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The Mitral Valve and Mitral Valve Disease

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Key Points:

- Mitral valve and left ventricular 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 (MS).

The Mitral Valve:

The mitral valve separates the left atrium from the left ventricle 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 (see Fig 1):

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 left ventricle.

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 peri-operative 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 3 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 5mm of overlap. This overlap at the edges seals the valve as the body of the leaflets push outwards like sails during systole (**see 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 left ventricle. The papillary muscles are antero-lateral and postero-medial 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 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 $>4\text{cm}^2$ 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), though it is relatively rare in developed countries where degenerative calcification or endocarditis are more likely causes. Disease progression is usually around $0.1\text{-}0.3\text{cm}^2$ per year¹ but this is greatly increased in cases of repeated valve inflammation such 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 commissural edges (**see video 2**). The chordae can also become shortened or thickened, and because blood flows around these cords on entering the left ventricle this can cause a sub-valvular 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 the development of atrial fibrillation 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 (see table 1):

MS is classified on the basis of valve area and pressure gradients across the valve. Pressure gradients are relatively simple to assess using echocardiography, with a mean pressure $>5\text{mmHg}$ indicating moderate stenosis, and $>10\text{mmHg}$ 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 $4\text{-}6\text{cm}^2$, symptoms may only develop once the valve area is $<2.0\text{cm}^2$, with $<1\text{cm}^2$ indicating severe disease.

Treatment of Mitral Stenosis: Medical

Atrial fibrillation:

In severe MS, up to 40% of patients will develop atrial fibrillation². 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 beta-blockers or calcium channel blockers³.

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 atrial fibrillation, prior embolic events or with a demonstrated left atrial thrombus should be anti-coagulated with warfarin (target INR 2-3) or heparin^{3 4}

Treatment of Mitral Stenosis: 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 $>1.5\text{cm}^2$ with no worse than moderate mitral regurgitation 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 (for example, bi-ventricular function, pulmonary pressures and atrial fibrillation).

Anaesthetic Management of Mitral Stenosis 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 PA pressures put strain on the right ventricle, and will eventually cause this to fail. Signs of right heart failure, such as a raised JVP and peripheral oedema, should be sought on examination.

As the left atrium dilates, atrial fibrillation is common. Those patients on warfarin may need to be converted to heparin preoperatively, depending on a number of factors – although if the warfarin is for atrial fibrillation 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:

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

In asymptomatic patients, where the systolic pulmonary artery pressure is less than 50mmHg, non-cardiac surgery is considered safe⁵.

ECG:

Atrial fibrillation is common, though 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/min) 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 fall in coronary perfusion pressures. Afterload maintenance is therefore crucial. Pulmonary vascular pressures should be optimised by avoiding hypoxia, hypercapnia and acidosis to prevent acute right ventricular decompensation. Nitrous oxide should not be used as it can cause further rises in pulmonary vascular resistance in those patients where it is already elevated.

Contractility:

The left ventricle 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 drop 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 post-operative 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, mean that the left heart is also at risk of failure during this period.

Management of Mitral Stenosis in pregnancy:

Due 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%, though 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 <1.5cm²) frequently results in heart failure, which usually develops in the second or third trimester and is progressive⁶

Medical management through pregnancy involves aggressive control of tachycardia and atrial fibrillation, with beta-blockers the mainstay of treatment. Diuretics are also added if symptoms of pulmonary congestion develop. If symptoms are not controlled with medical therapy, percutaneous commissurotomy 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. Blockade is achieved slowly and any hypotension is managed with alpha-agonists. Epidurals are titratable, with a mixture utilising 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 IVC 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 positive end-expiratory pressure (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:

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 left ventricular 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 atrial fibrillation 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 left ventricle is volume overloaded in MR, with dilation of the ventricle further worsening MR through annular dilatation.

Acute Mitral Regurgitation

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 a 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 stabilisation and treatment.

Chronic Mitral Regurgitation

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 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 left ventricular pathology. This may be as a result of dilation of the mitral annulus with left ventricular dilatation such that the leaflets can't meet adequately, or due to abnormal movement (dyskinesia) of the left ventricle following ischaemia or infarction. Systolic anterior motion (SAM) of the anterior mitral leaflet into the left ventricular outflow tract can occur in conditions such as hypertrophic cardiomyopathy, especially when hypovolaemic. This results in obstruction to outflow into the aorta, and also simultaneous regurgitation across the distorted mitral valve.

Classification (see table 2):

Grading the severity of the MR is complex and involves several techniques.

