# ADVANCES IN MITRAL VALVE REPAIR

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## INTRODUCTION

The development of surgical techniques to preserve the diseased mitral valve has challenged surgeons for more than a century. Enhanced understanding of its anatomy, physiology and pathology over the last 50 years have led to rapid development of reproducible, durable techniques for mitral valve repair that apply to almost all mitral valve pathology. Despite this, recent data from the Society of Thoracic Surgeons National Cardiac Database documents that only 37.7% of U.S.-based surgeries for mitral regurgitation were repairs. The repair rate for patients with combined regurgitation and stenosis fell to 19.7%.1 In contrast, reparative techniques developed in our center have produced a current repair rate in excess of 95% for mitral regurgitation (Figure 1).

## APPLIED ANATOMY AND PHYSIOLOGY

Detailed knowledge of the anatomy and physiology is essential for any surgeon or cardiologist contemplating mitral valve intervention. The mitral valve consists of an anterior and posterior leaflet attached to the heart at the mitral annulus. The anterior leaflet, through the aorric-mitral fibrous continuity, is closely related to the aortic root. The leaflets are stabilized by chordae tendinae attached from them to the left ventricle papillary muscles. The mitral annulus is a weak and distensible structure that is incomplete in many patients. The posterior two-thirds of the annulus attaches to the muscular ostium of the left ventricle and dilates when the left ventricle enlarges.

The anterior leaflet is rhomboidal in shape and highly mobile. The posterior leaflet is flatter, elongated horizontally around the posterior annulus, and less mobile than the anterior leaflet. Three separate sections allow it to lie flat against che wall of the left ventricle during diastole. The anterior and posterior leaflets are not fully separated at the anterolateral and posreromedial commissures. The commissural separation of the leaflets usually begins three to eight millimeters from the annulus toward the mitral orifice, which appears important for maintaining mitral competence.

The papillary muscles are located below the commissures of the mitral valve rather than the center of the leaflets, often creating a mechanical disadvantage for the chordae radiating to the center of the leaflets at an almost horizontal angle. The papillary muscles account for up to 25% of left ventricular mass and function. Their anatomy is highly variable, ranging from the classical two well-formed structures to no identifiable muscles at all. The annular-papillary distance and the alignment of the muscles are critical for mitral competence. Widening of the interpapillary distance between the bases of the papillary muscles or an increase in the distance from the posterior papillary muscle $ti_p$ to the annulus - as seen in dilated cardiomyopathie - routinely produces mirral regurgitation.

The chordae tendinae stabilize nor only the leaflets but also the adjacent myocardium of the free walls. The thin, first-order chordae attached to the free edges of the leaflets maintain apposition of rhe free edge at the start of systole, after which their role diminishes. The second-order chordae are thick and prevent leaflet prolapse into the left atrium during peak systole. The third-order chordae on the posterior leaflet maintain the position of the posterior annulus relative to the tips of the papillary muscles. The chordal-papillary complex also maintains positioning of the left ventricular free wall during left ventricular systole to help stabilize the ventricle's conical shape. Severing these connections, as occurs in prosthetic mitral valve replacement, creates a more spherical ventricle.

Left atrial contraction causes mild constriction and elevation of the mitral annulus just prior to the onset of left ventricular systole. During left ventricular systole, the effects of left atrial and ventricular contraction reduce the mitral orifice by 25-30%, which reduces the length of the annulus's posterior two-thirds and elevates the center of the mitral annulus's antero-posterior dimension. These changes shorten the antero-posterior dimension of the annulus and bring the posterior mitral leaflet into closer apposition with the anterior leaflet. Total leaflet area is about 150% of the mirral orifice area, and the leaflets $overla_p$ by about 1 cm at the free edges during systolic apposition.

## PATHOLOGY OF MITRAL VALVE STENOSIS

The most common cause of mitral stenosis in the United States is rheumatic fever. Following the initial episode of rheumatic carditis, the inflamed leaflets adhere to each other on their atrial surfaces at the commissures, leaving a central orifice and causing mitral stenosis. In some cases this represents the main pathological change, and the leaflets, chordae and papillary muscles remain otherwise relatively normal - findings most commonly seen in young patients. This represents the ideal pathology for closed mitral commissurotomy or catheter-based interventions because the leaflet fusion will usually separate along the original anatomic lines of the commissures and the chordae, and papillary muscles will allow good motion of the still-flexible leaflets. The resultant valve after commissurotomy is relatively normal.

