Chronic Ischemic Mitral Regurgitation: Repair, Replace or Rethink?

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Ischemic mitral regurgitation (IMR) is a common complication of coronary artery disease and is the focus of a rapidly increasing amount of research. Mechanistic studies have determined that IMR is caused by apical displacement and tethering of the mitral valve leaflets after myocardial infarction, resulting in incomplete coaptation. Despite the relatively high prevalence of IMR, most centers have only a small surgical experience with this disorder. The result is that a number of different procedures have been recently developed without clear improvement in patient outcomes. The current review will examine the myriad surgical options for IMR with a focus on clinical outcomes.

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Chronic ischemic mitral regurgitation (IMR) is becoming the focus of an increasing amount of cardiovascular research. Chronic IMR is also referred to as functional mitral regurgitation (MR), as opposed to organic MR, because of the normal appearance of the mitral valve (MV) leaflets. Chronic IMR is associated with a markedly worse prognosis after myocardial infarction (MI) and coronary revascularization [1–6], and is a common cause of post-MI congestive heart failure [7, 8]. Chronic IMR is present in 10% to 20% of patients with coronary artery disease (CAD) [9–11]. Chronic IMR has been called the "last frontier" in MV repair surgery and one of the few therapeutic opportunities in heart failure patients [12].

Despite the high prevalence of chronic IMR, few patients are referred for cardiac surgery. The result is that no surgical center has a large experience with these complex patients. A number of surgical techniques have been developed for IMR, but none of these strategies has resulted in clearly improved patient outcomes. Long-term survival rates for IMR remain worse than for many types of cancer [13].

The goal of this paper is to review recent advancements in the understanding of chronic IMR pathogenesis and to examine the numerous surgical techniques that are currently being employed for this complex problem.

Material and Methods

The current paper is intended as a practical review of the different surgical (and new percutaneous) procedures for IMR, with a brief review of pertinent studies on diagnosis, prevalence, pathophysiology, and natural history. We performed a PubMed search of the term "ischemic mitral regurgitation" in order to identify studies, with focus on those published within the last 5 years.

Definition of Chronic IMR

One of the main limitations of previous clinical studies on chronic IMR is the lack of a clear definition. Different descriptions have resulted in heterogeneous patient groups, which in turn complicate comparisons between studies. The following is a summary of conditions that are often misclassified as chronic IMR, followed by our suggestion for a simple definition of chronic IMR.

Acute Ischemic Mitral Regurgitation

Ischemic mitral regurgitation may present acutely secondary to papillary muscle (PM) infarction and rupture, a condition known as acute IMR. Such patients usually present in cardiogenic shock because of the limited ability of the LV to adapt to acute volume overload. Surgical therapy usually consists of MV replacement with substantial operative mortality rates. As stated above, this review article will focus on chronic IMR only, and therefore acute IMR will not be discussed any further.

Organic MR Plus Coronary Artery Disease

In chronic IMR, the MV leaks and yet the leaflets and subvalvular apparatus appear normal. Chronic IMR is therefore not a disease of the valve per se, but rather a disease of the ventricle [12–14]. Patients with organic MV leaflet pathology (myxomatous, rheumatic, or other) and incidental CAD should not be classified as having chronic IMR. This is an important distinction because patients with organic MR and concomitant

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CAD have a much better long-term prognosis than patients with chronic IMR.

CHRONIC IMR DEFINITION. Chronic IMR should be defined as mitral regurgitation occurring more than 1 week after MI with (1) one or more left ventricular segmental wall motion abnormalities; (2) significant coronary disease in the territory supplying the wall motion abnormality; and (3) structurally normal MV leaflets and chordae tendinae. The third criterion is particularly important as it excludes patients with organic MR and associated CAD. Adoption of this definition should ensure homogeneity of patient populations and facilitate comparisons between studies.

Diagnosis of Chronic IMR

Patients with chronic IMR often present with symptoms of congestive heart failure, but may also complain of angina. A holosystolic murmur may not be heard as the degree of MR is variable under different hemodynamic states. The diagnosis of chronic IMR is confirmed with echocardiography. Color M-mode shows early systolic or bimodal (early and late systolic) peak MR, corresponding to those time points when there is maximal imbalance between closing and tethering forces on the MV leaflets.

