Mitral regurgitation

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Summary

Mitral regurgitation (MR) involves systolic retrograde flow from the left ventricle into the left atrium. While trivial MR is frequent in healthy subjects, moderate to severe MR constitutes the second most prevalent valve disease after aortic valve stenosis. Major causes of severe MR in Western countries include degenerative valve disease (myxomatous disease, flail leaflet, annular calcification) and ischaemic heart disease, while rheumatic disease remains a major cause of MR in developing countries. Chronic MR typically progresses insidiously over many years. Once established, however, severe MR portends a poor prognosis. The severity of MR can be assessed by various techniques, Doppler echocardiography being the most widely used. Mitral valve surgery is the only treatment of proven efficacy. It alleviates clinical symptoms and prevents ventricular dilatation and heart failure (or, at least, it attenuates further progression of these abnormalities). Valve repair significantly improves clinical outcomes compared with valve replacement, reducing mortality by approximately 70%. Reverse LV remodelling after valve repair occurs in half of patients with functional MR. Percutaneous, catheter-based mitral valve repair is a novel approach currently under clinical scrutiny, with encouraging preliminary results. This modality may provide a valuable alternative to mitral valve surgery, especially in critically ill patients.

Key words: mitral valve; mitral regurgitation; mitral valve repair; mitral valve replacement; mitral leaflets; heart failure

Introduction

Over four million Europeans and a similar number of Americans suffer from significant mitral regurgitation (MR). Approximately 250 000 new patients are diagnosed with the disease annually [1, 2]. The disorder generally evolves insidiously over many years because the heart compensates for the regurgitant volume by left atrial enlargement, left ventricular (LV) volume overload, and progressive LV dilatation. Older patients (>50 years) with severe organic MR, defined as an effective regurgitant orifice (ERO) area $\geq$40 mm², have 6% annual mortality (as compared with 3% for moderate MR) [3].

The most common causes of MR include ischaemic heart disease, nonischaemic heart diseases and valve degeneration. Both ischaemic (coronary artery disease) and nonischaemic heart diseases (e.g., idiopathic dilated cardiomyopathy) cause "functional" MR via multiple different mechanisms including impaired LV wall motion, LV dilatation, and papillary muscle displacement and dysfunction. In contrast, degenerative ("organic")

MR is caused by structural abnormalities of the valve leaflets and the subvalvular apparatus, including stretching or rupture of tendinous chords *(chordae tendineae)*.

Echocardiography plays a central role in the assessment of MR. Valve surgery is the only treatment for which sustained relief from symptoms and prevention (or significant improvement) of heart failure have been demonstrated. However, elderly patients with MR fare less well after valve surgery than those with aortic stenosis. Mortality from mitral valve surgery, especially valve replacement, is increased in older patients or in those with concomitant coronary artery disease. Surgical mortality in the elderly (>75 years) is low in experienced centres but exceeds 20% in less experienced centres [4].

Percutaneous catheter-based mitral valve repair procedures are currently under clinical evaluation. The preliminary results are encouraging [5].

Valve anatomy

The mitral valve apparatus consists of the annulus, leaflets and tendinous chords attaching to them, as well as papillary muscles anchoring the chords (fig. 1). The normal mitral valve has two leaflets: anterior and posterior. From an atrial view (fig. 2a), the mitral annulus can be recognised as a roughly elliptical line where the leaflets are anchored to the atrioventricular junction in a D-shaped configuration. The two leaflets are in close contact at the closure line forming a single zone of apposition which determines systolic coaptation between the leaflets (fig. 2b). The two leaflets are divided by two commisures. Small indentations most often partition the posterior leaflet in three scallops (fig. 2c-d); however, four or more scallops also are observed in a minority of patients. The tendinous chords are fibrous string-like structures attached to the ventricular surface of the leaflets. They are classified as finer marginal (primary) chords and thicker basal (secondary) strut chords [6, 7]. Primary chords position the leaflet tips and prevent prolapse. Secondary chords insert symmetrically near the anterior leaflet base. This classification has implications for mitral valve repair.

