# ANAESTHESIA FOR PATIENTS WITH CARDIAC DISEASE UNDERGOING NON-CARDIAC SURGERY

Dr Sock Huang Koh, Queen Elizabeth Hospital, Birmingham,
Dr James Rogers, Frenchay Hospital, Bristol

## INTRODUCTION

Major surgery stresses the cardiovascular system in the perioperative period. This stress leads to an increase in cardiac output which can be achieved easily by normal patients, but which results in substantial morbidity and mortality in those with cardiac disease. Postoperative events which cause death include myocardial infarction (MI), arrhythmias, and multiple organ failure secondary to low cardiac output. If the different mechanisms involved in different cardiac disease states are understood, then the most suitable anaesthetic can be given. The skill with which the anaesthetic is selected and delivered is more important than the drugs used. Previous articles on cardiovascular physiology and pharmacology (*Updates 10 & 11*) provide background information and should be read in conjunction with this article.

## ASSESSMENT OF PERIOPERATIVE RISK

The Goldman Cardiac Risk Index attempts to quantify the risk of adverse perioperative cardiac events (Table 1). The index scores each of a range of various conditions including cardiac disease, age and the nature and urgency of the proposed surgery. The total score predicts the likelihood of complications and death. For certain operations this risk can be minimised by avoiding general anaesthesia and using local anaesthetic techniques. Examples include peribulbar eye blocks for cataract surgery and brachial plexus blocks for upper limb surgery.

There have been more recent indices of risk, including one study of patients undergoing major elective non-cardiac surgery[1]. This identified six independent predictors of complications: high-risk type of surgery, history of ischaemic heart disease, history of congestive cardiac failure, history of cerebrovascular disease, preoperative treatment with insulin, and a raised serum creatinine.

*Table 1. Goldman Cardiac Risk Index*

| | |
|---|---|
| 3rd heart sound / elevated JVP | 11 points |
| MI within 6 months | 10 points |
| Ventricular ectopic beats >5/min | 7 points |
| Age > 70 years | 5 points |
| Emergency operation | 4 points |
| Severe aortic stenosis | 3 points |
| Poor medical condition | 3 points |
| Abdominal or thoracic operation | 3 points |

| Score | Incidence of death | Incidence of severe CVS complications |
|---|---|---|
| < 6 | 0.2% | 0.7% |
| < 26 | 4% | 17% |
| > 25 | 56% | 22% |

The American Heart Association and College of Cardiology have issued guidelines for perioperative cardiovascular evaluation for non-cardiac surgery[2], giving levels of risk to certain clinical markers, functional capacity, and types of surgery (Table 2). In addition to identifying the presence of cardiac disease, it is essential to determine severity, stability, and prior treatment of the disease.

It is important to remember that the above schemes to identify populations of high risk patients will not predict accurately the perioperative problems facing any particular individual. However, they do allow planning of perioperative care. Depending on available resources, this includes: (a) optimisation of medical treatment and specific perioperative drug therapy, (b) preoperative surgical treatment of ischaemic and valvular disease, and (c) use of postoperative intensive care facilities.

## ISCHAEMIC HEART DISEASE (IHD)

In developed countries 5-10% of patients presenting for surgery have some degree of ischaemic heart disease. Patients with IHD are at increased risk of perioperative myocardial infarction (MI), which is associated with an in-hospital mortality of some 30%. This is usually the consequence of "silent" myocardial ischaemia, that is ischaemia without the characteristic symptoms of angina. The strong association between postoperative silent ischaemia and other adverse cardiac events makes it important to use anaesthetic techniques which minimise the chance of such ischaemia developing.

### *Pathophysiology*

Ischaemic heart disease is the result of the build-up in larger coronary arteries of plaques of atheroma - consisting of cholesterol and other lipids. This causes narrowing of the vessels, restricting coronary blood flow. There may be insufficient myocardial blood supply during times of high demand eg exercise, leading to the effort related chest pain of stable angina. The more serious conditions of unstable angina (pain at rest), silent ischaemia and myocardial infarction are thought to be due to rupture of the atheromatous plaques causing thrombus formation, as well as vasoconstriction of the coronary vessels. There are several factors in the perioperative period which make these more likely:

- high levels of adrenaline and other catecholamines as a consequence of surgery, causing tachycardia, coronary vasoconstriction, and increasing platelet "stickiness".
- an increased tendency for blood to coagulate, making thrombosis in coronary vessels more likely.

