Mitral Valve Repair: the Multimodal Approach and the Role of Minimally Invasive Procedures

GERALD M. LAWRIE, M.D., F.A.C.S.
CLINICAL PROFESSOR OF SURGERY
BAYLOR COLLEGE OF MEDICINE
ATTENDING SURGEON, THE METHODIST HOSPITAL
PARTNER, TEXAS SURGICAL ASSOCIATES

HOUSTON, TX

In recent years mitral valve repair has gained increasing acceptance as the preferred treatment for mitral insufficiency. Mitral valve repair has many advantages. The surgical risk is lower than prosthetic valve replacement. Improved preservation of left ventricular function due to maintenance of the papillary muscle - mitral annular continuity - has been demonstrated. Thromboembolism is rare after mitral repair in patients in sinus rhythm. These patients receive no coumadin and thus are free from bleeding complications. The repaired valves have been shown to have durability comparable to or better than prosthetic valves.

HISTORY

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,[1] who proposed notching the orifice of the stenotic mitral valve. In 1902, Brunton[2] 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.[3] 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.[3]

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 good experience with 811 cases.[3] 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.[3]

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 earliest techniques used for direct repair of mitral regurgitation involved attempts to correct dilatation

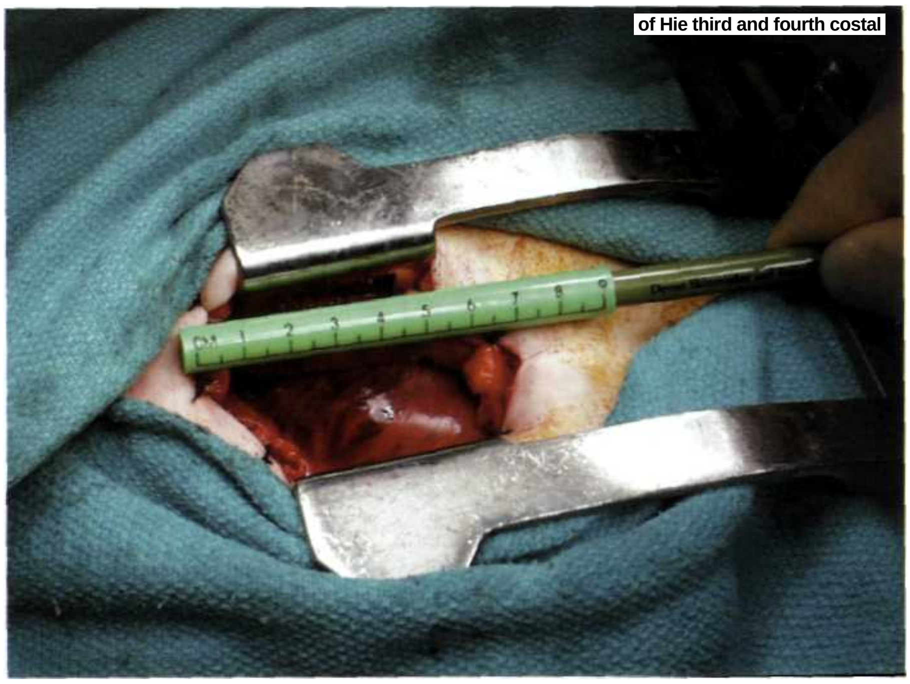

Figure 1. Right parastemal **incision for mitral** valve **repair.** Small **cartilage have been** resected. **The right atrium is visible.**

of the posterior mitral annulus by suture placation.[4-6] 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 sub-valvular apparatus of chordae and papillary muscles.[7-9] He described three types of abnormalities: type I, normal leaflet motion; type II, prolapsed leaflet; and type III, restricted leaflet motion. The mitral regurgitation in type I was ascribed to either annular dilatation 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.[7-9] The techniques described by Carpentier involved rigid ring annuloplasty based on the size of the anterior leaflet for repair of annular dilatation, 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, 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

Table 1. Factors determining the choice of whether to perform a mitral valve replacement or mitral valve repair.