Transesophageal echocardiography is helpful to assess MV morphology and to rule out organic causes of MR, but is not required in patients with chronic IMR. In fact, chronic IMR is frequently underestimated during intraoperative transesophageal echocardiography examination because of the afterload reduction induced by anesthesia. It is for this reason that decisions regarding surgical intervention on the MV should be made before patients undergo intraoperative transesophageal echocardiography. Similarly dobutamine stress echocardiography is not helpful in determining surgical indications for patients with IMR, as dobutamine may paradoxically decrease the amount of insufficiency as LV volumes decrease under pharmacologic stress. Transthoracic echocardiography is more likely to give an estimate of the degree of MR under normal conditions, but is also subject to underestimation. For these reasons, exercise echocardiography is becoming the "gold standard" for the diagnosis of chronic IMR [15]. Patients with pulmonary edema secondary to chronic IMR may demonstrate significant valvular regurgitation only during exercise [16].

The precise echocardiographic definition of "significant" IMR is open to debate, and traditional methods of MR quantification may not be applicable [14]. Color-flow mapping of the regurgitant jet area is highly susceptible to load conditions and is therefore unreliable. Regurgitant volumes and effective regurgitant orifice are less load dependent and more clinically relevant. Future studies of IMR should optimally report regurgitant volumes and effective regurgitant orifice areas, rather than the 1+ to 4+ scale that is often used in the cardiac surgery literature. Effective regurgitant orifice measurements require excellent quality images and significant
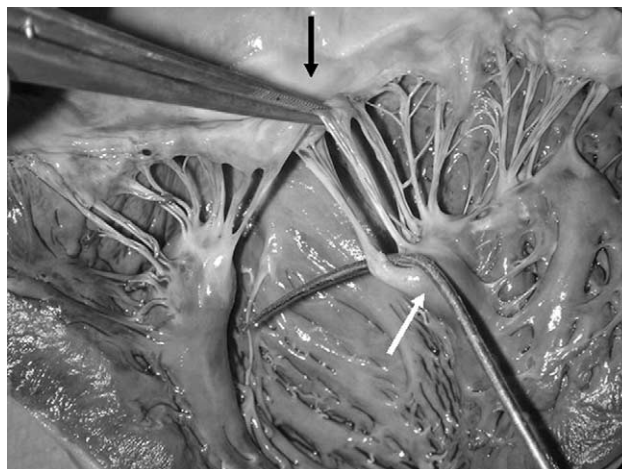

Fig 1. Secondary chordae tendinae attaching the posteromedial papillary muscle (white arrow) to the belly of anterior mitral valve leaflet (black arrow).

sonographer time, however, and that may limit their feasibility.

Lower thresholds for diagnosis of "severe" MR may be required for ischemic regurgitation. An effective regurgitant orifice of more than 20 mm², during rest or exercise, may be considered severe in patients with chronic IMR because of an associated worse long-term prognosis [7]. In contrast, the threshold effective regurgitant orifice for severe MR in organic mitral disease is 40 mm².

Pathophysiology of Chronic IMR

Normal MV function requires coordinated operation of all components including the annulus, leaflets, chordae tendinae, and PMs. The annulus is a saddlelike structure whose main function is to act as a fulcrum for the mitral leaflets and to decrease the size of the mitral orifice (by 10% to 20%) during late diastole and systole [17–19]. A dilated mitral annulus is often reported as an important etiologic factor in chronic IMR [13], but isolated annular dilation does not cause significant MR [20].

The second component of the MV apparatus is the leaflets. The leaflets are morphologically normal in IMR, but there is tethering and retraction of the leaflet bodies. As stated before, it is important to exclude patients with organic MV leaflet pathology and associated CAD from IMR studies, as these patients have a much better prognosis.