Transthoracic echocardiography (TTE) remains the gold standard for defining the severity of the MR, though transthoracic echocardiography is sufficient if good images are acquired. Determination of the severity is made using several measurements including effective regurgitant orifice area (EROA), 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 Mitral Regurgitation: 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 beta-blockers, ACE 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 left ventricular dysfunction, and generally when such dysfunction does develop it is an indication for surgery.

Treatment of Mitral Regurgitation: Surgical

The decision on when to operate can be complex.³ 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 atrial fibrillation.

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 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 is 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 revascularisation of viable myocardium.

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

With regards to 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 mortality³ and better preservation of LV function when compared to 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 versus replacement for ischaemic MR are similar⁷.

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 shorter⁸. Robotic arms can be used in all types of mitral surgery, although long-term outcome data is awaited.

Percutaneous mitral valve repair:

Recent trials have shown efficacy of the Mitraclip® system for percutaneous repair in moderate-severe or severe MR⁹ 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 left ventricle (see 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 to baseline. Although in the randomised 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 Mitral Regurgitation 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 left ventricle 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 left ventricular function is more difficult to assess. As blood is being ejected in systole through both the aortic *and* regurgitant mitral valves, the ejection fraction *appears to be* higher than normal. Therefore, a left ventricle ejection fraction of around 70% is thought to be normal in MR (cf. 55% 'normally'), with levels below 60% representing left ventricular 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. 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 complications¹⁰.

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 utility if the echo findings don't 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 severe¹¹. In fact, the risk associated with non-cardiac surgery rises only when the left ventricle function is severely impaired (<30%) or the patient is symptomatic. In these cases medical optimisation must be assured before proceeding.

Perioperative management:

As with all regurgitant lesions, the haemodynamic goals can be summarised as “full, fast and forward flow”.

Rate/rhythm:

A high normal heart rate (80-100bpm) reduces filling time of the left ventricle. 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 rises, so does the regurgitant fraction. Falls in blood 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 left ventricular failure. In this case, consideration should be given to the insertion of an IABP pre-operatively.

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 peri-operative principles should be adhered to.

Post cardiac surgery:

After replacement of the valve, there is no low-pressure system for the left ventricle 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 minimised if possible to allow forward flow and to 'offload' the ventricle. If an IABP was needed pre-operatively this is generally left in place post-operatively until haemodynamic and metabolic stability have been achieved.

Management of Mitral Regurgitation in pregnancy:

Pregnancy mimics the haemodynamic goals for regurgitant lesions with an increase in heart rate and blood volume, and a fall in systemic vascular resistance promoting forward flow. Because of this, pregnancy is generally well

tolerated, even if MR is severe, as long as left ventricular function is preserved³. Patients whose left ventricular 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.

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Figures and Tables

Mitral Stenosis			
	Mild	Moderate	Severe
Mean Pressure Drop	<5mmHg	6 - 10mmHg	>10mmHg
Pressure Half Time	<139ms	140 - 219ms	≥220ms
Valve Area	1.6 - 2.0cm ²	1.5 - 1.0cm ²	<1.0cm ²

Table 1. Criteria for echocardiographic diagnosis of mitral stenosis severity¹²

Mitral Regurgitation			
	Mild	Moderate	Severe
Regurgitant Fraction	<30%	30-49	≥50%
Regurgitant Orifice Area	<0.20cm ²	0.2 - 0.39 cm ²	≥0.40cm ²
Regurgitant Volume	<30ml/beat	30 - 59ml/beat	≥60ml/beat

Table 2. Criteria for echocardiographic diagnosis of mitral regurgitation severity¹².