	Mitral valve replacement	**Mitral valve repair**
Hospital mortality	**3-15%**	1-3%
Thromboembolism (annualized)	**1-3%**	0-1%
Need for 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

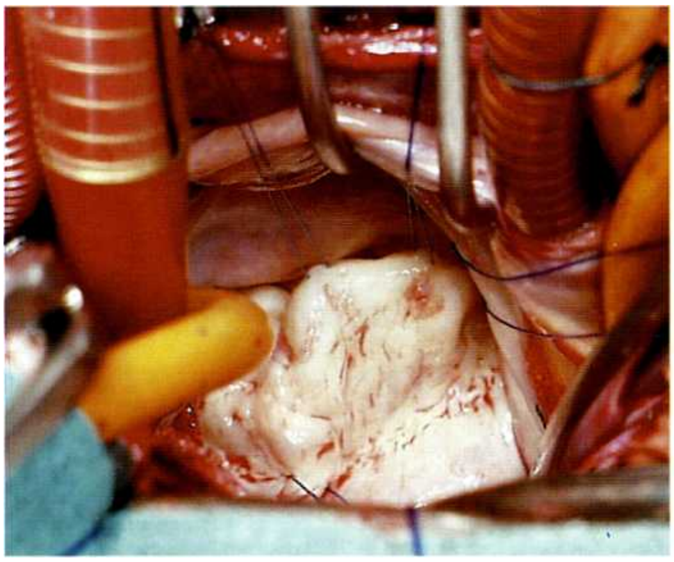

Figure 2. Marked prolapse of the middle scallop of the posterior mitral leaflet due to chorda! elongation and redundant myxomatous posterior leaflet tissue. The left atrium is dilated.

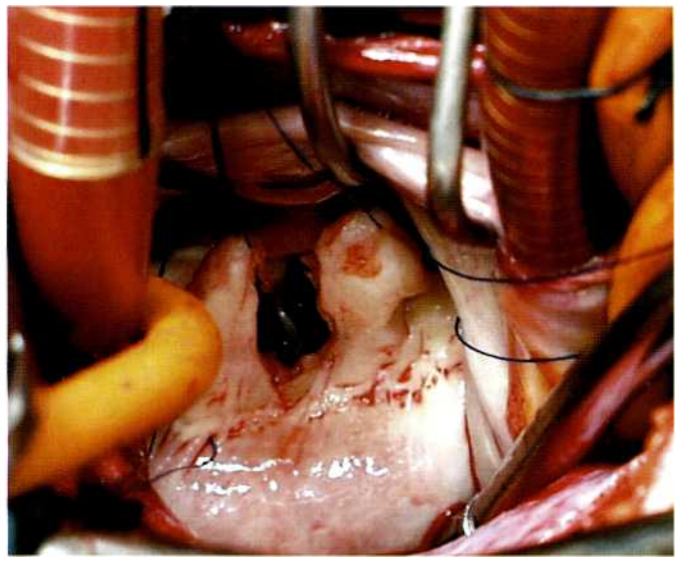

Figure 3. The redundant prolapsing portion of the posterior leaflet has been resected leaving a rectangular or "quadrarttic" defect based on the posterior annulus.

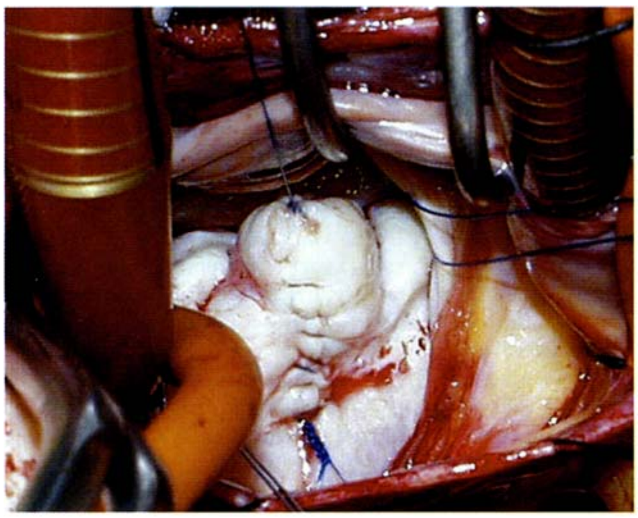

Figure 4. The defect is closed with 4-0 polypropylene suture. The annulus in the base of the defect is plicated by this suture line.

