Mitral valve repair offers superior long-term survival, freedom from cardiac morbidity, and quality of life in patients with severe mitral regurgitation compared to medical management and mitral valve replacement [1–3]; it is the treatment of choice in these patients [4]. Mitral valve repair is the only treatment that restores normal life expectancy to otherwise healthy patients with advanced degenerative mitral valve disease [1]. Although several series report repair rates of 90% [13–17], repair rates in developed countries continue to average around 50% [8–10]. This review outlines current concepts in the pathophysiology, assessment, and repair of degenerative mitral regurgitation, focusing on the role of valve anatomy in guiding both surgical referral and operative approach: such a strategy will play an important role in increasing the rate of successful and durable repairs in degenerative mitral valve disease.

Introduction

Mitral valve repair offers superior long-term survival, freedom from cardiac morbidity, and quality of life in patients with severe mitral regurgitation compared to medical management and mitral valve replacement [1–3]; it is the treatment of choice in these patients [4]. Mitral valve repair is the only treatment that restores normal life expectancy to otherwise healthy patients with advanced degenerative mitral valve disease [1]. Although several series report repair rates of 90% [13–17], repair rates in developed countries continue to average around 50% [8–10]. This review outlines current concepts in the pathophysiology, assessment, and repair of degenerative mitral regurgitation, focusing on the role of valve anatomy in guiding both surgical referral and operative approach: such a strategy will play an important role in increasing the rate of successful and durable repairs in degenerative mitral valve disease.

Definitions and Carpentier’s Pathophysiologic Triad

Lack of universally agreed terminology is a major barrier to interpreting studies assessing surgical repair [11]. Terms such as “billowing”, “prolapse”, “papillary muscle”, “myxomatous disease”, “floppy valve”, “Barlow’s disease” and “fibroelastic degeneration” are used interchangeably, often without an effort to distinguish between aetiology and lesion, and failing to offer much insight into the valve dysfunction causing regurgitation [12]. Carpenter originally classified leaflet dysfunction into three types: Type I—normal leaflet motion, Type II—excessive leaflet motion, and Type III—restricted leaflet motion (Fig. 1) [13,14]. In Carpenter’s original classification Type III mitral regurgitation was described as “restricted leaflet motion...a condition in which a leaflet does not open normally during diastole”, and attributed to commissural fusion, leaflet thickening and chordal fusion or thickening [13]. Rheumatic valve disease is the commonest cause of these lesions. Following recognition of the role of ischaemic ventricular remodelling in the pathogenesis of mitral regurgitation, Type III mitral regurgitation was subsequently subdivided into Type IIIa resulting from restricted leaflet motion in diastole most commonly due to rheumatic valve disease; and Type IIIb caused by restricted leaflet motion in systole and most commonly due to papillary muscle displacement as a result of ischaemic ventricular dysfunction and dilatation [15]. He combined this classification with segmental valve anatomy (Fig. 2), to provide a system of nomenclature and valve analysis that is central to any real understanding of mitral valve repair. Type II leaflet dysfunction is by far the commonest finding in mitral regurgitation due to degenerative disease. Within the spectrum of degenerative disease, it is important to emphasise that patients may present with different aetiologies (e.g. Barlow’s or Fibroelastic Deficiency), multiple different lesions (e.g. chordal elongation or rupture or less commonly restriction, leaflet...
Table 1. Anatomical Lesions and Resultant Leaflet Dysfunction causing Mitral Regurgitation.

<table>
<thead>
<tr>
<th>Leaflet dysfunction</th>
<th>Anatomical lesion</th>
<th>Repair Techniques</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type I</td>
<td>Normal leaflet motion</td>
<td>Annular dilatation, Leaflet perforation</td>
</tr>
<tr>
<td></td>
<td>Leaflet prolapse</td>
<td>Chondal rupture, Chondal elongation, Papillary muscle rupture</td>
</tr>
<tr>
<td>Type II</td>
<td>A: Restricted leaflet opening</td>
<td>Commissural fusion, Leaflet thickening, Leaflet calcification, Chondal fusion</td>
</tr>
<tr>
<td></td>
<td>B: Restricted leaflet closure</td>
<td>Chondal thickening, Chondal shortening, Ventricular dilatation</td>
</tr>
</tbody>
</table>

Figure 1. Carpenter’s functional classification. Type I: normal leaflet motion. Type II: increased leaflet motion. Type IIIa: restricted leaflet motion during diastole and systole. Type IIIb: restricted leaflet motion during systole (modified from Ref. [42]).

