MITRAL VALVE REPAIR vs REPLACEMENT

Current Recommendations and Long-Term Results

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The earliest attempts at reconstruction of the mitral valve were for relief of mitral stenosis. The first such procedures for the mitral valve were suggested in 1898 by Samways,[78] who proposed notching the orifice of the stenotic mitral valve. In 1902, Brunton[7] suggested that incisions in the mitral valve may relieve mitral stenosis. Cutler performed a series of operations consisting of nonanatomic leaflet incisions for mitral stenosis in 1923, but despite initial success, poor subsequent results led to his abandoning further attempts.[36] Souttar performed the first clinically successful closed anatomic mitral commissurotomy by finger fracture in 1925 but because of extreme criticism did not perform any further operations.[36]

In 1946, Bailey, using the technique used by Souttar in 1925, split open a heavily calcified mitral valve. Because of poor results with almost immediate restenosis, Bailey in 1948 developed a technique for closed incisional commissurotomies and by 1955 was able to report a good experience with 811 cases.[36] In 1947, Harken performed his initial sharp dissections of the mitral leaflets, and although the early results were poor, subsequent experience led to rapid improvement in the surgical outcomes.[36]

The introduction of cardiopulmonary bypass by Gibbon in 1953 led to efforts to perform mitral valve repair for mitral insufficiency using direct access through the left atrium. The implantation of the first clinically successful mitral valve prosthesis by Starr in 1960 was a major advance in the treatment of mitral regurgitation and calcific mitral stenosis.

The complications encountered then by Starr persist to some degree to the present, including transvalvular gradients, thromboembolism, hemolysis, infection, mechanical failure, and periprosthetic leaks. Bioprostheses have had significant late rates of structural deterioration in the mitral position. Burdon et al[8] reported on the experience at Stanford with 793 patients who had received porcine bioprostheses. Only 45% of valves were free of structural deterioration at 15 years. Thus, interest in preservation of the native mitral valve has persisted.

The earliest techniques used for direct repair of mitral regurgitation involved attempts to correct dilation of the posterior mitral annulus by suture plication.[49–51] These techniques used alone produced variable results. The single most important advance in valve repair was the description by Carpentier of a systematic approach to the classification of abnormalities of the mitral valve leaflets, annulus, and subvalvular apparatus of chordae and papillary muscles.[9, 10, 21] He de-

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scribed three types of abnormalities: type I, normal leaflet motion; type II, prolapsed leaflet; and type III, restricted leaflet motion (Fig. 1). The mitral regurgitation in type I was ascribed to either annular dilation or leaflet perforation; in type II to overriding or prolapse of one leaflet more than the other, leading to an asymmetric jet caused by ruptured chordae, elongated chordae, or a ruptured papillary muscle; in type III to commissural fusion and leaflet thickening or associated fused chordae.

A series of techniques were described for the correction of these individual anomalies that could be applied systematically with consistent results.[9, 10, 21] The techniques described by Carpentier involved rigid ring annuloplasty based on the size of the anterior leaflet for repair of annular dilation, leaflet resection or patching for leaflet abnormalities, and native chordal shortening or transposition of native chordae for chordal and papillary muscle abnormalities.

Although these techniques have been applied successfully in many thousands of patients, some problems have persisted. Inability to repair the valve successfully leading to intraoperative conversion to prosthetic replacement has been reported in some series in as many as 10% to 15% of patients. When the chordae of the anterior leaflet are extensively involved and especially if the posterior leaflet is simultaneously affected, valve repair is difficult with Carpentier techniques.

Use of the original Carpentier rigid rings has been associated with systolic anterior motion (SAM) of the anterior leaflet in 15% of cases perioperatively but has been clinically significant long-term in few patients.[32, 33, 38, 40, 45, 58] SAM is thought to have occurred because of excessive anterior displacement of the commissural line. Thus, in some cases, use of the size of the anterior leaflet to guide selection of the annuloplasty ring can lead to overconstriction of the posterior annulus, especially when the posterior leaflet is large. Furthermore, the rigid ring produces a symmetric posterior annuloplasty regardless of the degree of asymmetry of the posterior leaflet defect. Carpentier has developed additional surgical techniques directed at reducing the vertical dimensions of the posterior leaflet to correct the problem of SAM.[21] Reports have suggested that some late failures of the Carpentier techniques have been due to further deterioration of native chordae used in the initial repair.