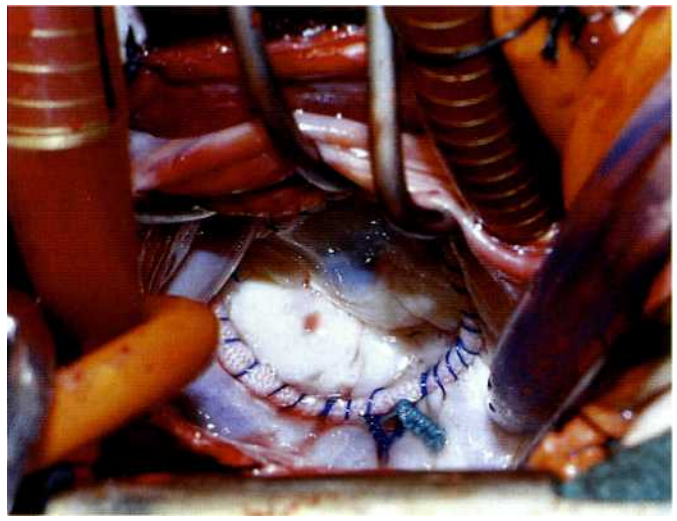

Figure 5. The repair is completed with an adjustable flexible ring annuloplasty held in place by a running 2-0 polypropylene suture. Excellent competence was achieved intraoperatively.

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.[10-13]

CURRENT SURGICAL TECHNIQUES

The mitral valve can now be approached using a minimally invasive surgical technique in most cases. The technique we use involves a right parasternal incision about 8 cm in length (Figure 1), through which the 3rd and 4th costal cartilages are divided. The lung is retracted and the pericardium is opened. Arterial return is into the ascending aorta and venous return is via a long catheter inserted through the femoral vein into the superior vena cava and augmented with venous suction drainage. The ascending aorta is clamped and cardioplegia given. The mitral valve is visualized through either an inter-atrial groove incision or via a right atrial transseptal approach.

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

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 polytetrafluoroethylene (PTFE) suture material.

Some cases of endocarditis are ideal for repair using localized debridement and pericardial patch repair with or without PTFE chordal replacement. True ischemic mitral regurgitation of the Carpentier type I category is still sometimes difficult to repair. Because it is a restrictive leaflet motion problem, annuloplasty alone is not always effective, and the outcome of any given

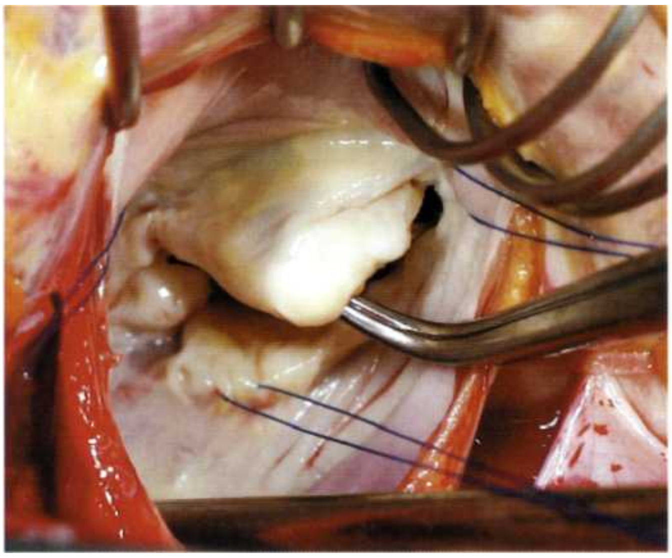

Figure G. Marked prolapse of the anterior mitral leaflet secondary to elongation of multiple chordae tendinae. The anterior mitral leaflet itself is relatively normal.