Unlike the tricuspid valve, the mitral valve does not have chords anchoring the leaflets to the ventricular septum. The papillary muscles are muscular protuberances of the LV wall anchoring the chords to the wall itself. The anterolateral papillary muscle is larger than the posteromedial and is supplied with blood from either the left circumflex or the left anterior descending coronary artery. Because most individuals exhibit a right-dominant pattern of the coronary anatomy, the posteromedial papillary muscle is in most cases supplied with blood from the right coronary artery. Thus, ischaemia in the territory of the left coronary artery frequently affects the anterolateral papillary muscle, whereas ischaemia in the territory of the right coronary artery most often affects the posteromedial papillary muscle.

Figure 2

Real-time 3D TEE view of the mitral annulus and leaflets (atrial view); a) Mitral annulus (black arrows); b) Mitral valve; closed position (black arrows: line of coaptation between the two leaflets; asterisks: indentations between scallops). PML = posterior mitral leaflet; AML= anterior mitral leaflet; Ao = aorta; c1) Real-time 3D TEE image of mitral valve in mid-diastole; c2 corresponding anatomical specimen (atrial view). According to Carpentier, scallops of the posterior and anterior leaflets are labelled P1, P2, P3 (from lateral to medial) and A1, A2, A3, respectively (arrows: commissures at the extremities of the coaptation line).

Figure 1

Anatomical specimen of human mitral valve consisting of the papillary muscles, tendinous chords, and the anterior and posterior leaflet (courtesy of Dr Edgardo Bonacina, Departmant of Pathology, Ospedale Niguarda-Cà Granda, Milano, Italy; reproduced with permission from: Faletra FF. Insufficienza mitralica. Quaderni di ecografia clinica. 2004, vol. 2, ERA Edizioni, Castelseprio VA, Italy).
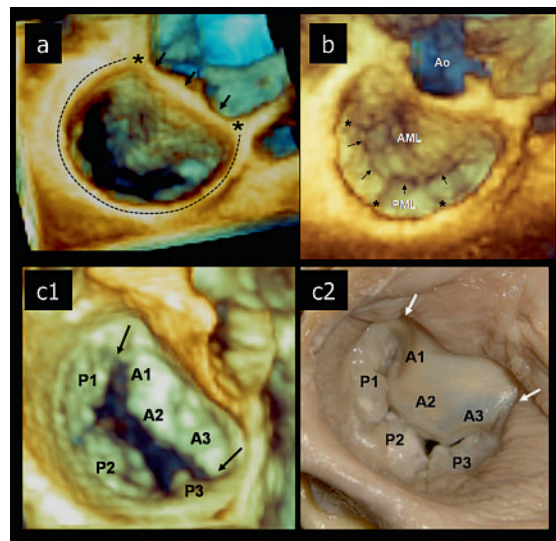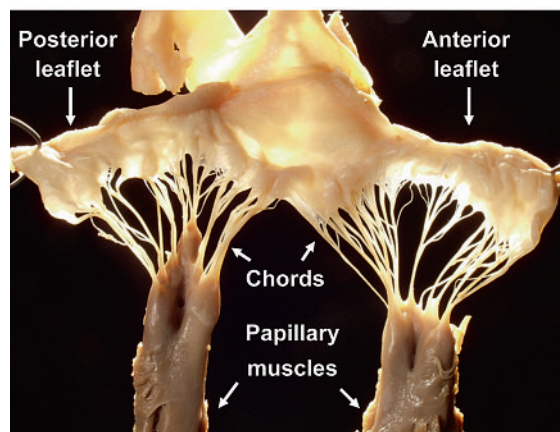
Causes and mechanisms of MR

Both functional and organic abnormalities causing MR do so by impairing the systolic coaptation between the anterior and posterior leaflet. There are a multiplicity of different causes and mechanisms for MR. A given cause is not invariably linked to the same mechanism; rather, it can affect leaflet coaptation through different mechanisms.

