Mitral valve and mitral valve disease

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Key points

- Mitral valve and left ventricular (LV) function are interdependent on each other.

- In mitral regurgitation (MR), measured LV ejection fractions are falsely high.

- Vasopressors should be used with caution in patients with severe MR.

- New minimally invasive and percutaneous techniques are now available to repair the mitral valve.

- Vasodilating drugs should be used with caution in mitral stenosis.

The mitral valve

The mitral valve separates the left atrium from the left ventricle (LV) and, as such, is an integral part of the high-pressure systemic circulation. Therefore, any pathology of the valve can have critical physiological effects that the anaesthetist must be aware of. Anaesthesia alters the way in which the heart, valve, and circulation interact and control of these changes needs to be both understood and anticipated.

Anatomy

The mitral valve is a complex structure that incorporates not only the two main leaflets, but an annulus, tendinous cords, and papillary muscles connecting the valve leaflets to the LV (Fig. 1).

Annulus

The mitral valve's annulus is a saddle-shaped structure which is shaped more like a 'D' than an 'O'. The aortic valve is wedged into the straight side of the 'D' and there is a fibrous continuity between the two valves. This means that one can be easily affected by surgery on the other.

The circumflex artery follows the line of the annulus as it passes anteriorly around the base of the heart, a position that places it at risk during any mitral valve surgery. Damage or ligation of the artery will result in lateral wall ischaemia, and this is an important area to assess on perioperative echocardiography.

Leaflets

The anterior leaflet hangs down from the straight side of the 'D'-shaped annulus and is more superior and medial than truly anterior. The posterior leaflet is a C-shaped leaflet divided into three sections by indentations that give it a scalloped appearance. These three segments are called P1, P2, and P3, with their opposing anterior leaflet segments termed A1, A2, and A3. The free edges of the leaflets do not meet exactly, but instead have around 5 mm of overlap. This overlap at the edges seals the valve as the body of the leaflets push outwards like sails during systole (see Online video 1). At the corners of the leaflets, there are two commissures which ensure a seal in the corners of the valve.

Papillary muscles and chordae

During systole, the leaflets are prevented from billowing back into the left atrium by the chordae. These tether the free edges of the leaflets to the two papillary muscles which grow out from the wall of the LV. The papillary muscles are anterolateral and posteromedial and sit under the commissures of the valve, with each muscle passing chordae to both leaflets. While the anterolateral muscle receives a dual blood supply from the left anterior
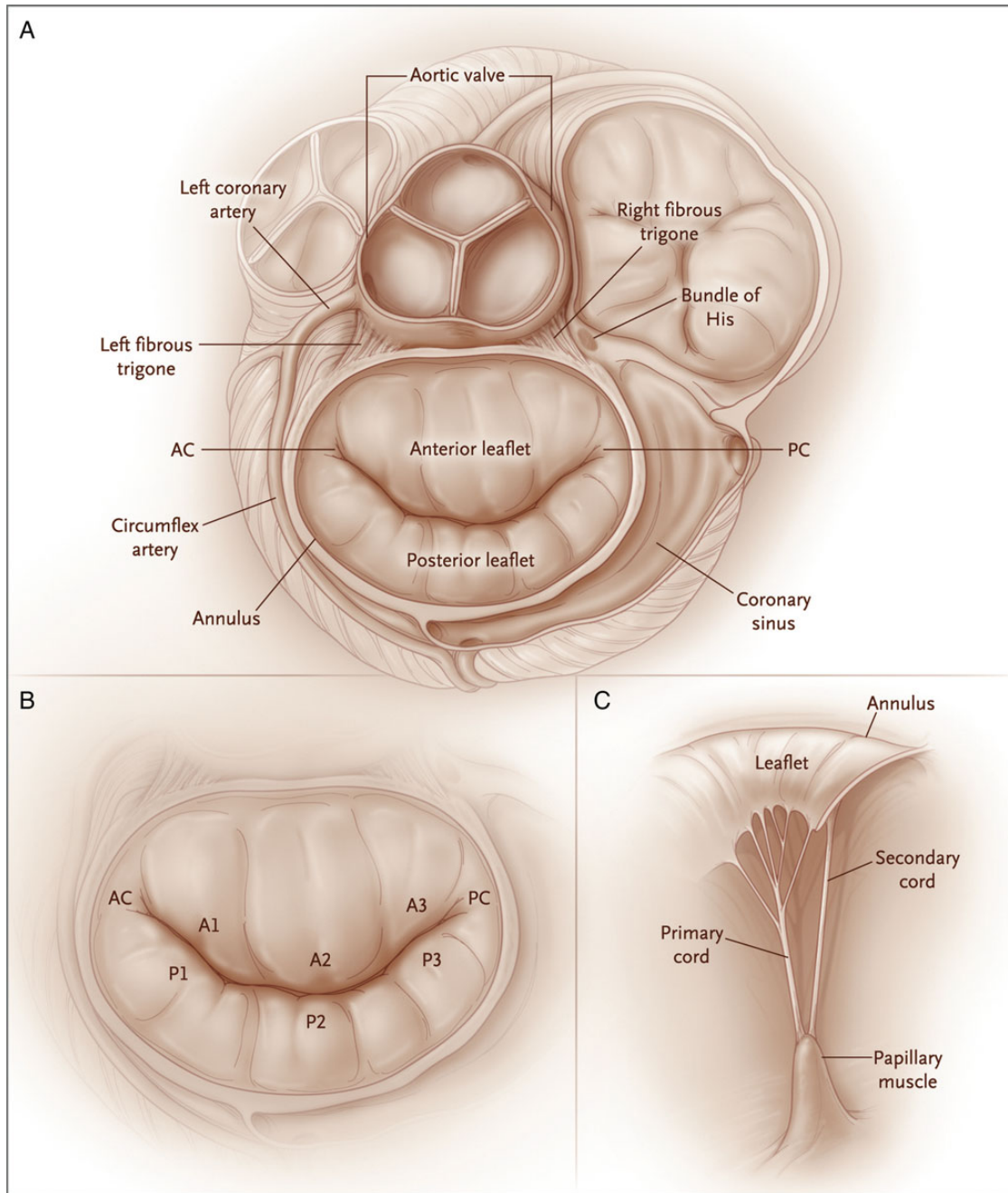
Fig 1 The mitral valve and surrounding structures. The valve is in the 'surgeon's view' orientation with the heart tilted to the left. (A) AC, anterior commissure; PC, posterior commissure. (B) The mitral valve leaflets, each of which usually consists of three discrete segments or scallops. These are designated A1, A2, and A3 for the anterior leaflet and P1, P2, and P3 for the posterior leaflet. (c) Primary chordae are attached to the free edge of the valve leaflet, and secondary chordae are attached to the ventricular surface of the leaflet. (From)[1]. Reprinted with permission from Massachusetts Medical Society.