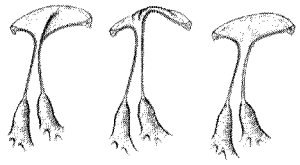

Figure 1. Classification of the basic types of abnormalities of the mitral valve.

In 1988, the author began a systematic attempt to repair mitral valves whenever possible. To try to avoid these problems and achieve competent repair in all patients, the author reviewed the results of all techniques reported to that date and restudied the literature regarding the contribution of the dynamic behavior of the mitral annulus to mitral valve competence. As a result of these studies and earlier experiences with valve repair, the author decided to adapt the analytic approach of Carpentier for the diagnosis of the components requiring correction but to use a different combination of available surgical techniques.

SURGICAL ANATOMY

These observations are derived from a number of reports.[3, 55, 56, 90, 92] The mitral annulus is a poorly defined structure consisting primarily of the fibrous condensation of the line of attachment of the mitral leaflets to the left ventricle. In the anterior one third of the mitral orifice, the anterior leaflet is attached to the fibrous aortic-mitral continuity and trigones of the heart and is relatively nondistensible except in patients with connective tissue disorders, such as those with Marfan Syndrome. In contrast, the posterior two thirds of the annulus consists of the attachment of the posterior leaflet either directly to the muscle of the muscular ostium of the left ventricle or to a subvalvular membrane, which, in turn, attaches directly to the myocardium.

The anterior and posterior portions of the annulus are in continuity medially at the right fibrous trigone or central fibrous body of the heart. The right fibrous trigone is formed by the junction of the mitral annulus, mitral valve leaflets, aortic root, and membranous septum. The left fibrous trigone is less well developed and is formed from the fusion of

the aortic root and mitral valve leaflets. These areas can be appreciated by the surgeon because of their denser texture when passing sutures through the annulus, and they form important structural *anchor points* when the posterior annulus is poorly developed.

The anterior mitral leaflet is roughly a truncated triangle and is much more mobile than the posterior leaflet. Because of its shape, there is usually little or no redundant tissue, and so resections of damaged or flail anterior leaflet segments are rarely done. The posterior leaflet is more elongated and rectangular. It usually has three scallops or sections to allow it to lay against the wall of the ventricle. Abnormal segments of up to about one third of the posterior leaflet can be resected without difficulty. The leaflets are fused medially and laterally, usually for a distance of about 3 to 8 mm from the annulus.

The chordae tendinae arise from the papillary muscles and insert into the mural surface of the leaflets. The chordal insertions into the posterior leaflet are in three layers: marginal attached to the edge, intermediate, and mural. Some chordae may also arise from ventricular trabeculae. The chordae of the anterior leaflet insert primarily into the free margin of the leaflet, and there are fewer chordae attaching to other portions of the underside of the anterior leaflet.

The chordae arising from each papillary muscle are distributed to both leaflets. The chordal patterns vary widely within these parameters.[92] The papillary muscles typically consist of two prominent structures, an anterolateral and a posteromedial papillary muscle. Papillary muscle morphology, however, can vary widely from this form.[92] Flattened sheets of muscle can give rise to the chordae, and in some cases the chordae appear to arise from the wall of the ventricle itself. Usually the papillary muscles and their attachment to the walls of the left ventricle are substantial structures, however, accounting for as much as 25% of left ventricular mass.

FUNCTIONAL ANATOMY

The area of the mitral orifice defined by the mitral annulus is 6.5 cm^2 in women and 8 cm^2 in men.[90] The effective orifice area through the level of the leaflets measured clinically is some 30% less than these values. As shown in Figure 2, the area of the mitral orifice in diastole is reduced by 23% to 40% during ventricular systole. This occurs because of reduction of the size of the posterior portion of the annulus during late atrial systole then ventricular systole, by contraction of its muscular ventricular attachment.[88] The mitral annulus is in the shape of a saddle oriented in the anteroposterior axis. During systole, the height of the saddle is increased as the annular dimensions decrease.[93] These changes are shown in Figure 3.

