Mitral Valve:
Toward Complete Repairability

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ABSTRACT

During the last 50 years, there has been an exponential increase in our understanding of the structure and function of the mitral valve in health and disease. Large numbers of patients have undergone mitral-valve reparative procedures worldwide with variable results. In our initial 10-year experience in which traditional techniques were used for repair of mitral regurgitation, a 70% success rate was achieved. In 1995, a critical reappraisal of the common causes of failure to repair, or failure of repair and developed improved surgical approaches for these patients began. The conditions addressed were massive bileaflet prolapse ("Barlow's valves"); anterior leaflet prolapse; multisegment chordal failure; commissural accessory leaflet tissue prolapse; and leaflet destruction by endocarditis. A widely applicable standard technique was developed that the author and colleagues called the "American Correction." It emphasizes chordal replacement with polytetrafluoroethylene (PTFE) artificial chordae, importance of the line of apposition of the leaflets, and importance of simultaneous dynamic adjustment of the chordal length and anteroposterior dimension of the mitral annulus. In addition, use of multiple techniques in 30% of patients to complete the repair has been emphasized. These techniques resulted in a 90% repairability rate for all pathologies and 100% for myxomatous-mitral insufficiency. These techniques also have led to a decline in reoperation rates, and improved long-term durability. Continued evaluation with intraoperative and postoperative three-dimensional (3-D) echo provides further insights and refinement of mitral-repair techniques.

INTRODUCTION

Preservation of the native mitral valve is generally accepted currently as the optimal approach in patients with severe mitral regurgitation. In addition to the obvious benefits of avoidance of warfarin and thromboembolism, mitral-valve repair has a mortality risk of one-half of replacement and preserves left ventricular morphology and function.

The last four decades of the 20th century were characterized by an exponential increase in knowledge of the structure and function of the mitral valve. However, despite this, repairability rates plateaued in the mid nineties at approximately 70% in the most experienced centers, including our own.[1,2] Even recently in the USA, only approximately one-third of mitral-valve surgeries have been repairs, and mostly of the simplest type.[3] In 1995, a critical appraisal of why certain categories of disease were difficult to repair and why repairs had failed began. The disease states identified were massive bileaflet prolapse ("Barlow's valves"), anterior leaflet prolapse, and multisegment chordal failure, commissural accessory leaflet tissue prolapse, leaflet destruction by endocarditis. A widely applicable technique was developed based on our improved understanding of the causes of mitral regurgitation and the physiology of normal and diseased mitral valve,[4] which combined with other techniques has produced a marked increase in repairability rates.

APPLIED ANATOMY AND PHYSIOLOGY

Complete familiarity with the anatomy and physiology is an essential prerequisite for the Surgeon or Cardiologist contemplating an intervention on the mitral valve. Several publications are especially useful summaries.[5-8] The mitral valve consists of an anterior and posterior leaflet attached to the heart at the mitral annulus. The anterior leaflet, through the aortic-mitral fibrous continuity, is closely related to the aortic root. The leaflets are stabilized by chordae tendinae attached from them to the papillary muscles of the left ventricle. The mitral annulus is a weak and distensible structure and is incomplete in many patients.[7] The posterior two-thirds of the annulus is attached to the muscular ostium of the left ventricle and dilates when the left ventricle enlarges.[5-8]

The aortic and mitral valves fill the ostium of the left ventricle, the roughly circular opening at the base of the cone-shaped left ventricle. The aortic-mitral continuity is a curtain of connective tissue that divides the ostium of the left ventricle into an inflow (mitral) and outflow (aortic) portion. This curtain is attached strongly to the left ventricular muscle medially and laterally by dense fibrous condensations, which can be identified as the dimples at each end of the aortic portion of the mitral annulus. These areas are named the right and left fibrous trigones and also receive fibers from the two dominant strut chordae inserted onto the aortic surface of the anterior mitral leaflet. They traverse the anterior leaflet obliquely and cross each other to blend into the opposite fibrous trigone. The plane of the aortic annulus lies at a higher level than the mitral annulus. The aortic annulus is attached to the top edge of the aortic-mitral continuity, which supports the posterior one-third of the aortic annulus. The anterior leaflet of the mitral valve is attached to the lower edge of the aortic-mitral continuity, which forms the well-defined fibrous portion of the mitral annulus and constitutes the anterior one-third to one-half of the mitral annulus.

This anterior portion of the annulus was thought to be non-distensible. Currently recognized is that it can stretch over time, particularly in disease states such as Marfan's syndrome.[9-11] This finding has led to a renewed recognition of the need for full-ring annuloplasty in patients with myxomatous-valve disease, or Marfan's syndrome. The posterior two-thirds of the annulus, the line of attachment of the posterior mitral leaflet to the muscular ostium, has a highly variable structure. It ranges from a well-developed line of fibrous condensation of the leaflet onto the muscle of the left ventricle to areas of almost non-existent support.[7] The corresponding basal postero-medial portion of the left ventricle also is sometimes thin and occasionally membranous.