descending and the circumflex artery, the posteromedial is supplied solely by the right coronary artery in the more common right dominant circulation. As it is usually only supplied by a single artery, infarction and rupture of the posteromedial muscle is much more likely, as could be seen in the context of an inferior myocardial infarction.

Mitral stenosis

A normal mitral valve will have an area of >4 cm^2, but symptoms are usually only present once the stenosis is moderate–severe.

Grading into mild, moderate, or severe disease is based on both the pressure needed to drive blood across the valve (mean pressure gradient) and the valve area.

Aetiology/epidemiology

Rheumatic fever is by far the largest cause of mitral stenosis (MS), although it is relatively rare in developed countries where degenerative calcification or endocarditis are more likely causes. Disease progression is usually around 0.1–0.3 cm^2 yr^{-1},[2] but this is greatly increased in cases of repeated valve inflammation such

Video 1 Trans-oesophageal echo of normal mitral valve (mid-oesophageal five-chamber view). The left atrium is at the top of the image and the mitral valve is seen separating the left atrium from the left ventricle. Part of the aortic valve is seen in the centre of the picture. If reading the pdf online, click on the image to view the video.
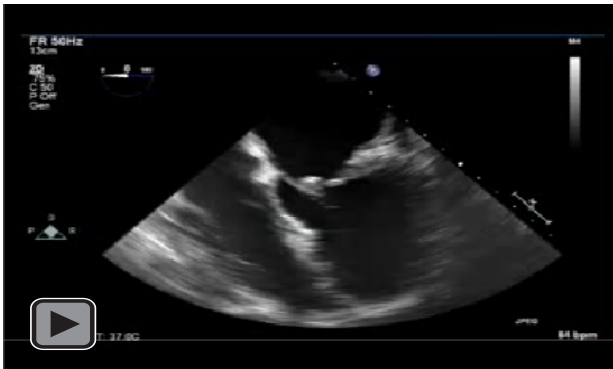
Video 2 Trans-oesophageal echo of stenotic mitral valve (mid-oesophageal four-chamber view). The leaflet tips are thickened and do no open widely. Note the dilated left atrium (at the top of the image). If reading the pdf online, click on the image to view the video.

as may occur with recurrent bouts of rheumatic fever. This is one reason why rheumatic heart disease follows a more indolent course when contracted in developed countries where the disease is less common. The inflammation results in a thickened, nodular valve that can become fused at the leaflet edges and into the commisural edges (see Online video 2). The chordae can also become shortened or thickened, and because blood flows around these cords on entering the LV, this can cause a subvalvular obstruction to flow.

