16

Mitral valve repair

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HISTORY

The first documented mitral valve operation was performed by Elliot Cutler at the Peter Bent Brigham Hospital (Boston) in 1923. Cutler used a tenotomy knife to access the left ventricle and partially incised both mitral leaflets on a patient with rheumatic disease and consequent severe mitral stenosis (MS). Two years later, in 1925, Henry Souttar performed the first digital dilation of the mitral valve on a patient with a similar clinical profile at the London Hospital. Unfortunately, although successful, the procedure was not well received by the medical community due to its significant perioperative complications and the skepticism around this surgery did not trigger further referrals. In 1948, after multiple failed attempts, Charles Bailey introduced the modern era of mitral commissurotomy at the Episcopal Hospital in Philadelphia. Soon after, other surgeons including Charles Dubost in Paris and Dwight Harken in Boston perfected the technique and launched mitral valve repair as a therapeutic alternative for mitral valve disease. In 1957, Walton Lillehei performed the first open mitral annuloplasty under extracorporeal circulation.

At that time, although mitral valve repair had an exponentially increasing acceptance among cardiovascular specialists, the advent of prosthetic valves in the early 1960s led most to consider valve replacement as the primary therapy for mitral valve disease. However, early publications reported mortality rates as high as 30% as well as numerous perioperative complications. Pioneering surgical leaders including Dwight McGoon, Robert Frater, and Alain Carpentier continued to research and work on the development of mitral valve repair. Carpentier, considered by most to be the "father of mitral reconstructive surgery," described the principles of mitral valve repair, combining leaflet resection, ring annuloplasty, and chordal techniques. In addition, he established a systematic analytic approach to patients with mitral disease known as the "pathophysiologic triad of mitral valve regurgitation." The triad emphasized the importance of distinguishing between the medical condition causing the disease (etiology), the resulting lesions, and finally how these lesions affect leaflet motion (dysfunction).

Currently, mitral valve repair is the gold standard for patients with mitral valve disease, especially in the setting of degenerative disease. Recent data have demonstrated that it is possible to repair practically all prolapsing valves with a mortality risk of less than 1% in expert valve centers.

PRINCIPLES AND JUSTIFICATION

Mitral regurgitation (MR) predisposes the left ventricle to a volume overload in order to compensate for the volume lost to the regurgitant jet. While mild to moderate MR might be well tolerated for long periods of time, severe MR is fatal at a determined stage. At an early stage, the pure volume overload of MR is compensated for by eccentric left ventricular hypertrophy, which enables rapid left ventricular diastolic filling and an increase in stroke volume. However, this remodeling eventually encumbers systolic emptying. This maladaptive geometry together with the adrenergic overactivation results in decreased contractility. Severe MR is often divided into three clinical stages: acute, chronic compensated, and chronic decompensated, and each stage will have a different management as well as different surgical triggers. If MR is corrected in a timely fashion, this progression in stage can be reversed.

Severe MR is a mechanical problem with surgery as the only definitive solution, either by mitral valve repair or mitral valve replacement. Although the lack of randomized trials has led to controversy, especially in the setting of functional MR, repair is favored over replacement, particularly in patients with degenerative mitral valve disease, for multiple reasons including:

- a likely lower perioperative risk
- improved event-free survival
- freedom from the numerous complications of prosthetic heart valves
- better postoperative left ventricular function.

Despite the general consensus regarding the clinical superiority of mitral valve repair versus mitral valve replacement, it is sobering to note that many patients still undergo unnecessary valve replacement due to lack of surgical skill or experience. It is important to estimate the reparability of

a valve based on the identified echocardiographic lesions in order to inform the patient about surgical expectations. Recent data form high-volume centers have shown a near 100% repair rate for degenerative mitral valve disease, with an operative risk of less than 1%. Matching surgical expertise and experience to the complexity of a specific valve morphology and patient is important in the modern era to assure high repair rates.

PREOPERATIVE ASSESSMENT AND PREPARATION

In general, transthoracic evaluation is adequate to assess mitral valve lesions and left ventricular function. A workup of coronary anatomy should generally be performed in patients over the age of 45 years, and a chest computed tomography (CT) scan done in elderly patients to rule out aortic or lung pathology. Otherwise, no other specific studies are mandatory in otherwise healthy patients.

ANESTHESIA

There are no specific changes to anesthetic management versus other open-heart procedures.

OPERATION

Classification of mitral valve disease

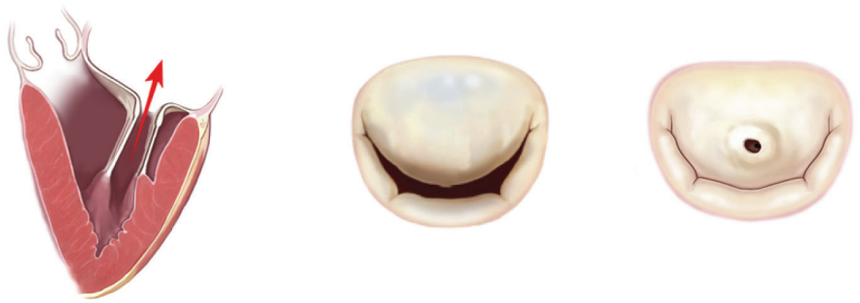
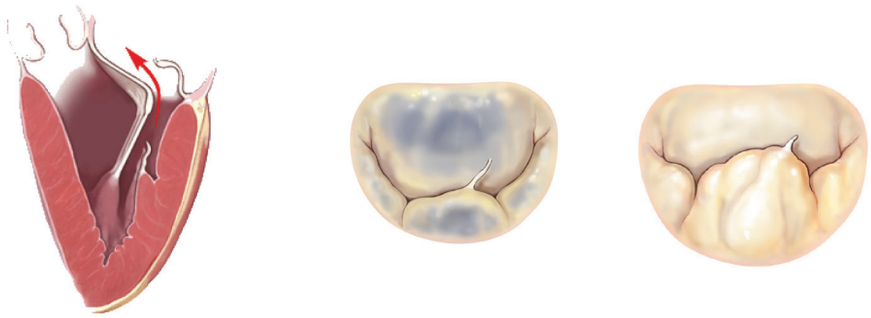
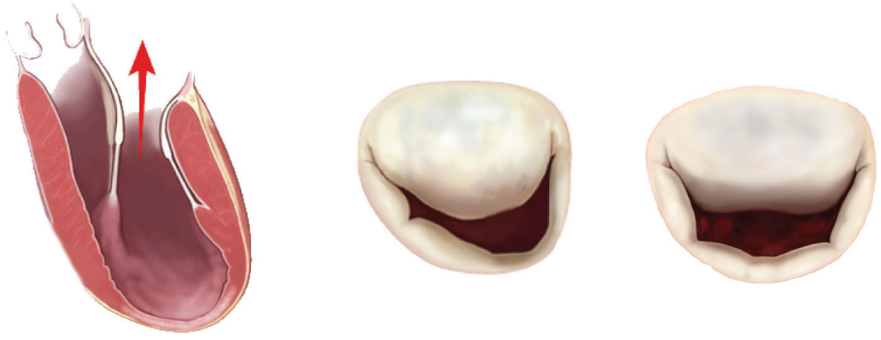
Structural lesions cause MR or MS by reducing leaflet coaptation or impairing leaflet opening respectively. An exhaustive interrogation (identification, localization, and magnitude) for mitral lesions is essential to determine the chances of successful valve repair and to proceed with a tailored therapeutic plan for each patient. As mentioned above, Carpentier described a systematic analytic approach to patients with MR known as the "pathophysiologic triad of mitral valve regurgitation." Besides emphasizing the importance of distinguishing between etiology, lesions, and dysfunction, the triad also represents a very consistent way to elucidate which are the most appropriate techniques to achieve a successful repair. Given the very generic characteristics of mitral valve repair as an operative procedure, in part due to the vast armamentarium of techniques available, we use the classification of mitral valve disease based on the presence of abnormal leaflet motion (dysfunction) (**Figure 16.1**) in order to describe and analyze every technique in an organized fashion.