Degenerative Mitral Regurgitation

Degenerative disease is defined as a spectrum of conditions in which infiltrative or dysplastic tissue changes prolapse, papillary or annular calcification, and annular dilatation), and multiple dysfunctions (Type I, Type II, and Type III), although the primary dysfunction is almost always type II (excess leaflet motion).

Definition of Degenerative Mitral Regurgitation

Degenerative disease is defined as a spectrum of conditions in which infiltrative or dysplastic tissue changes cause elongation or rupture of the mitral valve chordae resulting in leaflet prolapse and usually associated annular dilatation [16]. At one end of the spectrum of degenerative mitral disease is fibroelastic deficiency, characterised by insufficient tissue in a normal sized valve (Fig. 3): leaflets are thinned, almost transparent, and chordae are flimsy and thin. Regurgitation is most frequently caused by rupture of a single chord associated with a single prolapsing segment, usually P2, resulting in Type II leaflet dysfunction. The prolapsing segment may become distended and thickened by a localised myxomatous process in the chronic setting. Tissue resection must be approached with caution, as the surgeon runs the risk of leaving insufficient leaflet tissue to complete a successful repair. At the opposite end of the spectrum of degenerative mitral regurgitation is Barlow’s disease, characterised by marked excess tissue involving multiple leaflet segments in an otherwise large valve (Fig. 4). Leaflet tissue is thickened and redundant, with thick, elongated, mesh-like chordae which may or may not be ruptured [16]. Regurgitation is due to the multiple areas of prolapse (Type II leaflet dysfunction). Aggressive tissue resection and chordal reconstruction are mainstays of repair in these complex and challenging valves. Some regurgitant valves
Figure 3. Fibroelastic deficiency. An intra-operative photograph and drawings of typical valves, showing thinned leaflets in a small valve with single segment prolapse, which may or may not be thickened, and commonly used surgical techniques in valves without excess tissue. A: neochordae, B: chordal transfer, C: triangular resection.

Figure 4. Barlow’s disease. An intra-operative photograph and drawings of typical valves showing thickened leaflets with excess tissue resulting in multisegment prolapse in large valves. Commonly used surgical techniques in these valves are shown. A: Resection line for quadrangular resection and leaflet sliding-plasty. B: Vertical compression. C: Leaflet sliding plasty. D: The completed suture lines.

Table 2. Semi-quantitative and Quantitative Echocardiographic Grading of Mitral Regurgitation.

<table>
<thead>
<tr>
<th>MR Grade</th>
<th>Descriptive</th>
<th>Regurgitant Jet (% of LA Area)</th>
<th>Regurgitant Fraction</th>
<th>RVol (ml)</th>
<th>ERO (mm²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No-MR: 0</td>
<td></td>
<td>&lt;20%</td>
<td>&lt;30%</td>
<td>&lt;30</td>
<td>&lt;20</td>
</tr>
<tr>
<td>Trace: 1+</td>
<td>Small, central jet</td>
<td>20–40%</td>
<td>30–49%</td>
<td>30–59</td>
<td>20–39</td>
</tr>
<tr>
<td>Mild: 2+</td>
<td>Jet impinging on far wall of LA</td>
<td>&gt;40%</td>
<td>&gt;50%</td>
<td>≥60</td>
<td>≥40</td>
</tr>
</tbody>
</table>

occupy a position within the spectrum of fibroelastic defi-
ciency and Barlow’s disease, and we refer to these as Forme
Fruste of Barlow’s disease, as they show areas of excessive
and thickened tissue in somewhat smaller valves.