*Table 2. Predictors of Cardiac Risk*

**Clinical Markers**

*Major predictors*: recent MI, unstable angina, untreated heart failure, significant arrhythmias and severe valvular disease.

*Intermediate predictors*: mild angina, history of MI, treated heart failure, and diabetes.

*Minor predictors*: old age, abnormal ECG, non-sinus rhythm, history of stroke, and uncontrolled hypertension.

**Functional Capacity**

This is a measure of the metabolic demands of various daily activities on the heart. For example, a patient who was breathless at rest, or after walking a short distance, would have a low functional capacity, which is a predictor of increased risk.

**Type of Surgery**

*High risk surgery*: major emergencies, aortic and vascular, peripheral vascular, and prolonged procedures particularly with fluid shifts and blood loss.

*Intermediate risk surgery*: carotid endarterectomy, head and neck, abdominal, thoracic and orthopaedic.

*Low risk surgery*: cataract, breast, and superficial procedures.

***Anaesthesia***

All anaesthetic techniques must aim to keep myocardial oxygen supply greater than demand, and therefore avoid ischaemia. The relevant factors are summarized in Table 3.

The essential requirements of **general anaesthesia** for IHD are avoiding tachycardia and extremes of blood pressure, both of which adversely affect the balance between oxygen supply and demand. These are discussed in detail below during each phase of an operation.

- **Pre-medication**. A nervous patient may be tachycardic and require an anxiolytic premedication. Beta-blockers also reduce tachycardia, and prevent perioperative myocardial ischaemia. A regime of intravenous atenolol followed by postoperative oral treatment resulted in a reduction in both morbidity and mortality for two years after surgery in IHD patients[3]. In a similar fashion, alpha2-agonist drugs such as clonidine reduce noradrenaline release from synapses, causing both sedation and analgesia, also a reduction in intraoperative myocardial ischaemia.

- **Induction**. All intravenous anaesthetic agents have a direct depressant action on the myocardium, and may also reduce vascular tone. This causes hypotension (especially in the hypovolaemic patient), often with a compensatory tachycardia,, which may cause myocardial ischaemia. In general all agents can be used safely if given slowly in small increments. However, ketamine is unique in causing indirect stimulation of the sympathetic nervous system, leading to both hypertension (increased afterload) and tachycardia. This will be dangerous for a patient with IHD and should be avoided.

- **Intubation**. Laryngoscopy is a powerful stressor, causing hypertension and tachycardia. This can be avoided with a supplemental dose of intravenous induction agent or opioid eg alfentanil, just prior to laryngoscopy.

- **Maintenance**. Volatile agents have minimal effects on cardiac output, although they do reduce myocardial contractility, especially halothane. They cause vasodilation, and isoflurane has been implicated in the 'coronary steal' syndrome. The theory is that pre-stenotic vasodilation diverts blood away from already ischaemic areas of the myocardium. However, there is doubt as to the clinical significance of this phenomenon. Vagal stimulation due to halothane can cause bradycardias and nodal rhythms. Bradycardias can be beneficial by allowing greater coronary diastolic filling, providing blood pressure is maintained. Ether, despite being a direct myocardial depressant, causes indirect sympathetic stimulation with tachycardia, and therefore can aggravate ischaemia.

*Table 3. Factors affecting myocardial oxygen supply and demand*

| Oxygen supply | Oxygen demand |
|---|---|
| *Heart rate* | *Heart rate* |
| - diastolic time | |
| *Coronary perfusion pressure* | *Ventricular wall tension* |
| - aortic diastolic blood pressure | - preload |
| | - afterload |
| - ventricular end-diastolic blood pressure | *Contractility* |
| *Arterial oxygen content* | |
| - arterial oxygen partial pressure | |
| - haemoglobin concentration | |
| *Coronary artery diameter* | |