The differentiation of leaflet dysfunction is based on the position of the leaflet margins with respect to the mitral annular plane.

- *Type I dysfunction* implies normal leaflet motion, and the most common cause of significant MR is the perforation (e.g. endocarditis) of one of the leaflets or severe annular dilatation with a central regurgitant jet (e.g. long-standing atrial fibrillation).
- *Type II dysfunction* denotes excess leaflet motion generally secondary to chordal elongation or rupture (e.g. fibroelastic deficiency) or myxomatous degeneration (e.g. Barlow's disease) of the leaflets (regurgitant jet directed to the opposite site of the prolapsing leaflet).
- *Type III dysfunction* designates restricted leaflet motion and results typically from retraction of the subvalvular apparatus (IIIa, rheumatic valve disease or other inflammatory scenarios that lead to scarring and calcification) or papillary muscle displacement (leaflet tethering) from ventricular remodeling or dilatation (IIIb, ischemic or dilated cardiomyopathy).

Exposure of the mitral valve

Perfect exposure of the mitral valve is essential before any procedure is attempted and plays a key role in the success of the operation. Although multiple approaches have been described, including horizontal biatrial transeptal, superior biatrial transeptal, and interatrial approach through Sondergaard's groove, the latter remains the most efficient (better view and less tissue damage) when performing mitral valve repair.

DYSFUNCTION	FREQUENTLY ENCOUNTERED LESIONS
Type I Normal leaflet motion	
Type II Increased leaflet motion (leaflet prolapse)	
Type IIIA Restricted leaflet motion (restricted opening)	
Type IIIB Restricted leaflet motion (restricted closure)	

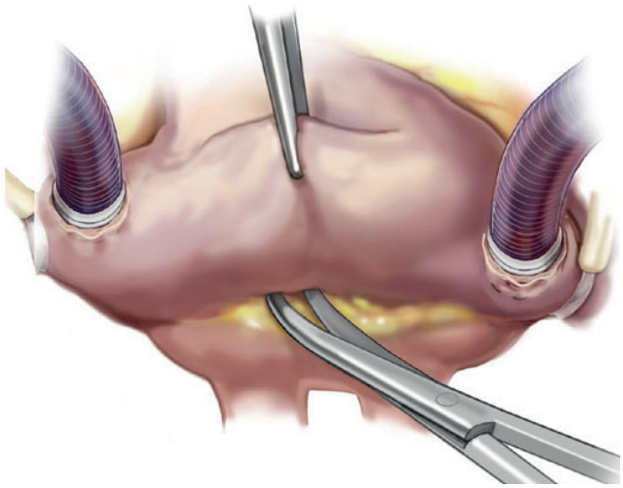
16.1

The interatrial groove is dissected and both atria are divided up to the fossa ovalis. The right atrium can be retracted medially and anteriorly. The right superior pulmonary vein at its junction to the left atrium is exposed (**Figure 16.2a**).

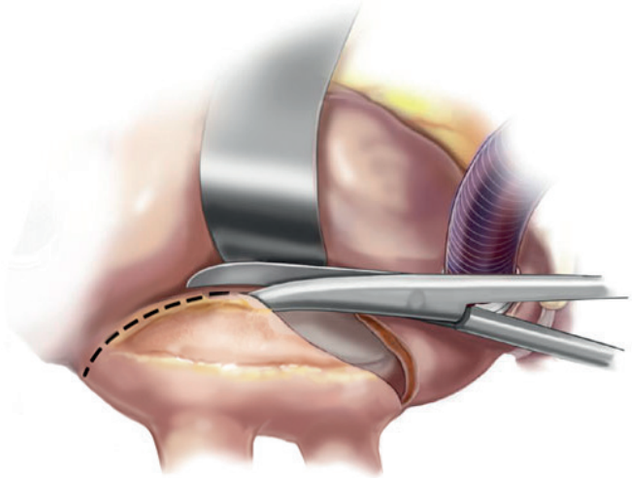
The dissection exposes the roof of the left atrium, which is opened at the midpoint between the right superior pulmonary vein insertion and the groove. During this maneuver, it is important not to inadvertently injure the posterior wall of the left atrium (**Figure 16.2b**).

The curvilinear incision is extended longitudinally both superiorly to 1 cm from the superior vena cava and inferiorly to the midpoint between the right inferior pulmonary vein and the inferior vena cava (**Figure 16.2c**).

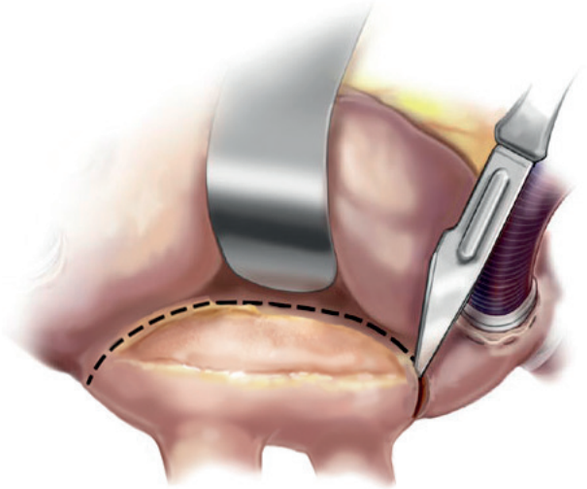
If further exposure of the left atrium is required, the pericardial reflection on both vena cavae can be released and blunt dissection can be used to free the lateral aspects of both veins for about 2–3 cm (**Figure 16.2d**).



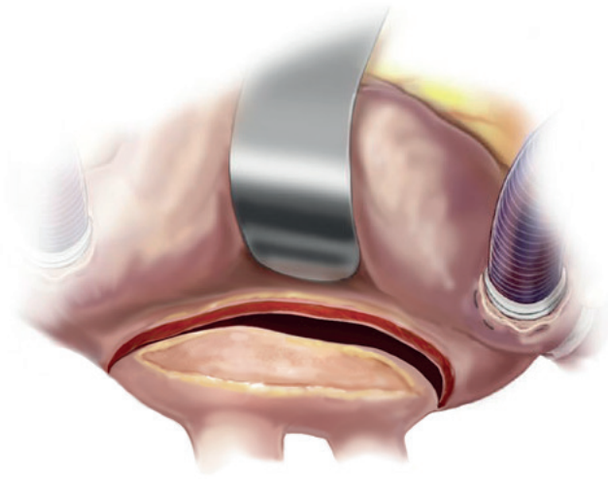
16.2a



16.2c



16.2b



16.2d

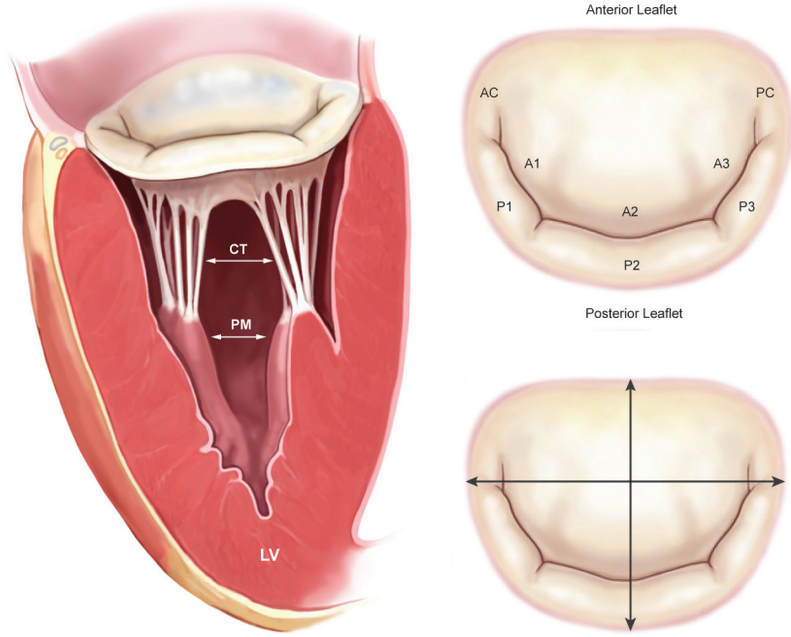
Valve analysis

Valve analysis starts with the echocardiogram. Intraoperative valve inspection, performed in a systematic fashion (annulus, leaflets, chordae, and papillary muscles), confirms echocardiographic findings or further characterizes the existent lesions (**Figure 16.3**). This allows an accurate assessment of repair feasibility and helps to plan a surgical strategy and to choose the most appropriate techniques. Initially, the endocardium of the left atrium is carefully examined for jet lesions, thickening, thrombus, and areas of calcification. In the presence of large distended leaflets, placement of posterior annular sutures might be undertaken first to allow adequate exposure for valve analysis.