Other causes are much less common. These include infiltrating diseases, and congenital deformities, or diseases that affect multiple systems, such as sarcoidosis.

Signs/symptoms

MS is generally well tolerated, and patients will remain asymptomatic for many years. This compensation is due to the compliant left atrium dilating and keeping pulmonary venous pressures stable. As the disease progresses, this compensation is gradually overcome and pulmonary hypertension starts to develop. Pulmonary oedema and dyspnoea worsen, and patients will complain of fatigue and more frequent lower respiratory tract infections. Subtle signs are often further delayed by a reduction in activity and may need to be carefully sought. Pregnancy and

Table 1 Criteria for echocardiographic diagnosis of MS severity[3]

	Mild	Moderate	Severe
Mitral stenosis			
Mean pressure decrease	<5 mm Hg	6–10 mm Hg	>10 mm Hg
Pressure half-time	<139 ms	140–219 ms	≥220 ms
Valve area	1.6–2.0 cm^2	1.5–1.0 cm^2	<1.0 cm^2

the development of atrial fibrillation (AF) will lead to a rapid progression of the pathophysiology, and development of symptoms. In pregnancy, any compensation is overcome by the large increase in blood volume, as discussed later.

Classification

MS is classified on the basis of valve area and pressure gradients across the valve (Table 1). Pressure gradients are relatively simple to assess using echocardiography, with a mean pressure >5 mm Hg indicating moderate stenosis, and >10 mm Hg indicating severe stenosis. Valve area is usually derived using several techniques as simply tracing the area out using the echo software can be unreliable. While a normal mitral valve has an area of 4–6 cm^2, symptoms may only develop once the valve area is <2.0 cm^2, with <1 cm^2 indicating severe disease.

Treatment of MS: medical

Atrial fibrillation
In severe MS, up to 40% of patients will develop AF.[4] An increased ventricular response rate decreases diastolic time and increases atrial pressure, further worsening symptoms. Rate control reduces symptoms and is generally easier to achieve than rhythm control in a heart with dilated atria and fibrosis from previous inflammation. It can be achieved with either β-blockers or calcium channel blockers.[5]

Dyspnoea
Dyspnoea is a common presenting complaint with symptomatic MS. Diuretics are used to reduce this, and long-acting nitrates can also provide some relief of symptoms.

Anticoagulation
While there is little evidence to justify anticoagulation in MS alone, patients with AF, prior embolic events, or with a demonstrated left atrial thrombus should be anti-coagulated with warfarin (target INR 2–3) or heparin.[5,6]

Treatment of MS: surgical

Surgical options, either open or percutaneous, are the treatment of choice in severe symptomatic MS. In patients with valves with mobile leaflets that are free of calcium, percutaneous mitral commissurotomy (PMC) is the preferred option. This is performed by passing a balloon across the valve and inflating it, thereby splitting the fused commissural edges. PMC achieves an MV area >1.5 cm^2 with no worse than moderate mitral regurgitation (MR) in 80% of cases, with emergency surgery rates of <1%. Surgery is recommended in the presence of atrial thrombus, heavy valve calcification, or when another open cardiac procedure needs to be performed. Mitral valve replacement is now the preferred treatment due to improved valve and clinical outcomes. Mitral valve replacement has an operative mortality of 3–5%, but long-term outcomes are highly variable and related to

multiple patient-related factors (e.g. bi-ventricular function, pulmonary pressures, and AF).

Anaesthetic management of MS in non-cardiac surgery

Preoperative assessment

As mentioned earlier, MS is generally well tolerated and insidious signs need to be carefully sought. Symptoms of dyspnoea, fatigue, and a history of frequent lower respiratory tract symptoms are suggestive. The indicative low rumbling diastolic murmur is best heard in expiration at the apex, with the bell of the stethoscope.

Ongoing elevated pulmonary artery (PA) pressures put strain on the right ventricle, and will eventually cause this to fail. Signs of right heart failure, such as a raised jugular venous pressure (JVP) and peripheral oedema, should be sought on examination.

