (Table 2) (Figure 7). Several studies reveal that approximately 95% of patients remain free from thromboembolic events 5–10 years after mitral valve reconstruction. This is a most significant contrast to the fact that 10–35% of patients with a mechanical prosthesis have signifi-
cant thromboembolic episodes within 5-10 years after operation.16

In the NYU series, warfarin (Coumadin) and dipyridamole treatment (Persantine) were begun on the third postoperative day and continued for 3 months. After this time, warfarin treatment was stopped, but antplatelet therapy alone was continued. With this regimen, 95% of patients have been free of thromboemboli 7 years after operation, whereas the incidence of anticoagulant-related complications has been near zero.19 Again, this contrasts markedly to a recent study of valve replacements that found significant bleeding to be the most common postoperative complication.36

Thus, the available data indicate that patients undergoing mitral valve reconstruction have fewer thromboembolic and anticoagulant-related complications than patients undergoing prosthetic mitral valve replacement.

Endocarditis

Late endocarditis is extremely rare, approaching zero in all reported series of mitral valve reconstruction. De novo postreconstructive mitral valve endocarditis was not seen in our experience.19 This differs from the 3-6% incidence of late endocarditis consistently reported after mitral valve replacement.37,38 We currently recommend standard antibacterial prophylaxis for dental and other invasive procedures after mitral valve reconstruction.

The relative resistance to infection conferred upon valve reconstruction patients is presumably related to the extended use of autologous tissue. Thus, another apparent advantage of mitral valve reconstruction over mitral valve replacement is a decreased frequency of late endocarditis in patients undergoing valve reconstruction.

Conclusions

The available data indicate that the operative results, late survival, and late valve durability obtained after mitral valve reconstruction are equal or superior to those obtained after prosthetic mitral valve replacement. Significantly, patients receiving mitral valve reconstruction clearly have fewer late complications than patients undergoing prosthetic mitral valve replacement; therefore, mitral valve reconstruction is the operative procedure of choice for many patients with mitral regurgitation.

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References

1. Lillehei CW, Gott VL, DeWall RA, Varco RL: Surgical correction of pure mitral insufficiency by anuloplasty under direct vision. J Lancet 1957;77:446-449
2. Merendino KA, Bruce RA: One hundred seventeen surgically treated cases of valvular rheumatic heart diseases: With preliminary report of two cases of mitral regurgitation treated under direct vision with aid of a pump-oxygenator. JAMA 1957;64:749-755

**KEY WORDS** • mitral repair • mitral regurgitation • mitral reconstruction • mitral valve surgery
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