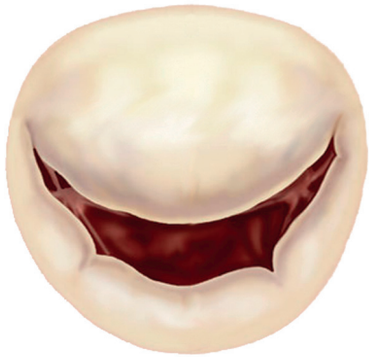
The mitral annulus is then evaluated first to assess shape, symmetry, and any degree of dilation, as well as areas of severe calcification that may require special consideration when placing annular sutures. The leaflets are examined with a nerve hook. We first identify and document the pathologic segments (A1-3; P1-3, anterior and posterior commissures). Filling the ventricle with saline helps identify "functional" leaflet lesions. This may also identify migration of the leaflet hinges onto the left atrium. Subsequently, chordae tendinae are commonly interrogated for elongation, rupture, or a combination of both. Thickened, fibrotic, fused, or calcified chords should also be noted in patients with more complex scenarios. Finally, the papillary muscles are examined for calcification, fusion, and/or abnormal ventricular insertion.

Type I dysfunction – annular dilation

Every patient undergoing mitral valve repair requires a remodeling annuloplasty in order to restore the native annular size and shape allowing full leaflet motion, and preventing any risk of recurrence by stabilizing the annulus (especially the posterior aspect) with a prosthetic device. Those valves with type I dysfunction and isolated annular dilation can be successfully repaired with only a remodeling annuloplasty (**Figure 16.4a**).



16.3

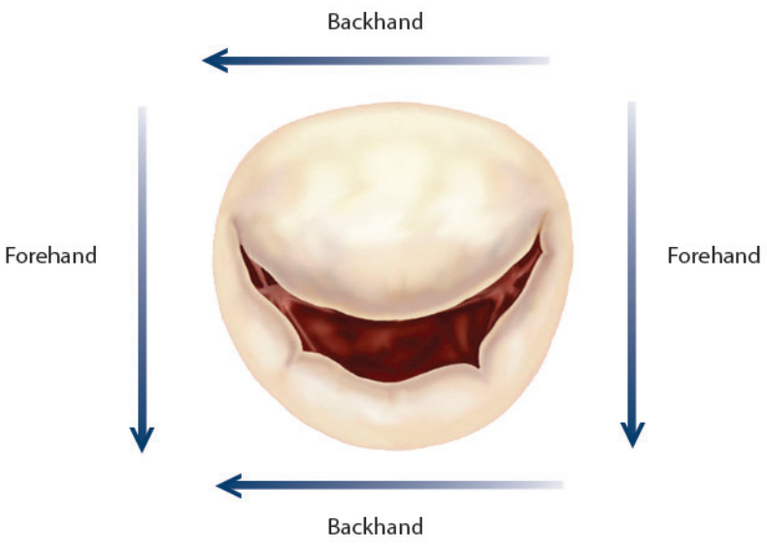


16.4a

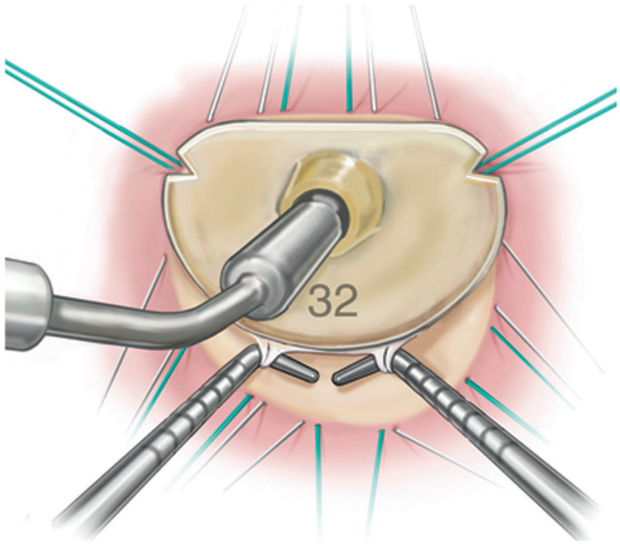
Placement of the annular sutures (12-15 2-0 braided polyester mattress sutures) mandates complete visualization and identification of the mitral annulus (located approximately 2 mm away from the leaflet hinge). Grasping the leaflets transversally (as close to the annulus as possible) while applying traction towards the ventricle provides exposure and facilitates positioning of the sutures. Placing sutures along the anterior leaflet should be done using a backhand position with the needle tip oriented towards the ventricle (avoiding the aortic cusps). Sutures within the posterior annulus follow the same premises but require slightly deeper bites to reach the fibrous skeleton. For sutures along the posterior commissure, sutures are placed using a forehand position and oriented downwards. Finally, placing sutures within the anterior commissure requires a forehand position and the needle tip must be oriented towards the ventricle in order to avoid the circumflex artery (**Figure 16.4b**).

Selection of the ring size is based on the assessment of the base (intercommissural distance) and height of the anterior leaflet (**Figure 16.4c**).

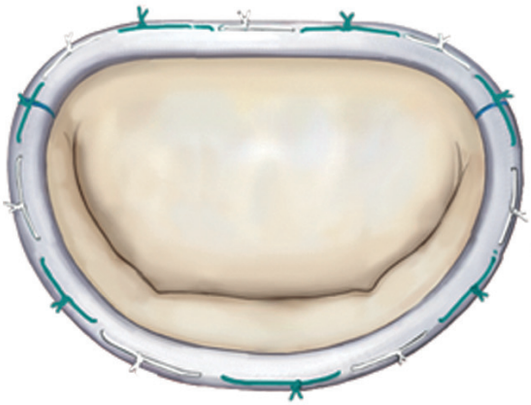
After remodeling annuloplasty, saline testing should show a competent valve, with a symmetric and posterior line of coaptation (**Figure 16.4d**).



16.4b



16.4c



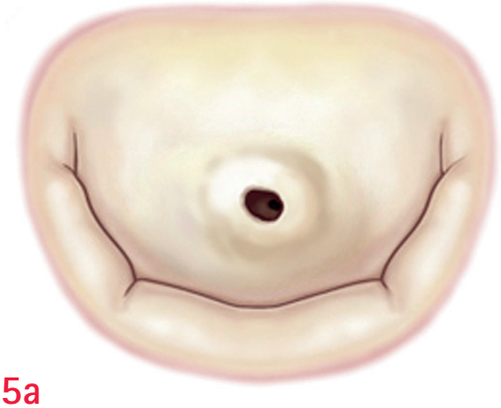
16.4d

Type I dysfunction – leaflet perforation

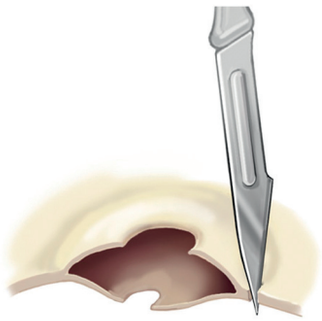
The second most common lesion leading to type I dysfunction is leaflet perforation, mostly due to bacterial endocarditis. The infection process usually leads to the formation of a vegetation-abscess on the body of the anterior leaflet (atrial side) that eventually becomes a true aneurysm, often with perforation (**Figure 16.5a**).