Causes of MR can be subdivided into non-ischaemic and ischaemic (i.e., related to coronary artery disease).

Mechanisms of MR can be subdivided into functional (i.e., MR through a structurally normal valve; e.g., caused by segmental LV wall motion abnormalities, LV dilatation, papillary muscle displacement and dysfunction) and organic (i.e., structural abnormalities of the leaflets or the subvalvular apparatus including the chords).

Carpentier introduced a functional classification of MR based on leaflet movement (table 1): Type I with normal leaflet movement (e.g., MR caused by annular dilatation or leaflet perfora-

tion); Type II with exaggerated leaflet movement (e.g., mitral valve prolapse); and Type IIIa and IIIb with restricted leaflet movement in diastole and systole, respectively.

Ischaemic MR is usually functional in nature and chronic in presentation. Occasionally it is caused by papillary muscle rupture, which is manifested in acute clinical symptoms. After infarction, the papillary muscles are displaced laterally, apically and posteriorly, pulling the leaflet into the left ventricle. Distortion is prominent in the basal anterior leaflet, creating a bend (the "seagull sign") [7]. Papillary muscle dysfunction plays only a minor role compared with apical and inferior papillary muscle displacement caused by ischaemic LV remodelling and dilatation. Because tendinous chords are not extensible, papillary muscle displacement exerts traction on the leaflet, causing tethering, apical leaflet displacement, and impaired coaptation between the two leaflets. Together with annular flattening, enlargement, and reduced contraction, mitral valve tenting affects leaflet coaptation and causes functional MR.

Degenerative (organic) MR involves mitral valve prolapse or, less frequently, calcifications of the mitral annulus.

Mitral valve prolapse is an abnormal systolic valve movement of one or both of the mitral leaflets towards the left atrium (≥2 mm beyond the saddle-shaped annular plane). Mitral valve prolapse is twice as prevalent in females as in males; however, severe MR caused by prolapse is more frequent in older males than young females. Prolapse is considered to be moderate when the leaflet tip remains in the left ventricle (billowing valve), and severe when the leaflet tip bulges into left atrium (flail leaflet). The latter is usually a consequence of chordal rupture [8]. Structural abnormalities causing mitral prolapse include diffuse myxomatous degeneration and primary flail leaflet with ruptured chords. The latter affects the posterior leaflet in 70% of cases. It can be associated with myxomatous degeneration restricted to the flail portion of the leaflet.

Rheumatic MR causes retraction of tendinous chords and leaflets, as well as annular dilatation, thus compromising coaptation between the two leaflets. Similar changes are observed in postinflammatory and postradiotherapy MR. Infectious endocarditis can cause MR through chordal rupture or leaflet perforation.

Table 1

Causes and mechanisms of mitral regurgitation.

Cause	Mechanism			
	Organic			**Functional**
	Type I	**Type II**	**Type IIIa**	**Type I/IIIb**
Nonischaemic	Endocarditis (perforation); degenerative (annular calcification); congenital (cleft leaflet)	Degenerative (mitral valve prolapse, flail leaflet); endocarditis (ruptured chords)	Rheumatic; iatrogenic (radiation/drug); inflammatory (lupus, anticardiolipin); etc.	Cardiomyopathy; myocarditis; left ventricular dysfunction (any cause)
Ischaemic		Ruptured PM		Functional ischaemic

MR = mitral regurgitation. PM = papillary muscle. Classification according to Carpentier: Type I: normal leaflet movement; Type II: excessive leaflet movement; Type III: restricted leaflet movement (IIIa in diastole, IIIb in systole).