**Preoperative assessment of IHD**

- The aim is to assess the severity of disease and the degree of impairment of myocardial function. The patient's exercise tolerance (functional capacity) and frequency of angina attacks are an indication of the severity of disease. Non-cardiac surgery is generally safe for patients with good exercise tolerance, even if they have minor or intermediate predictors of clinical risk (Table 1).
- The patient may already be on medication for angina or hypertension. These drugs include beta-blockers, nitrates, and calcium antagonists. These protect against the haemodynamic stresses of surgery and should be continued through the perioperative period. However, general anaesthesia may exaggerate the hypotensive actions of such drugs.
- An electrocardiogram (ECG) may show changes of a previous MI such as Q waves, or ST segment depression suggestive of ischaemia. However, a resting ECG may be normal in 50% of patients with IHD, and therefore cannot exclude serious underlying disease. An ECG will also detect conduction defects, ventricular hypertrophy, and arrhythmias such as atrial fibrillation. (*See "ECG Monitoring in Theatre" Update 11*).
- Anaemia is well tolerated in the general population, but can cause a critical reduction in myocardial oxygen supply in those with IHD - a haematocrit of 30% or more is recommended.
- Other investigations may be performed to supplement clinical findings, but may not be readily available
  - (a) an exercise ECG involves the patient exercising on a treadmill, therefore increasing myocardial oxygen demand and with ischaemia showing as ST segment depression
  - (b) patients who are unable to exercise may undergo pharmacologic stress testing - drugs are used to increase myocardial oxygen demand and radioisotope imaging techniques detect ischaemic areas
  - (c) an echocardiogram can detect abnormalities of ventricular wall movements, which are a sensitive indicator of ischaemia.

● **Analgesia**. High doses of opioids reduce the stressor response to surgery. Theoretically, non-steroidal anti-inflammtory drugs (NSAIDs) may have both a useful postoperative analgesic action and an anti-platelet effect which may reduce coronary thrombosis.

● **Reversal and recovery**. Reversal of muscle relaxation with a combined anti-cholinesterase/anti-muscarinic causes tachy-cardias, and extubation in itself is a stressor. Problems in the recovery phase which can cause ischaemia include; tachycardia, pain, hypothermia, shivering, hypoxia, and anaemia. These should be treated not just in the immediate postoperative period, but throughout the hospital admission. The use of supplemental oxygen in the postoperative period is one of the simplest, yet most effective measures in preventing myocardial ischaemia.

● **Monitoring**. As discussed above, the prime anaesthetic goals are to avoid tachycardias and extremes of blood pressure. It follows that it is most useful to monitor heart rate and blood pressure, also pulse oximetry to detect hypoxia. An ECG, if available, will give indications of arrhythmias, and ST segment depression may indicate ischaemia, although an observer will usually only detect the minority of such events. Rarely used techniques to detect ischaemia involve intraoperative trans-oesophageal echocardiography to assess ventricular wall motion abnormalities, and measuring serum troponin levels in the postoperative period.

The use of **regional anaesthetic** techniques has theoretical advantages: epidural anaesthesia reduces preload and afterload, coagulation responses, and in the case of thoracic epidurals, causes coronary vasodilation. These effects should reduce perioperative myocardial ischaemia, but this is not supported by research. However, good epidural analgesia may reduce the incidence of tachycardias arising due to postoperative pain. In a patient with IHD, local anaesthetic techniques such as brachial plexus block should be encouraged in order that the haemodynamic responses to general anaesthesia are avoided. However, even under local anaesthesia, the patient will be subject to the stresses of the surgical procedure itself, which can have marked haemodynamic effects.

## HEART FAILURE

Heart failure is the inability of the heart to pump enough blood to match tissue requirements. It occurs in 1-2 % of the population, rising to 10% in the over 75 year old age group, and is associated with increased mortality following anaesthesia. The commonest cause is ischaemic heart disease. Other causes include hypertension, valvular heart disease and cardiomyopathies. One third of untreated patients with an ejection fraction of less than 40% will die within a year.

### *Pathophysiology*

Cardiac output is lower in heart failure because stroke volume is reduced for the same left ventricular end-diastolic volume as compared to a normal heart. Starling's law of the heart demonstrates the relationship between ventricular end-diastolic volume and stroke volume (Figure 1). Since the failing heart has a limited ability to increase stroke volume, the only response to a greater preload is an increase in heart rate, which in turn can cause ischaemia. In addition, high end-diastolic ventricular pressures tend to oppose blood flow to the endocardium.