In addition to the absolute reduction in the size of the mitral orifice in need of occlusion by the leaflets, the forward movement of the posterior annulus produced first by atrial then ventricular systole increases the degree of apposition of the anterior and posterior

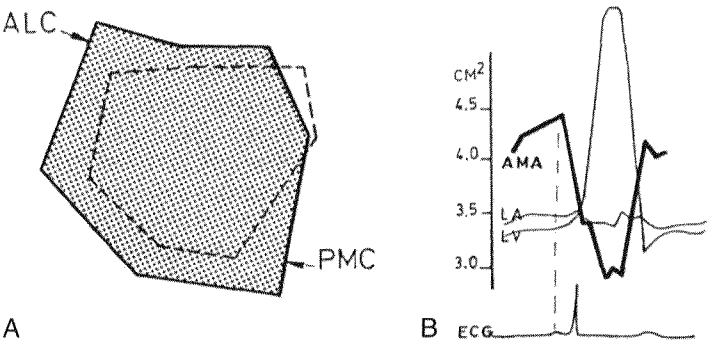

Figure 2. *A* and *B*, The physiologic reduction in mitral annular area during ventricular systole. LA = Left atrium; LV = left ventricle; ALC = anterolateral commissure; PMC = posteromedial commissure; AMA = area of mitral annulus. (*Data from* Tsakiris AG, von Bernuth G, Rastelli GC, et al: Size and motion of the mitral valve annulus in anesthetized intact dogs. J Appl Physiol 30:611, 1971.)

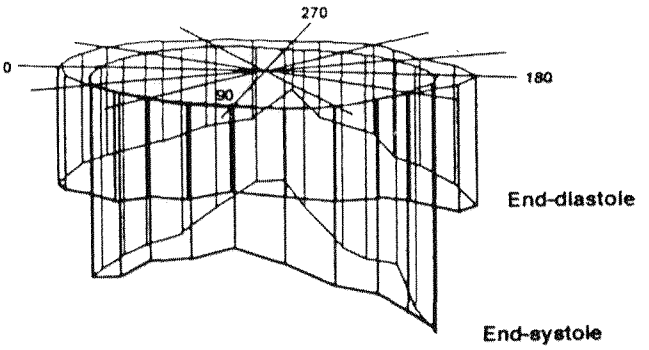

Figure 3. Data from the study of Yamaura et al[93] showing the three-dimensional changes in mitral annular shape and area in systole and diastole. The pronounced saddle shape of the central part of the annulus at end systole is well seen.

leaflets during systole. Examination of the opposing surfaces of the anterior and posterior leaflets shows a zone of apposition on the atrial side of the leaflets extending about 5 to 10 mm from the free edges of the leaflets producing the rough zone, an irregular thickened area. The remainder of the valves' atrial surfaces are smooth. This overlap is important in producing the so-called keystone effect, in which the leaflets are pushed together in systole, reducing greatly the load on the chordae.

In normal valves, posterior movement of the much more mobile anterior leaflet into apposition with the much less mobile posterior leaflet is responsible for the bulk of the systolic closure of the mitral orifice. In some cases of myxomatous degeneration, however, the posterior leaflet and chordae may be so redundant that the posterior leaflet has significant anterior movement and the line of apposition in systole is more anterior than usual. The importance of this phenomenon is that severe SAM can occur if the posterior portion of an annuloplasty inadvertently displaces the anterior leaflet too far forward. This problem is avoidable by appropriate use of annuloplasty.

The papillary muscles and their chordal-mitral leaflet annular continuity have an important role in ventricular systole, in which they have been shown experimentally to account for as much as 25% of the force of ventricular contraction.[20, 42, 52, 53, 67, 79, 83, 85, 86] Furthermore, they help maintain the optimal position of the subjacent ventricular wall during systole. This helps to preserve the conical shape of the ventricle during systole. Division of the papillary muscles causes a significant reduction in contractility and results in a more spherical left ventricle during systole.[88] Chordal elongation can also cause similar problems because of lack of an attachment point to the annulus via the chordae against which the papillary muscles can act.

SURGICAL TECHNIQUES

Assessment of the mitral valve structure and function by transesophageal echocardiography (TEE) is an indispensable step in the author's approach to mitral valve repair. In addition to general evaluation of the atria, ventricles, and other valves, TEE provides highly accurate information about the mitral valve and left ventricle of great importance to the surgeon. This information includes the size of the annulus and any associated calcification; the extent, site, and severity of leaflet prolapse or fixity, the relative size of the anterior and posterior leaflets and their flexibility or fixity; the extent of systolic opposition of the leaflets; and the direction of the regurgitation jet.