Surgical closed mitral commissurotomy **with** or without cardiopulmonary bypass has been highly successful for more than fifty years; the reported characteristics most favorable for this surgery include young age, no thickening or calcification of the leaflets, mobile anterior leaflet, minimal chordal shortening fusion and chickening. 2-6

Unforcunarely in many cases, especially in older patients or patients experiencing severe cardiris, changes in the leaflets, chordae tendinae, papillary muscles and left atrium are more severe and progressive. These valves have very severe fibrous leaflet fusion at the commissures that is often calcified and will not separate without sharp dissection. The leaflets are thickened, shrunken, calcified and relatively immobile, especially at the free edge. The chordae are shortened, thickened, fused together along with the papillary muscles and may almost disappear, and the tip of the papillary muscle fuses with the leaflet. The orifice's atrial side becomes deep and conical, with a fixed stenotic orifice that causes mitral regurgitation. The left atrium enlarges over time with patchy fibrosis, hypertrophy and other cellular and electrical changes leading to chronic atrial fibrillation.

These types of valves cannot be treated successfully by closed methods. Indeed, in some cases the valve destruction by fibrosis, calcification and ulceration is so extensive that prosthetic valve replacement is indicated.

## IMPLICATIONS FOR CHOICE OF THERAPY N MITRAL STENOSIS - ROLE OF EXTENDED OPEN MITRAL COMMISSUROTOMY

The pathological severity of the stenotic mitral valve can be identified reasonably accurately by TEE. The grading system that examines leaflet mobility, valvular thickening, subvalvular thickening and valvular calcification on scales of 1-4 is useful.? A number of studies have shown poorer outcomes in patients **with** valves graded at 7-8 **or** above. 2 The average score in the randomized studies comparing closed commissurocomy with balloon valvocomy was 6.7 and age was 30 years.2

Patients with higher scores and of older age require a combination of traditional open mitral commissurotomy with advanced techniques of mirral valve reconstruction. These may involve sharp dissection of the commissures, splitting apart of papillary muscles and chordae, chordal division and synthetic PTFE chordal replacement; leaflet debridement resection with or without a pericardia! patch; ring and/or Kay annuloplasty. B9

When mitral regurgitation severity is already more than 2+, catheter techniques may be contraindicated since in many patients they have exacerbated mitral regurgitation in mitral stenosis by at least one grade.2, [10,11]

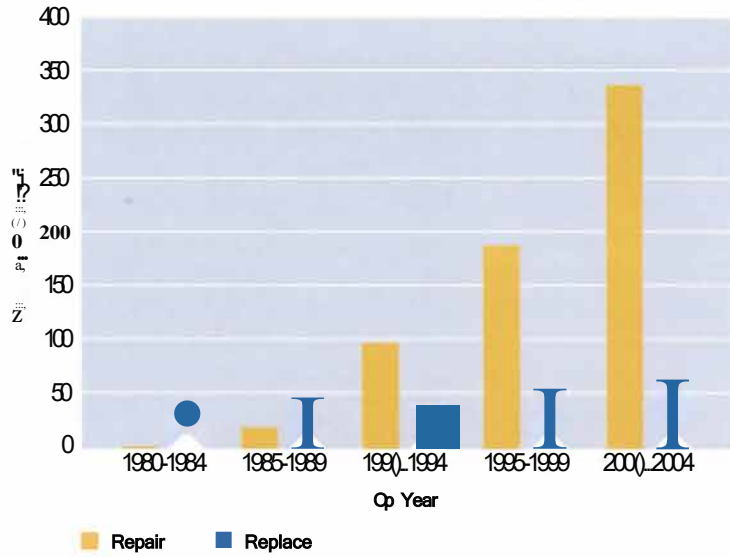


**Figure 1.**
Comparison of rates of mitral valve replacement and repair over time on one service at the Methodist DeBakey Heart Center. Currently, 95% of valves are repaired.