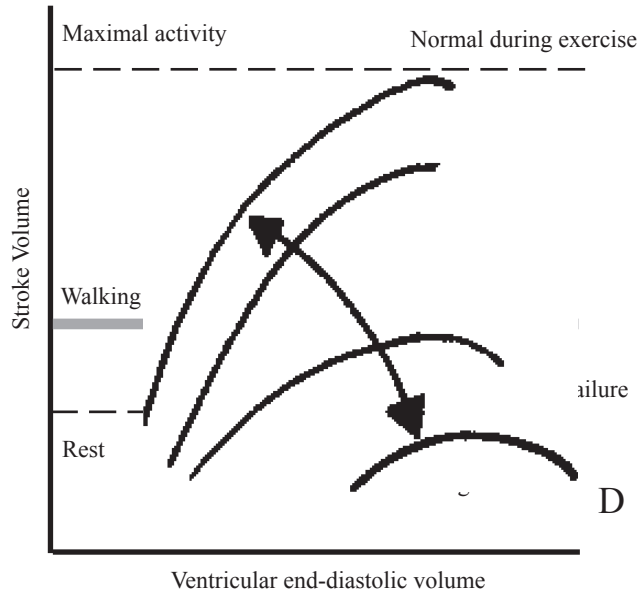


*Figure 1: Ventricular function*

Curves A and B illustrate the rise in cardiac output with increases in ventricular end-diastolic volume (pre-load) in the normal heart. Note that with an increase in contractility there is a greater cardiac output for the same ventricular end- diastolic volume.

In the diseased heart (C and D), cardiac output is less, and falls if ventricular end-diastolic volume rises to high levels, as in heart failure or overload.

***Preoperative assessment***

The aim is to assess disease severity and myocardial contractility. Limited exercise tolerance, orthopnoea, and paroyxsmal nocturnal dyspnoea are indicators of disease severity. Drug treatments may include ACE (angiotensin converting enzyme) inhibitors, diuretics and nitrates. In some patients with mild to moderate heart failure, cardioselective beta blockers may be used in an attempt to control the heart rate, but the risk is that they may block the low level sympathetic nervous activity which maintains contractility in the failing heart. Useful investigations are an ECG (looking for evidence of ischaemia), CXR, and, if available, an echocardiogram to assess ejection fraction. This is the proportion of end-diastolic blood volume ejected by the left ventricle during systole, and values of less than 30% equate to severe heart failure.

***Anaesthesia***

Safe anaesthesia for a patient in heart failure involves:

- Optimisation of ventricular filling - preload can be reduced with diuretics and nitrates, and both central venous and pulmonary artery pressures can be monitored. Trans-oesophageal echocardiography, if available, is a useful tool to visualize overall cardiac performance.
- Maintenance of myocardial contractility - in particular inotropes may be needed to oppose the cardiodepressant action of anaesthetic agents.
- Reduction of afterload by vasodilation, for example as a secondary effect of spinal or epidural anaesthesia. This not only reduces myocardial work, but helps maintain cardiac output. However, the benefit of such actions may be limited by falls in blood pressure which can compromise blood flow to vital organs such as the brain and kidneys.

**VALVULAR HEART DISEASE**

Pathology affecting valves on the left side of the heart is more common than on the right side. The same general principles of providing a haemodynamically stable anaesthetic apply as outlined in the section on ischaemic heart disease. In general, patients with valvular disease require antibiotic prophylaxis against infective endocarditis when undergoing certain operations (Table 4). Symptomatic regurgitant disease is usually better tolerated in the perioperative period than stenotic lesions, which sometimes need treatment such as valvotomy prior to non-cardiac surgery.

**AORTIC STENOSIS**

With a narrowed aortic valve, left ventricular (LV) outflow obstruction occurs. LV hypertrophy develops in compensation. This leads to reduced compliance, which is a reduction in ventricular wall movement for a fixed end-diastolic pressure. The eventual result is a fixed low cardiac output and inability to cope with systemic vasodilation. Coronary blood flow is also compromised due to the raised LV end-diastolic pressure. In addition to hypertrophy LV dilatation may also result, further reducing cardiac output. Aortic stenosis may result from rheumatic fever, often in association with the mitral valve. It may also be congenital, presenting in middle age, or be degenerative due to calcification in the elderly.