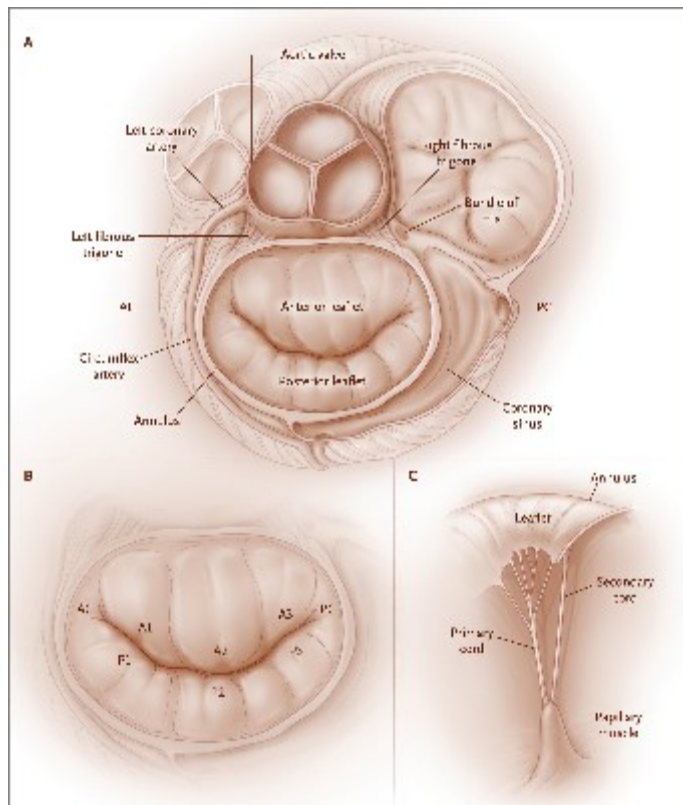


Figure 1. The mitral valve and surrounding structures. The valve is in the ‘surgeon’s view’ orientation with the heart tilted to the left. (Panel A): AC - Anterior commissure. PC - posterior commissure. Panel B shows the mitral-valve leaflets, each of which usually consists of three discrete segments or scallops. These are designated A1, A2, and A3 for the anterior leaflet and P1, P2, and P3 for the posterior leaflet. Panel 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. (Used with permission from the NEJM)¹³

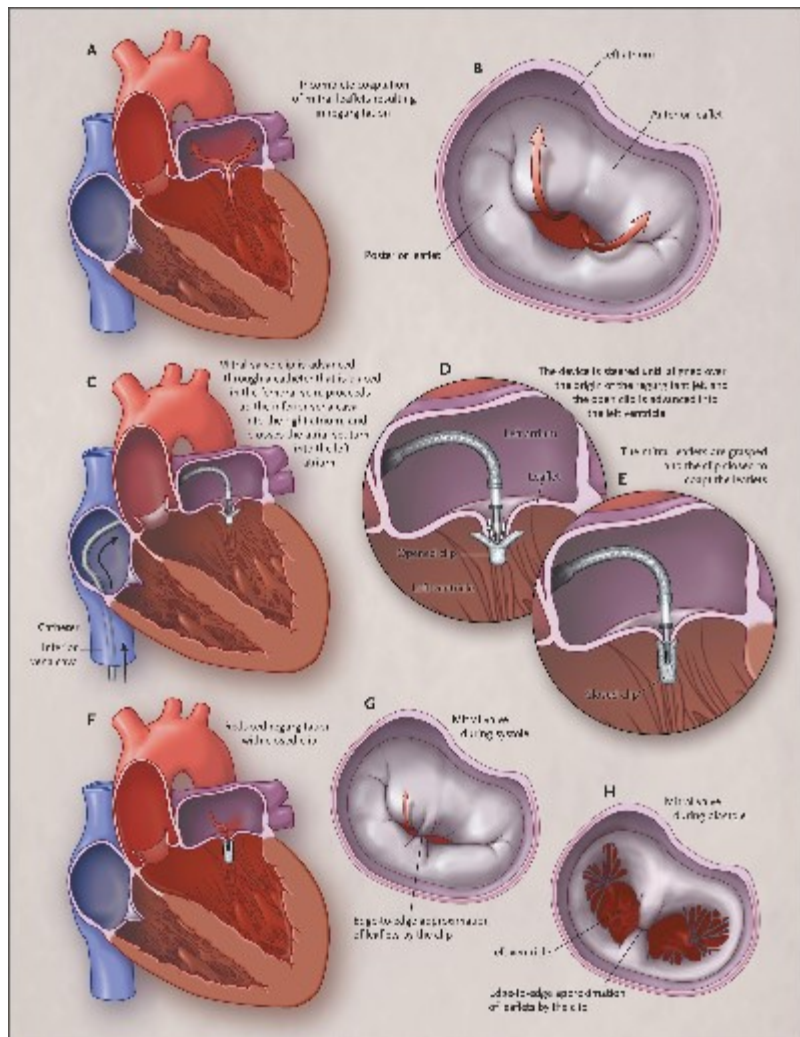


Figure 2: Repair of mitral regurgitation with the Mitraclip system. A dual inlet mitral valve results, with reduced regurgitation. In patients with mitral regurgitation resulting from incomplete leaflet co-aptation (Panels A and B), percutaneous mitral-valve repair is performed by means of femoral venous and trans-septal access to the left atrium to steer the device toward the origin of the regurgitant jet (Panel C). The Mitraclip® is passed through the mitral orifice from the left atrium to the left ventricle and pulled back to grasp the leaflet edges (Panels D and E). If reduction of the mitral regurgitation is satisfactory, the device can be locked and then released (Panel F). A double orifice is created in

conjunction with reduction in mitral regurgitation (Panels G and H). (Used with Permission from the NEJM)⁹

Video 1. Trans-oesophageal echo of normal mitral valve (mid-oesophageal 5-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.