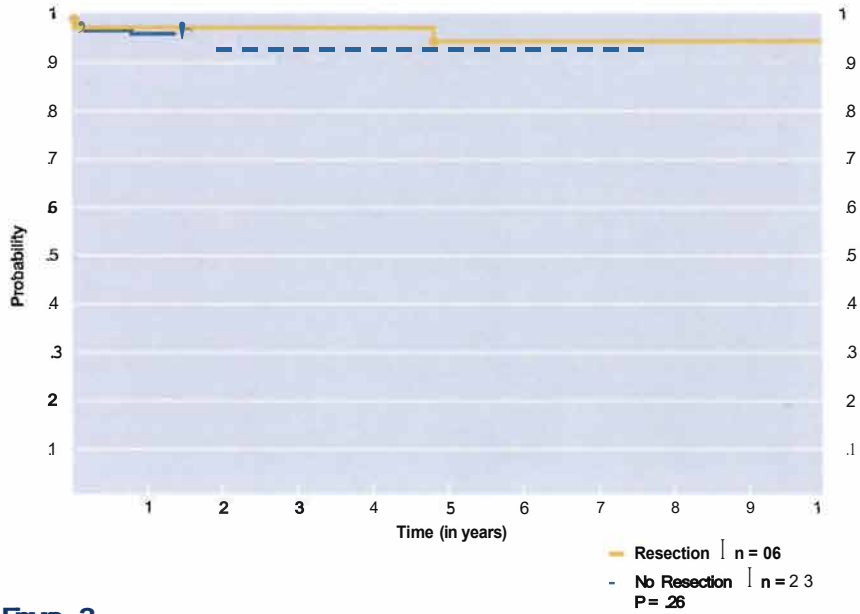


**Figure 2**

Comparison reoperation rates for "American correction" performed at Methodist DeBakey Heart Center versus traditional Carpentier techniques.

This is usually easy to correct when micral commissurotomy is being treated by direct surgery. Acute, severe mitral regurgitation after catheter interventions usually can be corrected by open mitral valve repair.

## PATHOLOGY OF MITRAL VALVE INSUFFICIENCY

In this country, the rwo most common causes of mitral insufficiency are myxomarous degeneration of the mitral valve and acute or chronic ischemic cardiomyopathy. $M_{y\,x}$omarous degeneration is characterized by enlarged, thickened prolapsing leaflets with elongated chordae and annular dilatation, purportedly due to structural deterioration of the leaflets and chordae secondary to abnormality of fibrillin synthesis.12 Despite these changes, the majority of myxomacous valves are competent.[13,14] Mitral regurgitation develops because of severe annular dilatation and/or asymmetrical leaflet apposition from chordal elongation or rupture. Mose of these valves have a combination of problems.

## SURGERY FOR MITRAL VALVE INSUFFICIENCY - THE AMERICAN CORRECTION

Surgical techniques for myxomatous mitral valve repair have undergone substantial modification at the Methodist $DeBak_{e\,y}$ Heart Center over the last 30 years. Initially, the "The French Correction" techniques described by Carpentier were used,15 which involved a rigid annuloplascy ring designed to restore the annular proportions of a normal mitral valve based on rhe size of rhe patient's anterior mitral leaAer. The chordal abnormalities were corrected by resecting leaflet segments affected by diseased chordae (quadranric resection) or transposing less-affected chordae from ocher more normal segments.

These techniques have a number of intrinsic disadvantages. They can be applied only to about 70% of diseased valves. Anterior leaflet repair is difficult. The annulus is immobilized by the rigid ring and usually mildly stenotic. Systolic anterior motion of the anterior mitral leaflet is seen in 15% of patients. Distortion of che remaining chordae usually immobilizes the posterior leaflet - the so-called "toilet sear" deformity.

Two important developments led us to completely abandon the Carpentier techniques. The first was the inrroducrion of PTFE suture material as an artificial chordal substitute, which we have used successfully for the past 20 years. The PTFE closely mimics the mechanical characteristics of natural chordae. It allows correction of chordal abnormalities at all sites on both leaflets and produces restoration of the physiological mitral valve function.

The second development was the demonstration chat myxomatous degeneration affects the leaflets and chordae in different ways. $M_{y\,x}$omatous mitral valves are characterized by large annular dimensions, large chickened leaflets and chickened and elongated chordae. Recent studies of the valves' mechanical characteristics have shown that the leaflets are abnormally elastic but retain normal strength. The chordae are also abnormally elastic but are mechanically weak. This makes them prone co over-elongation and rupture.16 Thus, correcting the chordae *by* artificial chordal replacement, while preserving the leaflets, seems a logical approach.