As the left atrium dilates, AF is common. Those patients on warfarin may need to be converted to heparin before operation, depending on a number of factors—although if the warfarin is for AF alone, a short period with no anti-coagulation carries minimal risk.

Investigations

Exercise tolerance testing

This provides an objective measure that may unmask poor functional ability in patients who have decreased their activity with the onset of symptoms.

Echocardiography

In addition to providing information on the severity of the lesion, echo may give information on any developing pulmonary hypertension and right heart dysfunction. In general, the LV continues to function well.

In asymptomatic patients, where the systolic PA pressure is <50 mm Hg, non-cardiac surgery is considered safe.[7]

ECG

AF is common, although p-mitrale may be noted if still in sinus rhythm.

Perioperative management

Like aortic stenosis, MS represents a fixed cardiac output state.

Rate/rhythm

If possible, sinus rhythm should be maintained, and a low normal heart rate (<70 beats min^{-1}) is critical regardless of the rhythm to allow sufficient diastolic time for ventricular filling.

Preload

Aim for normovolaemia, keeping in mind that fluid boluses can worsen pulmonary oedema.

Afterload

Because the cardiac output is fixed, any reduction in systemic vascular resistance can cause a decrease in coronary perfusion pressures. Afterload maintenance is therefore crucial. Pulmonary vascular pressures should be optimized by avoiding hypoxia, hypercapnia, and acidosis to prevent acute right ventricular decompensation. Nitrous oxide should not be used as it

can cause further increases in pulmonary vascular resistance in those patients where it is already elevated.

Contractility

The LV will usually be unaffected. The right ventricle may need support if signs of failure are present. After ensuring pulmonary vascular resistance is as low as possible, inotropes such as phosphodiesterase inhibitors or catecholamine agonists may be required to augment the right ventricular contractility. Care should be taken with phosphodiesterase inhibitors due to their vasodilating effects on the systemic system resulting in afterload reduction and hypotension.

Neuraxial anaesthesia

Neuraxial anaesthesia causes a decrease in afterload, and so has the potential for profound hypotension in MS. This may lead to a spiral of poor myocardial perfusion and worsening cardiac function. As such, the decision to undertake neuraxial anaesthesia in MS should not be made lightly. Consideration should also be given to the fact that the patient may be anticoagulated.

Postoperative management

Care needs to be taken to observe the same fundamentals of management in the postoperative period. This is generally best achieved in a high-dependency unit unless surgery is extremely minor. Instructions to avoid fluid boluses should be given to the recovery staff, and any hypotension should mandate anaesthetic review and aggressive treatment.

Post-cardiac surgery

After repair or replacement, preload should be well maintained, and afterload reduced to promote forward flow. The right heart may still need support in the form of pulmonary vascular resistance reduction and inotrope infusion. Surgical alterations to the mitral annulus can affect how the left heart contracts. This, combined with the effects of open heart surgery and cross-clamping with cardioplegia, means that the left heart is also at risk of failure during this period.

Management of MS in pregnancy

Owing to the increase in blood volume and heart rate in pregnancy, MS is poorly tolerated, and frequently will first present at this time. In normal pregnancy, cardiac output increases ~50%, although the fixed cardiac output state of MS results in a worsening of pressure through the pulmonary circulation and into the right heart. The risk of decompensation depends on severity. Anything worse than moderate disease (valve area <1.5 cm^2) frequently results in heart failure, which usually develops in the second or third trimester and is progressive.[8]

Medical management through pregnancy involves aggressive control of tachycardia and AF, with β-blockers the mainstay of treatment. Diuretics are also added if symptoms of pulmonary congestion develop. If symptoms are not controlled with medical therapy, percutaneous commisurotomy is the surgical treatment of choice as it avoids bypass with the attendant risk to the fetus.

Delivery

Haemodynamic goals for delivery remain afterload maintenance, heart rate and rhythm control, and care with fluid administration. Vaginal delivery is well described, but is carefully managed. Neuraxial analgesia is placed early to block any

tachycardia due to sympathetic stimulation. Block is achieved slowly and any hypotension is managed with α-agonists. Epidurals are titratable, with a mixture utilizing opioids achieving analgesia with fewer haemodynamic effects. The second stage is assisted, limiting labour duration and valsalva. In patients with pulmonary hypertension, or NYHA III/IV symptoms despite maximal medical therapy, Caesarean section is considered.