After precise diagnosis of the problem, a variety of surgical techniques can be used to correct abnormalities of the annulus, leaflets, chordae, and papillary muscles. Good exposure of the valve is essential.[69] Annuloplasty is used to support or reduce in size the posterior portion of the mitral annulus and to provide a stable foundation for appropriate alignment of the leaflets. If the abnormalities are localized to the region of the commissures alone, the Kay suture plication technique is

still a useful approach.[50, 51] Most commonly, however, some form of partial or complete prosthetic ring annuloplasty is performed. A ring shorter than the existing posterior annulus is sewn to it in a manner that plicates the annulus and shortens the intertrigonal distance. This maneuver in effect *bowstrings* the posterior annulus forward.

Although the rigid Carpentier ring has been used widely, many surgeons now prefer to use a flexible ring, which allows the occurrence of the systolic reduction of area of the mitral annulus by as much as 25%. Early left ventricular performance seems to be enhanced by use of a flexible ring but long-term ventricular function is similar with flexible and rigid rings.[11, 17, 22, 24, 37, 66, 77, 91, 93]

The author has used the flexible and adjustable Puig-Massana annuloplasty ring.[57, 65] The independent adjustability of the length of the posterior medial and lateral one thirds of these rings allows almost total elimination of mitral regurgitation. It also helps avoid overconstriction of the posterior annulus and SAM because only the minimal amount of tension necessary to achieve appropriate leaflet opposition is applied after the ring is sewn to the mitral annulus. In addition, because leaflet repairs are often asymmetric, involving one commissure more than another, the ring allows the annuloplasty to be localized mainly to that side. Other fixed-diameter but flexible rings include the Duran ring and the Cosgrove-Edwards ring (a partial annuloplasty ring).[22, 24, 68]

Asymmetric leaflet prolapse as a result of myxomatous degeneration causing an asymmetric regurgitant jet is the most commonly encountered pathology in the United States.[12, 13, 46, 89] Prolapse is caused by stretched or ruptured chordae.[63, 64] Prolapse of a segment of the posterior leaflet can be managed by resection of a segment of up to one third of the posterior leaflet, if localized prolapse is present. If it involves the anterior leaflet or most of the posterior leaflet, some form of chordal reconstruction is needed. Carpentier described multiple different techniques using more normal native chordae to replace stretched or broken native chordae.[9, 10, 21, 76] In addition to the technical complexity of these maneuvers, data indicate an incidence of failure because of elongation of native chordae used in this way. Since the inception of the author's experience, polytetrafluoroethylene (PTFE) suture material has been used for chordal replacement. This material has been investigated experimentally and now has more than 10 years of clinical use with sustained good results.[18, 19, 31, 75, 94, 95] The surgical technique is simple. The PTFE suture is passed through the head of the papillary muscle, and the two strands are then brought up and passed through the free edge of the leaflet. The suture is placed in such a way that the orientation of the original chordae is duplicated. The author has replaced up to 16 chordae in this way.

Small leaflet defects as seen in bacterial endocarditis can be repaired with pericardial patches sutured in place after débridement of the native leaflet.[43] The pericardium can be supported if necessary with PTFE chordae. Many variations of the foregoing techniques have been reported, but the basic principles of mitral valve repair are those described here.

Repair of ischemic mitral regurgitation deserves special consideration because of its more complex aspects.[80–82] The most common cause of mitral regurgitation in chronic myocardial ischemia is myxomatous degeneration of the mitral valve actually unrelated to the ischemic heart disease. True ischemic mitral regurgitation has been classified by Rankin et al.[71–73] Rankin type I is due to abnormal posterior leaflet function because of an inferior myocardial infarction with involvement of the posterior papillary muscle. Involvement of both the papillary muscle and ventricle is required to cause mitral regurgitation.[5, 41, 47, 54, 61] Type II is due to papillary muscle rupture after myocardial infarction. Type III is due to diffuse ischemic cardiomyopathy. The left ventricular dilation produces downward and outward displacement of the papillary muscles. Even in the presence of normal annular dimensions, this papillary muscle displacement may produce a restrictive defect because of the increased distance from the papillary muscle to the annulus with failure of leaflet apposition (Carpentier type I defect).