Assessment

Clinical Features
History and examination alone is of limited use to even
experienced clinicians in the diagnosis of mitral regur-
gitation [17], but the presence of symptoms or evidence of
atrial fibrillation, pulmonary hypertension or embolic
disease is of direct relevance to the decision of whether
and when to refer for surgery. Chronic mitral regurgi-
tation may be asymptomatic for many years. The most
common presenting symptoms are fatigue, decreased
exercise capacity, dyspnea and palpitations. It is important
to enquire directly about any history of atrial fibrilla-
tion, which may not be present at the time of clinical
examination. Pulmonary hypertension is suggested by the
presence of a parasternal heave on examination, and fluid
retention is a sign of cor pulmonale. In advanced cases
the patient may appear cachexic. Clinical features may be
helpful in distinguishing between the subtypes of degen-
erative mitral regurgitation: patients with Barlow’s disease
are typically younger, with a long history of a murmur,
compared to those with fibroelastic deficiency who are
usually older, with a relatively short history of mitral regur-
gitation.

Echocardiography
Echocardiography is diagnostic, although it must be
emphasised that mitral regurgitation is a dynamic con-
dition highly dependent on preload and afterload: under
conditions of reduced preload and afterload, such as
occurs under general anaesthesia during intra-operative
transoesophageal echocardiography, mitral regurgitation
is frequently downgraded [18], and stress echocardiog-
raphy may be the only means of detecting severe mitral
regurgitation in some patients. This is particularly true in
the setting of chordal elongation and mid-systolic mitral
regurgitation. When mitral regurgitation appears insignif-
icant on pre-bypass intra-operative echocardiography it
may be helpful to administer vasopressors and volume
to recreate physiological conditions in the awake patient,
and to review the pre-operative trans-thoracic echo-
cardiogram.

Two-dimensional echocardiography with colour
Doppler permits accurate determination of the leaflet
dysfunction, and semi-quantitative assessment of the
severity of mitral regurgitation by maximal jet length and
area, the ratio of jet to left atrial area, and vena contracta
mapping (Table 2). Regurgitant jet geometry is assessed
in multiple views and mitral regurgitation is graded on
a scale of 0 to 4+, where 1+ is trace mitral regurgitation
and 4+ severe mitral regurgitation. Definitions of the
intermediate grades vary between echocardiographers,
and patients classified by one practitioner as 3+ mitral
regurgitation may be re-classified as severe (4+) by
another. Colour Doppler jet area is affected by jet eccen-
tricity, user and instrumentation factors, and left atrial
size [19], potentially leading to variation in estimates of
the severity of mitral regurgitation [20].

Quantitative Doppler grading of mitral regurgitation
seeks to standardise assessment of mitral regurgitation.
Calculation of regurgitant volume using proximal iso-
electric surface area (PISA), vena contracta width and
effective regurgitant orifice (ERO), are essential adjuncts
in a detailed assessment of mitral regurgitation. An
ERO > 40 mm² is accepted as the cut-off for severe mitral
regurgitation in the setting of degenerative disease [21].

Reproducible and reliable echocardiographic assessment
of the severity of mitral regurgitation, however, remains
challenging and there is no single gold standard measure-
ment to quantify the degree of regurgitation [22].

Real-time three-dimensional echocardiography is a
newer modality that has already yielded insights into
mechanisms of mitral regurgitation such as the differ-
ences in between leaflet tenting, tethering and stresses
in anterior infarction in which tenting is more symmet-
ic, compared to posterior infarction where these changes
tend to occur in the P3 region [23]. Three-dimensional
echocardiography provides a surgical view of the mitral
valve where even subtle areas of prolapse or restriction
may be immediately identified [16].

