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Valvular heart disease for non-cardiac surgery – anaesthetic management

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Introduction

The incidence of heart disease ranges from 0.7% in the 18–44 age group and up to 13.3% in individuals 75 years or older.[1] In developed countries, the prevalence of valvular heart disease is estimated at 2.5%.[1]

A more recent review of the current evidence for the global burden of rheumatic fever (RF) and RHD estimates that 15.6–19.6 million people have rheumatic heart disease (RHD) (2.4 million children aged 5–14 years).[2] With the decline in RHD and the ageing population in the developed world, there has been a change in the disease patterns of valve lesions over the last few decades. Western populations are experiencing greater numbers of degenerative valve disease.

In the developing world, however, RHD remains an important cause of valve pathology.

Classification of valvular heart disease based upon the aetiology:

1. Congenital

Atresia, stenosis, malposition, abnormalities of valve structure (bicuspid valve)

2. Acquired

a. Endocarditis (regurgitation more common)
b. RHD: mitral stenosis (MS), mitral regurgitation (MR), aortic stenosis (AS), aortic regurgitation (AR)
c. Senile calcific AS
d. Myxomatous mitral valve prolapse leading to regurgitation

AS due to calcific disease and MR due to primary causes such as degenerative disease, or secondary causes such as ischaemic heart disease, are the most commonly encountered valvular lesions in Western countries.[3]

Aortic stenosis

AS can be congenital or acquired. Idiopathic senile degeneration with sclerosis and calcification of the valve due to chronic inflammation accounts for the majority of acquired segment of AS.

There is a clear association between the clinical risk factors for atherosclerotic disease and the development of AS, including the process of chronic inflammation.[4]

Greater mechanical stress with age and risk factors such as hypertension, smoking, diabetes and hypercholesterolaemia contribute to 2–4% of adults > 65 years of age suffering from acquired AS.[1]

Other causes are RHD and bicuspid aortic valve.

Pathophysiology

The normal aortic valve area (AVA) is 2.6–3.5 cm^2, with haemodynamically significant obstruction usually occurring at cross-sectional valve areas of 1 cm^2 or less.[5]

Haemodynamic consequences

The increased pressure load imposed by AS results in compensatory hypertrophy of the left ventricle (LV) without cavity enlargement (concentric hypertrophy), leading to diastolic dysfunction, increased left ventricular end-diastolic pressure (LVEDP) and subendocardial ischaemia.

With time, the ventricle can no longer compensate, causing secondary LV cavity enlargement, LV failure, reduced ejection fraction (EF), decreased cardiac output, and a misleadingly low gradient across the aortic valve (low-gradient severe AS).

Generally accepted criteria for critical outflow obstruction include a systolic pressure gradient greater than 50 mm Hg, with a normal cardiac output, and an AVA of less than 0.4 cm^2.[5]

Table I: Severity of aortic stenosis by valve area

Normal	2.6–3.5 cm^2
Mild	1.2–1.8 cm^2
Moderate	0.8–1.2 cm^2
Significant	0.6–0.8 cm^2
Critical	< 0.5 cm^2

Signs and symptoms

Patients with mild to moderate AS are usually asymptomatic as long as the hypertrophied LV compensates for an increasing pressure gradient.

Symptoms do not correlate well with the severity of the disease – patients with small valve areas may be asymptomatic.

However, once symptomatic, the prognosis is much worse, with increased risk of sudden death (survival < 2 years without surgery).

Classic symptoms

Exertional syncope.

Exertional dyspnoea.

Angina.

Congestive heart failure.

Examination findings

Pulsus parvus et tardus, soft S2.

Ejection systolic crescendo-decrescendo murmur, along left sternal border (LSB) radiating to upper right sternal border (RSB) and into carotids.

The pressure–volume correlation of AS is shown in Figure 1, which shows that due to an increase in aortic resistance, the LV pressures are increased during ejection with an increase in end-systolic volume and right shift of the loop.

Investigations

X-ray

Prominent ascending aorta due to post-stenotic dilatation.

