Mitral valve surgery was first attempted in the early 1920s for repair of mitral stenosis. There followed an increasing trend towards mitral valve replacement in the 1960s with the introduction of mechanical valves and homografts. More recently, however, with growing knowledge of the sophisticated functioning and interaction of the entire mitral valvular apparatus, there has been a gradual realization that repair rather than replacement of the mitral valve may be a more ideal operation in order to preserve some of its more complex functions.\(^1\) The increased use of perioperative transoesophageal echocardiography (TOE) now allows the anaesthetist to provide information on the aetiology, severity and likely natural history of the mitral valve lesions. This, along with the greater reliance of the surgeon on the anaesthetist to assist with the surgical decision-making process during mitral valve surgery, including gauging adequacy of repair, has encouraged anaesthetists to gain a greater understanding of mitral valve anatomy and function.

### Anatomy of the mitral valve

The mitral valve can be thought of as having five separate components, namely the mitral valve annulus, valve leaflets, chordae tendinae, papillary muscles and underlying left ventricular wall.\(^2\) All of these components work together to form both an anatomical and functional unit. The normal mitral valve area is 4–6 cm\(^2\). It forms the major component of the left ventricular inflow tract and therefore plays a vital role in different aspects of left ventricular performance.

The valve itself consists of two leaflets, which are asymmetrical (Fig. 1). The anterior leaflet makes up about two-thirds of the valve area but only one-third of the circumference. The posterior leaflet inserts into two thirds of the annulus and consists of three main scallops (P1, P2 and P3). The anterior leaflet is not scalloped but the portions of the leaflet adjacent to P1, P2 and P3 are referred to as A1, A2 and A3, respectively. The leaflets join at the anterolateral and posteromedial commissures. The anterior leaflet of the mitral valve and the non-coronary cusp of the aortic valve share the same fibrous attachment. The leaflets and annulus make up the valvular apparatus and the chordae tendinae and papillary muscles make up the tensor apparatus. The chordae have a primary attachment to the free margin of the valve leaflets with secondary and tertiary attachments away from the free margin. The anterolateral and posteromedial papillary muscles supply both leaflets.

### Mitral stenosis

Mitral stenosis (MS) is defined as a valve area of <2 cm\(^2\) and is considered severe when the valve area is <1 cm\(^2\). Most cases are caused by rheumatic valve disease, which can lead to inflammation of the valve leaflets resulting in leaflet thickening and fusion of the commissures. There may be elements of both stenosis and regurgitation present.

In patients with suitable valvular anatomy, percutaneous transeptal balloon valvuloplasty has become the treatment of choice for mitral stenosis, delaying the need for surgery. It may also be of particular value in those patients who are considered high risk for surgical intervention such as patients who are pregnant, or have coexisting pulmonary or renal disease. The procedure is considered successful if the valve area increases to >1.5 cm\(^2\) without a substantial increase in mitral regurgitation and results in significant symptomatic improvement. Significant mitral regurgitation and the presence of left atrial thrombus are relative contraindications.\(^3\) It is a palliative procedure and commissural stenosis commonly recurs, necessitating another percutaneous intervention or surgery.

### Mitral regurgitation

Since the vast majority of mitral valve surgery in the UK is now indicated mostly for pure or predominant mitral regurgitation, the...
The remainder of this article will concentrate solely on surgery for correction of mitral regurgitation.

Aetiology

Mitral regurgitation may be thought of as being either primary or secondary. The causes of primary mitral regurgitation include myxomatous degeneration of the mitral valve, rheumatic disease, endocarditis and mitral valve prolapse. Secondary mitral valve disease may be due to ischaemia affecting the papillary muscles and left ventricle or a dilated cardiomyopathy.

Diagnosis

Chronic mitral regurgitation may remain asymptomatic for a prolonged period and the detection of a pansystolic murmur at the apex radiating to the axilla in an asymptomatic patient may be the first indication of mitral valve disease. In others, the diagnosis may be made only after the onset of atrial fibrillation or the symptoms of congestive cardiac failure. It may be several years before the onset of any symptoms, depending on the aetiology and severity of the disease process.

However, there are two major concerns in patients with asymptomatic primary mitral valve disease. First, these patients are at risk of sudden death (which is responsible for approximately a quarter of the deaths occurring under medical treatment). A study of 348 patients with mitral regurgitation showed that sudden death occurred at a rate of 1.8% yr\(^{-1}\). This would suggest that surgery should be considered early in the course of the disease.

