Heart failure: pathophysiology, risk assessment, community management and anaesthesia

Alwyn Kotze MB ChB FRCA
Simon J. Howell MA FRCA MRCP(UK) MSc MD

Heart failure is a complex clinical syndrome caused by impaired ventricular performance. It is the final common pathway for a variety of cardiovascular disease processes, leading to potentially disabling symptoms and shortened life expectancy. Heart failure is common; currently, 1% of the population aged 50–59 yr, and 10% of those over 80 yr, have heart failure. Heart failure is the only major cardiovascular condition that is increasing in prevalence, because of an ageing population and improved survival from other cardiovascular diseases. Therefore, increasing numbers of patients with established heart failure will present for surgery.

A diagnosis of heart failure has serious implications. If the underlying cause cannot be rectified, heart failure alone has a 50% 4 yr-mortality, and 50% of patients with severe heart failure (those who are symptomatic and require frequent medical attention) will die within 1 yr of diagnosis. Decompensated heart failure is a major clinical predictor of perioperative risk. If heart failure is present at the time of major vascular surgery, the risk of cardiac death is 12 times higher than in patients without heart failure.

This article will describe the pathophysiology, diagnosis and anesthetic implications of heart failure in the setting of non-cardiac surgery.

Pathophysiology

Ischaemic heart disease and hypertension are the two most important causes of heart failure in the Western world. Other common causes include diabetes mellitus, valvular heart disease (especially aortic stenosis and mitral regurgitation) and other cardiomyopathies. Infective and nutritional causes are still common in the developing world. Advanced age and male gender are also risk factors (Table 1). The mechanisms underlying ventricular dysfunction are death or dysfunction of cardiac myocytes and longstanding pressure or volume overload. As myocardial contractility decreases, the stroke volume drops and the end-diastolic volume and pressure increase. This, according to the Frank-Starling law, will restore myocardial contractility and thus cardiac output. If sustained in the long-term, this volume increase leads to what is termed cardiac remodelling. This involves myocardial hypertrophy, chamber enlargement and an increase in ventricular wall stress, and increases oxygen demand. An increase in ventricular stiffness also occurs due to increased collagen deposition in the heart, which impairs filling and exacerbates the situation.

The decrease in cardiac output leads to sympathetic activation and stimulation of the renin–angiotensin–aldosterone (RAAS) system, with salt and water retention and an increase in circulating volume. This will initially restore cardiac output, again in accordance with the Frank–Starling law. However, it also compounds the injury to the heart and leads to a vicious cycle of increased and inefficient cardiac energy expenditure and reduced myocardial perfusion, particularly in the subendocardial region.

The combination of cardiac remodelling and the vicious neurohumoral cycle results in a myocardium vulnerable to ischaemia and a circulation which is dependent on sympathetic tone (Fig. 1).

Diagnosis and staging

The diagnosis of heart failure requires the presence of clinical signs and symptoms and objective evidence of ventricular dysfunction. The symptoms and signs may be difficult to interpret, and are not in themselves enough to make a diagnosis of heart failure. Furthermore, the pattern of symptoms is not always the same.
Some patients have functional limitation but little evidence of fluid retention, whereas others complain primarily of oedema and report few symptoms of dyspnoea or fatigue. The patient with symptoms suggestive of ventricular dysfunction should be investigated as a matter of course.

A normal ECG is very unusual in patients with heart failure, and should prompt review of the diagnosis. Key ECG findings are chamber hypertrophy, ventricular strain patterns and signs of ischaemia or previous infarction. Atrial fibrillation is also common in heart failure patients.

Echocardiography is a key investigation, as it allows assessment of severity of myocardial dysfunction, and may also diagnose the cause (e.g. valvular disease, hypertrophic cardiomyopathy). It is technically challenging and very operator-dependent. In particular, assessment of ventricular function is often subjective.