Natural Life History
The average interval from diagnosis of mitral valve pro-
lapse to the onset of symptoms is approximately 15
years [24], but left ventricular dysfunction frequently
occurs before symptoms develop. In patients with severe
mitral regurgitation estimates of long-term survival vary
widely, with five-year survivals quoted between 30 and
95% [24,25], reflecting the very heterogeneous population
included in most series. A review of 348 consecutive
patients with chronic mitral regurgitation due to flail
leaflet seen at the Mayo Clinic between 1980 and 1989
showed that the incidence of surgery or death at 10 years
was 90%, with excess mortality and morbidity in medi-
cally treated patients [26]. While most deaths are related
to heart failure, ventricular arrhythmias have been impli-
cated as an important cause of sudden death, particularly
(although not exclusively) in symptomatic patients and
those with documented left ventricular dysfunction [27].

Indications for Surgery
Factors determining timing of surgery for isolated mitral
regurgitation in the current American College of Car-
diology and American Heart Association (ACC/AHA)
guidelines include symptoms, left ventricular ejection
fraction, left ventricular end systolic dimension, atrial fib-
rillation, and pulmonary hypertension [28]. While there is
a consensus that patients in whom mitral repair is feasible
should undergo surgery before evidence of left ventricu-
lar decompensation, the poorer outcomes associated with
mitral replacement mean that asymptomatic patients with
severe mitral regurgitation but preserved left ventricular
Left Ventricular Ejection Fraction

The strongest predictor of outcome after surgery for chronic mitral regurgitation is the pre-operative left ventricular ejection fraction [29,30]. Despite the fact that the presence of mitral regurgitation means left ventricular dysfunction [29], severe symptoms of congestive heart failure, whether transient or persistent, almost invariably, therefore, indicate the need for surgical intervention, and a cogent case has been made for operating earlier in less symptomatic patients [29]. Stress testing may be required to demonstrate true NYHA status in a limited subset of apparently asymptomatic patients [28]. Current ACC/AHA guidelines recommend that asymptomatic and NYHA class I and II patients with severe mitral regurgitation should be referred for surgery based on deterioration in left ventricular function (class I), or atrial fibrillation or raised pulmonary artery pressures (class IIa) (see below).

Elevated Pulmonary Artery Pressures

Pulmonary artery hypertension is associated with significant increase in post-operative early mortality, poorer functional status and reduced term survival [38]. A marker of severe left ventricular diastolic dysfunction and of the severity of mitral regurgitation [39], it is associated with poorer post-operative indices of left ventricular function [30]. ACC/AHA guidelines recommend consideration of surgery in patients with severe mitral regurgitation and atrial fibrillation irrespective of left ventricular function and symptomatic status (class IIa). Surgical ablation is now a standard adjunct to mitral repair as it increases the number of patients that are in sinus rhythm post-operatively, as well as freedom from anti-coagulation and its associated morbidity and mortality [35–37].

Watchful Waiting Versus Early Surgery

The contrast between the substantial morbidity and mortality associated with chronic severe mitral regurgitation and the excellent outcomes associated with mitral valve repair in degenerative disease has led experienced groups to adopt a policy of early intervention in patients with degenerative disease, before onset of symptoms, atrial fibrillation, which occurs in almost a third of medically treated patients with mitral valve regurgitation [26], carries significant excess morbidity and mortality. Chronic atrial fibrillation occurs almost exclusively in patients with a left atrial diameter > 40 mm, and is less likely than recent onset or intermittent atrial fibrillation to be successfully converted to sinus rhythm by surgery [34]. For this reason ACC/AHA guidelines recommend consideration of surgery in patients with severe mitral regurgitation and atrial fibrillation irrespective of left ventricular function and symptomatic status (class IIa). Surgical ablation is now a standard adjunct to mitral repair as it increases the number of patients that are in sinus rhythm post-operatively, as well as freedom from anti-coagulation and its associated morbidity and mortality [35–37].

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Left Ventricular End Systolic Diameter

Left ventricular end systolic diameter and left ventricular end systolic volume index are simple, reproducible measures of left ventricular function that can be obtained non-invasively, and have the advantage that they are relatively independent of haemodynamic status. Pre-operative left ventricular end systolic diameter of 45 mm represents the value above which patients are highly likely to have abnormal post-operative left ventricular function [33]. Pre-operative left ventricular end systolic volume index >50 ml/m² predictive of persistent post-operative left ventricular dilatation [30]. ACC/AHA guidelines recommend surgery for patients with severe mitral regurgitation and left ventricular end systolic diameter >40 mm, irrespective of symptom status (class I) [28].