Electrocardiographic (ECG)

Left ventricular hypertrophy (LVH)

Right bundle branch block (RBBB)

Left bundle branch Block (LBBB)

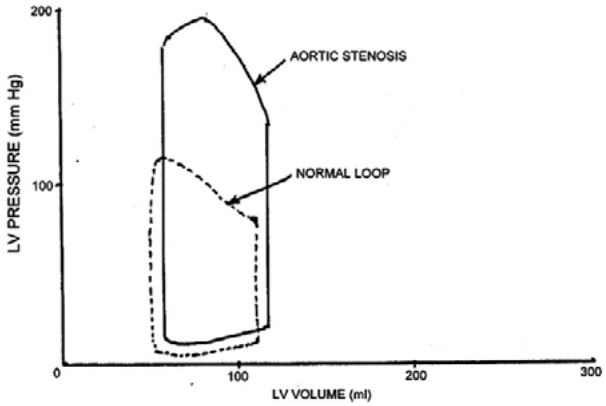

Figure 1: Pressure–volume loop in aortic stenosis[5]
LV – left ventricular

Echo

LVH; pressure gradient across the aortic valve is used to determine the severity of disease.

Value area measured.

Table II: Severity of AS by mean pressure gradient

Mild	12–25 mmHg
Moderate	25–40 mmHg
Severe	40–50 mmHg
Critical	> 50 mmHg

Anaesthetic considerations

Symptomatic patients with severe AS should have a cardiology referral for aortic valve repair (AVR) before elective non-cardiac surgery.[6]

Asymptomatic patients with moderate or severe AS and high-risk non-cardiac surgery should have a cardiology referral for AVR.[6]

In asymptomatic patients with moderate or severe AS and low-risk non-cardiac surgery, AVR risk may outweigh the risks of non-cardiac surgery.[6]

Anaesthetic management

Avoidance of systemic hypotension, maintenance of sinus rhythm and an adequate intravascular volume, and awareness of the potential for myocardial ischaemia.[5] (AS is a fixed cardiac output state and hypertrophied LV is sensitive to ischaemia).

Haemodynamic goals

Heart rate

Slow normal heart rate (HR) for adequate diastolic filling time.

Rhythm

Sinus rhythm – dependence of atrial kick – arrhythmias poorly tolerated.

Defibrillator and beta blocker should be ready.

Preload

Maintain to keep a filling pressure gradient, optimise intravascular fluid volume to maintain venous return and left ventricular filling.

Afterload

Maintain – difficult to compensate for decrease in systemic vascular resistance (SVR)

Intraoperative monitoring should include a standard five-lead ECG system, including a V 5 lead, because of the LV's vulnerability to ischaemia.[5]

Invasive monitoring such as arterial line placed before induction should be routine.

General anaesthesia (GA) offers good haemodynamic control. Neuraxial anaesthesia should only be used with extreme caution.

Intraoperative hypotension, regardless of the primary cause, should be treated immediately and aggressively with a direct α-adrenergic agonist such as phenylephrine.[5]

Patients should be monitored postoperatively in the intensive care unit (ICU) or high care (HC). Infusions of vasoconstrictors may be required postoperatively to maintain haemodynamic stability.

Aortic regurgitation

Aetiology

Most common – idiopathic.[7]

Other causes include congenital lesions, degenerative processes, and rheumatic disease, connective tissue disorders and chronic hypertension. These processes cause malcoaptation of the aortic valve leaflets by producing abnormalities in the leaflets themselves or dilation of the aortic valve annulus, the aortic root, or both.[7]

Acute AR: infective endocarditis (IE), aortic dissection, trauma.[7]

Haemodynamic consequences

Acute AR poorly tolerated can lead to acute left heart failure and pulmonary oedema.

Chronic AR develops slowly.

Left ventricular volume overload is the pathognomonic feature of chronic AR. The degree of volume overload is determined by the magnitude of the regurgitant flow.[4]

Volume-loaded LV leads to LV eccentric hypertrophy and massive LV dilatation, eventually decreased LV function.

Increased LVEDP, increased left atrial pressure (LAP) increased pulmonary artery capillary wedge pressure.

Symptoms of left heart failure.

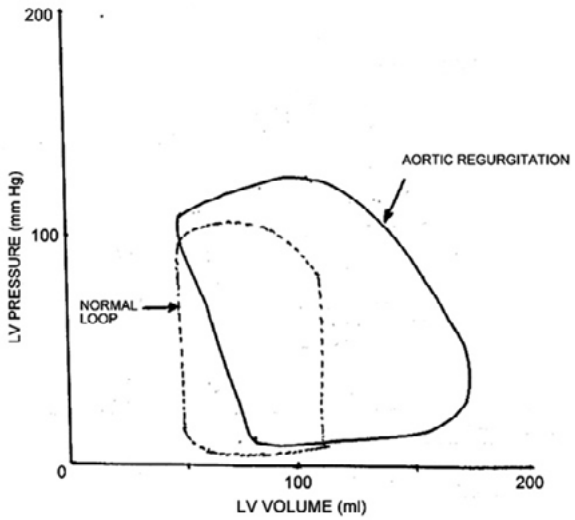

Figure 2: Left ventricular pressure–volume loop in aortic regurgitation[5]
LV – left ventricular

Signs and symptoms

Chronic AR is asymptomatic for many years.