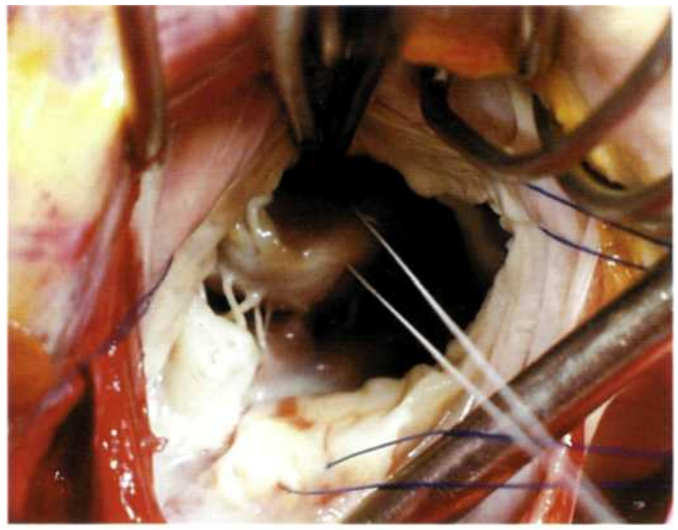

Figure 7. A double armed number 5-FTFE suture has been passed in a figure of 8, through the postero-medial papillary muscle. This will produce two artificial chordae which are aligned to match the chordae which they are replacing. Another FIFE suture is then inserted (not shown].

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.

The factors to consider when deciding on which therapy to choose for mitral valve disease are summarized in Table 1. Patient selection, accurate evaluation of the 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.

In our own experience, we have used multiple techniques in many patients. Assessment of the mitral valve structure and function by trans-esophageal echocardiography (TEE) is an indispensable step in my approach to mitral valve repair. In addition to general evaluation of the atria, ventricles, and other valves, TEE provides highly accurate information about die 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 die 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 die valve is essential.[14] 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.[5,6] 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 die 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.[15-23]

The author has used the flexible and adjustable Puig-Massana annuloplasty ring.[24,25] 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).[17,18,26]

Asymmetric leaflet prolapse as a result of myxomatous degeneration causing an asymmetric regurgitant jet is the most commonly encountered pathology in the US.[27-30] Prolapse is caused by stretched or ruptured chordae (Figure 2).[31] 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 (Figures 3 to 5). If it involves the anterior leaflet or most of the posterior leaflet, some form of chordal reconstruction is needed. Carpentier described various techniques using more normal native chordae to replace stretched or broken native chordae.[7-9,32] In addition to the technical complexity of these maneuvers, the data indicate an incidence of failure because of elongation of native chordae used in this way.

My own experience is of using PTFE suture material for chordal replacement. This material has been investigated experimentally and now has more than 10 years of clinical use with sustained good results.[33-38] The surgical technique is simple (Figures 6-9). The PTFE suture is passed through the head

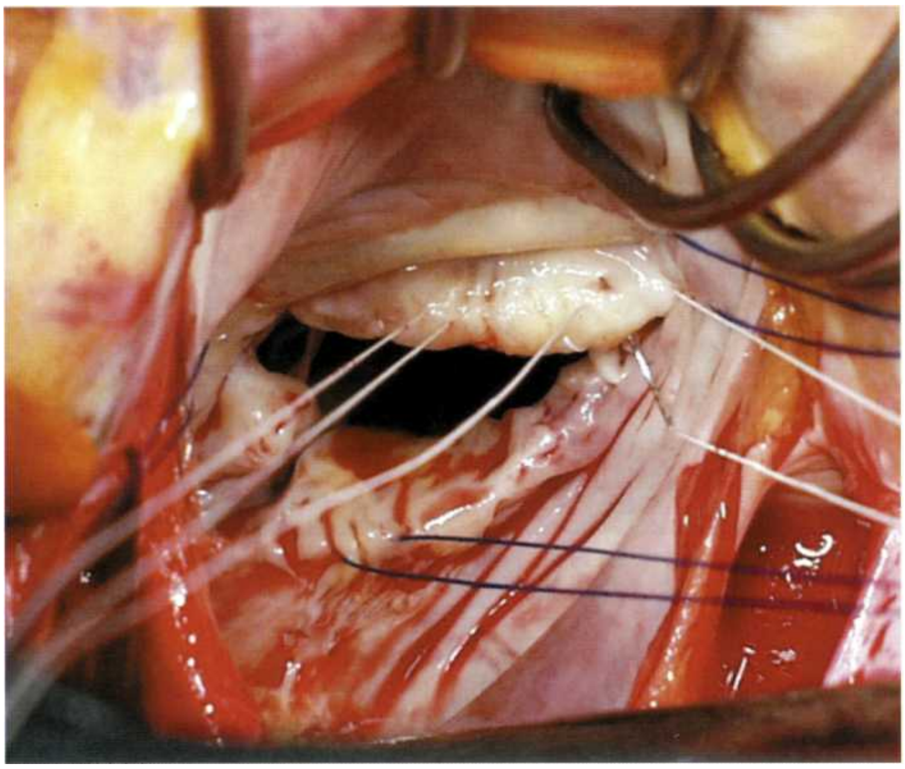

Figure 8. The sutures are brought up and inserted into the free margin of the anterior leaflet. A total of four artificial chordae were placed in the anterior leafelet of the patient.