Chordae tendinae are the third component of the MV apparatus, connecting the PMs to the leaflets. Primary chordae attach to the free edge of the leaflets and prevent prolapse during systole. Secondary chords attach to the belly of the leaflet and are thicker than primary chords (see Fig 1). Apical displacement of the PMs leads to tethering of the secondary chords and decreased leaflet coaptation. Chordal tethering can cause kinking of the anterior leaflet in its mid-belly, resulting in the characteristic "seagull" sign on echocardiography (see Fig 2).
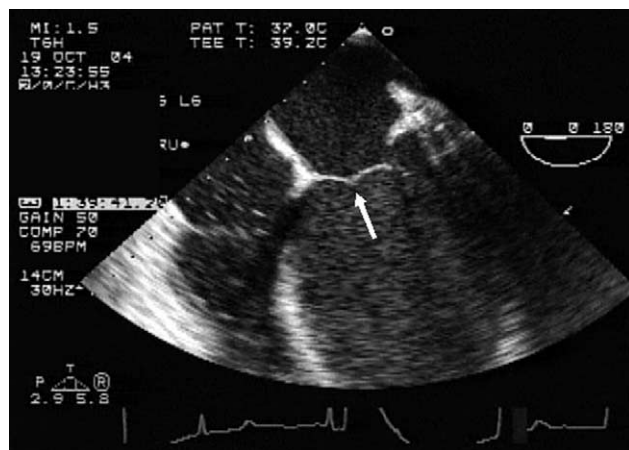
Fig 2. "Seagull" sign of ischemic mitral regurgitation caused by tethering of the anterior mitral valve leaflet (arrow) by secondary chordae tendinae.

The final component of the MV apparatus is the PMs, named according to their relationship with the commissural area. The anterolateral PM has a dual blood supply from branches of the circumflex and left anterior descending arteries. The posteromedial PM, in contrast, is supplied by a single artery arising from either the right coronary or from the terminal circumflex artery and is therefore more prone to infarction. Clinical studies have confirmed that IMR is more likely to occur after posteroinferior than anterolateral MI [14, 21]. However, the previously held notion that IMR is due to isolated PM dysfunction is incorrect. Large animal studies have revealed that isolated PM infarction does not cause significant MR [22], and may in fact paradoxically improve IMR [23].

Left ventricular distortion and remodeling after MI displace PMs away from the mitral annulus [14, 24, 25]. The displacement puts excessive tension on the chordae, resulting in apical mitral leaflet tethering, restricting their coaptation during systole [24, 26–29]. Leaflet tethering is compounded by LV contractile dysfunction, which decreases the closing force on the leaflets [28]. Once IMR is initiated, end-diastolic LV volume and wall stress increase in tandem with preload [5, 10, 29 30]. Left ventricular mass also increases progressively without a concomitant increase in end-diastolic wall thickness [30], resulting in generalized loss of myocardial contractile function [10, 31]. Increased wall stress causes more LV dysfunction [32], which in turn results in further PM displacement and leaflet tenting. If LV dilation occurs, it leads to annular enlargement and dysfunction thereby increasing valvular incompetence [33]. Chronic IMR therefore begets MR in a self-perpetuating manner.

Prevalence of Chronic IMR

The prevalence of chronic IMR has been difficult to calculate as a result of the heterogeneity of MR patients presented in previous studies [13, 14, 34]. Studies have reported a high incidence of IMR (11% to 19%) in patients undergoing cardiac catheterization for symptomatic CAD [9–11]. Hickey and colleagues [11] projected the incidence of moderate-to-severe IMR to be 7% within this patient cohort. Approximately 14.6 million Americans have angina pectoris (chest pain) or a history of MI [35]. Combining the above statistics, it can be estimated that the prevalence of IMR in the United States is 1.6 million to 2.8 million patients [10, 11, 35], a one-third increase from 1995 [13]. These statistics show that chronic IMR continues to be a widespread problem and may increase as survival rates for acute MI improve.

Natural History of Chronic IMR

Patients with IMR have a worse natural history than patients with CAD and no IMR. A study of 11,748 cardiac catheterization patients revealed that severe IMR was associated with a 1-year mortality of 40%, moderate IMR 17%, and mild IMR 10% [11]. If no IMR was present at catheterization, the 1-year mortality rate was 6%. Data from trials of thrombolysis for acute MI showed similarly poor prognosis for IMR [9]. Post–myocardial infarction patients have a 1-year mortality rate of 52% if they have severe IMR, 22% if they have mild-moderate IMR, and 11% if they have no IMR.