Eventually presents with left heart failure.

Exercise intolerance, dyspnoea, paroxysmal nocturnal dyspnoea, orthopnoea.

Signs are due to increased stroke volume and widened pulse pressure.[8]

Collapsing pulse, (water hammer pulse), pistol shots over femoral arteries, capillary pulsations over tongue, lips and fingertips, wide pulse pressure and low diastolic pressures.

High-pitched diastolic murmur loudest in 2nd intercostal space (ICS) at the right sternal border, increased by manoeuvres that increase SVR such as squatting.

Associated Austin Flint murmur (physiological MS murmur due to AR jet hindering opening of mitral valve leaflets) – apical, mid-late diastolic rumble.[8]

Investigations

ECG

LVH

Chest X-ray

LVH, pulmonary congestion, may have dilated ascending aorta.

Echo

Defines mechanism of AR, severity, and repercussions.

Regurgitant volumes.

Mild AR < 20% of LV SV.

Moderate AR 20–40% of LV SV.

Severe AR > 60% of LV SV.

Risk

If asymptomatic and preserved LV function – little risk.

Symptoms of LV dysfunction – consider cardiology evaluation.

If LV EF < 30%, only perform non-cardiac surgery if strictly necessary and after optimising medical therapy for heart failure.[9]

Haemodynamic goals

Rate and rhythm

High normal HR to minimise time spent in diastole, which leads to a decreased regurgitant fraction. Maintain sinus rhythm.

Preload

Adequate volume loading.

Afterload

Controlled reduction in SVR (reduce regurgitant volume).[5]

Contractility

Maintain contractility and maintain adequate diastolic pressure.

No absolute contraindication to any anaesthetic technique.

Arterial line if CHF.

Mitral stenosis

Aetiology

Females, childbearing age group.

Most common cause of MS is RHD.

Other causes

Primary age-related degenerative valves and congenital mitral valvular abnormalities.

Pathophysiology

Initially asymptomatic for more than 10 years.

Symptoms rarely appear until the normal mitral valve area of 4–6 cm^2 has been reduced to 2.5 cm^2 or less.[5]

When the mitral valve area reaches 1.5–2.5 cm^2, symptoms usually occur only in association with exercise or other conditions such as fever, pregnancy, or AF, that lead to increases in HR or cardiac output.[5]

After the mitral valve area decreases to less than 1.5 cm^2, symptoms may develop at rest. Some patients are able to remain asymptomatic for long periods by gradually reducing their level of activity.[5]

Haemodynamic consequence

Obstructed flow across mitral valve.

Increased LAP.

Left atrial (LA) dilatation.

Atrial fibrillation (AF), thromboembolic complications.

Obstructed flow can lead to decreased filling of LV, Muscle atrophy, scarring

Left ventricular end-diastolic volume (LVEDV) and LVEDP are reduced with an accompanying decline in SV (Figure 3).

Decreased EF.

Increased LA pressure.

Increase in pulmonary venous and pulmonary arterial pressure.

Reactive pulmonary vasoconstriction with histological changes in medial and intimal layers.

Elevated pulmonary pressures.

Right ventricular hypertrophy (RVH).

Right ventricle (RV) dilatation and failure.

Table III: Severity of mitral stenosis by valve area

Normal valve area	4–6 cm^2
Symptom free until	1.5–2.5 cm^2
Moderate stenosis	1–1.5 cm^2
Critical stenosis	< 1 cm^2

Signs and symptoms

Usually well tolerated.

Symptoms include exertional dyspnoea (due to pulmonary congestion), fatigue (decreased cardiac output), palpitations AF, haemoptysis, and recurrent bronchitis.

Enlarged LA can cause dysphagia, hoarse voice.