Chronic ischemic mitral regurgitation of the Rankin types I and II are routinely amenable to successful repair.[82] Type III defects are sometimes amenable to repair by annuloplasty. However unpredictably, annuloplasty may worsen the defect and require prosthetic mitral replacement despite apparently normal leaflets. Because the ejection fraction in the type III patients is often in the range of 20% to 30%, surgery is associated with a significant risk in this subgroup of patients.[74] Thus, variable results have been reported.[44, 48, 49, 51, 59, 62, 70, 74, 87]

After completion of the repair, intraoperative testing is performed using saline insufflation through the mitral orifice. The aortic valve can also be made incompetent with a catheter and the aorta unclamped, causing aortic insufficiency and inflating the left ventricle. Transesophageal echocardiography is used after resumption of cardiac rhythm during weaning from cardiopulmonary bypass. It allows immediate recognition of any technical problems with the repair. It is also useful in ensuring that all air has been evacuated from the heart.

RESULTS

The results of mitral valve repair have been documented in a number of series, and certain generalizations now can be made.* Despite its somewhat greater technical complexity, mitral valve repair is associated with a surgical mortality less than that of prosthetic mitral valve replacement. Although possibly reflective of patient selection biases, this may also relate to the absence of risk of atrioventricular groove hematoma, a potentially fatal complication of mitral valve replacement, and to the enhanced early left ventricular performance seen when the papillary-annular connection is maintained.

Thromboembolism is uncommon after mitral valve repair in contrast with prosthetic mitral replacement. The author has had no confirmed late thromboembolism in more than 200 cases followed up to 10 years. The author does not use warfarin (Coumadin) early or late after surgery unless chronic atrial fibrillation is present (about one third of patients). Thus, anticoagulant-related hemorrhage is almost nonexistent. Furthermore, repaired valves experience bacterial endocarditis much less frequently than prosthetic valves.

Reoperation rates for repaired valve are low in myxomatous disease but are somewhat higher in rheumatic disease.[4, 6, 22, 23, 27, 84] The good durability of repair of valves with insufficiency because of degenerative disease seems paradoxic. The reason for it appears, however, to be due to the fact that certain areas of the mitral leaflets and chordae are under the greatest stress in systole, and it is these areas that routinely fail. These are most commonly the central portions of the edges of the leaflets, which are furthest from the papillary muscles. Proper repair of these areas is durable, and the other, lower-stress areas rarely fail. Thus, reoperation rates are routinely reported to be lower than those for prosthetic valves. In most series, reoperation rates are highest early after surgery, reflecting inappropriate patient selection or the learning curve associated with mastery of the techniques of repair. Late failure is uncommon and usually relates to progression of intrinsic valve disease rather than breakdown of the repair.

The author's experience with mitral valve repair for mitral insufficiency consists of 200 consecutive patients operated on over the 15-year period between 1983 and 1998. Earlier results have been reported previously.[57] The mean age was 62.5 ± 14.7 years, and 103 were men (49.5%).

Preoperative rhythm was sinus in 59.0% of patients and atrial fibrillation in 31.3% of patients. New York Heart Association (NYHA) class was III–IV for 71.8% patients. Mitral regurgitation was moderate to severe in 77.7% of the patients. The most common leaflet pathology was myxomatous degeneration (52.7%), followed by rheumatic (14.4%), degenerative (12.8%), and ischemic (10.1%). Seventeen percent of patients had previous cardiac surgery. Surgical procedures used were leaflet resection (19.7%), leaflet plication (12.8%), PTFE artificial chordal replacement (40%), PTFE chordae and leaflet resection/plication (13.3%), and annuloplasty (91.5%). The types of annuloplasty were adjustable, flexible ring annuloplasty (Puig-Massana) in 86.2% and Kay annuloplasty in 9.0%. Pericardial patch and other miscellaneous procedures were performed in the rest. Associated cardiac procedures were done in 39.9% of patients: coronary artery bypass (20.7%) or aortic valve replacement (10%). Systolic anterior motion of any severity was found in only five patients: Three were due to small annulus and left ventricular cavity, and two were due to large posterior leaflet; three were improved by loosening the annuloplasty, one resolved, and one required mitral valve replacement. Postoperatively 83.5% of patients were NYHA class I or II, and mitral regurgitation was absent or mild in 96.0% patients, moderate in 3.7%, and severe in 0.5% (one patient). The 30-day mortality was 2.5% (five patients). There were no deaths in elective, isolated mitral valve repairs. Survival

*References 1, 2, 4, 6, 9, 10, 13–16, 21–30, 34, 35, 39, 57, 60.