Symptoms

The annual mortality of medically managed patients in New York Heart Association (NYHA) functional class III or IV, even if the patient only transiently deteriorated into one of these classes, was shown in one study to be as high as 34% [26]. NYHA functional class is also an independent predictor of post-operative mortality and left ventricular dysfunction [30]. Severe symptoms of congestive heart failure, whether transient or persistent, almost invariably, therefore, indicate the need for surgical intervention, and a cogent case has been made for operating earlier in less symptomatic patients [29]. Stress testing may be required to demonstrate true NYHA status in a limited subset of apparently asymptomatic patients [28]. Current ACC/AHA guidelines recommend that asymptomatic and NYHA class I and II patients with severe mitral regurgitation should be referred for surgery based on deterioration in left ventricular function (class I), or atrial fibrillation or raised pulmonary artery pressures (class IIa) (see below).
fibrillation, left ventricular dysfunction or pulmonary hypertension. This approach is supported by a prospective observational study of over 400 patients with severe degenerative mitral regurgitation which identified an effective regurgitant orifice area of >40 mm² as the single most important predictor of adverse outcome during follow-up [1]. The conclusion of Enríquez-Sarano and colleagues was that these patients should “promptly be considered for cardiac surgery, since surgery considerably reduces the rate of death from cardiac causes...and normalises life expectancy”. The results largely of this study lead the ACC/AHA Guidelines to recommend strong consideration of surgery in asymptomatic patients with normal ventricular function and severe chronic mitral regurgitation as long as mitral valve repair was highly likely (class IIa).

Shortly after this paper was published, a smaller observational study including 132 patients with severe mitral valve regurgitation was reported which appeared to contradict this approach [40]. Rosenhek et al. found that over half of patients with severe mitral regurgitation survived for eight years before developing accepted indications for surgery, with overall survival not significantly different to expected survival with this approach [40]. This led the European Guideline committee to down grade the indication for surgery in these patients to class IIb.

There are several potential reasons for the differences in findings between these two papers. The primary reason is probably the difference in aetiology in the patient cohorts. Enríquez-Sarano’s group had a mean age of 63 years, with less than 10% of those undergoing surgery before developing symptoms, atrial fibrillation, left ventricular dysfunction or pulmonary hypertension, and watchful waiting is therefore perhaps a more appropriate strategy in this group. In contrast patients with fibroelastic deficiency. In contrast Rosenhek’s patients had a mean age of 58 years, and 17% of those undergoing surgery received a valve replacement, which is more suggestive of Barlow’s disease. Patients with Barlow’s disease are more likely to experience a long period of event free survival before developing symptoms, atrial fibrillation, ventricular dysfunction or pulmonary hypertension, and watchful waiting is therefore perhaps a more appropriate strategy in this group. In contrast patients with fibroelastic deficiency are more likely to experience adverse events early during follow-up (and more likely to undergo successful valve repair) and a strategy of early mitral valve repair is certainly reasonable.

Surgical Techniques

Carpentier described three basic tenets of mitral valve repair over 30 years ago, which remain fundamental to successful repair today: preserve leaflet mobility, restore a large coaptation surface, and remodel the annulus [13]. The nature and combination of repair techniques is dictated by the lesions identified in a comprehensive evaluation that begins with the pre-operative and intra-operative echocardiograms, and is completed by systematic surgical examination of the mitral apparatus to confirm the lesions and dysfunctions. The endocardium of the left atrium is inspected first for jet lesions that indicate contralateral leaflet prolapse or ipsilateral leaflet restriction. The annulus is then assessed for the presence of annular dilation and calcification. Segmental valve analysis is then carried out with reference to a non-prolapsing segment, normally the anterior paracommissural scallop of the posterior leaflet (P1), to locate and quantify leaflet prolapse and restriction, assesses pliability, and identify ruptured and elongated chordae and papillary muscles, as well as areas of leaflet tethering. Finally, inserting saline into the left ventricle, allows direct visualisation of the location, size and direction of the regurgitant jets, complete the valve analysis by confirming echocardiographic and operative findings.