Chest pain that simulates angina is present in a small number of patients with MS and may result from RVH rather than coronary artery disease (CAD).[5]

Systemic thromboembolisation occurs in 10–20% of patients with MS and does not appear to be correlated with the mitral valve area or LA size.[5]

On examination, there is low volume pulse with or without AF.

Mitral facies, raised jugular venous pressure (JVP.)

Pulmonary hypertension can lead to right ventricular heave.

On auscultation, there is tapping apex beat, loud S1 with opening snap and loud P2.

Mid diastolic murmur (MDM) with presystolic accentuation best heard at the apex at lateral decubitus position.

Investigations

ECG

AF, P mitrale.

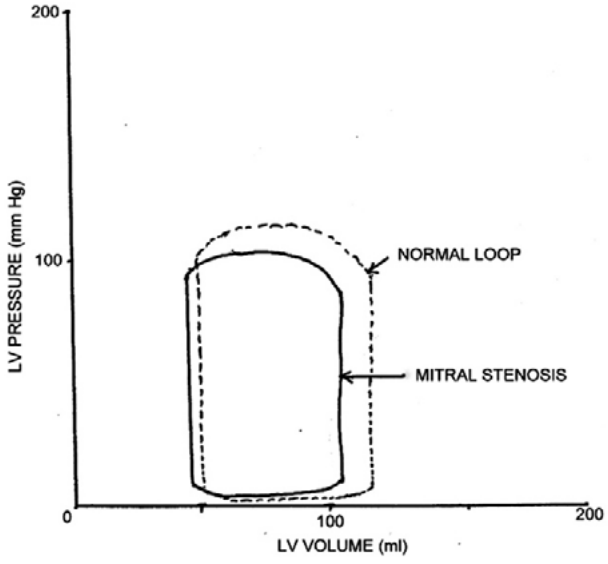

Figure 3: Left ventricular pressure–volume loop in mitral stenosis[5]
LV – left ventricular

Chest X-ray

LA enlargement.

Advanced disease enlarged RV and right atrium (RA).

Double shadow of LA (2nd heart shadow).

Displacement of barium-filled oesophagus on lateral film.

Pulmonary oedema.

Calcification of mitral valve (late sign).

Echo

Assess valve anatomy, calcification and measure gradient across the valve.

Examine for thrombus in LA.

Hockey stick-shaped valve.

Risk

Mild MS and good effort tolerance (ET), no pulmonary hypertension (PHPT)(pulmonary artery pressure < 50 mmHg) – minimal risk.[9] Can undergo less than high-risk surgery as long as HR controlled perioperatively with beta blockers.

Anaesthetic management

Like AS, MS represents a fixed cardiac output state.

GA offers the advantage of haemodynamic control, neuraxial anaesthesia can be hazardous.

Haemodynamic goal

Rate and rhythm

Sinus rhythm should be maintained.

Low normal HR for sufficient diastolic time for ventricular filling.

Preload

Aim for normovolaemia, keeping in mind that fluid boluses can worsen pulmonary oedema. Appropriate replacement of blood loss.[5]

Afterload

Maintain afterload.

Any reduction in SVR can cause a decrease in coronary perfusion pressures. Avoiding hypoxia, hypercapnia, and acidosis to prevent acute right ventricular decompensation.

Contractility

The LV will usually be unaffected.

The RV may need support if signs of failure are present.

Intraoperative management

Blunt intubation response.

Avoid ketamine – tachycardia.

Avoid N_2O – pulmonary hypertension.

Invasive haemodynamic monitoring – allows maintaining adequate preload while avoiding excessive fluid administration that could aggravate pulmonary vascular congestion.[5]

Symptomatic patients with pulmonary hypertension may benefit from pulmonary artery catheter (PAC) and/or transoesophageal echo (TOE) to assess biventricular function, pulmonary artery pressure and LA pressure.

Drugs to keep beta blockers, amiodarone – for sinus rhythm and slow HR.

Inotropes to support failing right heart: adrenaline, milrinone.

Maintain SVR with phenylephrine.

Inhaled nitric oxide or prostacyclin for pulmonary hypertension.

Mitral regurgitation

Aetiology

Organic and functional.

Organic

Degenerative processes that lead to leaflet prolapse with or without chordal rupture represent the most common cause of MR.[5]

Other causes of organic MR include IE, mitral annular calcification, rheumatic valve disease, and connective tissue disorders such as Marfan or Ehlers-Danlos syndrome.[5]

Functional

MR occurs despite structurally normal leaflets and chordae tendineae

CAD, idiopathic dilated cardiomyopathy, and mitral annular dilatation.