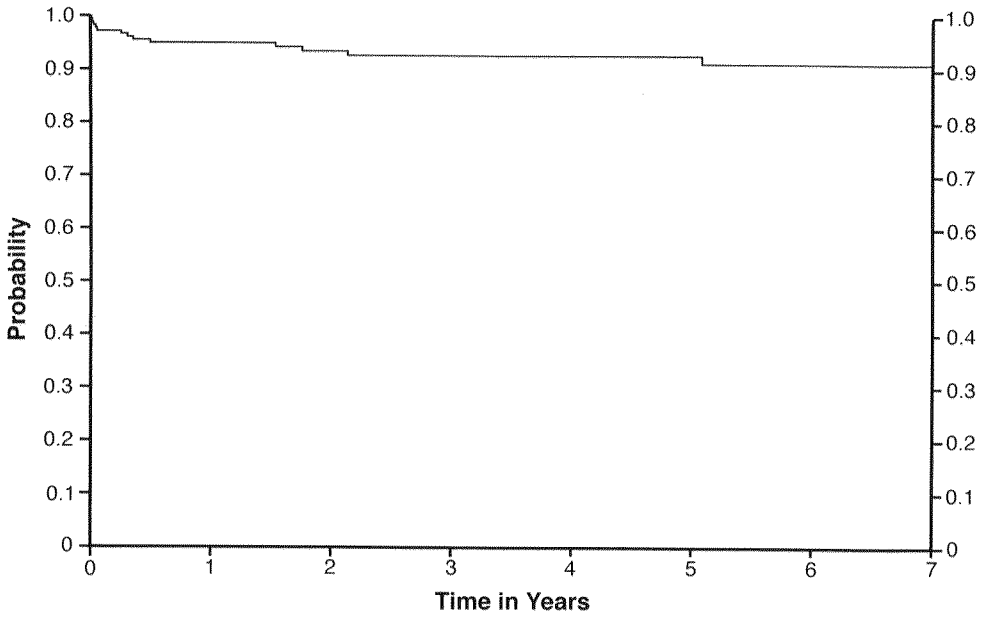

Figure 4. Kaplan-Meier curve of overall survival for 188 mitral valve repair patients with a minimal follow-up of 1 year, operated on by the author.

(Kaplan-Meier) at 7 years was 91.3%, and freedom from reoperation was 92.1% and no late thromboembolism (Figs. 4 and 5). Thus, use of PTFE, adjustable annuloplasty, and other procedures produced excellent results. Use of a variety of techniques increased the reparability rate to 98% of attempts and eliminated the adverse influence of anterior leaflet pathology.