Leaflets

It is crucial to distinguish between Type II lesions characterised by excess leaflet tissue and those with insufficient leaflet tissue: leaflet prolapse in valves with limited leaflet tissue is best addressed with very conservative (e.g. triangular) resection or chordal reconstruction, rather than aggressive resection which is the technique of choice for Type II lesions with excess tissue (Fig. 3). Posterior leaflet prolapse may be treated by no resection (neo chordae (Fig. 3A) or chordal transfer (Fig. 3B)), or triangular resection of the prolapsing segments or possible limited quadrangular resection and concomitant annular plication (Fig. 3C). In contrast, Barlow’s disease is managed by removing excess tissue including correcting the height of retained segments by resection and sliding plasty (Fig. 4A-D). Quadrangular resection with leaflet detachment and repositioning into the true atrioventricular groove is the mainstay of Carpentier’s approach to this disease (Fig. 4). More recently some have advocated a non-resection approach, but using neo chordae to pull the leaflet into the ventricle still effectively “removes” leaflet tissue from the valve orifice.

As anterior leaflet prolapse is less often characterised by excess tissue, a small triangular resection of the prolapsing segment or simple reuspension should be performed to preserve as much of the body of the leaflet as possible.

Chordae and Papillary Muscles

Chordal techniques may be applied to flail segments caused by ruptured chordae, particularly of either leaflet when there is insufficient tissue to permit leaflet resection; or to correct leaflet restriction caused by chordal shortening or ventricular dilation [13,41,42]. Where there is a flail segment the two main options are chordal reconstruction with polytetrafluoroethylene (PTFE) [43] (Fig. 3A) or chordal transfer [41] (Fig. 3B). Intermittently testing valve competency with normal saline as the neo-chord is adjusted is one method of ensuring the proper length to correct prolapse [12]. An alternative is selecting pre-formed PTFE loops based on measurement of distance between a reference leaflet margin point to the base of the nearest papillary muscle with calipers [44]. Chordal transfer (Fig. 3B) involves the selection of a normal looking nearby secondary chord, or adjacent chord from the opposite leaflet [45]. The chord is detached from its insertion on its leaflet, and reattached to the margin of the anterior
leaflet with 5/0 prolene. Although this is a reliable repair method using normal tissue, the technique is limited by the number of normal chordae available in valves affected by extensive disease.

Leaflet restriction, which is sometimes found primarily involving the anterior papillary muscle and the P1 leaflet segment in advanced cases of Barlow’s valve disease requires leaflet mobilisation as the primary treatment. An alternative is to use commissural sutures to advance the commissure in particular large valves.

Annulus

Annuloplasty techniques are a key component of mitral repair. The normal ratio between the antero-posterior and transverse diameter of the mitral annulus is 3:4 in systole (Fig. 6). This ratio inverts in patients with chronic mitral regurgitation, causing poor leaflet coaptation and regurgitation even in the absence of prolapse. The anterior aspect of the annulus is supported by the fibrous aortic mitral curtain and is thus less affected by increasing annular circumference versus the posterior aspect of the valve. The objective of remodelling annuloplasty, which was introduced in the 1970s by Carpentier, is to address this, restoring the mitral annulus to its systolic 3:4 ratio, preventing further annular dilatation, preserving leaflet mobility, relieving tension on the leaflets helping to stabilise the repair, and increasing the area of coaptation minimising mitral regurgitation [42]. The Carpentier-Edwards Classic ring (Edwards LifeSciences, Irvine, CA) is a rigid two-dimensional ring that, like the more modern semi-rigid Physio ring (Edwards LifeSciences, Irvine, CA), is designed to restore the 3:4 systolic ratio. A variety of flexible and rigid, incomplete and complete rings are widely used today. Duran originally proposed a totally flexible ring with the aim of preserving the three-dimensional saddle-shape and non-planar mobility that, under normal conditions, the annulus displays throughout the cardiac cycle [46]. Although there is some data suggesting better early left ventricular function with a flexible rather than a rigid ring, this advantage has only been shown to be present in the first few weeks following repair [47]. All of these rings are optimally sized by passing a nerve hook around the A2 marginal chords to fully extend the anterior leaflet, which may then be compared to the appropriate valve sizer.