Early surgical intervention should be performed after isolation and identification of the organism and initiation of the appropriate antibiotic therapy. Debridement of the infected tissue mandates a minimum of 2 mm margins of macroscopically healthy tissue (**Figure 16.5b**).

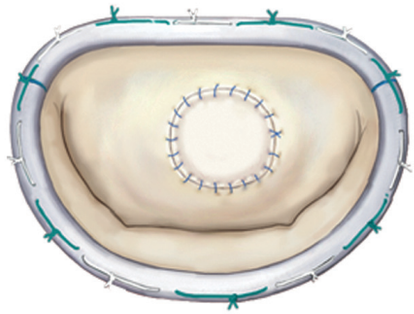
A piece of autologous pericardium is immersed in 0.625% buffered glutaraldehyde solution (Poly Scientific, Bay Shore, NY) for 10 minutes. Afterwards, the patch is rinsed in saline solution. The patch is then tailored to match the shape of the leaflet defect (accounting for 2–3 mm of margin for the suturing) and then secured using a 5-0 continuous polypropylene suture (**Figure 16.5c**).



16.5a



16.5b

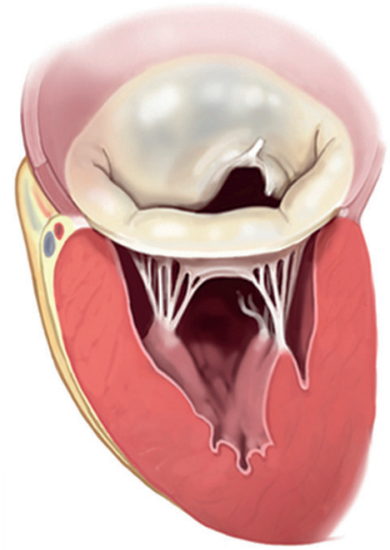


16.5c

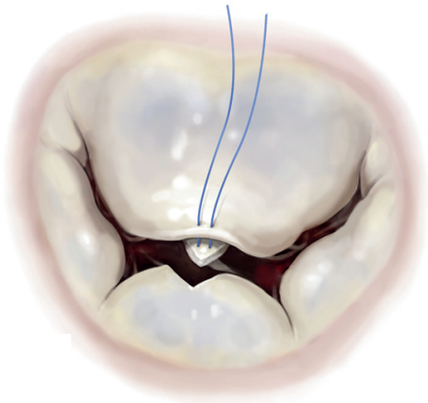
Type II dysfunction – anterior leaflet prolapse

Due to its anatomy, the anterior leaflet does not allow aggressive margin resections. Every surgical strategy to address anterior leaflet prolapse therefore includes minimal or non-resection techniques. Non-resection techniques mainly comprise chordal transfer, chordal transposition, and polytetrafluoroethylene neochordoplasty (loop technique and its variants). If resection is necessary, a triangular resection limited to the rough area of the leaflet (so as not to compromise the leaflet body) should be carefully performed (**Figure 16.6a**).

Chordal transfer describes the mobilization of strong secondary chordae to the free margin of the prolapsed area (using a 5-0 polypropylene suture). Alternatively, *chordal transposition or posterior leaflet flip technique* involves the mobilization of an isolated chord or a segment of the posterior leaflet (usually the one opposed to the prolapsing area of the anterior leaflet in order to match the chordal length) with its marginal chordae. After complete mobilization and inspection of the chordae, the strip of posterior leaflet is attached to the free edge of the anterior leaflet using 5-0 polypropylene sutures (**Figure 16.6b**).



16.6a

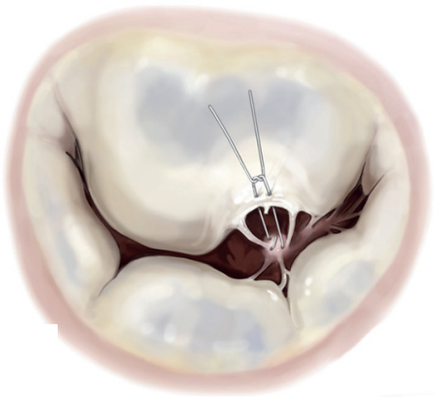


16.6b

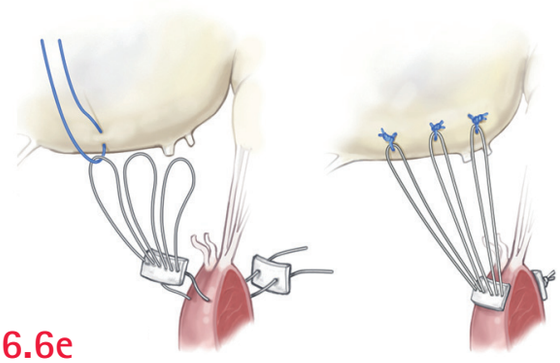
Polytetrafluoroethylene neochordoplasty has been increasingly used in mitral valve repair. First, a CV-5 double-armed polytetrafluoroethylene suture is passed and looped through the fibrous tip of the papillary muscle. Next, the two ends of the artificial chord are passed through the leaflet margin (with a distance of approximately 3 mm between them) and two slip knots are tied (**Figure 16.6c**).

In the setting of isolated anterior leaflet prolapse, we generally proceed with a remodeling annuloplasty first. Functional (using saline testing) adjustment of the final length of the neochordae can then be performed always after remodeling annuloplasty. A catheter is attached to a bulb syringe and saline is pushed into the ventricle. A nerve hook is used to adjust the length of the artificial chord. A minimum of six knots are then completed to secure the chord (**Figure 16.6d**).

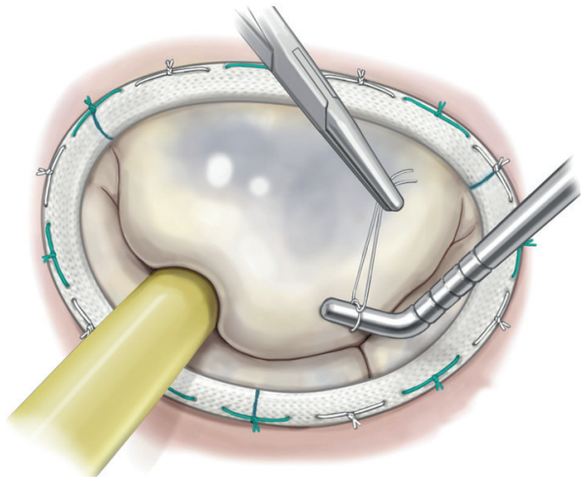
The loop technique was introduced to avoid problems of functional adjustment of the neochordae (inadvertent alteration of the chordal length during fixation). Three premeasured CV-5 polytetrafluoroethylene loops are attached to the body of the papillary muscle (mandatory use of pledgets) and the free margin of the prolapsing leaflet (using 5-0 polypropylene sutures) (**Figure 16.6e**) to complete the repair (**Figure 16.6f**).



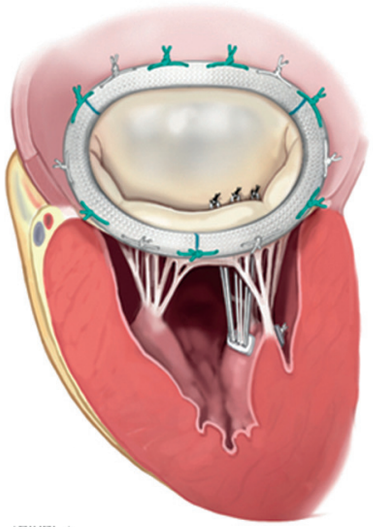
16.6c



16.6e



16.6d



16.6f

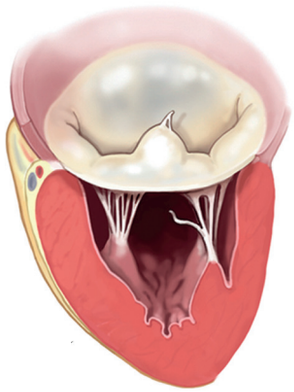
Type II dysfunction – P2 prolapse

Posterior leaflet prolapse (particularly isolated P2 prolapse) is the most common cause of mitral valve regurgitation (**Figure 16.7a**).