The mitral orifice defined by the annulus has a circumference of approximately 9 cm in women and 10 cm in men. The area is 6.5 cm^2 for women and 8 cm^2 for men. After addition of the leaflets, these areas are reduced by approximately 2 cm^2. The long axis of the orifice is approximately 5 cm between the commissures and 4 cm in the anteroposterior axis.[6]

The anterior mitral leaflet is rhomboidal in shape and highly mobile. The posterior leaflet is flatter and elongated horizontally around the posterior annulus. The posterior leaflet is relatively less mobile compared with the anterior
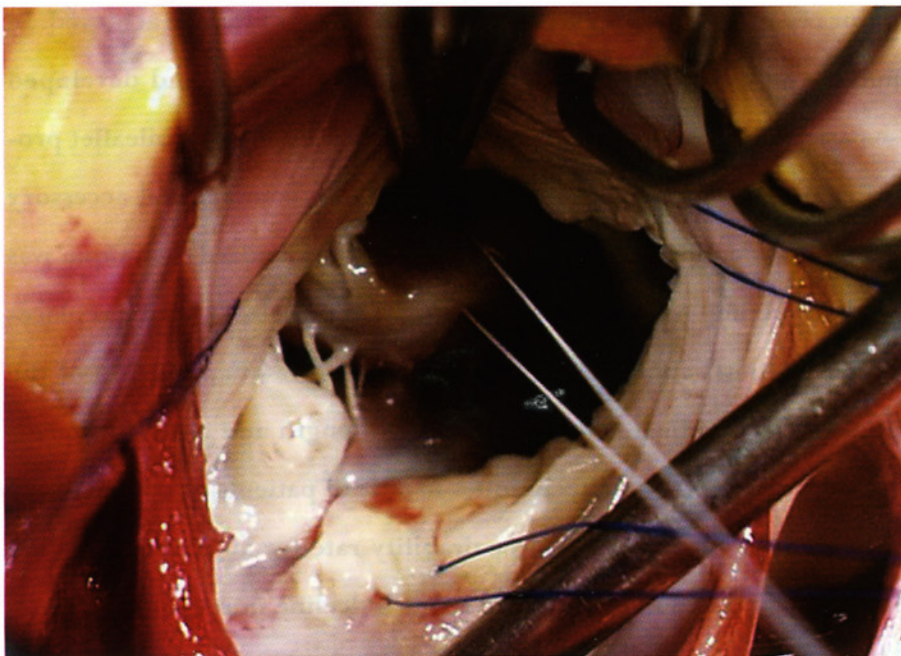

Figure 1. Details of the surgical technique USED. #5 PTFE suture is passed through the basal portion of the papillary muscle in a figure-of-eight fashion.

leaflet, because of a multiplicity of chordal attachment to its ventricular surface. It has three separate sections that allow it to lie flat against the wall of the left ventricle during diastole, which has been likened to the pleats in a skirt. The anterior and posterior leaflets are not fully separated at the anterolateral and posteromedial commissures. The commissural separation of the leaflets usually begins 3 mm to 8 mm from the annulus toward the mitral orifice, which appears to be important for maintenance of mitral competence because no chordae support these undivided commissural areas of the leaflets that can make repair of these areas difficult.

Unlike the posterior mitral leaflet, anatomy of the anterior mitral leaflet does not lend itself to repair of prolapsing segments by extensive leaflet resection. Although it is almost equal in surface area to the posterior leaflet, it has a base of fixed dimension (the aortic-mitral continuity) that occupies only one-third of the annular-mitral leaflet attachments. Little redundancy is present in its area. It has to swing freely through a wide arc, hinging on its attachment to the aortic mitral continuity, to achieve proper apposition with the U-shaped surface formed around the posterior two-thirds of the ostium of the left ventricle by the posterior leaflet. Even "triangular" resection, as described by Carpentier, is prone to cause loss of mobility of the leaflet due to the "drumhead" effect across the free edge of the leaflet.

The lower portions of the atrial surfaces of the leaflets are irregular due to insertion of the chordae onto their ventricular aspect. This appearance is particularly pronounced on the anterior leaflet where the lower third is markedly thickened, and is known as the "rough zone." The rough zone of the posterior leaflet is less constant and occupies approximately one-half of the depth of the leaflet centrally and gradually diminishes until absent at the commissures. The rough zones correspond to the lines of apposition of the leaflets. Whereas they extend equal distances from the free edges of the leaflets (approximately 8 mm to 10 mm), they occupy one-third of the depth of the anterior leaflet and more than one-half of the posterior leaflet due to the different shapes of the leaflets.

The papillary muscles are attached to the posterior left ventricle located
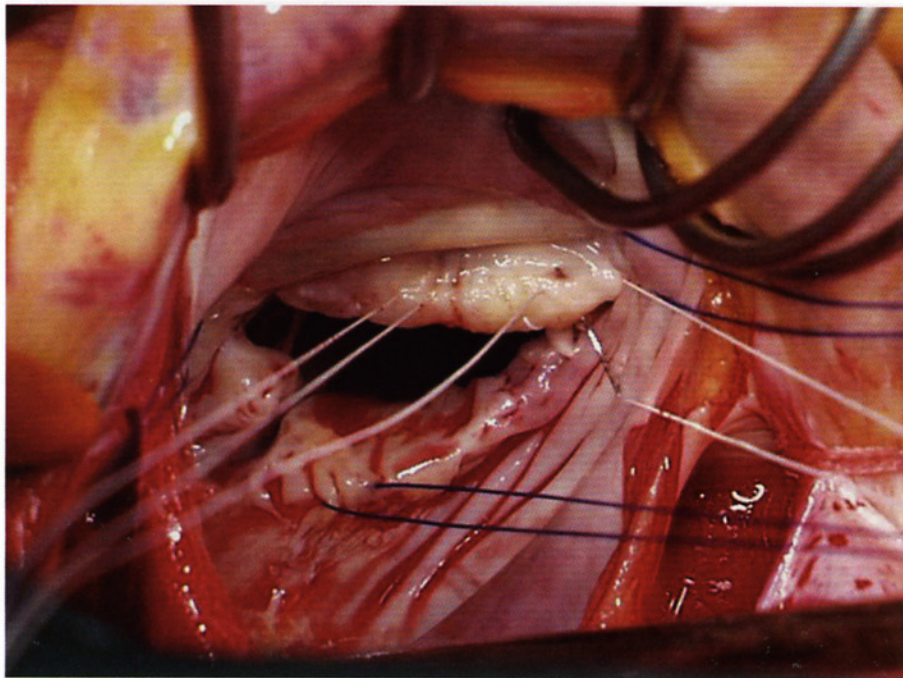
Figure 2. Each suture is passed through the edge of the leaflet twice, in a rolling fashion toward the center of each pair. The needle can be seen completing this for the medial pair of chordae.