More detailed investigations of cardiac function and structure (e.g. cardiac MRI, coronary catheterization, radionucleotide scans, assay of serum Brain Natriuretic Peptide) may provide additional information.

The American College of Cardiology and American Heart Association (ACC/AHA) guidelines for heart failure management include a four-stage classification that emphasizes interventions to slow disease progression (Table 2).

### Risk assessment

#### Surgery-specific risk factors

A commonly used classification of surgical risk is detailed in the ACC/AHA guidelines. Surgery is classified as low-risk and intermediate risk, corresponding to the potential for perioperative haemodynamic changes, fluid shifts and the likely severity of the stress response to surgery. In the 2007 guidelines, open aortic surgery and peripheral vascular surgery are considered as high-risk (Table 3).

#### Patient risk factors

Detailed enquiry into the patient’s functional capacity gives an indication of their cardiovascular reserve, unless exercise capacity is compromised by disease of other systems (e.g. pulmonary, peripheral vascular, musculoskeletal). Limitation of exercise tolerance to less than that required to climb one flight of stairs without rest (exercise that equates to four metabolic equivalents or METS) is a recognized marker for cardiovascular disease and a predictor of risk. Perioperative cardiovascular risk is inversely proportional to maximal functional capacity. Objective measurement of maximal oxygen consumption with treadmill or bicycle exercise testing may be useful in patients with poor functional capacity, or if high-risk surgery is planned.

An echocardiogram provides measurements of both systolic and diastolic function. The left ventricular ejection fraction (LVEF) is the most commonly used measure of systolic function. An LVEF >50% is regarded as good, <40% as abnormal and <30% as an indication of severe LV dysfunction. A decreased LVEF has been found to be a predictor of perioperative mortality and morbidity, with the highest risk group being those with an LVEF <35%. Accurate calculation of the LVEF is operator-dependent, and requires that the ventricle be uniform in shape. Subjective comments on ventricular motion may be all that is possible in some cases. Currently, the ACC and AHA do not recommend perioperative transoesophageal echocardiography solely to predict risk. It gives no additional information over transthoracic imaging, but may offer improved image quality if the transthoracic sonographic windows are poor (e.g. in the obese or in ventilated patients).

#### Medical management

The vicious neurohumoral cycle of RAA system activation, decreasing cardiac output and cardiac remodelling is the target for the long-term treatment of heart failure.
The only ‘cure’ for early heart failure remains definitive treatment of the cause, or heart transplantation if the disease has progressed. Medical management is directed at decreasing symptoms and preventing disease progression.3 Drugs used in the treatment of heart failure have significant implications for perioperative care.

Angiotensin-converting enzyme inhibitors

Angiotensin-converting enzyme (ACE) inhibitors are recommended as first-line treatment for the patient with left ventricular dysfunction, with or without symptoms. They have been shown to decrease the risk of myocardial infarction and death in patients with symptomatic heart failure.2 Patients may also be taking ACE inhibitors for the treatment of hypertension without heart failure.

There is continuing controversy over how to manage perioperative ACE inhibition.2 On the one hand, there is concern over severe hypotension in patients taking ACE inhibitors, when the raised sympathetic tone of heart failure is attenuated during anaesthesia. This was reported in 22% of patients in one study, if the ACE inhibitor was given less than 10 h before surgery.6 On the other hand, there is evidence that recovery of the RAA system can be quick, and deleterious to both the myocardium and kidneys,2 and that hypotension associated with ACE inhibitors and general anaesthesia is relatively easy to rectify.7 In addition, there is currently no parenteral formulation of an ACE-inhibitor available. 