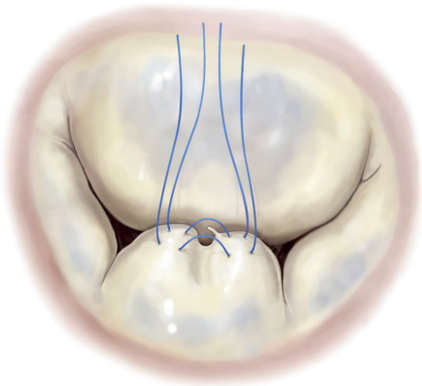
Several approaches have been suggested including chordal techniques (see previous section), leaflet plication techniques (McGoon's), and resection techniques (triangular and quadrangular resection). McGoon's technique is used to repair very limited prolapses and involves the placement of two imbricated "magic" 5-0 polypropylene sutures to plicate the prolapsing leaflet segment (**Figure 16.7b and c**).

Triangular resection is a very useful technique. The resection is generally limited to within a few millimeters of good marginal chords, and extends to the midpoint of the belly of the leaflet (**Figure 16.8a**).

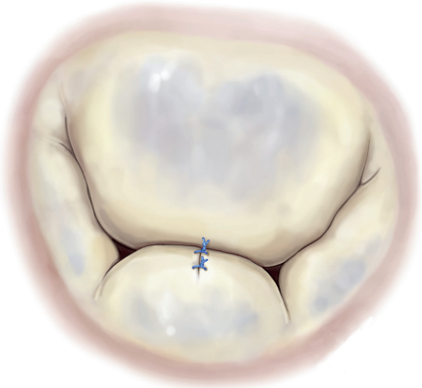
Leaflet continuity is restored using either interrupted or continuous 5-0 polypropylene sutures, depending on the pliability of the tissue (**Figure 16.8b**). A continuous suture technique is best avoided in the setting of leaflet calcification or retraction. After completion of the sutures, a nerve hook is utilized to assess leaflet continuity and to detect residual defects.



16.7a



16.7b



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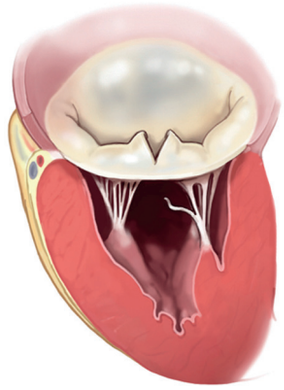
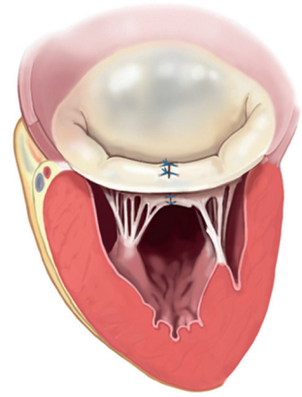


16.8a



16.8b

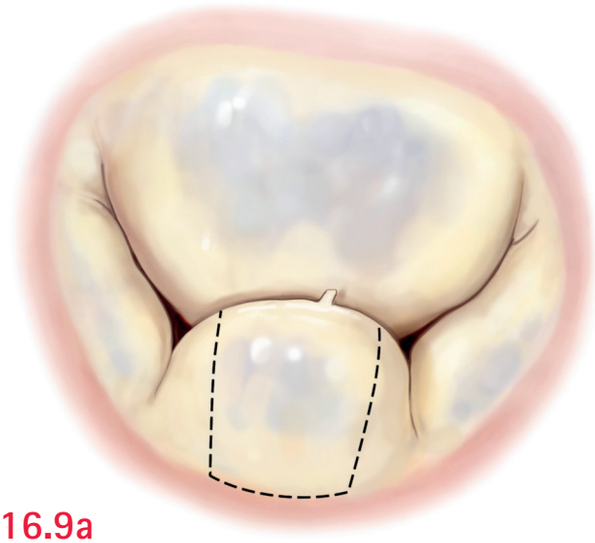
Type II dysfunction – posterior leaflet prolapse

In the presence of a more extensive (excess tissue) posterior leaflet prolapse, more aggressive resection techniques are needed including quadrangular resection, annular plication, and sliding leaflet plasty. In this regard, quadrangular resection is the most frequently used technique in posterior leaflet prolapse. However, with the growing adoption of chordal techniques, many surgeons have decided to respect as much tissue as possible and abandon more complex techniques that involve large resections. After valve analysis, a quadrangular resection of the prolapsing area is carried out to the annulus (**Figure 16.9a**).

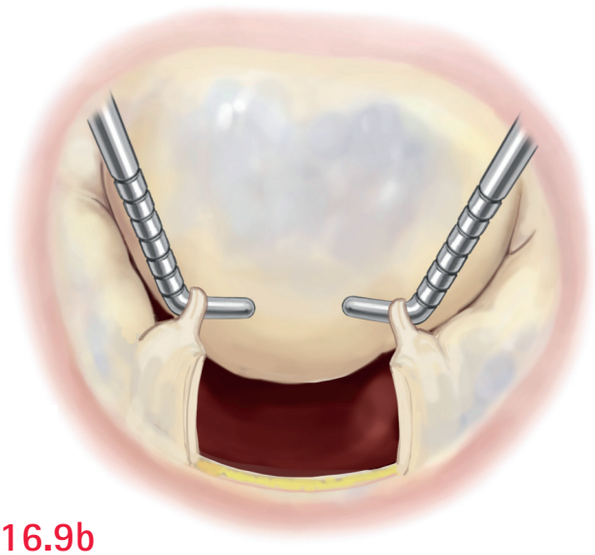
Generally, the tallest portion of the leaflet is excised, and well within the margin of normal chordae. Clefts or indentations are often targeted as one margin of the resection (**Figure 16.9b**).

If the residual leaflet defect is less than 2 cm, plication techniques are applied in order to avoid excess leaflet tension. Interrupted 2-0 braided polyester sutures are placed through the annulus at the limit of the resected area (**Figure 16.9c**).

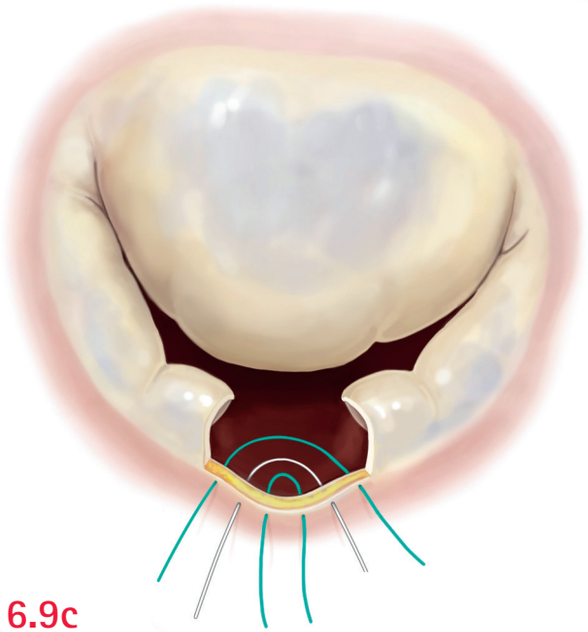
The leaflet continuity is restored using 5-0 polypropylene sutures (**Figure 16.9d**).