below the commissures of the mitral valve rather than the center of the leaflets. They are attached posterior to the transverse plane that joins the commissures. The papillary muscles account for up to 25% of left ventricular mass and function. Their anatomy is highly variable, and result from the classical two well-formed structures to no identifiable muscles.[12] The annular-papillary distance and alignment of the muscles

are critical for mitral competence and normal left ventricular-wall motion.[13] Widening of the interpapillary distance between the bases of the papillary muscles, or an increase in the distance from the posterior papillary muscle tip to the mitral annulus as seen in dilated cardiomyopathies, routinely produces mitral regurgitation.[14]

The chordae tendinae stabilize not only the leaflets but also the adjacent
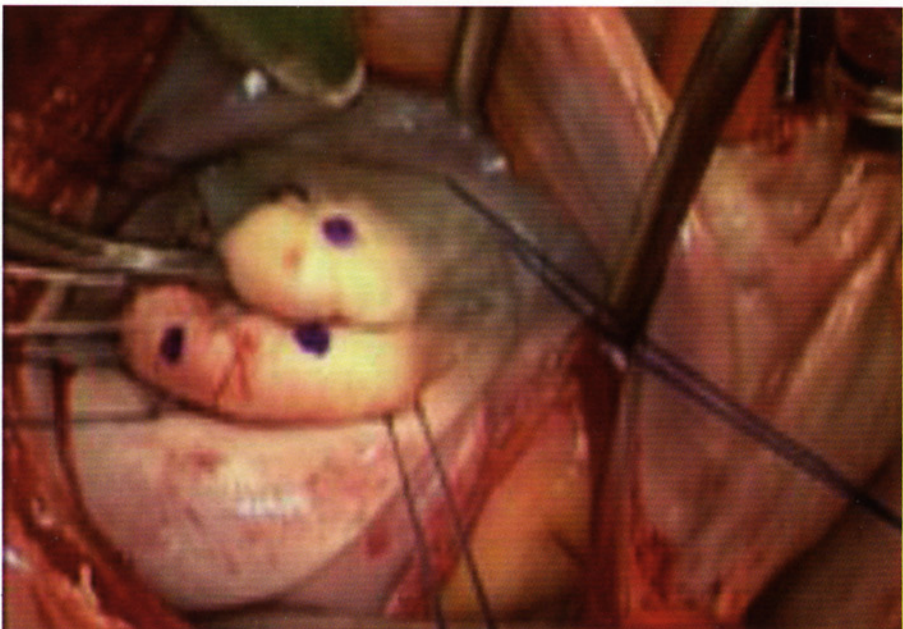
Figure 3. Dots are placed to mark the desired final line of leaflet apposition using a marker pen (top). Note also the four annular 2-0 polypropylene stay stitches.
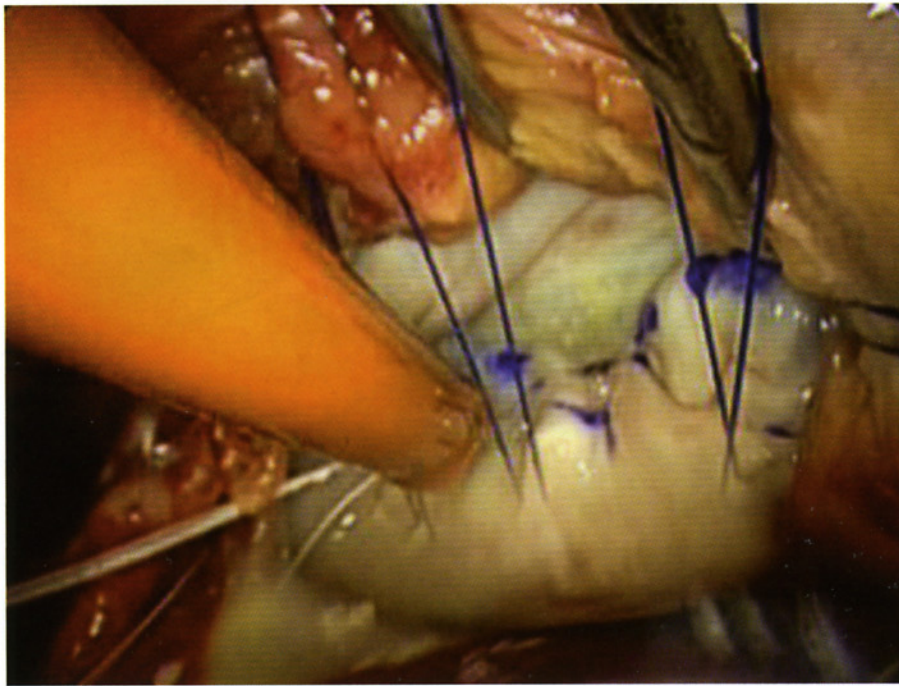
Figure 4. The left ventricle is inflated with saline and the chordal length is adjusted.