Video 2. Trans-oesophageal echo of stenotic mitral valve (mid-oesophageal 4-chamber view). The leaflet tips are thickened and do not open widely. Note the dilated left atrium (at the top of the image).

Video 3. Trans-oesophageal echo of posterior mitral valve leaflet prolapse and flail (mid-oesophageal 4-chamber view). The posterior mitral valve leaflet rises above the mitral valve annulus and results in regurgitation of blood into the left atrium.

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The Mitral Valve and Mitral Valve Disease MCQs

1.	A 72 year old man undergoes an elective mitral valve repair involving an annuloplasty ring and returns to the ICU. He rapidly becomes hypotensive and trans-oesophageal echo reveals a new regional wall motion abnormality of the lateral wall. Appropriate statements on this complication may include:
(a)	The right coronary artery is at risk in this procedure and has likely been damaged.
(b)	Starting a noradrenaline infusion will improve his coronary perfusion and resolve the regional wall motion abnormality.
(c)	Papillary muscle rupture is a recognised outcome from this complication.
(d)	Medical management is appropriate at this point.
(e)	The circumflex artery may have been damaged or ligated, and this patient needs surgical intervention.

1.	(a)	False	The right coronary artery is not near the surgical field, and damage is unlikely. The right coronary supplies the right ventricle and usually the inferior left ventricular wall, not the lateral wall.
	(b)	False	Starting a noradrenaline infusion may improve this patients coronary perfusion pressure. However, the cause of the regional wall motion abnormality is likely to be mechanical obstruction of his circumflex artery of a surgical aetiology. This will not be remedied by noradrenaline.
	(c)	False	Lateral wall ischaemia indicates circumflex artery pathology. The anterolateral papillary muscle receives dual blood supply from both the circumflex and the left anterior descending arteries and so is unlikely to infarct.
	(d)	False	This patient requires urgent surgical intervention as the causative lesion is almost certainly due to a surgical complication and could not be appropriately managed with medical therapy alone.
	(e)	True	The circumflex runs very close to the mitral annulus and may well have been ligated by a suture (and often even closer in those with left dominant systems). A return to theatre or catheter lab is indicated as the damage needs to be repaired, or the area bypassed with a graft or stent.

2.	A 54 year old previously fit and well lady presents to Accident and Emergency with a vague history of chest pain and worsening shortness of breath over the last 3 days. She is noted to have a systolic murmur, which the patient denies previous knowledge of. Troponins are elevated. Regarding further investigation and treatment for this patient:
(a)	ST elevation in II, III and aVF indicates an inferior myocardial infarction with potential for subsequent papillary muscle rupture.
(b)	Her ECG is likely to reveal ST segment changes in V2-V5
(c)	This lesion is likely to be amenable to percutaneous treatment.
(d)	This patient may become shocked and will require cautious resuscitation with fluid boluses and potentially inotrope support.
(e)	If hypotension develops, vasopressors should be used to support her blood pressure.

2.	(a)	True	Inferior myocardial infarction is the most likely to result in papillary muscle rupture due to the right coronary artery providing the sole blood supply to the posteromedial papillary muscle. When this occurs it results in severe mitral regurgitation.
	(b)	False	This would indicate an anterior myocardial infarction which is very unlikely to result in this pathology due to the dual blood supply of the anterolateral papillary muscle. Of note, ST segment reciprocal depression may be seen in the inferior leads with an anterior myocardial infarction.
	(c)	False	While the coronary artery stenosis may be amenable to percutaneous correction this patient requires surgical intervention for the ruptured papillary muscle. The causative stenosis can be bypassed at this time.
	(d)	True	As this is acute mitral regurgitation, haemodynamic support should include cautious fluid administration with inotropes.
	(e)	False	Vasopressor use alone will increase afterload and worsen the backflow from the left ventricle through the regurgitant mitral valve.