A decrease in venous return or co-administration of negative inotropic drugs such as amiodarone may exaggerate hypotension under anaesthesia, whether ACE inhibitors are withdrawn or not.2 Hypotensive episodes during anaesthesia are first treated with conventional vasopressors or sympathomimetics and cautious expansion of intravascular volume. Rarely, particularly in association with high-dose ACE inhibitor treatment and neuraxial blockade, an

---

**Table 2** ACC/AHA guidelines for long-term management of heart failure in adults. AT1, angiotensin 1 receptor

<table>
<thead>
<tr>
<th>Stage A</th>
<th>Stage B</th>
<th>Stage C</th>
<th>Stage D</th>
</tr>
</thead>
<tbody>
<tr>
<td>At high risk for HF but without structural heart disease or symptoms of HF.</td>
<td>Structural heart disease but without signs or symptoms of HF.</td>
<td>Structural heart disease with prior or current symptoms of HF.</td>
<td>Refractory HF requiring specialized interventions.</td>
</tr>
<tr>
<td>Patients with:</td>
<td>Patients with:</td>
<td>Patients with:</td>
<td>Patients who have marked symptoms at rest despite maximal medical therapy (e.g., those who are recurrently hospitalized or cannot be safely discharged from the hospital without specialized interventions).</td>
</tr>
<tr>
<td>- Hypertension</td>
<td>- Previous MI</td>
<td>- Known structural heart disease</td>
<td></td>
</tr>
<tr>
<td>- Atherosclerotic disease</td>
<td>- LV remodeling including LVH and low EF</td>
<td>- Shortness of breath and fatigue, reduced exercise tolerance</td>
<td></td>
</tr>
<tr>
<td>- Diabetes</td>
<td>- Asymptomatic valvular disease</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>

**GOALS** - Treat hypertension - Encourage smoking cessation - Treat lipid disorders - Encourage regular exercise - Discourage alcohol intake, illicit drug use - Control metabolic syndrome

**DRUGS** - ACEI or ARB in appropriate patients (see text) - Beta-blockers in appropriate patients (see text)

**THREATS**

**GOALS** - All measures under Stage A and B - Dietary salt restriction

**DRUGS FOR ROUTINE USE** - Diuretics for fluid retention - ACEI - Beta-blockers

**OPTIONS** - Appropriate measures under Stages A, B, C - Decision re: appropriate level of care

**TABLE 1** ACC/AHA guidelines for long-term management of heart failure in adults. AT1, angiotensin 1 receptor

Reprinted with permission.

Table 3 ACC/AHA classification of surgical risk

<table>
<thead>
<tr>
<th>Risk stratification</th>
<th>Procedure examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vascular (High) (reported cardiac risk often more than 5%)</td>
<td>Aortic and other major vascular surgery</td>
</tr>
<tr>
<td>Intermediate (reported cardiac risk generally 1% to 5%)</td>
<td>Peripheral vascular surgery, intraperitoneal and intrathoracic surgery, carotid endarterectomy, head and neck surgery, orthopaedic surgery, prostate surgery</td>
</tr>
<tr>
<td>Low (reported cardiac risk generally less than 1%)</td>
<td>Endoscopic procedures, superficial procedures, cataract surgery, breast surgery, ambulatory surgery</td>
</tr>
</tbody>
</table>


infusion of phenylephrine, norepinephrine or epinephrine may be needed.2

Another concern over the perioperative use of ACE inhibitors is ACE-inhibitor-induced acute renal failure. This occurs when glomerular afferent arteriolar blood flow is reduced (e.g. hypovolaemia), and the glomerular filtration rate (GFR) therefore becomes dependent on angiotensin-II-mediated efferent arteriolar vasoconstriction. GFR in an ACE-inhibitor treated kidney is blood pressure-dependent, and most ACE inhibitors are renally excreted. This can cause a vicious cycle of accumulation and further hypotension, resulting in renal failure.8 Regular monitoring of renal function is recommended, particularly on initiation of treatment and during hospital admissions.3

If significant fluid or blood loss is anticipated, or if the patient develops renal dysfunction in the postoperative period, stopping ACE inhibitors may theoretically be beneficial. In this situation, substitution with oral nitrate and hydralazine therapy may provide comparable cardiac benefits, although potentially at the cost of exacerbating hypotension.