myocardium of the free walls. The first order or marginal chordae attached to the free edges of the leaflets are the thinnest and structurally weakest. The marginal chordae attached to the center of the leaflets are thicker and stronger than those near the commissures. Their function is to maintain apposition of the free edge of the leaflets at the end of diastole and the start of systole, after which their role diminishes if leaflet apposition is normal. The second order,

or so-called "strut chordae," of the anterior leaflet are notably strong. The second order chordae arise lower on the papillary muscles than the other chordae. They prevent doming of the leaflets into the left ventricle during systole.[15,16] On the anterior leaflet, their collagen radiates obliquely across and upwards into the opposite fibrous trigone. They anchor the papillary muscles securely in relation to the fibrous trigones and help maintain left ventricular geometry.
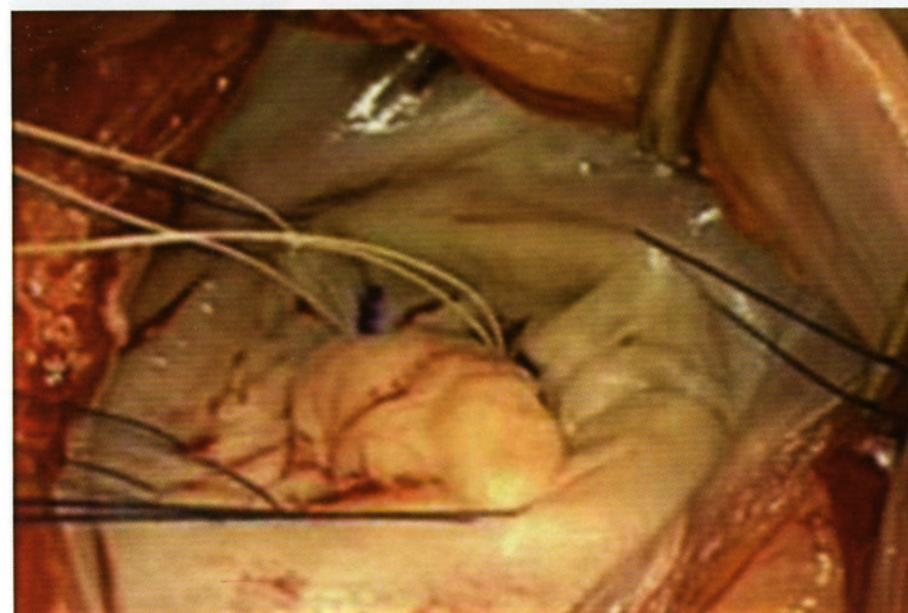

Figure 5. The annular stay stitches are used to simulate the correct annular size during further insufflation and chordal adjustment.

Their tensile strength is 10 times that of the thin marginal chordae. The third order chordae are seen mainly on the posterior leaflet and often arise directly from the wall of the left ventricle. They also are strong and maintain the position of the posterior annulus relative to the tips of the papillary muscles.

Transfer of load is present during systole away from the marginal chordae to the secondary or strut, chordae then tertiary chordae, and finally the annulus. The load on the marginal chordae also is reduced by good leaflet apposition.[13] If the mitral leaflets can oppose each other with 5 mm to 10 mm of apposition, the hydraulic force of blood acting on the ventricular surface of these leaflets causes them to be held in place, aligned against each other by friction. This phenomenon has been compared to the forces that keep the keystone of a Roman arch in position. Also, as the rough areas of the leaflets move to the line of apposition and the leaflets are rolled onto each other, the secondary chordae become aligned more directly below the line of apposition. Any condition in which the apposition is not preserved, such as annular or ventricular dilation, leads to increased loading of the marginal chordae. When this state is combined with chordal pathologic findings and unfavorable chordal anatomy, failure of the marginal chordae is prone to occur. The marginal chordae that radiate to the center of the leaflets are acting at a flat angle in many cases at a significant mechanical disadvantage, because the papillary muscles are located beneath and behind the commissures.

The corollary of these observations is that the central goals of mitral-valve repair must be to restore optimal overlap of the leaflets by proper annular reduction in combination with optimal artificial chordal alignment. This is dependent upon the chordal length and alignment of the papillary muscles in relation to the mitral annulus, annular dimensions, and the interpapillary distance. In the techniques used in this study, these three sets of dimensions are evaluated and adjusted simultaneously.

Attachment of the left atrium to the annulus and ostium is variable. The left atrial muscle continues downward over the annulus onto the atrial surface of the leaflets. This layer of muscle is known as the "atrialis muscle," and is most constant over the anterior leaflet where

it extends over the aortic mitral continuity onto the anterior leaflet. At its termination with the anterior leaflet, it forms the hinge point of the anterior leaflet seen on echocardiography.[7]

Left-atrial contraction causes mild constriction and elevation of the mitral annulus just before the onset of left-ventricular systole. During early left-ventricular systole, the area of the mitral orifice is reduced by approximately 25% to 30% by effects of the left-atrial contraction and left-ventricular contraction. This change not only reduces the length of the posterior two-thirds of the annulus, but also increases the height of the center of the antero-posterior dimension of the mitral annulus, in the shape of a saddle with the pommel anteriorly. These changes in the annulus shorten both the commissure-to-commissure and antero-posterior dimension of the annulus and bring the posterior mitral leaflet into closer apposition with the anterior leaflet. Total leaflet area is approximately 150% to 200% of the area of the mitral orifice in diastole and the leaflets overlap by approximately 1 cm at the free edges during systolic apposition of their rough area at peak systole. The anterior leaflet alone has sufficient area to occlude the entire mitral orifice, because of the reduced orifice area in systole.

The mitral annulus is elevated into the left atrium relative to the left-ventricular apex during diastole, and reaches its maximum atrial position during atrial systole. At the start of isovolumic ventricular systolic contraction, the annulus descends toward the apex.[17,18] The end-diastolic and isovolumic-systolic changes are simulated in the American Correction by progressive inflation of the left ventricle with saline.