Haemodynamic consequence

In the early stages of MR, LVEDP is relatively normal because of compliance changes of the LV.

LV function and pressure tend to be normal in patients with chronic MR. With time, however, compensatory eccentric hypertrophy fails to preserve LV systolic function, and gradual systolic failure ensues, as noted on pressure-volume loops.[7]

EF is often normal or supranormal unless the ventricle has decompensated.

The LA is exposed to increases in volume and pressure. The LA dilate, and in the early stages of MR, preservation of near-normal LAP and protection of the pulmonary vasculature is likely.

Progressive LA enlargement commonly leads to AF.[7]

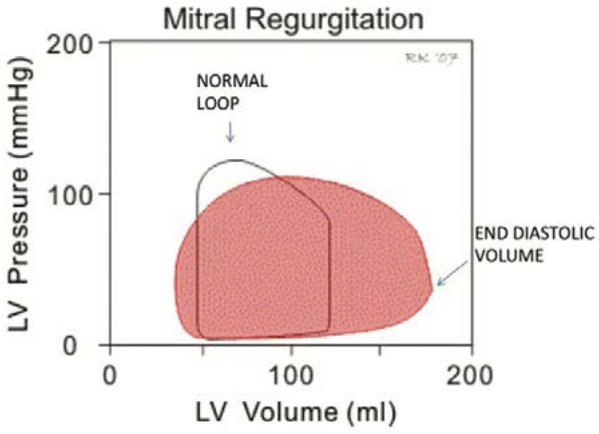

Figure 4: Left ventricular pressure–volume loop in mitral regurgitation
LV – left ventricular

Once LA compliance threshold reached, LA pressure increases leading to pulmonary vascular congestion, pulmonary hypertension, and right ventricular dysfunction.

Signs and symptoms

Mostly asymptomatic till significant cardiac damage.

Fatigue (low output), dyspnoea (pulmonary congestion), palpitations, AF, displaced apex (eccentric LVH).

Murmur: apical, pansystolic, radiating to axilla, +/- MDM, flow murmur into dilated LV in systole).

Soft S1 and prominent S3.

Signs of pulmonary hypertension and right heart failure (RHF), oedema.

Investigations

Chest X-ray

In patients with chronic MR, cardiomegaly due to LA or right-sided heart enlargement is visible.

ECG

AF is the most common ECG finding in patients with MR. Patients with AF can also present with more severe MR than patients without arrhythmia.

Echo

Assess degree of regurgitation, LA enlargement, look for thrombus in LA and evaluate for associated pulmonary hypertension in advanced disease.

Risk

Chronic MR and asymptomatic, no signs of CHF, usually tolerate non-cardiac surgery well.[10]

More severe MR and especially symptomatic patients scheduled for anything more than minor surgery should undergo further evaluation.[10]

MV surgery is rarely indicated in the patient with organic MR prior to elective non-cardiac surgery. However, patients with functional MR may warrant a different approach. If secondary to severe IHD, myocardial revascularisation and mitral annuloplasty may need to be considered prior to non-cardiac surgery.

Anaesthetic management

Rate and rhythm

High normal HR – reduced diastolic time, reduce regurgitant fraction and LV size.

Preload

Mild preload augmentation and maintenance is useful to ensure adequate forward stroke volume.

Afterload

Decrease afterload – decreases regurgitant volume and augments systemic perfusion.

Contractility

Should be maintained.

Early aggressive use of inotropes is needed if LVF is present.

Patients tolerate GA or neuraxial techniques. (Beware of patients on anticoagulation for AF.)

Avoid factors that may increase pulmonary hypertension.

Avoid bradycardia.

Care with cardio-depressant drugs: volatiles.

If very unstable, use an opioid-based anaesthetic.

Inotropes may be required if hypotension.

Use invasive monitoring in patients with preoperative symptoms and impaired myocardial function.

Monitor for CHF postoperatively especially if large intraoperative fluid shifts occurred.

Antibiotic prophylaxis

Patients with acquired valvular heart disease are at risk of developing IE. However, prophylaxis is no longer recommended for patients with underlying cardiac condition solely on an increased lifetime risk of acquisition of IE.

Prophylaxis is no longer recommended in patients with mitral valve prolapse or other valvular diseases without previous history of IE.[9]

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