Symptoms of aortic stenosis usually occur when the valve area falls below 1$cm^2$ (normal 2-3$cm^2$), including angina, syncope on exertion, and symptoms of heart failure (dyspnoea, orthopnoea and paroxysmal nocturnal dyspnoea). Symptoms suggestive of aortic stenosis are angina and syncope on exertion, and symptoms of LV failure (dyspnoea, orthopnoea and paroxysmal nocturnal dyspnoea). An ECG will show signs of LV hypertrophy and strain, and an echocardiogram will assess (a) the pressure gradient between the LV and the aortic root, and (b) the LV contractility.

Anaesthesia may precipitate myocardial ischaemia or arrhythmias in patients with aortic stenosis. LV failure may also result. In the absence of treatment, sudden death occurs in 15-20%..

***Anaesthesia***

The aim is to maintain haemodynamic stability, in particular perfusion of the coronary vessels which are dependent on aortic root diastolic blood pressure. It is important to avoid reducing the systemic vascular resistance by vasodilation, but also not to cause excessive vasoconstriction. Tachycardia, myocardial depression and non-sinus rhythm (with consequent loss of the atrial contribution to ventricular filling) are all adverse factors.

*Table 4. Prevention of endocarditis in patients with heart-valve lesion, septal defect, patent ductus, or prosthetic valve. Guidelines from the British National Formulary 2002.*

**Dental procedures** *under local or no anaesthesia*,

- patients who have not received more than a single dose of a penicillin in the previous month, including those with a prosthetic valve (but not those who have had **endocarditis**), oral amoxicillin 3 g 1 hour before procedure; CHILD under 5 years quarter adult dose; 5–10 years half adult dose
- patients who are penicillin-allergic or have received more than a single dose of a penicillin in the previous month, oral clindamycin 600 mg 1 hour before procedure; CHILD under 5 years clindamycin 150 mg *or* azithromycin 200 mg; 5–10 years clindamycin 300 mg *or* azithromycin 300 mg
- patients who have had **endocarditis**, amoxicillin + gentamicin, as under general anaesthesia

**Dental procedures** *under general anaesthesia*,

- *no special risk* (including patients who have not received more than a single dose of a penicillin in the previous month),
  - *either* i/v amoxicillin 1 g at induction, then oral amoxicillin 500 mg 6 hours later; CHILD under 5 years quarter adult dose; 5–10 years half adult dose
  - *or* oral amoxicillin 3 g 4 hours before induction then oral amoxicillin 3 g as soon as possible after procedure; CHILD under 5 years quarter adult dose; 5–10 years half adult dose
- *special risk* (patients with a prosthetic valve or who have had **endocarditis**), i/v amoxicillin 1 g + i/v gentamicin 120 mg at induction, then oral amoxicillin 500 mg 6 hours later; CHILD under 5 years amoxicillin quarter adult dose, gentamicin 2 mg/kg; 5–10 years amoxicillin half adult dose, gentamicin 2 mg/kg
- patients who are penicillin-allergic or who have received more than a single dose of a penicillin in the previous month,
  - *either* i/v vancomycin 1 g over at least 100 minutes then i/v gentamicin 120 mg at induction or 15 minutes before procedure; CHILD under 10 years vancomycin 20 mg/kg, gentamicin 2 mg/kg
  - *or* i/v teicoplanin 400 mg + gentamicin 120 mg at induction or 15 minutes before procedure; CHILD under 14 years teicoplanin 6 mg/kg, gentamicin 2 mg/kg
  - *or* i/v clindamycin 300 mg over at least 10 minutes at induction or 15 minutes before procedure then oral or i/v clindamycin 150 mg 6 hours later; CHILD under 5 years quarter adult dose; 5–10 years half adult dose

**Upper respiratory-tract procedures**, as for dental procedures; post-operative dose may be given parenterally if swallowing is painful.

**Genito-urinary procedures**, as for *special risk* patients undergoing dental procedures under general anaesthesia except that clindamycin is not given, see above; if urine infected, prophylaxis should also cover infective organism.

**Obstetric, gynaecological and gastro-intestinal procedures** (prophylaxis required for patients with prosthetic valves or those who have had **endocarditis** only), as for genito-urinary procedures.

Spinal and epidural anaesthesia causes falls in systemic vascular resistance, and is therefore relatively contraindicated.