Post-delivery, patients with MS are at risk of developing pulmonary oedema. This can occur precipitously as a result of decompression of the inferior vena cava along with autotransfusion due to uterine compression causing a sudden increase in preload. This 'flash' pulmonary oedema can be initially managed with head-up positioning, and the institution of 100% oxygen. This may necessitate intubation and ventilation if severe, with sufficient PEEP.

Also of note, oxytocin should be administered with great care in the MS population due to its vasodilating properties on the systemic circulation and propensity to increase pulmonary vascular resistance. Ergometrine is contraindicated due to its pulmonary vasoconstricting effects.

Mitral regurgitation

MR can be acute or chronic in onset, and primary or secondary in nature. Primary MR is due to pathology of the valve preventing normal closure, whereas secondary MR is caused by LV dysfunction that affects the closing of the mitral valve (this is also known as 'functional' MR).

In either case, the left atrium dilates as blood is ejected back into it. This dilation can be significant, and AF is common. Overload of the pulmonary circulation causes dyspnoea, although frank pulmonary oedema is usually only seen in acute MR and in severe chronic disease. The LV is volume overloaded in MR, with dilation of the ventricle further worsening MR through annular dilatation.

Acute MR

Acute MR can be caused by any disruption to the normal mechanism of the valve. Examples of pathology include the growth of vegetations on the leaflets in infective endocarditis, chordae rupture in patients with pre-existing degenerative disease, or papillary muscle rupture due an ST-elevation MI (usually affecting the inferior territory).

The left atrium is unable to compensate acutely for the increased pressure caused by blood refluxing back into it. As the pressure is transmitted back into the pulmonary circulation, patients can present with sudden-onset dyspnoea and require rapid stabilization and treatment.

Chronic MR

In chronic primary disease, there is an abnormality of the leaflets that prevents them from closing normally. The major cause for this is degenerative disease resulting in leaflet prolapse (see Online video 3).

Chronic secondary MR is also known as functional MR because the anatomy of the valve is normal, but its function is impaired due to LV pathology. This may be as a result of dilation of the mitral annulus with LV dilatation such that the leaflets cannot meet adequately, or due to abnormal movement (dyskinesia) of the LV after ischaemia or infarction. Systolic anterior motion of the anterior mitral leaflet into the LV outflow tract can occur in conditions such as hypertrophic cardiomyopathy, especially when
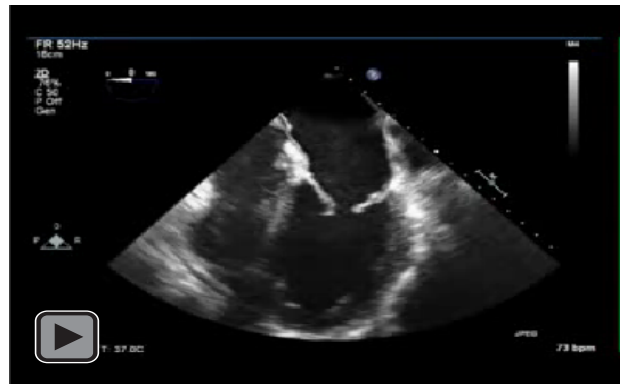
Video 3 Trans-oesophageal echo of posterior mitral valve leaflet prolapse and flail (mid-oesophageal four-chamber view). The posterior mitral valve leaflet rises above the mitral valve annulus and results in regurgitation of blood into the left atrium. If reading the pdf online, click on the image to view the video.

Table 2 Criteria for echocardiographic diagnosis of MR severity[3]

	Mild	Moderate	Severe
Mitral regurgitation			
Regurgitant fraction	<30%	30–49	≥50%
Regurgitant orifice area	<0.20 cm^2	0.2–0.39 cm^2	≥0.40 cm^2
Regurgitant volume	<30 ml beat^{-1}	30–59 ml beat^{-1}	≥60 ml beat^{-1}

hypovolaemic. This results in obstruction to outflow into the aorta, and also simultaneous regurgitation across the distorted mitral valve.

Classification

Grading the severity of the MR is complex and involves several techniques (Table 2).

Transoesophageal echocardiography remains the gold standard for defining the severity of the MR, although transthoracic echocardiography is sufficient if good images are acquired. Determination of the severity is made using several measurements including effective regurgitant orifice area, regurgitant volume, and regurgitant fraction. These are correlated with other measurements, such as the narrowest part of the regurgitant jet (vena contracta) to support the diagnosis.

Treatment of MR: medical

In acute MR, the goals include filling pressure reduction with nitrates or diuretics, and afterload reduction with vasodilators or an intra-aortic balloon pump (IABP) as bridging to definitive treatment.