Cohn et al[14] reported on 219 patients with

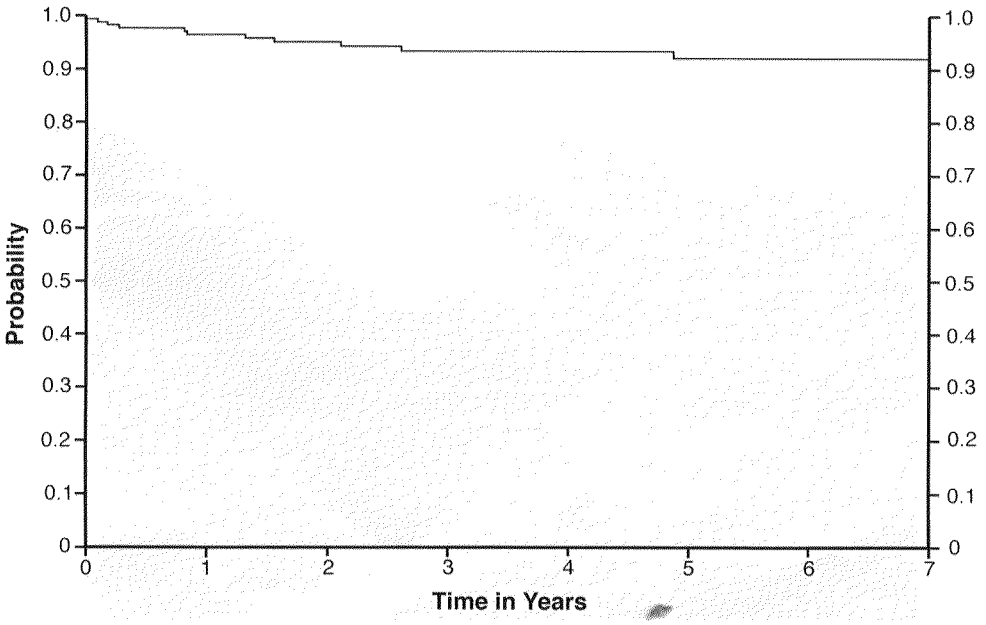

Figure 5. Kaplan-Meier curve of freedom from reoperation for the same patient group as shown in Figure 4.

myxomatous degeneration who underwent mitral valve repair. The mean age was 63 years (range, 23 to 84 years). Coronary bypass was performed in 29%. Posterior leaflet resection was the most common operation. The anterior leaflet was resected in 14 patients and both leaflets in 15 patients. The Duran flexible, nonadjustable ring was used in 111 patients (51%), Carpentier-Edwards ring in 44 patients (20%), and no ring in 64 patients (29%). Perioperative mortality was 2.3%. Over a follow-up of up to 9 years (mean, 2 years), 90% of patients were asymptomatic; two patients had endocarditis, and seven patients experienced thromboemboli. Reoperation was required in 12 patients (10%), of whom 6 had received no annuloplasty ring. The type of ring used did not affect outcome. Actuarial data at 5 years showed overall survival of 86% ± 5% versus freedom from valve failure of 83% ± 4% and freedom from thromboembolism of 94% ± 3%.

Deloche et al[21] reported the 15-year follow-up of 206 patients who underwent repair by Carpentier's group between 1972 and 1979. The valves were repaired using the techniques of Carpentier. The 15-year patient survival was 72.4%. The 15-year freedom from thromboembolism was 93.9%; 96.6% were free of endocarditis, 95.6% had no anticoagulant related hemorrhage, and 87.4% were free from reoperation. The freedom from reoperation was 92.7% for degenerative disease and 76.1% for rheumatic disease. Follow-up echocardiography showed that 91% of patients had trivial or absent mitral regurgitation.

Akins et al[1] compared 133 patients who had mitral reconstruction for degenerative or ischemic mitral regurgitation with 130 patients treated with mitral valve replacement. The techniques of Carpentier were used with a rigid annuloplasty ring. Hospital mortality was 3% for repair and 12% for replacement patients. Actuarial freedom from thromboembolism at 6 years was 92% for the reconstruction group and 85% for the replacement group. At 6 years, freedom from bleeding, endocarditis, and reoperation were similar.

Galloway et al[34] reported a comparison of 280 mitral valve repair operations using the techniques of Carpentier with 975 porcine mitral valve prostheses and 196 mechanical prostheses. Hospital mortality was 5.0%, 10.6%, and 16.6% for repair, porcine valves, and mechanical valves. Overall 5-year survival was 76% for repair, 69% for porcine replacement, and 72% for mechanical prostheses. The 5-year freedom from reoperation was 94.4% for nonrheumatic repair, 77.4% for repair of rheumatic patients, 96.6% for porcine valve replacement, and 96.4% for mechanical valve replacement. Freedom from thromboembolism was 94.1% after repair, 86.7% after porcine replacement, and 94.0% after mechanical replacement. Reflective of the much greater use of warfarin in the prosthetic groups, freedom from hemorrhage was 99.5% after valve reconstruction and 94.8%