Pathophysiology and natural history

The regurgitant volume depends upon the regurgitant orifice and the systolic pressure gradient between left ventricle and atrium [9]. Thus, the observed degree of MR depends on haemodynamic conditions at the time of examination. Any increase in preload or afterload, and any decrease in myocardial contractility, causes LV dilatation, enlargement of the mitral annulus, and an increase in ERO. In acute MR the atrium is noncompliant, and therefore mechanical energy generated by the left ventricle causes an increase in intra-atrial pressure. In chronic MR the atrium is more compliant, and therefore mechanical energy generated by the ventricle causes volume overload and atrial enlargement rather than an increase in intra-atrial pressure. Consequently, chronic MR may be associated with a small regurgitation wave (the so-called V-wave).

Progression of organic MR is associated with an increase in regurgitant volume by 5–7 ml per year. This is manifested by a progressive increase in ERO area due to valvular degeneration and annular enlargement [10]. Patients with chronic MR often remain asymptomatic for many years. Over time, however, the left ventricle dilates to accommodate the increased volume load and maintain cardiac output. Chronic LV volume overload leads to contractile dysfunction, heart failure and increased risk of sudden death. Patients with severe, symptomatic MR have a poor prognosis, with a 5% annual mortality if valve surgery is not performed. Progressive heart failure is the most common cause of death in these patients, while sudden death, stroke, and fatal endocarditis are less frequent. Asymptomatic or minimally symptomatic patients with severe MR secondary to mitral valve prolapse and normal LV function at rest have an annual risk of undergoing valve surgery of approximately 10%, mainly due to the appearance of clinical symptoms [11].

Assessment of MR

Echocardiography plays a central role in the assessment of the mechanism and the severity of MR, as well as of the feasibility of valve repair versus replacement. In severe MR, transthoracic echocardiography (TTE) shows left atrial and ventricular enlargement. LV systolic motion may be increased in the compensatory phase of chronic MR. Peak mitral flow velocity is increased and flow in the pulmonary veins during systole may be reversed. Echocardiography is very useful for identifying the mechanism of MR, e.g., chordal rupture, valve prolapse, rheumatic disease, a flail leaflet, endocarditis or LV dilatation. Calcification of the mitral annulus is visible as a high-density structure between the mitral apparatus and the posterior LV wall.

Transoesophageal echocardiography (TEE) is superior to TTE for assessing the precise anatomy of the valve and MR severity. TEE is mandatory when transthoracic images are of suboptimal quality (fig. 3) [12], as well as for pre- and perioperative valve assessment. Real-time three-dimensional (3D) TTE or TEE provides a comprehensive imaging of the mitral valve. 3D TEE images mimic the view of the valve through the left atrium at surgery (fig. 2). This method allows a precise assessment of anatomical structures (e.g., the mitral annulus), which cannot be easily evaluated by 2D modalities.

Quantitative methods for assessing the severity of MR include *vena contracta* and the proximal isovelocity surface area (PISA) method [13]. *Vena contracta* is defined as the narrowest cross-sectional area of the regurgitant jet determined by colour flow Doppler echocardiography. It correlates well with the severity of MR. The PISA method is based on the acceleration of the regurgitant flow towards the mitral orifice. This flow can be mapped as isovelocity hemispheric shells. ERO area ≥ 40 mm^2 and regurgitant volume ≥ 60 ml indicate severe MR. These methods have prognostic value.