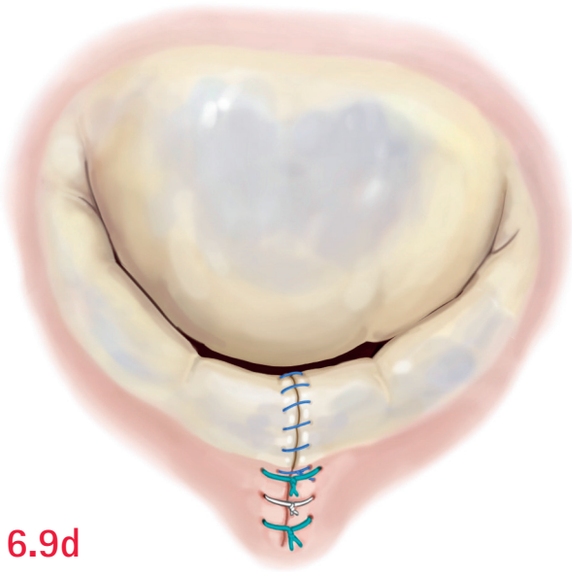
16.9a



16.9b



16.9c



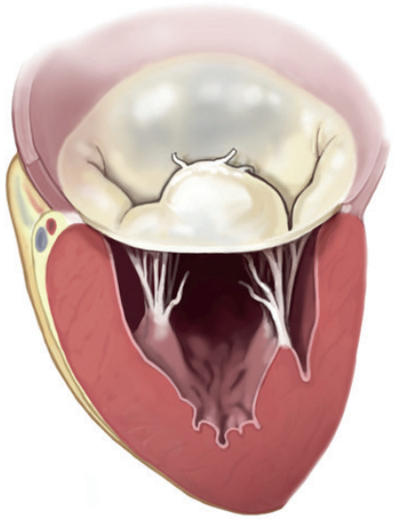
16.9d

Sliding leaflet plasty is a useful technique to avoid excess height and tension and is often employed in scenarios of more diffuse prolapse and leaflet myxomatous changes (**Figure 16.10a**).

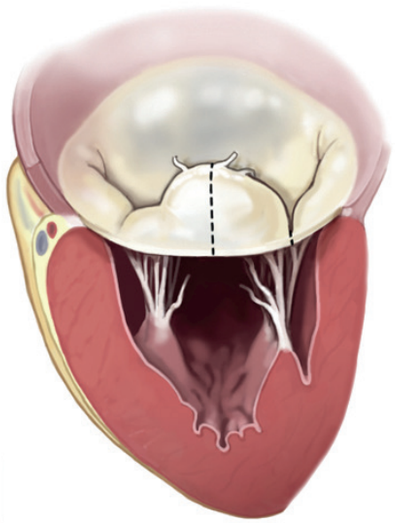
A targeted (including deep indentations if present) leaflet quadrangular resection is usually performed where the prolapse is greatest or leaflet is tallest (**Figure 16.10b**). This resection is typically about 1 cm wide (additional excess tissue can be removed later). It is important not to remove all abnormal tissue. In the setting of additional deep indentations, we close these first with a figure-eight suture in order to treat the segments as one.

If the height is more than 15 mm in any residual leaflet segment, a sliding leaflet plasty (asymmetric in this case) to reduce the residual leaflet height to 12–15 mm needs to be performed. Specially angled scissors are used to detach the leaflet remnant, starting from the remaining left position of P2 and going to the anterior commissure (**Figure 16.10c**). At this point, the leaflet is suspended by the primary and secondary chordae with the basal chordae remaining on the annular side. Secondary chordae are detached to maintain free mobility of segments after advancement. This prevents secondary chords from restricting the leaflet after leaflet advancement. Additionally, at this stage, further resection of the base of P1–P2 may be considered in order to re-establish a uniform leaflet height.

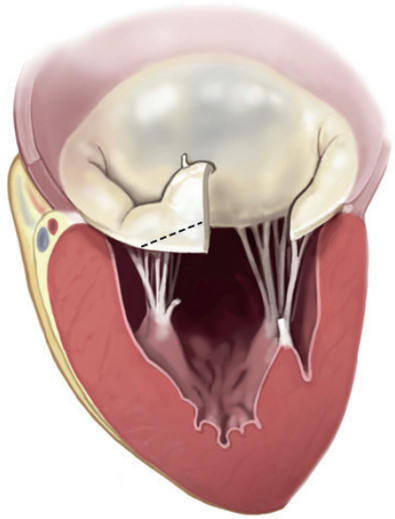
A double layer of 4-0 polypropylene running suture is used to reattach the leaflet to the annulus, assuring no excess tension on either segment. Excess height is compensated for by taking sutures up to 5 mm deep into the leaflet; in areas of adequate height, sutures are taken just 1–2 mm from the leaflet edge. The two leaflet margins are then joined using a running 5-0 polypropylene suture (**Figure 16.10d**). The margins of the reconstructed posterior leaflet are examined to ensure that all segments are adequately supported. Any gaps in support, or areas supported by thinned out chordate (even in the absence of prolapse), are reinforced by transposition of previously detached secondary chordate, or now more commonly implantation of artificial neochordae.



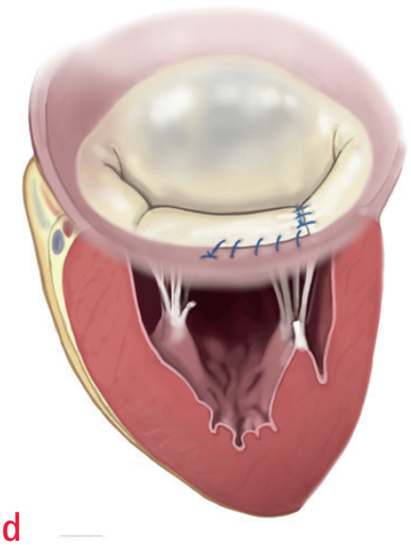
16.10a



16.10b



16.10c



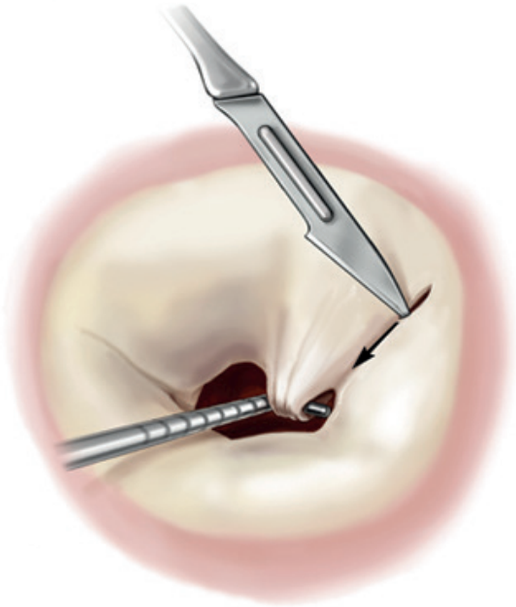
16.10d

Type IIIa dysfunction – commissural fusion

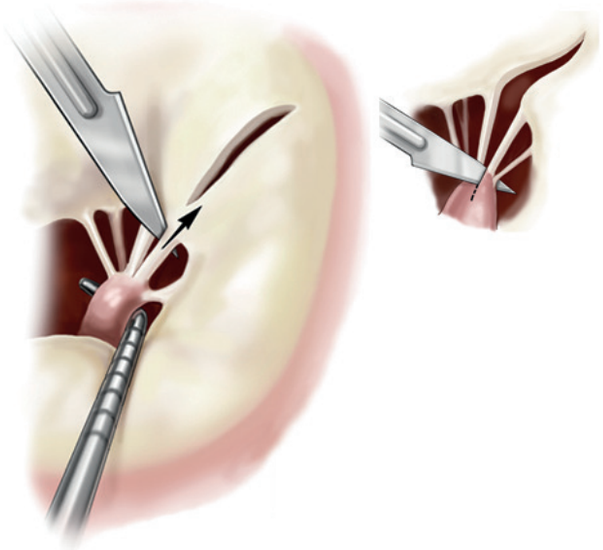
The majority of lesions leading to type IIIa (commissural fusion, leaflet thickening and retraction, and chordal thickening and shortening or fusion) have a rheumatic origin. The limitation of the leaflet excursion often results in combined valve stenosis and regurgitation. In this scenario, commissurotomy is the technique of choice. Traction of the commissure with a nerve hook helps to identify the commissural line (incision). A 11 blade is then used to make an incision about 5 mm from the annulus (**Figure 16.11a**).