We have developed surgical techniques to accomplish this in a systematic fashion.

Known as "The American Correction," these evolutionary techniques allow repair of almost 100% of myxomatous valves. Details of these techniques may be

viewed at www.geraldlawriemd.com.

The "French" and "American" corrections have contrasting principles. In the French Correction, the area of the anterior leaflet is used co establish an index dimension without considering the posterior leaflet size or preoperative annular dimensions.15 The size of rhe rigid annuloplasty ring is based on this measurement, and an attempt is made co restore the relationships of the annulus and the anterior and posterior leaflets to normal proportions. Leaflet prolapse is treated by resection of the prolapsing segment. The wide variations in the areas of myxomatous posterior leaflets relative to the anterior leaflet are compensated for by posterior leaf.. let-sliding maneuvers and "posterior leaflet height" adjustments. In the American Correction, the wide variations in the sizes and proportions of the annulus and anterior and posterior leaflets characteristic of myxomatous mitral valve disease are accepted. The weak myxomacous chordae are considered the primary cause of the leaflet prol$_{ap}$se, and the large, strong leaflets are preserved without resection to enhance restoration of the normal one-centimeter apposition of the leaflets' free edges. Once the free edges have been restored co 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 co close with a 1 cm overlap. All adjustments are made by dynamic manipulation of PTFE chordal length and choice of the coral circumferential annular dimension. Leaflet resection is not required.

In myxomatous disease, we routinely use a full flexible ring annuloplasty because of recent evidence chat the anterior one-third of che mitral annulus can enlarge despite being a fibrous structure. On average, ring annuloplasty reduces the annular diameter by 10-15 mm, and elimination of all mitral regurgitation is routinely achieved. In younger patients and especially females, a 7-10 cm incision low on the chest *is* empl$_{oye}$d with excellent long-term results. Despite the application of the American Correction co more complex groups of patients, reoperation races have been co$_{mp}$arable to the highly selected French Correction patients (Figure 2).

The term "ischemic mitral regurgitation" describes a complex group of conditions usually associated with intrinsically normal valve mitral leaflets and chordae. Acute micral regurgitation may occur after extensive inferior wall infarction. This may cause elongation of the mitral annular-papillary muscle distance due to compromise or rupture of the posterior papillary muscle. Chronic ischemic mitral regurgitation is seen in association wich inferior wall morion abnormalities that cause a re trict1ve defect. In patients with diffusely poor left ventricular function, spherical dilation of the left ventricle displaces the bases of their papillary muscles downward and outward away from each other, again causing restrictive mitral regurgitation. Surgical correction involves reducing the length of the posterior mitral annulus, especially the posteromedial portion, despite the fact chat the initial annular dimensions may be normal. The annuloplasty has been shown co bring the tips of the papillary muscles closer together and improve leaflet apposicion.17-2U

## CATHETER INTERVENTIONS FOR MITRAL INSUFFICIENCY

The earliest effort to correct mitral regurgitation befure che availability of cardiopulmonary bypass involved attempts to displace the posterior annulus forward with foam plastic wedges and sutures. In most cases, however, the magnitude required to reduce annular dimensions is so great that techniques utilizing the coronary sinus (usually located above the annulus) are unlikely to provide long-term benefit. The coronary sinus is usually separated by l cm from the coronary sinuses, and is also very delicate in elderly patients. However, in acutely ill patients with mitral regurgitation and low ejection fractions, even a modest reduction in mitral regurgitation may have a major beneficial i$_{mp}$act, and catheter-based therapies may have an important role in these patients.21 In myxomatous disease, the Alfieri technique has been used to suture together the center of the leaflets' free edges, producing a double outlet rnitral orifice.[22] Without including annuloplascy to stabilize the repair, results have been poor. A surgical clip to duplicate the Alfieri technique is now in clinical trials.23

## 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 ofvalve surgery is now performed in all our patients using the Maze procedure, which has a 98% drug-free cure rate ofatrial fibrillation at three months offollow-up. Excision ofthe leti: aerial appendage is an integral part of the procedure.[24,25]

## CONCLUSION

Open isolated repair of stenotic or insufficient mitral valves is now usually performed through limited acces exposures with a mortality of less than 1%. Open repair can be combined with other heart valve

surgeries, coronary bypass or Maze procedures. Surgery and evolving catheter-based therapies should be viewed as complementary.

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