Vasodilator use promotes forward flow into the aorta and so reduces the regurgitant fraction; however, their use can be limited by hypotension. In this case, inotropic agents or an IABP should be added. The IABP decreases afterload by deflating during systole (reducing afterload and therefore reducing the work of the heart). The inflation of the balloon during diastole increases the pressure in the aortic root which in turn increases coronary perfusion pressure.

In chronic MR with signs of heart failure, the treatment is in line with standard heart failure management including β-blockers, angiotensin-converting enzyme inhibitors, and

aldosterone antagonists, with diuretics used where heart failure is present. In addition, if cardioversion is attainable, it will often immediately reduce MR severity. However, there is no proven benefit to any medical therapy for MR without LV dysfunction, and generally when such dysfunction does develop, it is an indication for surgery.

Treatment of MR: surgical

The decision on when to operate can be complex.[5] It depends not only on whether the MR is primary or secondary in nature, but also on the condition of the patient, their response to therapy, and the development of sequelae such as heart failure or AF.

Primary MR

In primary MR, the evidence to operate is clear: surgery is indicated if the MR is severe and acute in nature, such as from a ruptured papillary muscle. If the MR is chronic and causing symptoms, and there are no contraindications to surgery, repair is also clearly indicated.

However, if the patient is asymptomatic, then clear consequences from the MR have to be seen, whether this be an LV ejection fraction (LVEF) <60% or the development of AF or pulmonary hypertension. Surgery is generally avoided if the heart failure is severe (LVEF <30%).

Secondary MR

The indications for surgical treatment of secondary MR are less evidence-based than with primary MR.

In patients already undergoing CABG, it appears clear that repair of severe MR is indicated. However, whether to repair moderate MR or not during CABG is less obvious.

In symptomatic patients, with severe LV failure, the benefits of surgery are felt to be questionable unless there is some reversibility of the underlying problem, such as revascularization of viable myocardium.

Surgery is also considered in symptomatic patients with moderate LV failure if medical therapy has failed.

With regard to the type of surgery, valve repair, rather than replacement, is invariably preferred for primary MR if at all possible. Repair has significantly reduced operative mortality[5] and better preservation of LV function when compared with replacement and avoids the long-term consequences of prosthetic valves.

For secondary disease, such as ischaemic MR, repair results in reduced early mortality but a higher degree of recurrent MR in the long term. Otherwise, the long-term outcomes of repair *vs* replacement for ischaemic MR are similar.[9]

Minimally invasive surgery

Mitral valve repair and replacement can also be performed through a small right thoracotomy and several small instrument ports with a femoral incision for bypass cannula insertion. Standard operative techniques are used under endoscopic view. The safety and efficacy of this approach has been well demonstrated and is similar to the traditional midline sternotomy approach in the hands of experienced surgeons.

Robotic telemanipulators are developing a place in mitral valve surgery since their first use in 1998. Their dexterity helps the surgeon place sutures in difficult to reach positions more precisely. While bypass and cross-clamp times are longer, times to discharge and return to work are shorter.[10] Robotic arms can be used in all types of mitral surgery, although long-term outcome data are awaited.

Percutaneous mitral valve repair

Recent trials have shown efficacy of the Mitraclip® system for percutaneous repair in moderate–severe or severe MR[11] in patients deemed too high risk for surgery. This technique works by clipping the two leading edges of the leaflets together and creating a double orifice into the LV (Fig. 2). The Mitraclip® system is less effective at reducing MR in the short term but has similar outcomes at 12 months. It results in improved LVEF, NYHA score, and quality of life compared with baseline. Although in the randomized EVEREST trial, 20% of the patients required further surgical intervention, the role of the Mitraclip® system is expanding in high-risk surgical patients.

Anaesthetic management of MR in non-cardiac surgery

Preoperative assessment

While acute MR can present as florid cardiac failure with severe pulmonary oedema, chronic MR can be tolerated for many years, with symptoms only developing as the LV starts to fail.

As with MS, signs of pulmonary hypertension and right heart failure should be sought as the increased pressure is transmitted back through the pulmonary circulation to the right heart. These signs include dyspnoea, fatigue, and frequent chest infections indicating pulmonary hypertension, or peripheral oedema, and a raised JVP to indicate right heart failure.

The pansystolic murmur of MR is frequently very soft, and its intensity is unrelated to the severity of the disease. There may be a displaced and forceful apex beat palpable.