The decision whether to continue or stop ACE inhibitors in the perioperative period must be judged on a case-by-case basis. It may be safe to continue therapy unless major blood loss is anticipated, the patient becomes hypovolaemic, or renal dysfunction develops.

Angiotensin receptor antagonists

These drugs are indicated as an alternative in symptomatic heart failure patients who are intolerant to ACE inhibitors, or in combination where ACE inhibitors and beta-blockers do not give a resolution of symptoms.3 Angiotensin receptor (AT) blockers are relatively long-acting competitive inhibitors of the actions of angiotensin II at the AT1 receptor.2

During anaesthesia, blood pressure may become angiotensin-dependent, particularly in hypovolaemic patients.9 As is true for patients on ACE inhibitors, those taking AT blockers are thus more prone to hypotension during anaesthesia. These hypotensive spells may be resistant to first-line sympathomimetics where high-dose AT blockers have been used preoperatively and drugs such as norepinephrine or vasopressin are sometimes required in this situation.2 The hypotension is also sometimes associated with bradycardia, which has led some authors to recommend prophylactic glycopyrrolate before anaesthesia in AT-blocked patients.2 11 The same concerns as with ACE inhibitors over ARF in patients with hypovolaemia or renal dysfunction apply.10

A decision to continue AT blockers in the perioperative period may be made subject to the same cautions as with ACE inhibitors. If an AT-blocker is withdrawn perioperatively, the drug should be stopped at least 24 h before surgery, as the duration of action is prolonged.2

Beta-adrenoceptor antagonists

All patients with chronic, stable heart failure and without contraindications should be considered for treatment with beta-adrenoceptor antagonists (beta-blockers). The beta-blockers now recommended for use in chronic heart failure are metoprolol, bisoprolol and carvedilol.11 These drugs reduce hospital admissions, prevent disease progression and improve long-term survival in heart failure after cardiac ischaemia.3

There is consensus that withdrawing long-term beta-blocker treatment in the perioperative period is harmful.2 4 Therefore, substitution with intravenous beta-blockers should be considered if prolonged fasting is anticipated. There is much less clarity about whether new beta-blockade in the perioperative period is indicated. While there is some evidence that it is useful for the prevention of cardiac ischaemia and arrhythmias in certain patient groups,4 there is no equivalent evidence for heart failure.

While beta-blockers are helpful in the long-term, in the short-term they limit the sympathetic response. This can prevent compensation for hypovolaemia, sepsis or the myocardial depressant effects of many anaesthetic agents. Therefore, hypotension is frequent and relatively resistant to simple sympathomimetics. Should inotropic support be necessary, phosphodiesterase inhibitors may prove useful as second line after beta-agonists.12

Diuretics

Diuretics are very useful for symptom control, and enhance the antihypertensive effects of ACE inhibitors and beta-blockade, but there is no strong evidence that diuretic therapy improves survival in heart failure.3 Chronic therapy can lead to electrolyte abnormalities, hypovolaemia and arrhythmias, particularly in the elderly.2
This can exacerbate heart failure. Conversely, patients who are nil by mouth and therefore omitting diuretics may retain fluid and also develop pulmonary congestion. Close attention to perioperative fluid and electrolyte balance is essential whether diuretics are continued or stopped.

**Digoxin**

Digoxin is most often used for symptom control in patients who are still symptomatic despite treatment with ACE inhibitors, beta-blockers and diuretics. It is also used for the management of the ventricular rate in AF, a common problem in heart failure. Digoxin is the only positively inotropic drug that does not increase mortality when used as maintenance heart failure treatment. Though digoxin confers no long-term survival benefit, withdrawal of digoxin therapy may increase the incidence of acute heart failure exacerbations.