Second, these patients are at risk of irreversible left ventricular dysfunction. The left ventricle becomes dilated as a result of the volume load which chronic mitral regurgitation imposes on it. Despite the usefulness of clinical and standard left ventricular and left atrial markers, none of these parameters precludes the possibility of incipient left ventricular dysfunction. Notably, symptoms may not precede deterioration of left ventricular function. LVEF is typically initially maintained despite the regurgitation into the left atrium during systole and may even remain in the normal range when function is impaired. This is attributable to the altered loading conditions imposed by the regurgitant lesion itself, i.e. a significant proportion of blood ejected from the left ventricle during systole in mitral regurgitation is offloaded ‘upstream’ into the lower pressure system of the left atrium rather than being ejected into the higher pressure system of the aorta. The fractional area change on echocardiography may therefore appear normal even if there is a reduced stroke volume owing to ventricular dysfunction. However, preoperative ejection fraction and end-systolic dimension have been found to be significant predictors of the postoperative left ventricular dysfunction in these patients. Therefore, either an ejection fraction <60% or an end-systolic diameter ≥45 mm are considered as demonstrating overt left ventricular dysfunction and these patients should be immediately considered for surgery. However, even patients with ejection fractions >60% have a notable long-term mortality when they are followed-up medically. Thus, it is essential to assess these patients at regular intervals with echocardiography to assist in the early detection of worsening mitral regurgitation and left ventricular dysfunction.

Other adverse consequences of chronic mitral regurgitation include left atrial enlargement, atrial fibrillation and pulmonary hypertension.

Secondary mitral regurgitation is most often diagnosed coincidentally during evaluation of the primary disease process. Treatment of this, such as coronary artery bypass grafting (CABG) or stenting for ischaemia, may improve the severity of the regurgitation.

Surgery for mitral regurgitation

The surgical options for treatment of mitral regurgitation include mitral valve replacement, with or without chordal preservation, and mitral valve repair.

Mitral valve repair

Mitral valve repair offers several advantages including the avoidance of long-term anticoagulation; it also allows the preservation of the continuity between the mitral annulus and papillary muscles. There are ample data demonstrating that preservation of the structural integrity of the mitral valve is essential for the
maintenance of normal left ventricular geometry and systolic function; it allows ejection fraction to remain stable or even improve postoperatively. A large study comparing short- and long-term outcome after mitral repair and replacement for mitral regurgitation showed that, adjusting for all differences, repair was associated with a lower operative mortality, better long-term survival, and better postoperative left ventricular function. Therefore, repair should be the preferred mode of surgical correction of mitral regurgitation.8

### Mitral valve replacement

When a repair is not feasible, the next option is a valve replacement with chordal preservation. A mechanical mitral valve is used when possible because these tend to last longer than tissue valves in the mitral position and because many of these patients will require long-term anticoagulation for chronic atrial fibrillation anyway. However, stented tissue valves can be used when anticoagulation is contraindicated.

### Perioperative haemodynamic management

The aim of the anaesthetist prior to repair of the mitral valve should be to reduce the regurgitant fraction by keeping systemic vascular resistance low, by maintaining a moderate tachycardia (or at least avoiding bradycardia) so that systolic duration is shortened (this reduces the regurgitant volume per beat) and optimising preload to ensure forward flow across the valve. Many of these patients will have lost the atrial contribution to ventricular filling in diastole because of atrial fibrillation and therefore they will require a higher heart rate to maintain cardiac output.

Predicting the need for inotropic support after cardiopulmonary bypass (CPB) is difficult as left ventricular dysfunction commonly occurs because of reperfusion injury (myocardial ‘stunning’). This contractile dysfunction may occur in patients with normal preoperative left ventricular function and typically peaks 4–6 h after surgery, usually resolving 24 h after surgery. A recent retrospective study by McKinley and colleagues, involving over 1000 patients, looked at predictors of inotropic use during separation from CPB.9 This study identified six independent predictors of inotropic usage:

1. ventricular wall motion abnormalities on TOE;
2. combined mitral valve and CABG surgery;
3. LVEF < 35%;
4. re-operation;
5. moderate to severe mitral regurgitation; and
6. aortic cross-clamp time.

It is our practice to site a pulmonary artery catheter (PAC) in anticipation of inotropic requirement in high risk mitral valve surgery patients, i.e. when there is impaired left ventricular function, severe pulmonary hypertension, a major co-morbidity such as renal impairment, combined CABG surgery and in redo-surgery cases. The use of a PAC allows rationalization of inotropes and fluids. The information gained from intraoperative TOE is also valuable in this context. However, PACs allow continuous monitoring of cardiac output and derived indices such as systemic vascular resistance and pulmonary vascular resistance and can prove invaluable in the haemodynamic management of these patients, both in the post-bypass period in theatre and during their postoperative intensive care treatment when TOE may not be available.