Figure 3

TEE long-axis view showing severe MR due to tethering of the posterior mitral leaflet (functional, ischaemic MR).
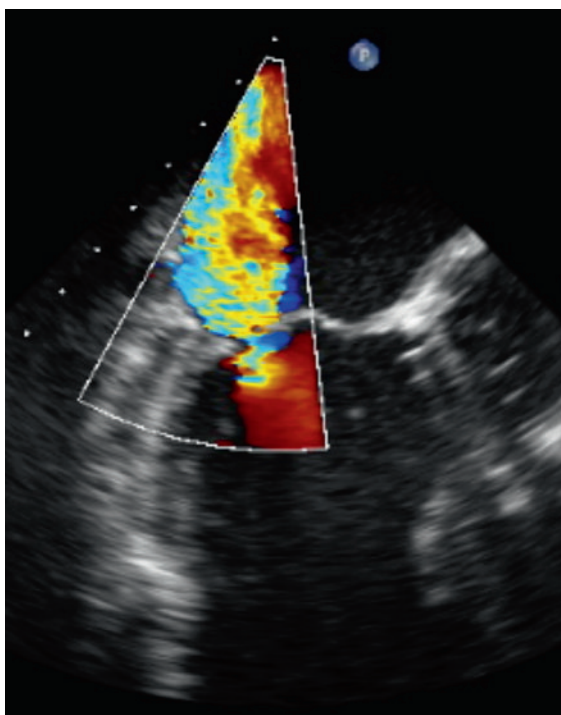
Treatment

The impact of different treatments on survival rates has not been evaluated in randomised clinical trials; hence, it can only be estimated from outcome studies [14].

Medical treatment

Pharmacological treatment aims to alleviate symptoms and slow down progression of LV dysfunction. In acute MR, nitrates and diuretics reduce filling pressures. Nitroprusside reduces afterload and hence regurgitant fraction. Inotropic agents may be beneficial in patients with heart failure and hypotension. Patients with MR and permanent or paroxysmal atrial fibrillation, and patients with a history of systemic thromboembolism or evidence for a left atrial thrombus should receive anticoagulant therapy with a target international normalised ratio (INR) of 2–3 units [14]. Anticoagulant therapy is also required after mitral valve repair (3 months). In contrast, there is no evidence to support the use of vasodilators, including ACE inhibitors, in chronic MR in the absence of heart failure [15]. Once heart failure is established, however, ACE inhibitors are beneficial. They can be used in patients with advanced MR and severe symptoms who are poor candidates for surgery and in patients who remain symptomatic after surgery due to LV dysfunction. Beta-blocker and spironolactone may also be beneficial in patients with heart failure. According to new guidelines, endocarditis prophylaxis for MR is no longer required; however, it is still mandatory after mitral valve repair (6 months) or mitral valve replacement [16].

Surgical treatment

Mitral valve surgery is the only treatment for MR which provides sustained relief of symptoms and prevents the development (or further progression) of heart failure [17]. However, no randomised trials have been carried out to define the precise impact of surgery on mortality and morbidity in these patients. Criteria for mitral valve surgery are summarised in table 2.

Mitral valve repair is superior to valve replacement in terms of both mortality and morbidity, and is therefore preferable whenever feasible [17]. This approach includes an array of valvular, subvalvular, and annular procedures aiming to restore leaflet coaptation, i.e., valvular normal function. Overall these techniques are more effective in the presence of redundant, as opposed to retracted or calcified, leaflets.

Surgical repair for valve prolapse

A typical repair technique for prolapse of a posterior leaflet consists of triangular or quadrangular resection of the prolapsed leaflet segment in conjunction with posterior annulus plication sutures (fig. 4a). Subvalvular support can be achieved by chordal transfer or artificial chords rather than chordal shortening. Annuloplasty is routinely per-formed by the use of annular bands and either flexible or rigid rings. Prosthetic rings reconstitute and stabilise the correct shape of the annulus and the repaired valve; they therefore represent a mandatory part of mitral valve reconstruction.

Surgical repair for anterior leaflet prolapse is more challenging than that for posterior leaflet prolapse. Reconstructive techniques for anterior leaflet prolapse include replacement, shortening or transposition of chords, reinforcement with artificial chords, leaflet-folding plasty and triangular resection [18]. Overall, long-term outcomes of mitral valve repair for anterior leaflet prolapse are satisfactory [18]. Early valve repair of MR caused by leaflet prolapse, before deterioration in LV size or function, increases the likelihood of postoperative normalisation of LV ejection fraction [19].

Table 2

Indications for mitral valve surgery.