The incision is completed leaving one chord on each side of the defect. The papillary muscle is then split in order to create a subcommissural orifice which will avoid future refusion (**Figure 16.11b**).

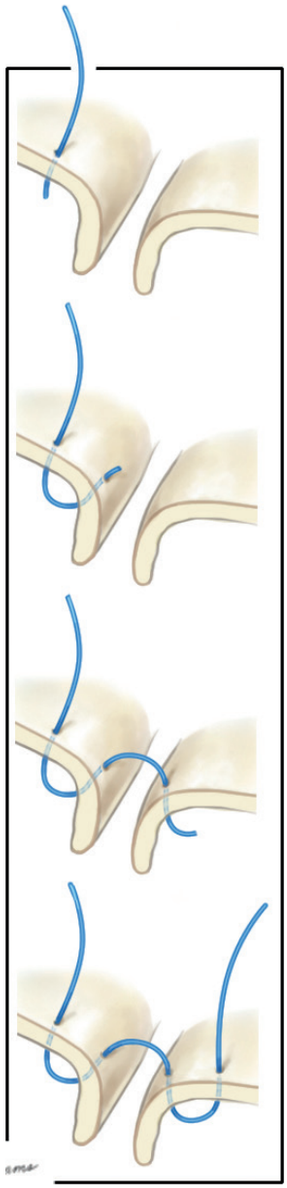
In cases of combined valve stenosis and regurgitation or pure stenosis and concomitant annular dilation, the commissures may need reconstruction (commissurotomy may contribute to further annular dilation and mitral regurgitation). In this scenario, also seen in diffuse leaflet prolapse or acute bacterial endocarditis, the optimal technique is the "magic suture" or commisuroplasty. This is performed using 5-0 polypropylene sutures (**Figure 16.11c**).



16.11a



16.11b



16.11c

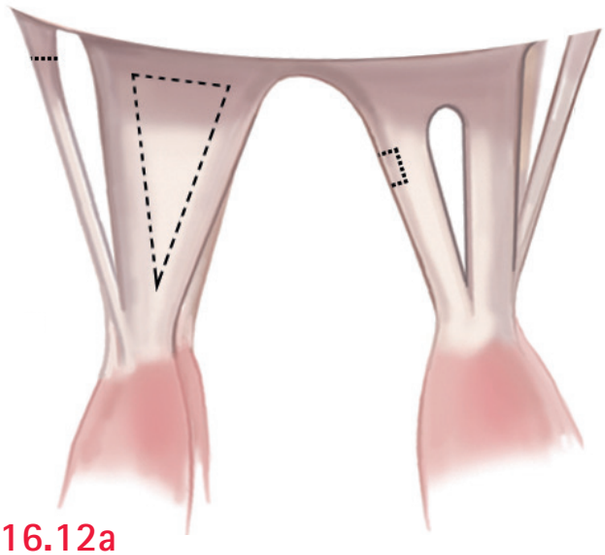
Type IIIa dysfunction – severe leaflet restriction

Leaflet retraction in the context of type IIIa dysfunction is mainly due to abnormalities of the subvalvular apparatus (chordal thickening and shortening or fusion). In this case, leaflet mobilization should be achieved by resecting secondary chords, splitting fused hypertrophic chords and fenestrating isolated thickened chords (**Figure 16.12a**).

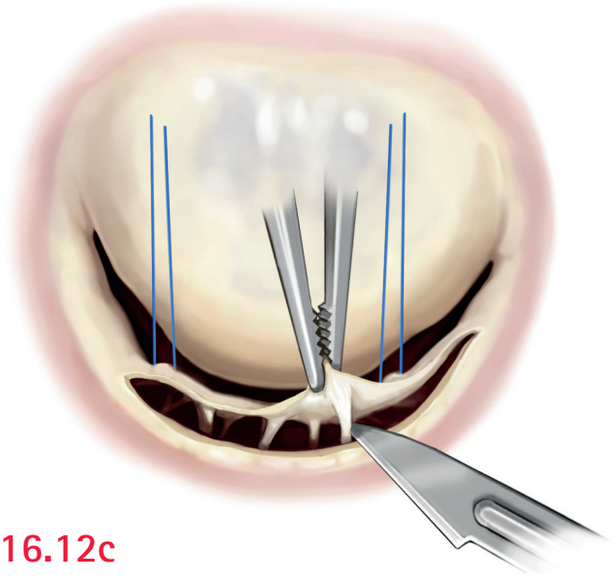
When leaflet mobilization cannot be achieved with subvalvular techniques, pericardial patch extension of the leaflet is indicated. Traction sutures (5-0 polypropylene) are placed in order to unfold the leaflet as much as possible and the incision is made about 5 mm from and parallel to the annulus (**Figure 16.12b**). The extent of the incision depends on the degree of leaflet retraction.

All secondary chordae are resected to free the leaflet and achieve adequate mobilization (**Figure 16.12c**).

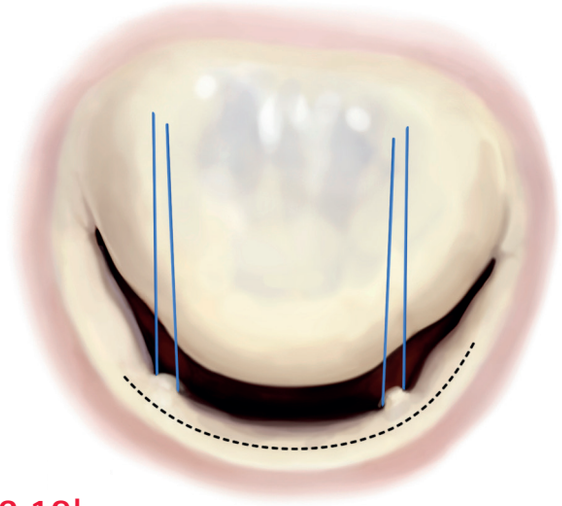
A semilunar autologous pericardial patch (see previous section for further details) is tailored to the leaflet defect adding a 2 mm margin for suturing. The patch is then sutured using continuous 4-0 polypropylene sutures. Interlocked bites are used on the leaflet side to prevent a potential purse-string effect (**Figure 16.12d**).