3.	A 74 year old gentleman is seen in pre-assessment clinic for a trans-urethral prostate resection. Exercise is limited by his knee pain and denies orthopnoea, though admits to poor sleep due to bladder symptoms. He takes warfarin for atrial fibrillation. You detect a murmur on exam, and limited echo reveals severe mitral stenosis. Regarding this patient:
(a)	Spinal anaesthesia would be a reasonable option.
(b)	As this patient is relatively asymptomatic, it is safe to proceed with this surgery.
(c)	When he presents for surgery, he will likely require fluid preloading prior to induction.
(d)	This patient is high risk, and requires further investigation including a stress echocardiogram and assessment of pulmonary artery pressures.
(e)	Cardioversion will be required prior to proceeding.

3.	(a)	False	Mitral stenosis is a fixed output state, and cardiac output would not be able to increase in the face of a reduction in afterload caused by spinal anaesthesia.
	(b)	False	Mitral stenosis typically has an insidious symptom profile. This patient's history is not reassuring, and further investigation is required.
	(c)	False	Fluid preloading is not indicated in mitral stenosis. The cardiac output cannot increase and so fluid loading will cause an increase in pulmonary vascular pressures and subsequent oedema.
	(d)	True	Symptoms of mitral stenosis are often difficult to illicit on history. A stress echocardiogram may provide an objective measure of functional ability of the heart to increase work in the face of increasing demand.
	(e)	False	Atrial fibrillation is very common in mitral stenosis. While rate control is important, cardioversion is unlikely to be successful.

4.	An active 72 year old lady is placed on the acute theatre list for hemiarthroplasty after falling and fracturing her neck of femur while ballroom dancing. A systolic murmur is detected during preoperative assessment. Echocardiography reveals severe mitral regurgitation with a left ventricular ejection fraction of 60% and preserved right ventricular function
(a)	Surgery needs to be delayed until a stress echocardiogram can be performed.
(b)	Spinal anaesthesia is unsafe in this patient.
(c)	Fluid boluses may be needed prior to induction, or to control hypotension.
(d)	Surgery can go ahead without further delay.
(e)	Surgery on the hip needs to be combined with mitral valve repair or replacement.

4.	(a)	False	While stress echocardiography will give a better idea of ventricular function. It is not necessary to delay this emergency surgery in an asymptomatic patient.
	(b)	False	Spinal anaesthesia causes a fall in afterload, which is beneficial in mitral regurgitation. It is generally well tolerated with the usual precautions, and would be a reasonable option for this surgery.
	(c)	True	The haemodynamic goals of mitral regurgitation include maintaining adequate preload and an assessment of the patient's fluid status is an important part of the preoperative consultation.
	(d)	True	While this patient likely has borderline mild LV impairment with an ejection fraction of 60%, the fact she is active and asymptomatic means she is fit for surgery.
	(e)	False	Mitral valve surgery does not need to be undertaken at this time. The patient is asymptomatic with a good LV, though she will require surveillance as any further fall in LV function may be an indication for surgery. This should not delay her hip surgery.

5.	<p>The obstetric registrar notifies you of a category 2 caesarean section on an 18 year old primip with an epidural in situ. She is known to have mitral stenosis from previous rheumatic fever. The delivery plan is for controlled epidural top-up which proceeds uneventfully. Immediately post-delivery she complains of difficulty breathing and becomes acutely dyspnoeic. Regarding this situation:</p>
(a)	Oxytocin has likely been administered too rapidly and this effect will be transient.
(b)	This patient requires immediate intubation and ventilation before she deteriorates further.
(c)	A fluid bolus should be one of the first measures to help stabilise the patient.
(d)	This patient should be placed head up and given 100% oxygen with positive end expiratory pressure.
(e)	This patient is likely to have a high block from the epidural top-up.

5.	(a)	False	While oxytocin needs to be administered cautiously in mitral stenosis, it's principal effect is vasodilation and hypotension which can be profound.
	(b)	False	This patient may require intubation as part of the management of this complication, but more conservative measures should be attempted first.
	(c)	False	This complication is the result of a massive autotransfusion overwhelming the ability of the heart to increase its cardiac output due to the mitral stenosis. Care always needs to be taken with fluid administration with any stenotic valve lesion.
	(d)	True	The release of inferior vena cava compression and autotransfusion from uterine contraction have combined to significantly increase preload. The presentation can be similar to amniotic fluid embolus, but given the patient's history the administration of oxygen with PEEP and a head up position should be the first line of treatment
	(e)	False	Epidural top-up needs to be conducted in a slow, steady fashion in patients with mitral stenosis. Unless the Anaesthetist has been overly enthusiastic with the boluses, this is an unlikely scenario. Of note, head up positioning will aid regression of the block if it cannot be excluded.