The Survival and Ventricular Enlargement (SAVE) study demonstrated that mild chronic IMR increases the risk of cardiovascular mortality, even in patients without congestive heart failure [4]. Patients with IMR had a higher incidence of cardiovascular mortality (29% versus 12%) and heart failure (24% versus 16%) than patients without IMR at a mean of 3.5 years after MI. Adjustment for differences in baseline characteristics revealed that mild-to-moderate (1+ or 2+) IMR strongly predicted mid-term mortality [4].

Surgical Treatment Options for Chronic IMR

Isolated CABG Versus MV Surgery and CABG

The indications for surgery in chronic IMR are not well defined. It is generally agreed that patients who have indications for coronary artery bypass grafting (CABG) with moderate-to-severe IMR (3+ or 4+) should also undergo concomitant MV surgery [18, 36]. Rarely, patients present with episodic IMR that occurs only during episodes of acute ischemia. Such patients can be treated with revascularization alone with good results.

It is controversial whether CABG patients with mild-to-moderate MR (1+ or 2+) should undergo concomitant MV surgery. Mallidi and coworkers [37] compared patients undergoing isolated CABG with mild-to-moderate MR to patients without MR. They found a higher prevalence of heart failure symptoms and decreased cardiac-event–free survival in MR patients during follow-up. In addition, 30% of patients progressed to 3+ or 4+ MR during a mean follow-up of 16 months.

Worsening of IMR after isolated CABG surgery has been demonstrated by other investigators and is associ-

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ated with decreased long-term survival [38, 39]. Aklog and coworkers [38] found persistent moderate or severe MR in 77% of patients treated with revascularization alone and were unable to identify predictors of postoperative improvement. Wong and coworkers [40] described more long-term MR, but no difference in survival, among patients with 3+ IMR who underwent isolated CABG versus patients who underwent CABG plus MV repair.

Such findings would suggest that MV surgery should be performed at the time of CABG in patients with mild-to-moderate IMR. However, the risk of long-term MR and heart failure progression must be balanced against the increased perioperative risk of the additional MV procedure. Data from the STS database suggests that concomitant MV surgery increases the perioperative risk of CABG by roughly twofold [37]. We therefore recommend that patients with mild-to-moderate IMR and multiple comorbidities, or a life expectancy of less than 5 years, should be treated conservatively and undergo CABG only. Low-risk patients with mild-to-moderate IMR should undergo concomitant MV surgery provided the procedure can be performed with low mortality rates, namely, 5% or lower.

Mitral Valve Replacement

The preferred surgical approach to severe IMR in early studies was MV replacement with a mechanical or tissue prosthesis. Mitral valve replacement involved complete excision of the subvalvular apparatus, but subsequent studies revealed a detrimental effect on LV function with this technique [41]. The negative effect on LV function may explain the high mortality rates in early IMR surgical series. Preservation of the entire subvalvular apparatus can usually be performed in IMR patients because the MV leaflets and chordae are thin and pliable (see Fig 3). Subvalvular preservation results in maintenance of annulo-ventricular continuity and improved preservation of LV function [42].

Mitral valve replacement is still a reasonable surgical option in many patients with IMR, predominantly because of its reliability and reproducibility. Mitral valve replacement should be considered for patients with acute IMR, and for patients with chronic IMR and multiple comorbidities, complex regurgitant jets (noncentral jet or more than one jet), or severe tethering of both MV leaflets [34, 43, 44]. Calafiore and coworkers [43] recommend MV replacement when the distance between the coaptation point of the leaflets and the plane of the mitral annulus exceeds 10 mm.