PATHOLOGY OF MITRAL-VALVE INSUFFICIENCY

In the United States, the two most common causes of mitral insufficiency are myxomatous degeneration of the mitral valve and acute or chronic ischemic cardiomyopathy. Myxomatous degeneration is characterized by enlarged, thickened prolapsing leaflets with elongated chordae, and annular dilatation, thought to be due to structural deterioration of the leaflets and chordae secondary to abnormality of
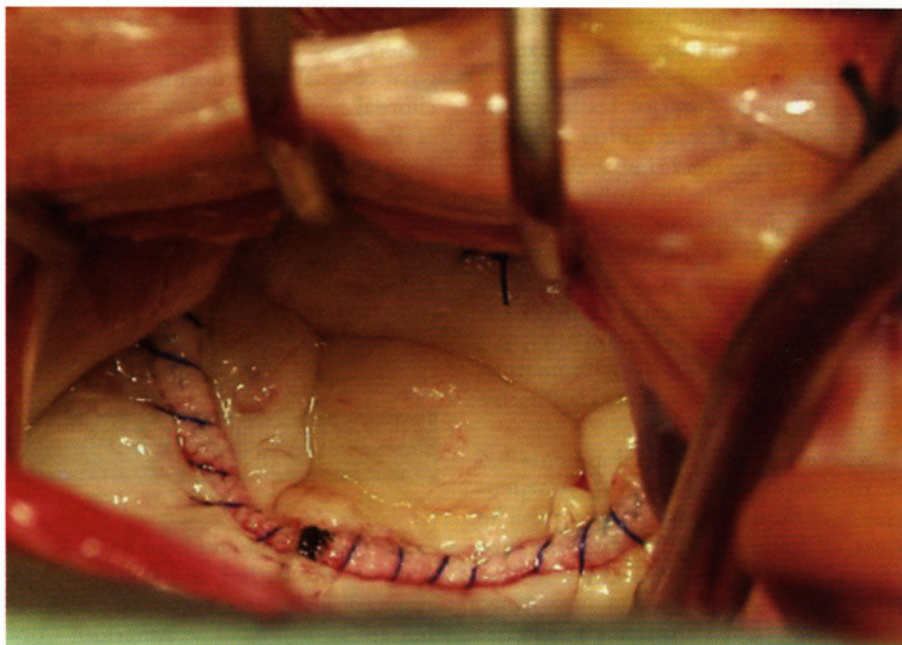
Figure 6. The chordae are tied down and the flexible annuloplasty ring is attached with a continuous 2-0 polypropylene suture. The dots and PTFE knots are now buried in the line of leaflet apposition.

fibrillin synthesis.[19] Despite these changes, the majority of myxomatous valves are competent.[20,21] Mitral regurgitation can develop because of severe annular dilatation alone, as is seen in "Barlow's Valves." Asymmetrical leaflet apposition due to chordal elongation or rupture is the most commonly encountered pathologic finding and usually associated with marked annular dilation. Currently, recognized is that the leaflets of myxomatous valves, while large, redundant, and excessively elastic are strong, whereas the chordae are seriously weakened.[22] Systolic anterior motion (SAM) of the anterior mitral leaflet is rarely seen in the unoperated state. Thus, large, redundant leaflets alone cannot be the cause of SAM. Resection of myxomatous-valve tissue not only is unnecessary, but is actually harmful because it reduces valuable leaflet area. This, in turn, demands a greater reduction in annular size by use of a smaller annuloplasty ring—the real cause of perioperative SAM.

Clearly, the ideal procedure should be directed at restoration of the proper line of apposition of the leaflets by replacement of diseased chordae and correction of excessive annular dilatation by the amount necessary to enhance leaflet apposition and reduce chordal stress. The problem has been that in the arrested, relaxed heart, assessment of artificial chordal length and extent of optimal annular reduction

have been difficult to predict. Adjacent valve tissue is not always an accurate guide, as leaflet symmetry is often lost in myxomatous disease.[17,18] This difficulty has largely existed because the left ventricle elongates during late diastole, and chordae placed in the relaxed heart tend to be too short, and cause leaflet restriction in the beating heart. Choice of the correct size of annuloplasty ring in the relaxed heart also is difficult and increases the risk of failure of the repair either from residual mitral insufficiency (too large) or SAM (too small).

PRINCIPLES OF SURGICAL REPAIR

To overcome these problems, surgical techniques for repair of myxomatous-mitral valves have undergone substantial modification in the author's center over the last 30 years. Initially, the techniques described by Carpentier were used. Carpentier called these "The French Correction",[22-28] which involved the use of a rigid annuloplasty ring designed to restore the annular proportions of a normal mitral valve based on the size of the anterior mitral leaflet of the patient. The chordal abnormalities were corrected by means of resection of leaflet segments affected by diseased chordae (quadrantic or triangular resection) or transposition of less-affected chordae from other more normal segments.

These techniques have a number of intrinsic disadvantages. They can be applied only to approximately 70% of diseased valves. Anterior leaflet repair is difficult.[24] The annulus is made immobile by the rigid ring, and is usually mildly stenotic. Systolic anterior motion of the anterior-mitral leaflet is present in 15% of patients. The posterior leaflet is usually made immobile by distortion of the remaining chordae, the socalled, "toilet seat" deformity. All chordae are affected by myxomatous disease and the use of diseased native chordae is not ideal.