Testing the Valve

In addition to the saline test, and prior to post-operative echocardiography an ink-test is useful to demonstrate the coaptation area [48]. The ventricle is filled with saline until the mitral leaflets are distended in the systolic position. Using tip of a gentian surgical marker (Codman, Raynham, Mass) held in a clamp, a line drawn along the closure line, which should be symmetrical and closer and parallel to the posterior annulus. The saline is then aspirated and the leaflet margins inspected: a coaptation depth of 4–10 mm suggests that the repair will be satisfactory and durable. Coaptation depth greater than 10 mm suggests the valve may be predisposed to abnormal systolic anterior motion, and this should be addressed by further reducing the leaflet height. Coaptation depth less than 3 mm may be potentially addressed by resecting restrictive secondary chordae, cleft closure, commissural sutures, or down-sizing the annuloplasty ring.

Outcomes

There is a particular need to minimise adverse outcomes in this population, as patients are frequently young, and increasingly often asymptomatic. Operative mortality is an important outcome measure, but additional performance indicators such as morbidity, long-term survival and quality of life; and improvement in left ventricular function, repair and failure rates must therefore also be considered when attempting to compare outcomes between techniques and surgeons. There are several problems associated with measuring outcomes in mitral valve repair [11].

Problems in Outcome Measurement

Accurate comparison of outcomes between techniques and surgeons presents several challenges. The lack of universally agreed nomenclature means that most series consist of a heterogeneous population of patients, aetiology and lesions, making it difficult to draw conclusions about individual techniques and specific lesions. The absence of a uniform approach to assessment and repair of the mitral valve amplifies this problem, as lesions may be unrecognized or ignored, or repaired suboptimally. Lack of echocardiographic data is a significant challenge. Even benchmark centers in outcomes measure-
Morbidity and Quality of Life

Complication rates are low for isolated mitral repair. Major neurological events occur in approximately 1% of patients [53], and freedom from cardiac events is almost 75% at 20 years. Mitral valve repair reduces the likelihood of the major complications associated with mitral valve replacement including those of long-term anticoagulation for mechanical prostheses, thromboembolism (stroke), bleeding, and prosthetic valve endocarditis. Current evidence from medically treated and surgically treated cohorts suggests that optimal mitral valve repair yields better freedom from cardiac events than medical management in patients with severe mitral regurgitation [1]. There is only one study including an objective assessment of quality of life following mitral repair; the evidence suggests it is similar to the general population [54].

Failure and Re-operation

Mitral valve repair failure rates, defined by recurrence of moderate or severe mitral regurgitation, or re-operation for mitral regurgitation are determined by the aetiology, lesion and repair technique(s). Most early failures are the result of technical issues related to the repair. Common reasons for an early failure include tissue disruption due to excess tension, residual uncorrected prolapse or restriction, or a less than optimal annuloplasty. The presence of residual mitral regurgitation greater than mild is a strong predictor of repair failure. Late failures primarily relate to progression of disease with new leaflet prolapse, but can also result from leaflet scarring and retraction, or infection.

A re-operation is actually uncommon after degenerative mitral valve repair, but recent studies have highlighted an approximate 1–2% per year risk of recurrence of moderate or severe mitral regurgitation in patients operated on in the past decade or longer, and the risk appears to be higher in patients with anterior or bi-leaflet prolapse [55,56]. What remains unclear is if continued refinement of mitral repair understanding and strategies will lower this rate in future studies.