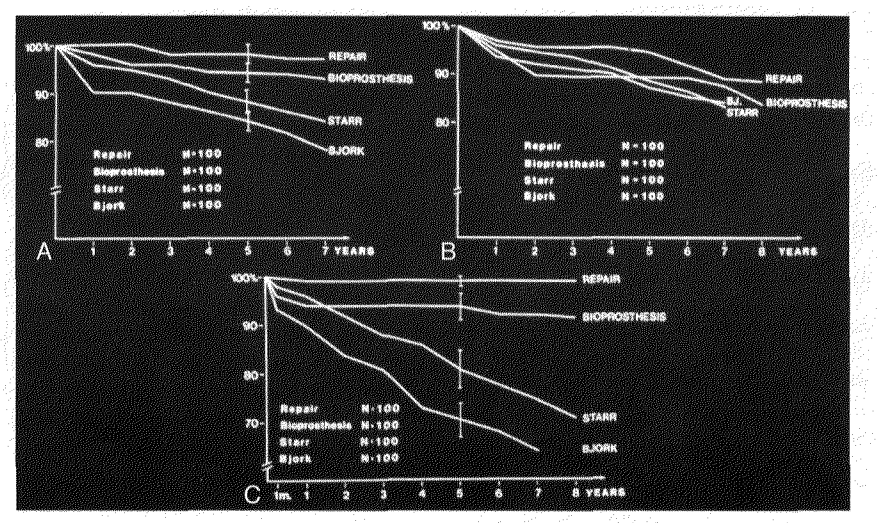

Figure 6. Comparative results of the long-term follow-up of valve repair with bioprosthetic and mechanical heart valve. (*Data from* Carpentier A: Cardiac valve surgery—the French correction. J Thorac Cardiovasc Surg 86:323, 1983).

and 94.6% after prosthetic replacement. Freedom from endocarditis was 100% after repair and 95.8% and 95.7% after prosthetic replacement.

Carpentier[9] reported on a comparison of 100 mitral valve repairs with 100 porcine valves, 100 Starr valves, and 100 Björk valves. The clinical characteristics of the patients were similar. Hospital mortality was 2% for repair and 12% to 13% for prostheses. Actuarial survival at 7 years was 82% for repair and 56% to 61% for replacement. Freedom from thromboembolism was 96% for repair, 94% for porcine bioprostheses, and 70% and 68% for mechanical devices. No fatal thromboembolism occurred in repair patients compared with a 20% to 28% fatal thromboembolism rate in the prosthetic groups (Fig. 6).

The risk of need for reoperation at 7 years was 13% for repair, 8.5% for bioprosthesis, 8% for Starr prostheses, and 18% for Björk valves. Cumulative event-free curves showed a 79% rate for repair patients versus 53% to 71% for prosthetic patients.

Table 1. COMPARISON OF MITRAL VALVE REPLACEMENT AND REPAIR

	Mitral Valve Replacement	Mitral Valve Repair
Hospital mortality	3%–15%	1%–3%
Thromboembolism (annualized)	1–3%	0%–1%
Needs warfarin (Coumadin) (assuming sinus rhythm)	Usually	Rarely
LV function reduced	Yes	No
LV anatomy affected	Yes	No
Papillary muscle function preserved	No	Yes
Annualized failure rates	1%–2%	<1%
Endocarditis (annualized)	1%–2%	<1%
Suitable for all mitral disease	Yes	No
Absolute contraindications	None	Calcified leaflets Severe rheumatic disease
Relative contraindications	None	Rheumatic disease Ischemic mitral regurgitation
Ideal pathology	All	Myxomatous disease

LV = Left ventricular

SUMMARY

Techniques now exist to correct abnormalities of all components of the mitral valvular apparatus except extensive loss of pliable leaflet area.

Thus, paradoxically, myxomatous valves with redundant leaflets represent the ideal candidates for mitral valve repair. Repair for mitral insufficiency can be performed for some rheumatic valves, but patient selection is critical. Loss of leaflet area, leaflet thickening, and extensive calcification of the leaflets or commissures are contraindications to repair. The abnormalities of the subvalvular apparatus are less important because a complete set of new chordae can be reconstructed using PTFE suture material.

Some cases of endocarditis are ideal for repair using localized débridement and pericardial patch repair with or without PTFE chordal replacement. True ischemic mitral regurgitation of the Carpentier type I category is still something of a surgical enigma. Because it is a restrictive leaflet motion problem, annuloplasty alone is not always effective, and the outcome of any given repair attempt is less predictable. Repairs in patients with small annuli and multiple leaflet defects requiring complex series of maneuvers have a low probability of success. Furthermore, such patients with small left ventricular cavities are more prone to experience SAM.

Several factors contributing to which therapy is chosen for mitral valve disease are summarized in Table 1. Patient selection, accurate evaluation of the cause or causes of mitral regurgitation, and well-executed application of the appropriate techniques for repair are all critical factors in the early and late success of mitral valve repair.

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