Mitral Valve Repair

The suboptimal early results for MV replacement led surgeons to start performing MV repair for IMR. There have been no randomized trials to date comparing MV repair to replacement for chronic IMR, but there have been a number of retrospective studies. The two largest and most methodologically sound studies are those per-
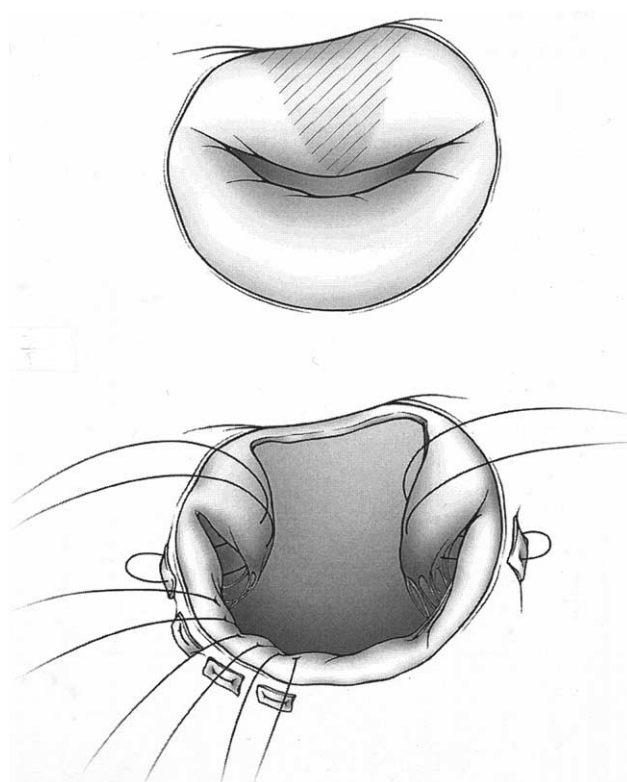

Fig 3. Retention of the subvalvular apparatus during mitral valve replacement for IMR, as described by Calafiore and coworkers [43]. Only a small portion of the anterior leaflet (shaded area) is resected. (Modified and reprinted from Ann Thorac Surg, 77, Calafiore AM et al, Mitral valve surgery for chronic ischemic mitral regurgitation, 1989–97, Copyright 2004, with permission from The Society of Thoracic Surgeons.)

formed by Gillinov and associates [44] and Grossi and colleagues [45]. These investigators demonstrated that both MV repair and replacement are effective at eliminating MR immediately postoperatively, but MV repair was associated with lower perioperative mortality. However, patients at the highest risk with the most severe MR did just as well, and possibly better, with MV replacement [34, 44]. Regardless of which surgical technique was employed, 5-year survival rates were uniformly disappointing at approximately 50% [44, 45].

Currently, there is general consensus in the cardiac surgery community that MV repair results in lower perioperative mortality than MV replacement and should therefore be performed whenever possible [43]. A recent study by Al-Radi and coworkers [46] confirmed a lower perioperative mortality for MV repair in patients with chronic IMR. The survival advantage decreased over time, however, and was no longer apparent by 5 years after surgery.

Undersized Mitral Annuloplasty

The most common surgical procedure currently performed for chronic IMR is undersized mitral annulo-

plasty. Bolling and colleagues [47] popularized this approach, using very small (size 24 to 26) mitral annuloplasty rings. The rationale is that undersizing the mitral annulus will result in increased leaflet coaptation and decreased regurgitation. Although this procedure does not fully address the ventricular causes of chronic IMR, it is simple to perform and reproducible, and therefore has been widely adopted by the cardiac surgery community. The type of ring that should be used for undersized annuloplasty is unclear, with several authors arguing the merits of rigid versus flexible and complete versus incomplete rings [48]. A close examination of the literature does not reveal any definite advantages for any specific type of annuloplasty ring, however, with the exception of poor long-term results for pericardial bands [49].

The initial results for undersized mitral annuloplasty seemed encouraging, with low perioperative mortality rates [50]. However, subsequent studies at various centers revealed a significant proportion of patients develop recurrent MR during follow-up. A recent article from the Cleveland Clinic examined 585 patients undergoing undersized annuloplasty surgery over a 17-year period [51]. In 28% of patients, moderate or more MR developed 6 months postoperatively, an incidence that is similar to that described by other investigators [51–53]. Mitral regurgitation recurrence tends to occur early postoperatively with relatively low recurrence rates thereafter [49]. The relatively high MR recurrence rate associated with undersized annuloplasty has led a number of investigators to examine alternative surgical therapies.

Alternative Surgical Procedures for Chronic IMR

Second-Order Chordal Cutting

Messas and coworkers [3] proposed to reduce leaflet tethering by cutting a limited number of critically positioned second-order chordae tendinae. Secondary chords are the most responsible chordae for leaflet restriction in chronic IMR, but are not required to prevent leaflet prolapse. Dividing secondary chordae in a sheep model of IMR resulted in improved leaflet coaptation and reduced MR, without leaflet prolapse or decline in LV ejection fraction [3]. Other investigators have demonstrated, however, that second-order chordal cutting may adversely affect LV function [54]. A recent case report described no MR and improved LV function in a patient undergoing chordal cutting surgery [55].