Digoxin has a narrow therapeutic index, and digoxin toxicity can be difficult to diagnose and treat. Conditions that make digoxin toxicity more likely include hypomagnesaemia, hypercalcemia, and hypokalaemia, all of which may occur during the perioperative period. Digoxin therapy has also been found to be associated with mortality and serious morbidity in patients undergoing urgent or emergency surgery, even after adjusting for the severity of heart failure.

There are published recommendations both to continue and stop digoxin therapy before surgery. If digoxin is stopped perioperatively, heart rate control and positive inotropy should be maintained with other drugs. Should perioperative digoxin therapy be considered essential, close monitoring of serum electrolytes and digoxin concentrations is necessary.

**Other inotropes**

An increase in intracellular cyclic AMP may be achieved with beta-agonists or phosphodiesterase inhibitors. As the long-term use of these drugs has been found to be associated with worse survival, they are reserved for patients with severe heart failure. Patients receiving inotropic support are likely only to present to the anaesthetist in the intensive care setting or in specialist centres, for consideration of transplant or ventricular reduction surgery.

**Timing of surgery**

The correct timing of elective non-cardiac surgery is important to decrease patient risk. The ACC/AHA guidelines identify decompensated or untreated heart failure as a major clinical predictor of risk. Where possible, surgery should be postponed for the purpose of medical therapy, modification of risk factors and further investigations (including coronary angiography, if appropriate). Compensated heart failure, or a history thereof, represents an intermediate clinical predictor. Low-risk surgical procedures can safely continue, but formal assessment of the patient’s functional capacity should be considered if intermediate risk or vascular surgery is planned.

**Anaesthetic management**

The anaesthetic management of patients with heart failure depends on (i) the degree of heart failure, (ii) the cause and nature of the cardiomyopathy and (iii) the surgical procedure.

Anaesthesia for patients with Stage A and B heart failure centres on avoiding drug interactions with their long-term therapy, and maintenance of the benefits of such therapy throughout the perioperative period. Drugs that worsen heart failure should be avoided if possible, and close attention should be paid to fluid balance, analgesia, maintenance of normothermia and sinus rhythm, and optimal treatment of any co-morbid conditions.

Patients with Stage C and D heart failure present significant challenges of perioperative fluid and medical management in addition to the challenges of anaesthesia. There is little evidence that any one anaesthetic technique is superior to another in this group.

‘Stable’ anaesthesia, with as little myocardial depression or change in afterload as possible, is a reasonable goal for patients with heart failure. Traditionally, cardiovascularly ‘stable’ agents such as etomidate and benzodiazepines have been advocated, both for induction and maintenance. There is little evidence for this, and the use of most i.v. induction drugs and volatile agents have been described in heart failure patients.

The majority of patients with disabling heart failure (Stage D, or symptomatic Stage C) are dependent on their preload to maintain ventricular filling. Many patients with heart failure also rely on increased sympathetic tone to maintain tissue perfusion and cardiac output. Induction, emergence and fluid shifts during the perioperative period may cause the pre- or afterload to move outside the narrow range in which the patient functions. Though there is no strong evidence published that invasive arterial and central venous monitoring improves outcome, information from these monitors may help the anaesthetist to anticipate or limit these changes. In addition, a means of estimating cardiac output may prove useful.

Regional techniques are an alternative to general anaesthesia for certain procedures. The sympatholysis associated with neuraxial blockade may cause severe hypotension, especially if ACE inhibitors or AT blockers have been continued preoperatively. However, a moderate reduction in afterload may increase cardiac output without increasing oxygen demand. The superior analgesia associated with well-functioning regional techniques may also limit hypertensive spells, tachycardia and cardiac ischaemia postoperatively. Peripheral nerve block techniques have similar advantages and disadvantages. Despite these considerations, there is no strong evidence that either regional or general anaesthesia leads to improved survival after surgery in patients with heart failure.

**References**


Please see multiple choice questions 10–13