If an inotropic agent is required, the choice is between catecholamine β-agonists and phosphodiesterase (PDE) inhibitors. Traditionally, β-agonists such as epinephrine have been used but these are associated with arrhythmias, increased myocardial oxygen consumption and peripheral vasoconstriction. Inodilators such as milrinone (PDE type-III inhibitor) may be preferred. Milrinone bypasses the β-receptor (down-regulated in chronic heart failure patients) to increase cAMP concentration and improve myocardial contractility and diastolic relaxation.10 The vasodilator effects of milrinone reduce afterload and pulmonary vascular resistance (elevated in patients with pulmonary hypertension associated with mitral valve disease). Milrinone is loaded into the CPB circuit after removal of the aortic cross-clamp at a dose of 50 μg kg⁻¹. Any subsequent infusion is at a rate of 0.375–0.75 μg kg⁻¹ min⁻¹. However, even with high-risk patients, a loading dose may be all that is required as the half-life of milrinone is >2 h. If a continuous infusion is required, there will be an inevitable need for a vasonstrictor infusion to counter the vasodilator effects of milrinone.

### The role of TOE in mitral valve repair

Intraoperative TOE is a mandatory requirement for mitral valve repair. Although this is not the case for mitral valve replacement, surgeons increasingly request TOE monitoring for this operation. The mitral valve is ideally suited for examination with TOE as it lies in close proximity to the oesophagus and is separated from the probe by a blood filled left atrium which acts as an excellent acoustic window (Fig. 2). It is important that valve repair/replacements are assessed by TOE in theatre as there is the potential for the surgical result to be suboptimal. If this is the case, prompt TOE evaluation post-bypass before protamine administration means that cardiopulmonary bypass can be re instituted easily and surgical correction performed. This is preferable to the discovery of a problem postoperatively that may require re-operation. There is a large body of evidence to support the positive impact of TOE on valvular surgery. Pre-bypass TOE provides new information or initiates changes in valve surgery in 9–13% of cases and post-bypass TOE identifies significant valve dysfunction in 6–11% of cases, prompting second pump runs in 3–10% of cases undergoing mitral valve repair.11

It is important for the anaesthetist to have a systematic approach to examining the mitral valve with TOE. By changing rotation of the two dimensional plane of interrogation, the mitral valve can be viewed in many different slices. This enables a detailed...
Mitral valve surgery

Fig. 2 Intraoperative two-dimensional TOE image showing prolapse of the P2 portion (arrow A) of the posterior mitral valve leaflet, left atrium (B), A2 portion of the anterior mitral valve leaflet (arrow C), aortic valve (D), left ventricle (E), interventricular septum (F) and right ventricle (G).

delineation of the anatomy of all parts of the valve and provides information on aetiology (myxomatous degeneration, ventricular dysfunction, endocarditis, rheumatic disease), the mechanism of regurgitation (prolapse, flail, restriction, perforation) and the location and extent of the lesion. Pre-bypass TOE examination focuses on the severity of regurgitation and whether the valve is repairable. Severity is assessed using colour flow Doppler to gauge the size of the regurgitant jet. It is important to realize that the reduced loading conditions associated with anaesthesia can lead to underestimation of severity. The anaesthetist must therefore replicate the patient’s normal pre-operative blood pressure to ensure accurate assessment. It is not only the size, but also the direction, of the colour jet that yields useful information. An anteriorly directed eccentric jet is associated with posterior valve leaflet prolapse and conversely a posteriorly directed eccentric jet is associated with anterior leaflet prolapse. Methods other than Colour Doppler can be used to assess severity of regurgitation such as analysis of left atrial size and the pattern of flow in the pulmonary veins using pulsed wave Doppler (systolic flow reversal back up the pulmonary veins being associated with severe regurgitation). Feasibility of repair depends upon the thickness and redundancy of the valve leaflets, which leaflet is involved (anterior leaflet repairs are more difficult than posterior), which scallop is involved (P2 prolapse is the commonest and most amenable to repair), whether there is associated chordal rupture and the expertise of the surgeon.

Pre-bypass assessment of left ventricular function and estimation of systolic pulmonary pressure (using pulsed wave Doppler analysis of any tricuspid regurgitation) also helps the anaesthetist to plan any post-bypass support. Post-bypass TOE examination focuses on the adequacy of repair. Again, it is important to match loading conditions to the baseline pre-bypass examination in order not to underestimate any residual regurgitation. An acceptable repair has no or minimal regurgitation. More severe residual regurgitation requires a second bypass run to re-repair or replace the valve. Invaluable information about ventricular function can also be obtained at this stage to guide haemodynamic management, including looking for left ventricular outflow obstruction, which is a known complication of mitral valve repair. It is caused by systolic anterior motion of a redundant portion of the anterior leaflet.

Conclusion

Mitral valve repair is an established and, if feasible, the preferred surgical treatment for mitral regurgitation because of its superiority versus replacement in terms of operative mortality, thromboembolism and long-term survival. Repair not only places greater demands on the surgeon but also on the anaesthetist who provides TOE data, which are vital for surgical intraoperative decision making and improves haemodynamic management. It is therefore extremely useful for anaesthetists involved in mitral surgery to understand the functional anatomy and nomenclature of the mitral valve as well as having a systematic approach to evaluating the mitral valve with TOE.

References


See multiple choice questions 147–151