We have recently adopted the technique of chordal cutting for patients with chronic IMR. We divide all second-order chords arising from the affected PM (usually the posteromedial muscle) at their insertion on the anterior and posterior leaflets, and insert a slightly undersized (size 28 to 30) flexible posterior annuloplasty ring. We have performed this operation in more than 30 patients, with a perioperative mortality rate of 6%. Follow-up echocardiography revealed trivial or mild MR in 97% of patients for as long as 24 months postoperatively. In addition, there has been no decrease in LV function in

the early postoperative period. Although we are enthusiastic about this procedure at this time, the long-term results and durability of the repair remain to be seen.

Alfieri Edge-to-Edge Repair

Alfieri and colleagues [56] and Maisano and associates [57] have described an edge-to-edge MV leaflet repair technique for many different causes of MR. The technique is quick and relatively simple to perform. A suture is used to join the center of the anterior and posterior leaflets, creating a double orifice MV. A mitral annuloplasty ring is inserted because long-term results without an annuloplasty ring are suboptimal [57]. Although many studies have indicated good results for the Alfieri repair technique, most had small numbers of patients with chronic IMR. An exception is the recent study by Bhudia and colleagues [58] that included 143 patients with ischemic cardiomyopathy. These investigators found a progressive increase in MR recurrence in chronic IMR patients, with more than 30% of patients having moderate-to-severe MR 1 year postoperatively. They concluded that other MV repair techniques are required for this patient population.

Infarct Plication

Liel-Cohen and associates [27] devised an infarct plication procedure to reverse LV remodeling in sheep. The infarcted region of the LV is plicated with mattressed sutures to reduce myocardial bulging and to bring the displaced PM tips back toward the anterior mitral annulus (see Fig 4). The plication process also reduces the proportion of LV circumference occupied by infarcted myocardium.

Infarct plication for IMR was recently described in humans. Ramadan and colleagues [59] performed plication of posterolateral myocardial scar without mitral annuloplasty in 3 patients. Two patients had no MR and 1 had trivial MR 7 months postoperatively. Although this technique appears promising, more data are required before it can be recommended for IMR patients.
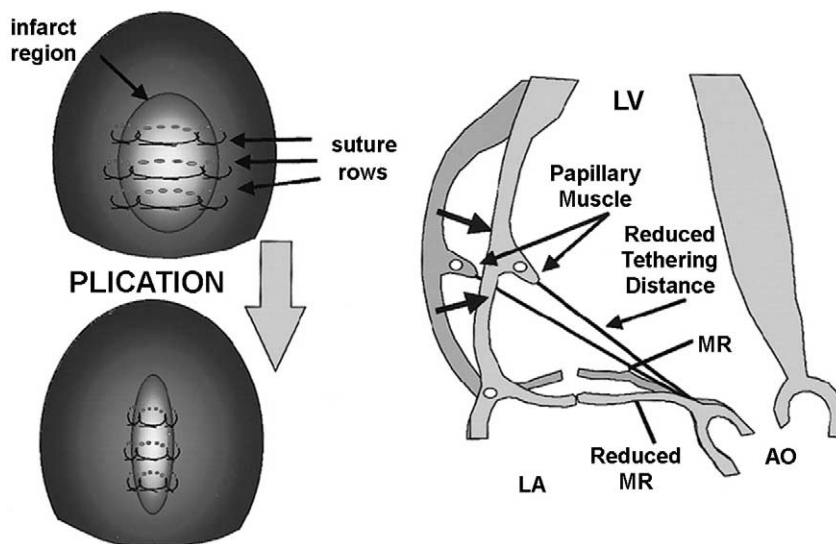
Papillary Muscle Imbrication

Menicanti and associates [60] devised an LV restoration procedure for the treatment of chronic IMR and dilated ischemic cardiomyopathy. The procedure consists of coronary revascularization, LV restoration with a Dor procedure, and imbrication of the PMs without a prosthetic ring. The Dor procedure may reduce MR in dilated ischemic cardiomyopathy by reducing LV size and improving PM orientation [61]. Menicanti and colleagues [60] have used this approach in 46 patients with previous anterior transmural MI and MR. Postoperative MR was mild or less in 84% of the patients [60].