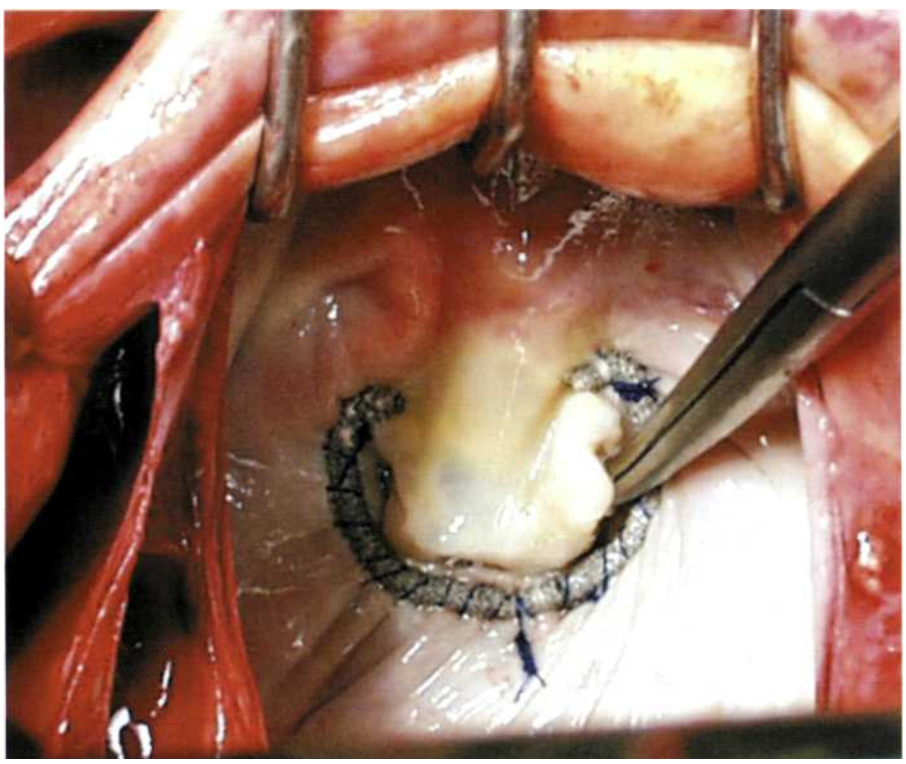

Figure 9. A flexible annuloplasty ring is sutured to the native mitral valve annulus with continuous 2-0 polypropylene to provide a stable dimension for the annulus.

of the papillary muscle (Figure 7), and the two strands are then brought up and passed through the free edge of the leaflet (Figure 8). 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. Residual commissural prolapse if present can be treated with a Kay annuloplasty (Figure 10).

Some leaflet defects as seen in bacterial endocarditis can be repaired with pericardial patches sutured in place after debridement of the native leaflet.[39] 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.[40-42] 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.[43-45] Rankin type 1 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.[46-50] 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.[42] 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.[51] Thus, variable results have been reported.[4,6,51-47]