Three important developments led us to completely abandon the Carpentier techniques many years ago. The first of these was introduction into clinical practice of PTFE suture material as an artificial chordal substitute. The author and colleagues have currently accumulated a large experience with this material during a 20-year period. Our favorable experience with chordal replacement is similar to that of others.[30-38] The PTFE closely mimics the mechanical characteristics of natural chordae.[39] PTFE allows correction of chordal abnormalities at all sites on both leaflets. It produces restoration of the physiologic mitral-valve function.

Secondly, the recognition that the chordal pathologic findings and annular dilatation are responsible for the mitral regurgitation, not the leaflets, leads one to believe that leaflet resection was unnecessary and correction of the marginal chordal abnormalities by artificial chordal replacement with preservation of the leaflets seemed a more logical approach.

Thirdly, the active role of the mitral annulus, and critical importance of the interplay between annular dimensions and chordal length and leaflet apposition, is currently more fully understood.

SURGICAL TECHNIQUE—THE AMERICAN CORRECTION

Surgical techniques have been developed by the author and colleagues to accomplish this in a systematic fashion, and are known as "The American Correction." It is these evolutionary techniques that currently allow repair of almost 100% of myxomatous valves.[40] The "French" and "American" corrections are based on entirely different concepts. In the French Correction, an attempt is made to restore normal proportions to the relationships of the annulus and anterior and posterior leaflets to each other. To accomplish this, the area of the anterior leaflet is used to establish an index dimension without consideration of the size of the posterior leaflet or preoperative annular dimensions.[23] The size of the rigid annuloplasty ring is based only on this measurement. The wide variations in the areas of myxomatous-posterior leaflets relative to the anterior leaflet are compensated for by posterior leaflet sliding maneuvers and "posterior leaflet height" adjustments in which posterior leaflets are reduced in size by resection. They are viewed as a problem in need of correction.[27,29] Leaflet prolapse from elongated or ruptured chordae is treated by resection of the prolapsing segment.

In the American Correction, the wide variations in sizes and proportions of the annulus, and anterior and posterior leaflets characteristic of myxomatous mitral-valve disease, are recognized as an integral part of the disease state and are viewed as advantageous. The weak myxomatous marginal chordae are viewed as the primary cause of the leaflet prolapse along with the annular dilatation and large, strong leaflets, are preserved without any resection to enhance restoration of the normal 1-cm apposition of the free edges of the leaflets. After the free edges of the leaflets have been restored to proper alignment by PTFE chordal replacement, the left ventricle is distended with saline and a flexible annuloplasty ring is chosen of the largest size that aligns the leaflets to ensure closure with a 1-cm line of apposition. All adjustments are made by manipulation of PTFE chordal length and choice of the total circumferential annular dimension during left-ventricular distension. Leaflet resection is not required. No effort is made to restore "normal" morphology beyond restoring the "normal" line of apposition.

In myxomatous disease, a full flexible ring annuloplasty is used routinely, because of the evidence mentioned earlier that the anterior one-third of the mitral annulus can enlarge despite being a fibrous structure[9-11] On average, ring annuloplasty reduces the annular anteroposterior diameter by 10 mm to15 mm. Routinely, results are good. Elimination of all mitral regurgitation is achieved routinely. In younger patients, especially younger females, a 7-cm to 10-cm midline incision, low on the chest, is used. Despite application of the American Correction to more complex groups of patients, reoperation rates have been comparable to the highly selected French Correction patients.[39]
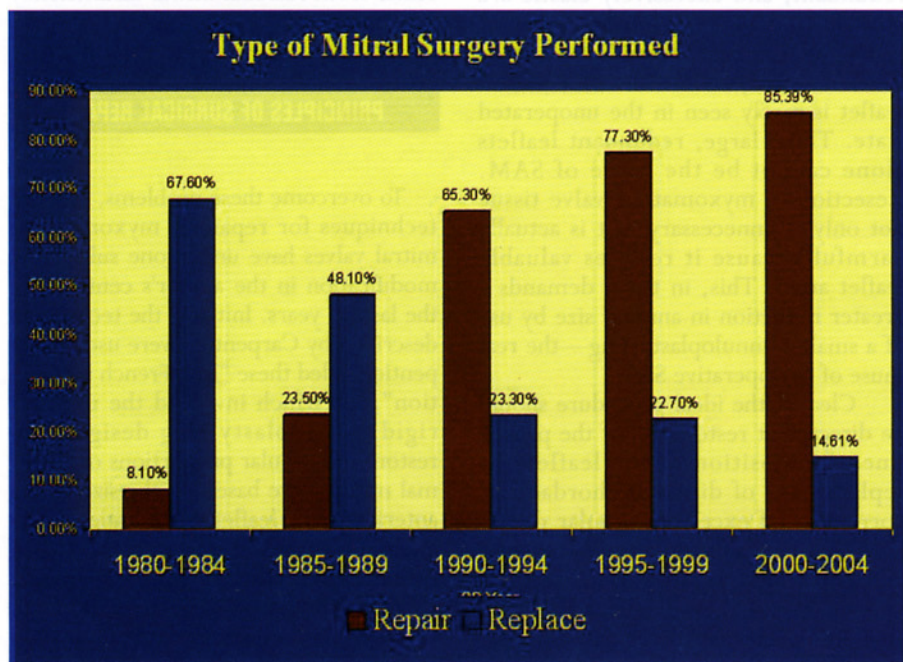

Figure 7. Trends in proportion of valves repaired according to time period of surgery.