Papillary Muscle Sling

Another complex surgical approach to IMR has been recently devised by Hvass and colleagues [62]. This technique consists of correcting abnormal PM displacement with an intraventricular Gore-Tex sling. The device is implanted through the left atrium and across the mitral

Fig 4. Infarct plication to restore papillary muscle position closer to the anterior mitral annulus and reduce tethering of the leaflets, as described by Liel-Cohen and associates [27]. (AO = aorta; LA = left atrium; LV = left ventricle; MR = mitral regurgitation.) (Modified and reprinted from Liel-Cohen N, et al, Design of a new approach for ventricular remodeling to relieve ischemic mitral regurgitation: insights from three-dimensional echocardiography, Circulation, 101, 23, 2756–63 [27], with permission.)

orifice, encircling the trabecular base of both PMs. Tightening the sling with sutures decreases the distance between the PMs, and a moderately undersized mitral annuloplasty ring is inserted. A double ring is thereby produced: one inside the ventricle and one on the mitral annulus. Hvass and associates [62] have applied this technique in 10 patients, with reduced mitral tenting and decreased MR in all patients immediately postoperatively. However, the long-term effects of this technique are unknown.

Surgical Relocation of the Posterior Papillary Muscle

Kron and associates [33] have described another technique for the treatment of chronic IMR, particularly in patients with severe restriction of the P3 segment of the MV. A suture is used to connect the posterior PM to the mitral annulus, adjacent to the right fibrous trigone, and a mitral annuloplasty ring is inserted (see Fig 5). The suture between the PM and the mitral annulus is shortened to alleviate tethering of the P3 segment and to increase leaflet coaptation. Kron and coworkers [33] have performed this procedure in 18 patients, with all patients having no or trace MR 8 weeks postoperatively.

Posterior MV Restoration

Fundaro and associates [63] devised a relatively simple technique to ameliorate posterior leaflet tethering and restore normal distance between the annulus and PM. An incision is made at the base of the posterior leaflet and the basal chordae are transected to increase posterior leaflet mobility (see Fig 6). The detached portion of the mitral annulus is then plicated and the resulting defect in the posterior leaflet is closed with a running suture. The plicated annulus is reinforced with a short Gore-Tex strip or posterior annuloplasty band. The data on this procedure are sparse.

Experimental Therapies for IMR

Percutaneous Alfieri Repair

The Alfieri MV repair technique has recently been applied percutaneously [64]. A double-armed clip device, deployed through the femoral vessels, was developed in a large animal model and is now undergoing trials in patients. The results for this device are pending, but MR recurrence rates may be higher compared with the conventional Alfieri operation because a mitral annuloplasty ring is not inserted [57].