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

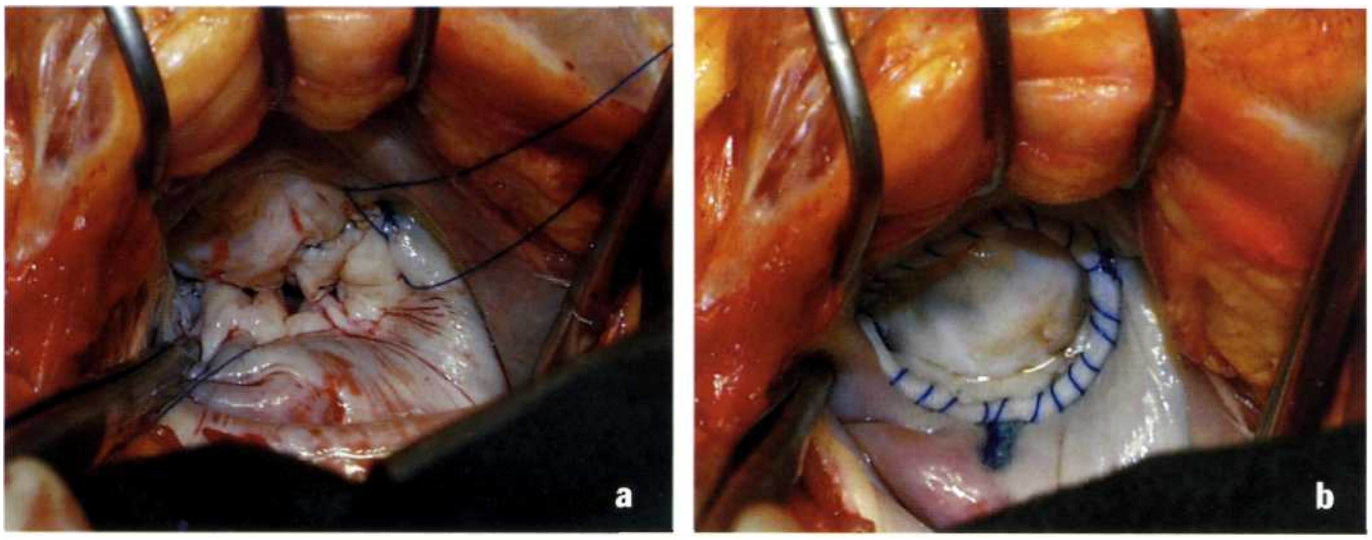

Figure 10. Illustration of how the Kay annuloplasty can be used to plicate one or both commissures. Here the posteromedial commissure (right of picture) is being plicated with 2-0 polypropylene.

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.[7-9,17,18,28,58-74] 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. I have 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.[17,60,61,65,68,75]

The good durability of repair of valves with insufficiency because of degenerative disease seems paradoxical. 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. In my experience with mitral valve repair for mitral insufficiency consists of 270 consecutive patients operated on over the 15-year period between 1983 and 1998. Earlier results have been reported previously.[24] The mean age was 62.5 +/- 14.7 years.

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 patches 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 leaflets; three were improved by loosening the annuloplasty, one resolved, and one required mitral valve replacement. Post operatively 83.5% of patient were NYHA class I or II, and mitral regurgitation was absent or mild in 96.0% patients, moderate in 3.7%, and severe in one patient. The 30-day mortality was 2.5% (five patients). There was only one death in elective, isolated mitral valve repairs. Survival (Kaplan - Meier) at 7 years was 91.3%, and freedom from reoperation was 92.1% and no late thromboembolism occurred.

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.

Other authors have had similar good experiences. Cohn et al.[62] reported on 219 patients with 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%.

Deloch et al.[9] 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.[58] compared 133 patients who had mitral reconstruction for degenerative or ischemic mitral regurgitation with 1 30 patients treated with mitral valve replacement. The techniques of Carpentier were used with 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.[72] 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 the prosthetic groups, freedom from hemorrhage was 99.5% after valve reconstruction and 94.8% and 94.6% after prosthetic replacement. Freedom from endocarditis was 100% after repair and 95.8% and 95.7% after prosthetic replacement.

Carpentier[7] reported on a comparison of 100 mitral valve repairs with 100 porcine valves respectively, 100 Starr valves, and 100 Bjork 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. The risk of need for reoperation at 7 years was 13% for repair, 8.5% for bioprosthesis, 8% for Starr prostheses, and 18% for Bjork valves. Cumulative event-free curves showed a 79% rate for repair patients versus 53% to 71% for prosthetic patients.

CONCLUSION

The data presented suggest that mitral valve repair is the preferred therapy for patients with mitral regurgitation as it provides anatomical correction with minimal ongoing morbidity. Because of this, the advantages of correction of severe mitral regurgitation before significant clinical manifestations develop can now be realized in manv patients. Acceptance of this approach has been enhanced further by the combination of mitral valve repair with minimally invasive surgical approaches. This has allowed patients to avoid the risks associated with progressive left ventricular dilatation, congestive heart failure, atrial fibrillation and thromboembolism. With increasing experience we have progressively liberalized our indications for minimally invasive repair of severely regurgitant mitral valves.

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