

Perioperative cardiac care - not just systolic

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Introduction

Revised guidelines for the management of heart failure (HF) were published in 2013.¹ HF arises due to a reduction in cardiac output secondary to a reduction in stroke volume (SV). Systolic HF (SHF) arises due to overfilling of a dilated left ventricle (LV) and/or right ventricle with inadequate ejection, and thus a reduced ejection fraction (EF). SHF progressively limits activity and results in increasing back pressure and secondary congestion of the pulmonary and/or systemic venous circulation.² However, the majority of patients with HF have preserved or even exaggerated systolic function, with a normal or increased EF owing to diastolic heart failure (DHF). SV is reduced because of underfilling of a stiff LV. Sudden-onset dyspnoea in response to stress or exercise occurs as a result of raised LV end-diastolic pressure (LVEDP). Congestion owing to an intermittent or persistent increase in LVEDP is thus an early, prominent feature of diastolic dysfunction.³

Pathophysiology

Systolic heart failure

Therapy for SHF is reviewed comprehensively in the American Heart Association (AHA) guidelines.¹ Positive inotropes increase metabolic demand on remaining myocytes, resulting in accelerated apoptosis. Mortality is increased when positive inotropes are used in the management of acute or chronic SHF.⁴

Systolic function can be markedly improved by cardiac resynchronisation therapy by means of dual chamber pacing and/or placement of an implantable cardioverter-defibrillator. These devices should be interrogated preoperatively and a management plan devised in conjunction with the inserting cardiologist.⁵

Forward flow from the failing ventricle may be reliably improved by insertion of a ventricular assist device. Despite expense and morbidity, these devices are being used more frequently.⁶

Diastolic heart failure

The pathophysiology of DHF⁷ is markedly different from that of SHF. The LV in a patient with diastolic dysfunction is likely to be hypertrophied (because of hypertension) and prone to ischaemia (as a result of coronary artery disease), resulting in failure of relaxation (decreased lusitropy). SV is reduced by limitation of filling and is crucially dependent on preload. EF is maintained or even increased, leading to a possible misdiagnosis of good systolic function if diastolic dysfunction is not assessed.

Diagnosis of heart failure

Clinical history

A subtle feature of the clinical history⁸ is progressive dyspnoea and a reduction in the effort tolerance with SHF, compared with normal activity below a certain threshold in DHF. Assessment of effort tolerance should be made using the AHC/American College of Cardiology Foundation (ACCF) quantification of metabolic equivalents.¹

Examination

During examination,⁸ the presence of peripheral and pulmonary oedema should be assessed. Blood pressure (BP) is typically reduced with SHF, but normal or more commonly elevated in DHF. SHF results in dilation as a prominent feature with a diffuse apex. DHF will result in an undisplaced apex with a hyperdynamic quality.

Investigation

Basic investigations⁸ include:

- *Electrocardiogram (ECG)*: An ECG will reveal rhythm abnormalities, ventricular hypertrophy, as well as an old infarction.
- *Urea and electrolytes*: Hyponatraemia < 130 mmol/l predicts poor prognosis in SHF.
- Hypo- or hyperkalaemia may arise, either primarily or due to therapy.
- An independently raised urea indicates overdiuresis.
- *Chest X-ray*: A chest X-ray will reveal pulmonary oedema⁹ as well as other pathologies, such as pneumonia, tuberculosis or pericardial or pleural effusions.

Brain natriuretic peptide levels

Brain natriuretic peptide (BNP)¹⁰ and N-terminal pro-brain natriuretic peptide are raised in heart failure, but not in chronic obstructive pulmonary disease, thus differentiating cardiac from respiratory dyspnoea. Declining levels are a reassuring sign of effective therapy, whereas persistent or rising levels are a cause for concern.

Echocardiography

Systolic dysfunction will be easily revealed on echocardiography¹¹ (ECG) by a dilated and poorly contractile LV.

Diastolic dysfunction is much more difficult to diagnose relying on assessment of LV inflow (E/A ratio). A normal EF does not exclude a diagnosis of DHF, but is actually confirmatory.

Stress tests

A six-minute walk test may be performed in the ward. Distances < 200 m indicate poor prognosis, while > 300 m indicates good prognosis.¹²

Distances of 2-300 m require further testing, such as a dobutamine stress ECG. A positive response to dobutamine improves prognosis, while failure to respond, or a response with ischaemia, worsens prognosis.¹³

Treatment of diastolic heart failure

Beta blockers

Patients who are hypertensive and who have DHF require beta blockers which reduce myocardial contractility, reducing LVEDP. A slower heart rate lengthens diastolic time, maximises filling and improves ejection, thus reducing congestion.¹⁴ Beta blockers should not be commenced in the immediate perioperative period because of an increased risk of strokes.¹⁵

Vasodilation

Initial vasodilation is best achieved with nitrates. A positive response to nitrates is indicative of a favourable response to diuretics.¹⁶