LV Function

Current evidence from medically treated and surgically treated cohorts suggests that optimal mitral valve repair is associated with better left ventricular function both immediately post-operatively and in the long term than mitral valve replacement or medical management in patients with severe mitral regurgitation [51]. Pre-operative echocardiographic left ventricular ejection fraction has been shown to be the strongest independent predictor of both post-operative left ventricular ejection fraction and late survival [30,33]. This is the case irrespective of whether repair or replacement is performed, but valve replacement is an independent predictor of poor post-operative left ventricular function [30]. Preserved contractile reserve on exercise testing is associated with much better long-term outcome even in the setting of uniform mitral valve repair.

Who Should Operate?

Mitral valve repair is the treatment of choice for degenerative mitral regurgitation, because of the outcome benefits described above. Particularly in young or minimally symptomatic patients undergoing mitral valve repair for distant benefit, the surgery must be performed firstly with very low morbidity and mortality, and secondly with the very high likelihood of a successful and durable repair. Although repair rates are over 90% at some centres, American and European repair rates are less than 60% [9,53] despite clear guidelines advocating repair whatever feasible. Although referral bias may contribute, this wide variation and the overall disproportionately high replacement rate, primarily reflects that fact that mitral valve repair is highly dependent on mitral pathology and relevant surgeon experience [57].

Mortality

The operative mortality for mitral valve repair is low. The Society of Thoracic Surgery Database, which analysed over 15,000 isolated primary mitral valve operations between 2001 and 2004, reports mortality figures of 1–2% for centres submitting data on mitral valve repair, compared to 4–10% for mitral valve replacement [8]. Gammie et al. amplified the impact of volume on mortality further, noting a 3% operative mortality in low volume centres compared to a 1% operative mortality in high volume centres [50].

Long-term Survival

Current data on long-term survival reflects practice 10–20 years ago. Incremental change in timing of intervention, pre-operative assessment, myocardial protection, repair techniques, and peri-operative management appear to have improved early mortality, and parameters of left ventricular function, and can therefore reasonably be expected to contribute to improved long-term survival [12]. Current data suggests that if mitral valve repair is carried out before onset of symptoms and deterioration in ventricular function, the long-term survival is similar to that of the general population [32]. Conversely impaired left ventricular function or NYHA III-IV symptoms at time of surgery is associated with greatly reduced long-term survival [32]. Mitral valve replacement has been shown to be an independent predictor of mortality in patients undergoing mitral valve surgery at 5 and 10 years post-operatively [51]. Current evidence from medically treated and surgically treated cohorts suggests that optimal mitral valve repair yields better survival than medical management in patients with severe mitral regurgitation [1,52].
The echocardiogram must be considered, not only in the operative plan, but also in the initial surgical referral, and the repair rate and outcomes of an individual surgeon for the specific echocardiographic lesions and dysfunction should be taken into account on a case-by-case basis. Echocardiographic assessment is key in matching surgical expertise to valve complexity. Multisegment disease with excess tissue is increasingly seen as the preserve of surgeons with extensive experience in this specific aspect of degenerative disease, who are most likely to achieve successful and durable repair of this technically very challenging lesions [58]. In contrast the probability of successful repair of an isolated P2 prolapse secondary to chordal rupture due to fibroelastic deficiency, would be high in the hands of most cardiothoracic surgeons. This relationship between specific surgical experience and successful mitral repair is the rationale behind recent proposals to limit mitral repair to accredited surgeons with not only additional specialisation in mitral repair, but a demonstrated case-load above predetermined minimum limits [53,59].

Summary

Mitral valve repair is the treatment of choice for severe mitral regurgitation. It offers superior long-term survival, freedom from cardiac events and quality of life to mitral valve replacement, and medical management. As such a result increasing numbers of minimally symptomatic patients with preserved left ventricular function are being referred for mitral valve repair: long term outcomes in patients with preserved left ventricular function are being reported. The echocardiogram must be considered, not only in the valve surgery the "French concept" [20].

References


State of the Art: Degenerative Mitral Valve Disease