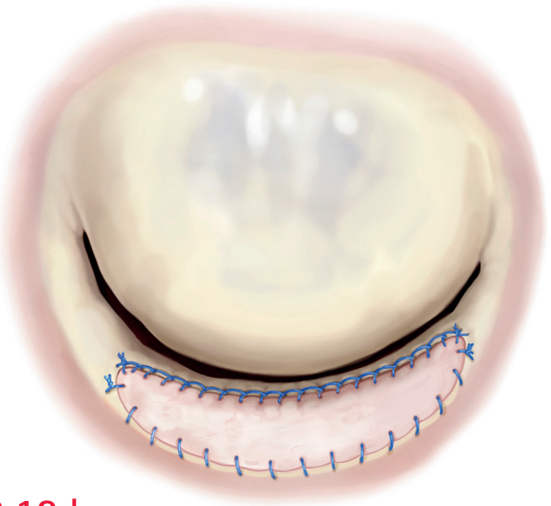
16.12a



16.12c



16.12b



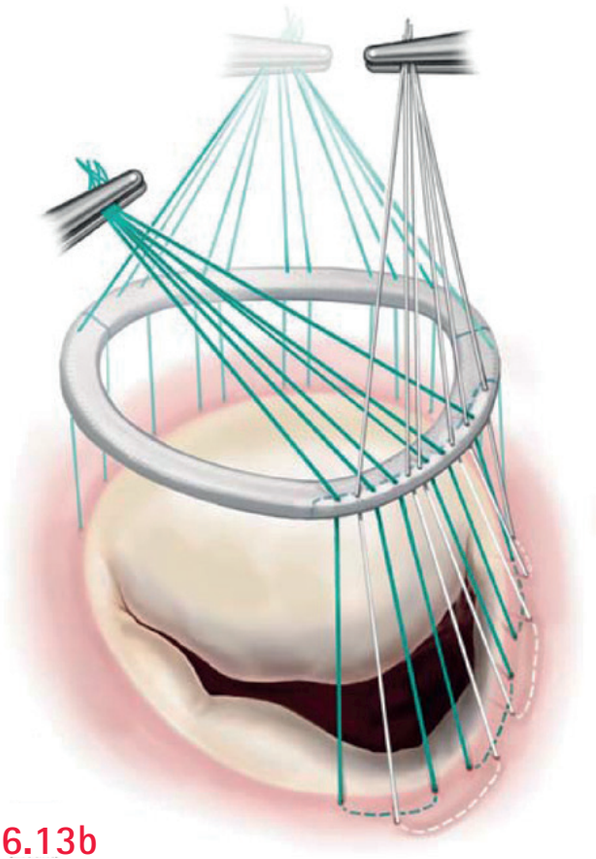
16.12d

Type IIIb dysfunction – posterior leaflet tethering

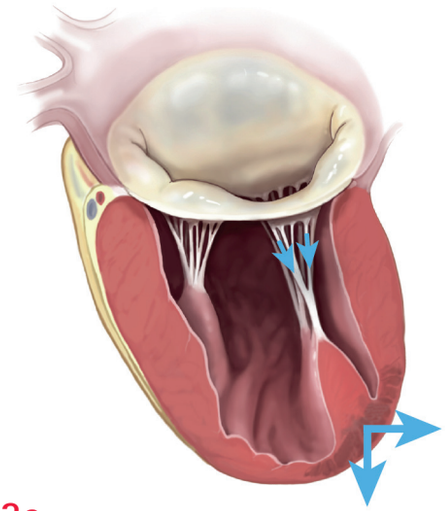
The primary ventricular alteration leading to ischemic MR is papillary muscle displacement. The papillary muscle tips are displaced away from the midseptal (anterior) annulus, i.e. posterolaterally, apically, and away from each other. Papillary muscle tethering leads to apical tenting of the leaflets (restriction of the motion of the free margins of the leaflets), which prevents them from rising to the plane of the annulus to provide good coaptation (**Figure 16.13a**).

Because leaflet restriction in ischemic MR results in less leaflet tissue available for coaptation, it is necessary to downsize a complete remodeling ring by one or two sizes or to use a true-sized asymmetric ring to ensure an adequate surface of coaptation following annuloplasty. The potential increased tension with associated annular dilation mandates placing the sutures very close together along the annulus, and suture crossover may be warranted, especially in the P3 area (**Figure 16.13b**).

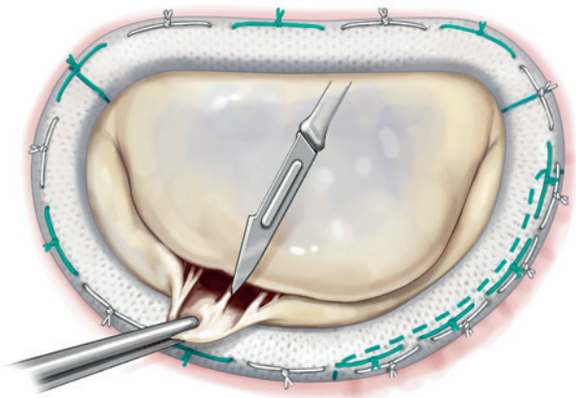
The battery of adjunct techniques to downsized rigid ring annuloplasty include cutting of the secondary strut chord to the anterior leaflet in the setting of a "hockey-stick" deformity of the closure line, closure of all clefts and indentations in the posterior leaflet if severe leaflet tethering is present, and cutting restricted marginal chords if a residual leak is still present (replacing them with chordal transfer or artificial neochordae) (**Figure 16.13c**).



16.13b



16.13a



16.13c

Objectives of mitral valve repair

The most important goal for patients with MR is to achieve a competent and durable mitral valve repair. In this regard, the procedure should mainly restore the native annular shape meeting the following criteria:

- competent valve on saline and ink testing
- good surface of coaptation
- symmetric line of closure where the anterior leaflet occupies ≥80% of the valve area
- no residual areas of billowing
- no tendency to systolic anterior motion.

Evaluation for all these points may require two different intraoperative tests including the saline test and the ink test. The saline test is performed by filling the ventricle with saline and confirming all the aforementioned prerequisites. The ink test is performed by drawing a line on the valve closure line during maximum saline insufflations. The coaptation zone beyond the ink is examined with the help of nerve hooks and should be at least 6 mm in length (this will transform to approximately 8 mm on echocardiography as some of the ink is within the coaptation zone). Also, there should be no more than 10 mm of anterior leaflet beyond the ink line as this would signify a risk for systolic anterior motion.

POSTOPERATIVE CARE

The application of rigorous postoperative care following strict protocols is critical to ensure optimal success of mitral valve repair. Major physiological and mechanical derangements can occur in patients recovering from a cardiac operation as a consequence of pre-existing conditions or due to changes secondary to cardiopulmonary bypass and the surgical procedure. In this regard, a system-based approach is necessary to deal with problems in an organized fashion. The cardiovascular system frequently deranged and primary determinant of recovery will be the main focus of attention. If patients have preserved ventricular function and consequently good urine output, adequate oxygenation (optimal arterial blood gases) and preserved neurological status, they can be extubated fairly early, within hours following the procedure. In the presence of ventricular dysfunction, vasodilators for afterload reduction (ACE inhibitors preferentially) should be initiated in combination with inotropic agents. If pulmonary hypertension persists after surgery, the administration of nitric oxide has been demonstrated to be very effective. In addition, volume overload may require the administration of diuretics for a few weeks after hospital discharge.

Chronic or paroxysmal atrial fibrillation is common in patients with chronic MR due to increased left atrial pressure and progressive atrial stretch and dilation. It is present in 20–40% of patients undergoing mitral valve repair and current trials have reported the restoration of sinus rhythm postoperatively in up to 90% of patients undergoing adjunct ablation procedures. New onset of postoperative atrial fibrillation has been shown to be present in around 20% of patients. In this scenario, the rate control strategy should include the use of beta blockers or amiodarone and oral anticoagulation in refractory cases. Note that intravenous coagulation should be used with caution to avoid possible mediastinal bleeding. In patients with persisting atrial fibrillation up to 2–3 months after surgery, cardioversion might be indicated; transesophageal echocardiography must demonstrate the absence of atrial thrombus before any cardioversion attempt.

Anticoagulation is a very important aspect of the early postoperative care, although different centers have different treatment plans. We tend to use aspirin therapy alone in patients in normal sinus rhythm with no risk factors, and warfarin therapy in patients with preoperative or postoperative atrial fibrillation. Rhythm should be reassessed at 3 months to determine who should continue on warfarin therapy. Cardioversion should be coordinated with the patient's cardiologist.

OUTCOME

Contemporary data have shown a trend towards very low operative mortality rates after mitral valve repair regardless of the etiology. Preoperative factors that might significantly affect mid-and long-term survival in patients with mitral regurgitation include the presence of left ventricular dysfunction (left ventricular ejection fraction <60%), functional class III or IV, effective regurgitant orifice of ≥40 mm^2, a left ventricular end systolic dimension of >40 mm, a left atrial index of ≥60 mL/m^2, a left atrial dimension of >55 mm, pulmonary hypertension or exercise pulmonary hypertension, and the presence of atrial fibrillation. Patients with preoperative symptoms have increased postoperative mortality despite symptom relief (especially those with a left ventricular ejection fraction that is <50%), whereas in those with no or few symptoms, restoration of life expectancy can be potentially achieved. Durability of repair (assessed as freedom from moderate or greater MR) in patients with degenerative mitral valve has been reported to be between 90% and 95% at 5 years in high volume centers, with a recurrence rate of 1–1.5% a year. The failure to use an annuloplasty ring, chordal shortening techniques (which are now uncommon), the presence of anterior leaflet pathology, and, of course, the unavailability of pliable leaflet tissue (more often seen in patients with rheumatic disease) have been associated with higher repair failure rates.

ACKNOWLEDGEMENT

We would like to thank M. Williams for preparation of the images in this chapter.

FURTHER READING

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