Class I
Symptomatic patient with acute severe MR
Chronic severe MR and NYHA functional class II, III or IV symptoms in the absence of severe LV dysfunction (LVEF <30%) and/or ESD >55 mm
Asymptomatic patients with chronic severe MR and mild to moderate LV dysfunction, LVEF 30–60%, and/or ESD >40 mm
Valve repair is recommended (superior to valve replacement)
Class IIa
Valve repair is reasonable in experienced surgical centres for asymptomatic patients with chronic severe MR with preserved LV function (LVEF >60% and ESD <40 mm) in whom the likelihood of successful repair without residual MR is >90% (IIb in the guidelines of the European Society of Cardiology)
Valve surgery is reasonable for asymptomatic patients with chronic severe MR, preserved LV function, and:
New onset of atrial fibrillation
Pulmonary hypertension (PASP >50 mm Hg at rest or >60 mm Hg during exercise)
Valve surgery is reasonable for patients with chronic severe MR due to primary abnormalities of the mitral apparatus, NYHA functional class III-IV, and severe LV dysfunction (LVEF <30% and/or ESD >55 mm) in whom valve repair is highly likely
Class IIb
Valve repair can be considered for patients with chronic severe secondary MR due to severe LV dysfunction (LVEF <30%) persistently in NYHA functional class III–IV despite optimum therapy for heart failure, including biventricular pacing
Class III
Valve surgery is not indicated for asymptomatic patients with MR and preserved LV function (LVEF >60% and ESD <40 mm) in whom significant doubt about the feasibility of repair exists
Isolated MV surgery is not indicated for patients with mild to moderate MR
Abbreviations: ESD: endsystolic diameter; LVEF: left ventricular ejection fraction; PASP: pulmonary artery systolic pressure

Figure 4

Left panel: Classical quadrangular resection for mitral valve prolapse. Right panel): New ring design aimed at improving annular support.

Valve repair for functional MR

In functional MR, valve repair rather uniformly includes remodelling of the distorted annulus with a prosthetic ring also aiming to reduce the antero-posterior annular diameter. Ring annuloplasty is usually sufficient for nonischaemic functional MR.

Surgery for ischaemic functional MR is more demanding. This is mainly due to the *sequelae* of ischaemic heart disease (regional or diffuse hypo-/akinesia, ventricular dilatation, ventricular dysfunction) and the tethering of the posterior leaflet usually present due to apical and posterolateral displacement of the papillary muscles following myocardial infarction and ventricular remodelling. The common denominator of all surgical approaches is restrictive (undersized) annuloplasty with a rigid or semirigid ring. Additional procedures specifically address the tethering of the posterior leaflet and include papillary muscle relocation by traction sutures or cutting of second order chordae of the posterior leaflet. So-called saddle-shaped annuloplasty rings (e.g., Geoform® rings) have recently been developed to address this problem [20]. These rings induce upward traction on the affected papillary muscle towards the mitral annulus. Initial data in patients with ischaemic MR show that mitral valve annuloplasty using the Geoform® ring restores leaflet coaptation and eliminates MR by effectively modifying the mitral annular geometry [21].

Centres with wide experience in mitral valve repair report 80–90% success rates [3]. LV reverse remodelling after mitral valve repair was observed in half of patients with functional MR and ischaemic or idiopathic dilated cardiomyopathy [22]. It was associated with longer repair durability and a better clinical outcome than in patients with persistence or progression of the remodelling process. Predictors of reverse remodelling are a preoperative LV enddiastolic diameter <65 mm and a generous coaptation surface between anterior and posterior leaflet after restrictive annuloplasty (coaptation height >8 mm) [23]. Continued LV remodelling contributes to recurrent regurgitation after annuloplasty [24], which is observed in 20–30% of patients after surgical valve repair for post-infarction MR [25]. Careful indication and choice of the surgical procedure, as well as attention to technical details, contribute to improved results [23].