Investigations
Echocardiography

Echo is useful when grading the severity of the lesion. However, the LV function is more difficult to assess. As blood is being ejected in systole through both the aortic *and* regurgitant mitral valves, the EF *appears to be* higher than normal. Therefore, an LVEF of around 70% is thought to be normal in MR (cf. 55% 'normally'), with levels below 60% representing LV dysfunction.

ECG

Signs of ischaemia should be sought, especially in functional MR. If the patient presents with acute or new-onset MR, this may be due to recent papillary muscle rupture. The history of the event may be vague and ECG may aid the diagnosis. As an inferior myocardial infarction is the most likely cause of papillary muscle rupture, ST changes in leads II, III, and aVF may suggest the diagnosis.

Blood tests

Brain natriuretic peptide (BNP) can be useful in prognostication, with low plasma BNP having a high negative predictive value for developing further complications.[12]

Exercise testing

Symptoms of chronic MR can often be masked by the patient simply reducing their exertion to a level they are capable of. Formal testing, such as cardiopulmonary exercise testing, is of clear
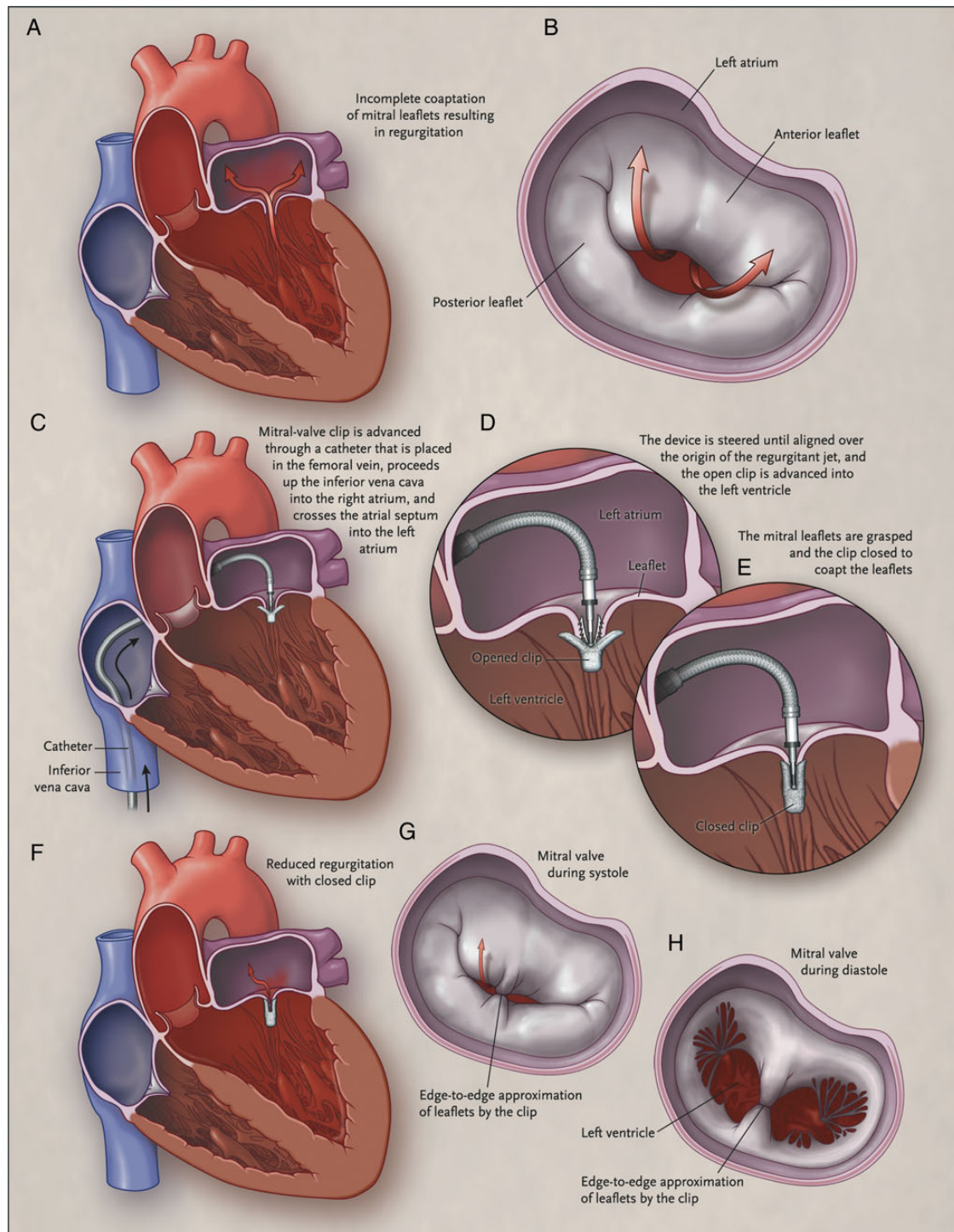
Fig 2 Repair of MR with the Mitraclip® system. A dual-inlet mitral valve results, with reduced regurgitation. In patients with MR resulting from incomplete leaflet co-aptation (A and B), percutaneous mitral valve repair is performed by means of femoral venous and trans-septal access to the left atrium to steer the device towards the origin of the regurgitant jet (C). The Mitraclip® is passed through the mitral orifice from the left atrium to the LV and pulled back to grasp the leaflet edges (D and E). If the reduction in the MR is satisfactory, the device can be locked and then released (F). A double orifice is created in conjunction with reduction in MR (G and H). (From)[11]. Reprinted with permission from Massachusetts Medical Society.