DETAILES OF SURGICAL TECHNIQUE

The first step in the American Correction is a careful review of the pre- and intraoperative transesophageal echocardiography. The chest is entered either through a full median sternotomy or a limited lower sternal incision. The superior and inferior vena cavas are mobilized extensively. Cardiopulmonary bypass is established with bicaval 24-French cannulas, vena caval tapes, and dynamic venous drainage. The heart is arrested with cardioplegia. The left atrium is entered through an extensive interatrial incision and the mitral valve is exposed. Stay stitches of 2-0 polypropylene are placed: two anterior to the commissures into the right and left fibrous trigones, and two in the posterior annulus. The annulus, valve leaflets, chordae, and papillary muscles are examined. The left ventricle is inflated with 75 mL to 100 mL of saline. An aortic-vent needle open to atmosphere is used to minimize the risk of air embolism to the right ventricle during these maneuvers. The abnormal structures are identified. Leaflet prolapse is corrected by insertion of #5 PTFE sutures. The sutures are first placed into the bases of the postero-medial, antero-lateral papillary muscles in a figure-of-eight fashion, or both (Fig. 1). An effort is made to select an attachment site that will duplicate the normal chordal geometrical orientation.

The PTFE chordae are brought through the free edge of the prolapsing segment as a rolling stitch (Fig. 2). This second passage through the tissue provides sufficient friction to hold the length of the chordae constant during adjustment of the realignment of abnormal leaflet segment with the adjacent and opposite leaflet edges. Dots are placed 5 mm to 10 mm from the free edges of the leaflets with a marker pen to mark the desired line of apposition (Fig. 3). The ventricle is again inflated with saline (Fig. 4). The two stay stitches in the posterior annulus are brought forward to simulate effects of the annuloplasty ring on the posterior annulus to bring the lines of dots on both leaflets into apposition. Leaflet alignment is checked and the PTFE chordae are further adjusted as needed (Fig. 5). In patients with symmetrically prolapsing leaflets and annular dilatation only ("Barlow's" valves), no PTFE is needed

and the annulus is adjusted after marking the leaflets, usually with a 33-mm or 31-mm ring. At this point, the mitral orifice is sized according to its circumference and the appropriate flexible ring chosen.

The PTFE is tied down with 10 throws and the knot is locked with a 6-polypropylene stitch tied over the end of the PTFE knot to prevent sliding of the PTFE knots. The annuloplasty ring is secured to the annulus with the previously placed trigonal 2-0 sutures in continuous fashion (Fig. 6). The valve is again tested by saline insufflation of the left ventricle. The left atrium is closed. The air is evacuated, warm blood cardioplegia given, and aorta unclamped. The operation is completed and repair checked by transesophageal echocardiography (TEE). Further technical details, including a video of this technique, are available at www.geraldlawriemd.com. All patients undergo postoperative transthoracic echocardiography before discharge.

ISCHEMIC MITRAL REGURGITATION

The term, ischemic mitral regurgitation, describes a complex group of conditions usually associated with intrinsically normal mitral-valve leaflets and chordae. Acute mitral regurgitation may occur after extensive inferior-wall infarction, which may cause elongation

of the mitral annular-papillary muscle distance due to compromise or rupture of the postero-medial papillary muscle.

Chronic ischemic mitral regurgitation is seen in association with inferior-wall motion abnormalities that cause a restrictive defect due to inferior and lateral displacement of the posteromedial papillary muscle.[41] In patients with diffusely poor left ventricular function, spherical dilation of the left ventricle causes displacement of the bases of the papillary muscles downward and outwards away from each other, again causing restrictive mitral regurgitation. Surgical correction of both the latter conditions involves marked reduction of the length of the posterior mitral annulus, especially the posteromedial portion as described originally by Kay and colleagues,[41-43] although the initial annular dimensions may be normal. The annuloplasty has been shown to bring the tips of the papillary muscles closer together and improve leaflet apposition.[44,45]

SURGICAL TECHNIQURE—ISCHEMIC MITRAL REGURGITATION

The author and colleagues have developed a two-layer annuloplasty technique for correction of this type of ischemic mitral regurgitation. The first layer consists of a Kay plication annuloplasty to abolish most of the P3 segment
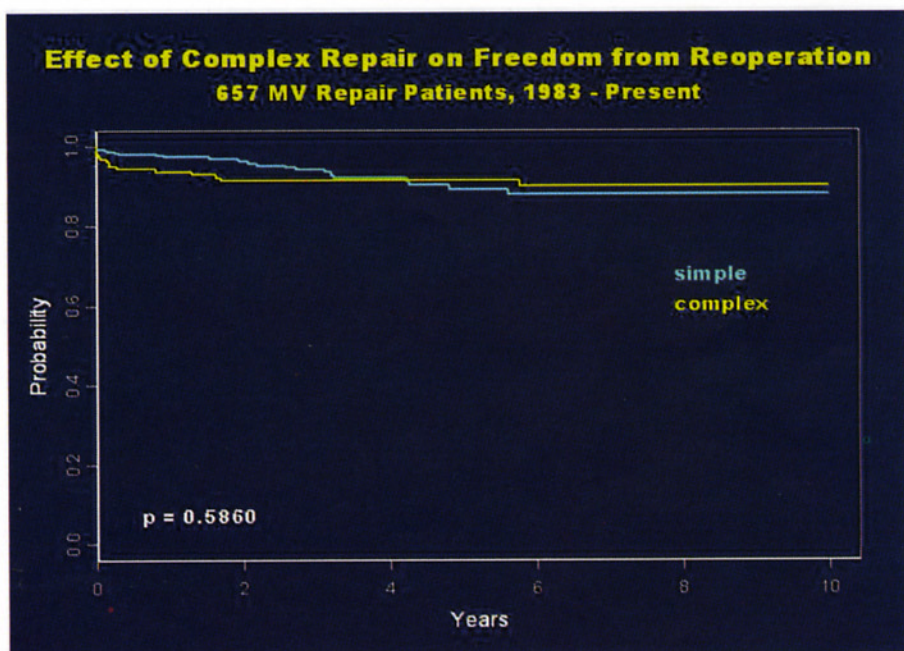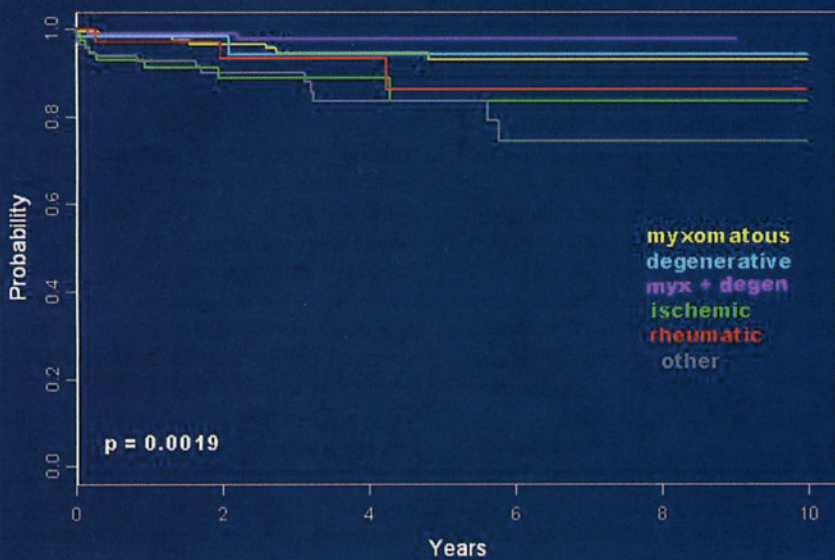