Valve replacement

Valve replacement involves implantation of a biological or mechanical prosthesis. Bioprosthetic valves are associated with lower thromboembolic risk but limited longevity compared with mechanical ones. On the other hand, mechanical valves are associated with increased risk of thrombo-embolism, which mandates chronic anticoagulant therapy, with an associated increased risk of haemorrhagic complications [14, 26]. Considering the theoretical longevity of prosthetic valves, bioprostheses are generally preferred in patients over 65. It is obviously important to discuss the choice of the prosthesis with the patient.

Outcome after surgery

Clinical outcomes after mitral valve surgery depend on patient- and disease-specific factors, as well as on surgery-related factors. In-hospital morbidity and mortality for first time mitral valve repair and replacement surgery are listed in table 3. Early postoperative mortality is significantly affected by age. However, improvement of surgical techniques in experienced centres has resulted in mortality rates of ≈1%, 2%, and 4–5% for patients under 65, aged 65–75, and over 75 respectively [27]. Major surgery-related determinants of the operative risk include the surgical procedure used (i.e., valve repair *versus* replacement) and the need for concomitant CABG surgery [28].

Patients with advanced heart failure generally have a poor prognosis, particularly in the case of functional MR. In a recent study, 5-year survival rates in non-operated patients with ischaemic MR were 38% and 29% in the presence of ERO area < or >20 mm² respectively [29]. In a separate high-risk population with post-infarction ischaemic cardiomyopathy treated by surgical ventricular restoration (SVR) and mitral valve repair, 5-year survival was 62% [30]. The effectiveness of SVR, alone or in conjunction with coronary artery bypass grafting (CABG) in patients with ischaemic heart disease and anterior LV dysfunction is under evaluation by the Surgical Treatment for Ischemic Heart Failure (STICH) trial [31].

Management of asymptomatic severe MR

The management of asymptomatic patients with severe MR remains controversial. A study in asymptomatic patients with floppy valves reported improved late outcomes after mitral valve repair [32]. On the other hand, a series of 132 consecutive asymptomatic patients (age 55 ± 15 years, 49 female) with severe degenerative MR treated by a "watchful waiting" approach showed 6% mortality at 5 years [32]. Survival free of any indication for surgery was 92 ± 2% at 2 years, 78 ± 4% at 4 years, 65 ± 5% at 6 years, and 55 ± 6% at 8 years. These data suggest that, in general, asymptomatic patients with severe degenerative MR can be safely followed up until either symptoms occur or currently recommended cutoff values for LV size, LV function, or pulmonary hypertension are

Table 3

In-hospital morbidity and mortality for first time isolated mitral valve surgery (STS 2008).

Complication	Mitral valve repair	Mitral valve replacement
Death	1.1%	6.3%
Stroke	2.2%	3.8%
Renal failure	1.8%	6.9%
Prolonged ventilation	9.5%	22.9%
Perioperative MI	1.4%	1.7%
Reoperation		
– any	5.2%	11.3%
– bleeding	2.3%	5.1%
– other cardiac	0.8%	2.2%