Percutaneous Annuloplasty

Percutaneous mitral annuloplasty has been recently described in large animal models of IMR by two separate groups. Devices were inserted percutaneously into the coronary sinus in sheep [65] and dogs [66]. Both studies
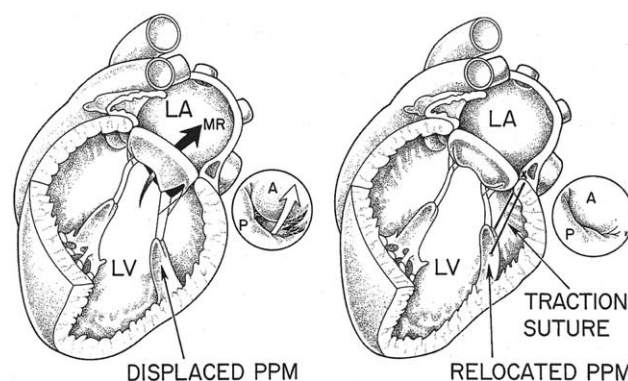
Fig 5. Relocation of the posterior papillary muscle (PPM), as described by Kron and associates [33]. (A = anterior mitral leaflet; LA = left atrium; LV = left ventricle; MR = mitral regurgitation [arrow]; P = posterior mitral leaflet.) (Reprinted from Ann Thorac Surg, 74, Kron IL, et al, Surgical relocation of the posterior papillary muscle in chronic ischemic mitral regurgitation, 600–1, Copyright 2002, with permission from The Society of Thoracic Surgeons.)
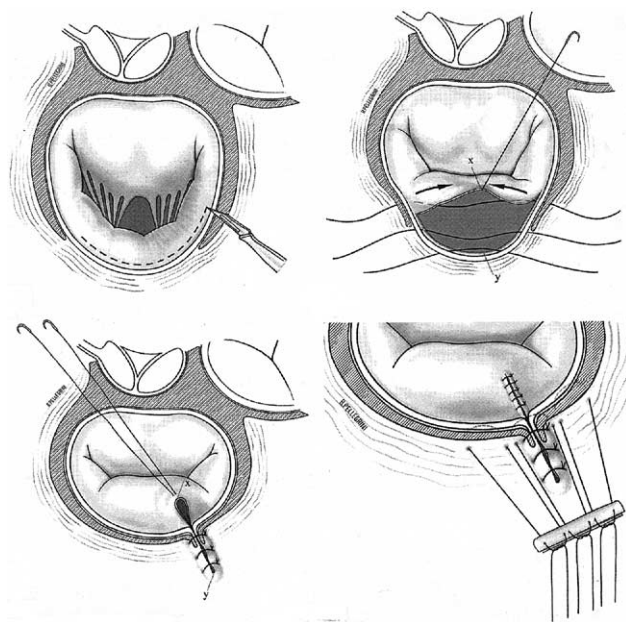
Fig 6. Posterior mitral valve restoration, as described by Fundaro and coworkers [63]. (Reprinted from Ann Thorac Surg, 77, Fundaro P et al, Posterior mitral valve restoration for ischemic regurgitation, 729–30, Copyright 2004, with permission from The Society of Thoracic Surgeons.)

revealed acute reductions in MR, but long-term animal studies are pending. Percutaneous annuloplasty offers the advantage of avoiding an operation, but has the potential disadvantage of coronary sinus perforation or thrombosis, or injury to the adjacent circumflex artery [67]. In addition, the durability of this approach may be compromised by the coronary sinus having no fibrous connections to the mitral annulus.

Septolateral Annular Cinching

Septolateral annular cinching was recently used by Timek and colleagues [68] in an ovine model of chronic IMR. Septolateral annular cinching is performed by anchoring a single suture to the midseptal annulus, then exteriorizing the suture through the posterior lateral annulus. The suture is tightened under echocardiographic guidance to reduce lateral displacement of the posterior PM. As opposed to undersized ring annuloplasty, septolateral annular cinching is not associated with impaired mobility of either the anterior or posterior leaflets [69]. Septolateral annular cinching decreased IMR and reduced the septolateral diameter in sheep [68], but clinical studies are pending.

Myocor Coapsys

The Coapsys device (Myocor, Maple Grove, Minnesota) is designed to restore septolateral annular geometry in patients with IMR [70]. It is inserted through a sternotomy but without cardiopulmonary bypass. The device consists of two epicardial pads and an expanded polytetrafluoroethylene-coated subvalvular chord. The poste-

rior pad has two heads that change the shape of the LV at the mitral annulus and PM level. The anterior pad is adjustable and is fixed after echocardiographically confirmed positioning of the posterior pad. The Coapsys device has been successfully used in a dog model [70] and is currently undergoing clinical assessment.

Dacron Patch-Inflatable Balloon Device

Hung and associates [2] devised a Dacron patch inflatable balloon to reverse LV remodeling and reduce IMR. The balloon device is secured externally onto the infarcted myocardium and is inflated under echocardiographic guidance. The infarcted myocardium and PM is displaced inward toward the anterior mitral annulus, thereby decreasing leaflet tethering. The inflatable balloon has the benefit of not requiring cardiopulmonary bypass, but is yet to be tested in humans.

Conclusions

Chronic IMR is a common clinical entity and is associated with poor long-term survival. Numerous surgical techniques have been developed for IMR, but none has resulted in clearly improved patient outcomes. Several recent advancements have led to important insights into the pathogenesis of IMR. A heightened understanding of the pathogenesis of IMR by the cardiac surgery community may lead to improved surgical techniques. Chronic IMR should remain an active area of basic and clinical research to define the optimal treatment strategy and to improve outcomes for a steadily growing patient population.

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