Long-term vasodilation is best achieved with angiotensin-converting enzyme (ACE) inhibitors or angiotensin-receptor blockers (ARBs).¹⁷

Labetalol is a very useful drug in the intensive care unit (ICU) setting, combining both alpha (vasodilation) and beta-blocking effects.¹⁸

Diuretics

Loop diuretics are effective decongestants, particularly of the pulmonary circulation, mainly through their venodilator effects.¹⁹ Excessive use of diuretics reduces preload, SV and forward flow. An adverse effect on the renal function is seen in the cardiorenal syndrome.²⁰

Warning of excessive diuresis is persistently elevated jugular venous pressure (JVP) or central venous pressure (CVP), confirmed by a rising urea with a static creatinine.

Perioperative heart failure

The ACCF/AHA guidelines for assessment of cardiac patients for non-cardiac surgery recognises the significant effect of heart failure²¹ on postoperative mortality.

The management strategy for DHF differs markedly to that for SHF in the coronary care unit and cardiac ward, so is also likely to differ in the operating room and ICU.

Preoperative decision-making

Stable patients with previous heart failure

Patients with a previous episode of HF who are currently stable on therapy should have their therapy continued in the perioperative period. There is some controversy surrounding the perioperative administration of ACE inhibitors and ARBs.²² Patients undergoing cardiac or major vascular surgery, who are likely to require inotropic and/or vasopressor support, should have these medications withheld. The majority of patients should continue to use these agents.

Beta blockers should not be stopped perioperatively owing to the risk of myocardial ischaemia.²³

Patients with acute heart failure requiring surgery

Patients with acute heart failure requiring surgery²⁴ should be treated as follows:

Patients with end-stage SHF who are not candidates for transplant or a LV assist device may not be able to have surgery, and may require palliative care.²⁵

Patients with acute DHF may respond very rapidly to therapy. A period of 4-6 hours is likely to be of great benefit, using therapy with labetalol, with the addition of nitroglycerine, if tolerated.

Intraoperative management

Systolic dysfunction

A neuraxial block in systolic dysfunction²⁶ results in vasodilation and sympathectomy that are beneficial to forward flow. Hypotension should be managed with vasopressors, rather than fluids. General anaesthetic drugs are cardiovascular depressants, so the lowest effective doses of the least depressant agents should be chosen. Propofol and the synthetic opioids must be avoided. Low-dose ketamine for induction and maintenance is extremely useful, especially with the addition of nitrous oxide. The volatile agent of choice is desflurane, owing to minimal tissue absorption.

Postoperative analgesia should encompass a multimodal approach, including local anaesthetics, paracetamol and an opioid (tramadol, morphine or oxycodone). Non-steroidal anti-inflammatory drugs (NSAIDs) should be avoided because of the risk of fluid overload and renal dysfunction.

Diastolic dysfunction

Neuraxial blocks are also beneficial for patients with diastolic dysfunction,²⁷ as long as preload is maintained. General anaesthetic agents should be chosen to limit sympathetic activation and provide systemic and pulmonary vasodilation. The alpha 2 agonists are ideal baseline drugs for DHF. Propofol and synthetic opioids such as remi- and sufentanil provide titratable anaesthesia and analgesia. Desflurane may cause a tachycardia if the underlying sympathetic tone is high. Sevoflurane is neutral, while isoflurane provides vasodilation. All of the volatile agents provide myocardial protection through preconditioning. Postoperative analgesia is similar to that with SHF. NSAIDs may be used in the absence of contraindications, particularly renal dysfunction.

Postoperative care

The management of systolic dysfunction in the postoperative period is a continuation of preoperative therapy.

Patients may develop diastolic dysfunction for the first time in the postoperative period. The presence of basal crepitations, signs of pulmonary oedema on chest X-ray, and a normal EF on ECG, often lead to the institution of high-dose diuretic therapy, despite a normal or elevated BP.

Diuretics reduce preload and thus filling of the LV, with worsening of ejection and persistence, or worsening of symptoms. JVP and CVP remain elevated, urea rises independent of creatinine, and BNP levels rise.

These patients require:

- Cessation of diuretics.
- Institution of labetalol 5-10 mg stat, followed by 1-5 mg/minute (60-300 mg/hour).

- The addition of nitroglycerine 5 µg/minute (300 µg/hour) to a maximum of 20 µg/minute (1 200 µg/hour) with normotension and without tachycardia.
- Oral therapy with a beta blocker and ACE inhibitor or ARB should be commenced in consultation with a cardiologist.
- As the oral drugs take effect, the labetalol and nitroglycerine are weaned off.
- After stabilisation, the patient should be further evaluated. Coronary artery disease may require revascularisation. A statin and low-dose aspirin should be commenced, smoking should be stopped and advice given on diet and exercise.

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