## AORTIC REGURGITATION

An incompetent aortic valve leads to retrograde flow of blood from the aorta to the LV during diastole. This results in LV dilatation and hypertrophy. Initially, there is an increase in stroke volume, but eventually aortic regurgitation results in LV failure and a low cardiac output state. Causes include ischaemic heart disease, degeneration, infection (rheumatic fever, syphilis, endocarditis), ankylosing spondylitis, aortic dissection, or it may be congenital. Symptoms of aortic regurgitation are the symptoms of LV failure. Angina may occur at a late stage. An ECG may show LV hypertrophy, with a large left ventricle on chest X-ray.

### *Anaesthesia*

It is important to avoid bradycardia as this increases the time for regurgitation and reduces forward flow and hence cardiac output. Peripheral vasoconstriction and increased diastolic pressure also increase the regurgitant flow. Conversely, vasodilation encourages forward flow of blood.

*Examples of causes of perioperative myocardial ischaemia*

| Preoperative | Intraoperative | Postoperative |
|---|---|---|
| Anxiety | Tachycardia | Pain |
| Pain | Extremes of blood pressure | Hypoxia |
| Hypovolaemia | Anaesthetic agents | Hypothermia |
| Inadequate drug treatment | Surgical stresses | Anaemia |
| | | Hypercoagulability |

**MITRAL STENOSIS**

Patients become symptomatic when the mitral valve area falls from the normal of 4-6cm$^2$ to 1-3cm$^2$. The obstruction leads to left atrial (LA) hypertrophy and dilation. LV filling is also reduced, hence reducing cardiac output. Within the pulmonary circulation, pulmonary vascular resistance increases due to pulmonary congestion, reducing lung compliance. Pulmonary hypertension results. Atrial fibrillation occurs in 50% of patients with mitral stenosis due to the LA enlargement. Causes of mitral stenosis are rheumatic fever, infection, inflammatory conditions and it may be congenital. Arrhythmias, pulmonary oedema and myocardial ischaemia can occur during anaesthesia.

Symptoms include those of LV failure, in particular dyspnoea and haemoptysis. ECG changes are those of LA (P mitrale) and perhaps RV hypertrophy. A chest X-ray may show LA enlargement and pulmonary oedema, and an echocardiogram can demonstrate the presence of left atrial thrombus. Patients with mitral stenosis may be on digoxin and warfarin, so a clotting screen should show appropriate values for the proposed surgery, and hypokalaemia should be avoided since it can cause digoxin toxicity.

***Anaesthesia***

The anaesthetic goals are to prevent tachycardia, which allows less time for diastolic flow through the stenosed valve, and to try to preserve sinus rhythm. In addition, it is important to maintain cardiac output and avoid hypovolaemia and vasodilation, which cause reduced atrial and ventricular filling. Raised pulmonary vascular resistance can inadvertently be further increased by hypercarbia and hypothermia, which should be avoided.

**MITRAL REGURGITATION**

The incompetent mitral valve allows retrograde flow of blood from the LV to the LA, resulting in LA dilation. Pulmonary oedema then develops. Atrial fibrillation occurs in severe cases. Rheumatic fever accounts for 50% of cases, with other causes including myocardial infarction (secondary to papillary muscle rupture), degenerative changes, bacterial endocarditis, ruptured chordae tendinae, and cardiomyopathies. The main symptom is dyspnoea. ECG changes are similar to those seen in mitral stenosis, and a chest X-ray may show cardiac enlargement and pulmonary oedema.

Mitral valve prolapse is the most common valvular abnormality, occurring in 3-8% of the population, in which one of the mitral valve leaflets prolapses into the left atrium. Mitral regurgitation and autonomic dysfunction may be associated with this condition, but otherwise there are no significant anaesthetic implications.

***Anaesthesia***

The aim is to maintain cardiac output and reduce the regurgitant flow. Therefore, myocardial depression and hypovolaemia should be avoided. Bradycardias and systemic vasoconstriction will increase regurgitant flow.

**Summary**

Patients with cardiac disease present for anaesthesia every day. Since their perioperative courses are associated with greater morbidity and mortality, it is important to provide a haemodynamically stable anaesthetic. This requires knowledge of the pathophysiology of the disease, and of the drugs and procedures and their effects on the patient.

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