Figure 6

Step-by-step percutaneous edge-to-edge repair (left panel: cartoon provided by the supplier; right panel: real-time 3D TEE); a) guide-wire introduced into the left atrium; b) clip delivery system deployed over the mitral orifice; c) arms of clip delivery system oriented perpendicularly to long axis of leaflet edge; d) clip delivery system anchored to mitral leaflets; e) final result (long axis view).
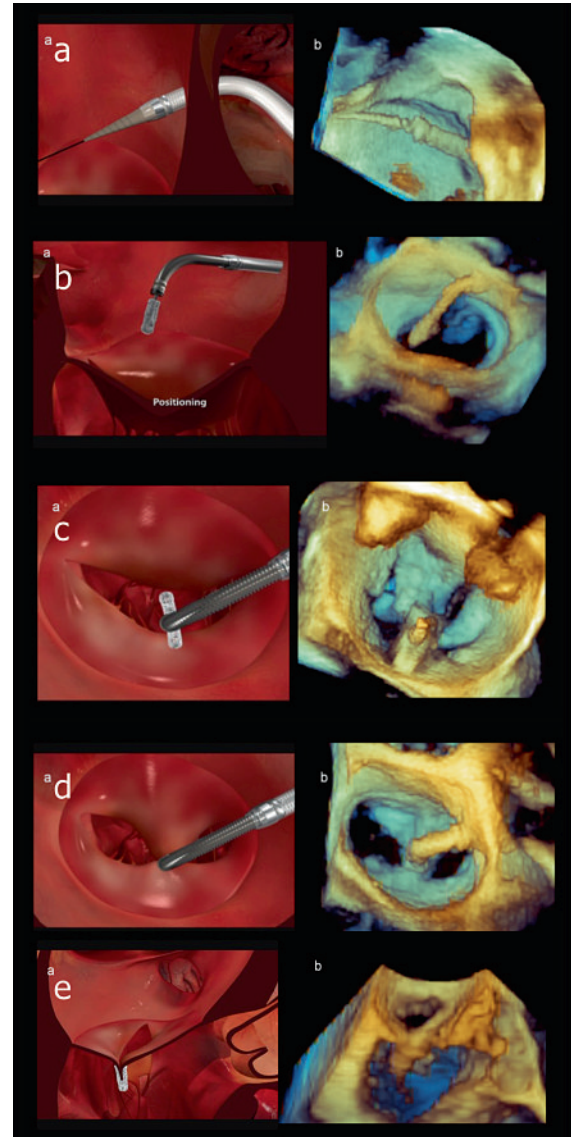
reached. However, this management requires careful follow-up.

In selected patients with advanced heart failure, Chronic resynchronisation therapy (CRT). attenuates MR, improves cardiac output and alleviates symptoms [34].

Percutaneous, catheter-based mitral valve repair includes leaflet (edge-to-edge) repair and mitral annular reduction. These approaches have not yet been approved for clinical use, but remain largely investigational.

Percutaneous edge-to-edge repair mimics the surgical procedure originally proposed by Alfieri [35]. It aims to create a tissue bridge between the anterior and the posterior leaflet by clip deployment via transseptal catheterisation (fig. 5 and 6). Preliminary data are encouraging, with ≈80% of patients showing mild or trace MR at hospital discharge [36]. Moreover, 12-month follow-up data from the High-Risk Registry Arm of the North American EVEREST II study suggest that high-risk patients with functional or degenerative MR may benefit substantially from mitral clips in terms of morbidity and mortality [37].

Percutaneous mitral annular reduction aims to reduce annular dilatation. This approach generally includes coronary sinus cinching. It has been shown to attenuate MR in animal models [38], but the clinical data are preliminary. Achieving a constraining force resulting in >20% diameter reduc-

tion is challenging from a technical standpoint. This procedure includes anchoring devices that are placed in the distal and proximal coronary sinus, and an intermediate tensioning or supporting element. A practical limitation is that these devices occupy the space where biventricular pacing leads are frequently positioned for CRT.

Conclusions

MR imposes volume overload on the left atrium and ventricle, eventually resulting in their progressive dilatation, atrial fibrillation, and heart failure. Pharmacological therapy provides limited symptomatic benefit. Timely mitral valve repair prevents development or further progression of LV dysfunction. Reverse LV remodelling after valve repair occurs in half of patients with functional MR. This approach is the treatment of choice for moderate to severe MR but is not feasible in patients with advanced degenerative abnormalities of the valve. Functional (secondary) MR results from regional LV dysfunction, most often in the context of ischaemic heart disease. Although

surgical mortality has declined in the past decade, long-term outcomes remain poor. The management of secondary MR includes pharmacological therapy of heart failure and CRT in selected patients. Surgical approaches to secondary MR need to be optimised. Ongoing clinical trials will probably provide new insights into the safety and efficacy of percutaneous mitral valve repair.

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