Figure 8. Comparison of simple and complex repairs on reoperation rates (Kaplan-Meier analyses).

Figure 9. Comparison of reoperation rates for various leaflet pathologies. The best durability is obtained in myxomatous valves.

of the mitral annulus at the postero-medial commissure.[41] The Kay technique allows much greater annular reduction than can be achieved with ring annuloplasty alone. This procedure is followed by a flexible partial ring annuloplasty of the entire mitral posterior annulus. This additional layer is designed to stabilize the plicated medial portion, as the Kay annuloplasty is less durable than a ring annuloplasty.[46] This technique has produced durable and stable reduction of the posteromedial annulus and elimination of the ischemic mitral regurgitation.

RESULTS

Use of these improved techniques have enabled us to increase our overall repairability rate to more than 90%, with a repair rate of close to 100% for myxomatous valves (Fig. 7). Analysis of long-term follow up has demonstrated good durability of the repairs despite the increased repair rate (Figs. 8 & 9). Analysis of reoperation rates over time has shown no increase recently despite the more complex repairs performed (Fig. 8). In fact, the rates of reoperation have declined (Fig. 10). The major problem remains the rheumatic valves with fibrotic and calcified leaflets. The major determinant of need for reoperation has been the preoperative pathologic findings (see Fig. 9). Patients with rheumatic and other chronic inflammatory leaflet pathologies continue to have lower repairability rates. Our clinical and echocardiographic results using PTFE chordae are similar to those documented by other authors.[33-37]
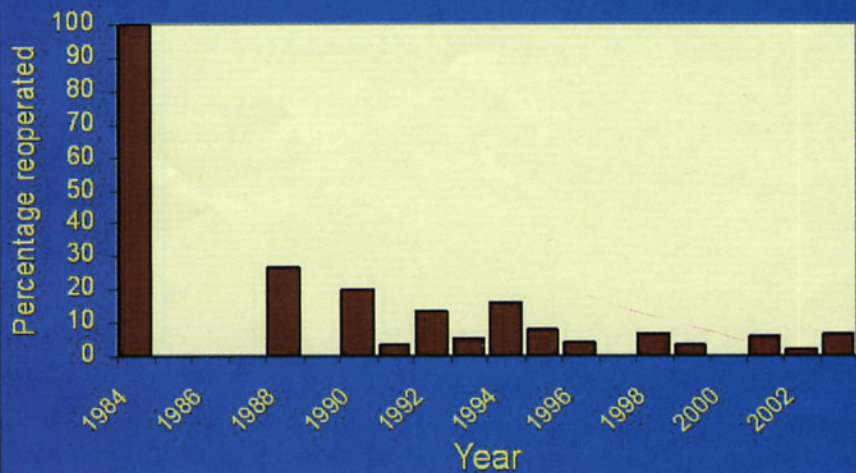
MANAGEMENT OF ATRIAL FIBRILLATION

Atrial fibrillation develops eventually in mitral stenosis and regurgitation as the left atrium dilates and develops myocardial and electrical changes. Atrial fibrillation leads to embolic complications, impaired cardiac function, and reduced long-term survival. Correction at the time of valve surgery is performed currently in all our patients. The procedure of choice is the Maze procedure that has a 98% drug-free cure rate of atrial fibrillation at three months of follow up. Excision of the left-atrial appendage is an integral part of the procedure.[47,48]

CONCLUSION

Open isolated repair of insufficient mitral valves is usually performed currently through limited-access exposures with a mortality rate of less than 1% and a high long-term success rate. Open repair can be combined with other heart-valve surgeries, coronary bypass or Maze procedures, with further enhancement of outcome. As the first decade of the 21st century reaches its midpoint, mitral-valve repair continues to evolve as the treatment of choice for the majority of patients with mitral insufficiency. ■

Figure 10. Overall number of patients requiring reoperation according to the year of surgery.

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