utility if the echo findings do not match with the symptom history, and may give a clearer picture of ventricular function.

If LV function is preserved and the patient is asymptomatic then non-cardiac surgery can be performed with no additional risk, even if the MR is severe.[13] In fact, the risk associated with non-cardiac surgery increases only when the LV function is severely impaired (<30%) or the patient is symptomatic. In these cases, medical optimization must be assured before proceeding.

Perioperative management

As with all regurgitant lesions, the haemodynamic goals can be summarized as 'full, fast and forward flow'.

Rate/rhythm

A high normal heart rate (80–100 beats min^{-1}) reduces filling time of the LV. This helps counteract overload of the ventricle and encourages forward flow. The desire for an elevated heart rate needs to be balanced with myocardial oxygen demand and supply considerations in the presence of ischaemic heart disease.

Preload

Assessment of preload can be difficult in MR. Erring on the side of well filled is preferable as it also promotes forward flow.

Afterload

As systemic vascular resistance increases, so does the regurgitant fraction. Decreases in arterial pressure should be treated with fluid and elevating the heart rate—vasoconstrictors can be used with care. Pulmonary vascular resistance should be kept as low as possible by strict avoidance of hypoxia, hypercapnia, and acidosis and avoiding the use of nitrous oxide.

Contractility

In acute MR, inotropes such as dobutamine or even inodilators such as enoximone or levosimendan may be needed—especially if there is evidence of LV failure. In this case, consideration should be given to the insertion of an IABP before operation.

Neuraxial anaesthesia

Neuraxial anaesthesia is generally well tolerated with MR as the afterload is reduced, which aids forward flow. Vasopressors need to be titrated with care.

Postoperative management

Recovery staff should be made aware of the cardiac diagnosis, and the same perioperative principles should be adhered to.

Post-cardiac surgery

After replacement of the valve, there is no low-pressure system for the LV to eject into and it will suddenly be subjected to a higher workload. Poor function may be unmasked, and inotropes and inodilators should be considered. Systemic vascular resistance should be minimized if possible to allow forward flow and to 'off-load' the ventricle. If an IABP was needed before operation, this is generally left in place after operation until haemodynamic and metabolic stability have been achieved.

Management of MR in pregnancy

Pregnancy mimics the haemodynamic goals for regurgitant lesions with an increase in heart rate and blood volume, and a decrease in systemic vascular resistance promoting forward flow. Because of this, pregnancy is generally well tolerated, even if MR is severe, as long as LV function is preserved.[5] Patients whose LV function is impaired, or have severe symptomatic MR, are at increased risk of heart failure. Diuresis is commonly used to limit fluid overload, and this is usually all the medical therapy that is needed.

Delivery

Vaginal delivery is generally possible with epidural anaesthesia and an assisted second stage to avoid spikes in the systemic vascular resistance seen with pushing.

Summary

Anaesthetists are an integral part of any team undertaking surgical management of a patient with mitral valve disease. An understanding of the pathophysiology allows the anaesthetist to not only engage in decisions regarding treatment, but to anticipate and manage the consequences of surgery and anaesthesia.

Online videos

The videos associated with this article can all be viewed from the article in *BJA Education* online.

Acknowledgements

MCQs

The associated MCQs (to support CME/CPD activity) can be accessed at https://access.oxfordjournals.org by subscribers to *BJA Education*.

Podcasts

This article has an associated podcast which can be accessed at http://www.oxfordjournals.org/podcasts/mitral_valve_disease_dr_gibbison_bjaeducation_jan2017.mp3.

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