The role of surgery in acute heart failure

Stephen Westaby

Abstract

Acute heart failure is a syndrome of myocardial dysfunction whereby systemic blood flow falls to levels inadequate to preserve end organ function. Many cases are superimposed upon chronic heart failure. Eleven per cent die during the acute hospital admission and 30% within 1 year. Urgent surgical treatment is used for valve disruption in endocarditis and for complications of myocardial infarction, including ventricular septal or free wall rupture, and mitral regurgitation. Coronary bypass grafting occasionally plays a role in the revascularization strategy during acute infarction. Increasingly surgeons play an important role in rescue from profound cardiogenic shock by deploying temporary left or biventricular assist devices as bridge to myocardial recovery, transplantation or a long term implantable blood pump. Some devices are easily portable allowing rescue in a district general hospital, then transportation to the tertiary care centre. Between 40 and 70% of patients who would otherwise die, can be salvaged with different pumps selected according to clinic indication. In contrast the widely used intra-aortic balloon pump provides no survival benefit in established cardiogenic shock.

Keywords Acute; blood pumps; heart failure; shock; surgery

Introduction

The normal heart beats around 120,000 times per day, ejecting 7000 L of blood into parallel systemic and pulmonary circulations. This enormous workload can be sustained for 100 years. Nonetheless, any sudden structural or dysrhythmic event can cause a precipitous fall in cardiac output with profound consequences for other organs. When the heart fails and dilates, myocardial wall tension, energy and oxygen consumption increase. Sub-endocardial blood flow is diminished, while reflex vasoconstriction elevates afterload. Irrespective of cause, medical treatment aims to restore systemic blood flow and perfusion pressure before there is irreversible damage to brain, liver, kidneys and gut. Adequate right ventricular function is necessary for lung perfusion, oxygenation and prevention of venous hypertension. High central venous pressure limits tissue perfusion gradient as systemic pressure falls.

Acute heart failure can follow any aspect of cardiac dysfunction and is immediately life threatening. In many cases it is superimposed on chronic cardiomyopathy. Alternatively, for those with acute coronary syndrome, dysrhythmia, acute aortic dissection, pulmonary embolism or valve disruption in bacterial endocarditis, acute heart failure may be a new syndrome. Presentation may be gradual with breathlessness on exertion and peripheral oedema, or sudden with pulmonary oedema and cardiogenic shock. The initial treatment algorithm from the European Society of Cardiology is shown in Figure 1.[1] Despite best medical efforts 11% of patients die during the acute hospital admission; mortality at 1 year in those who survive is 30%. When acute heart failure progresses to cardiogenic shock, 30-day mortality ranges from 40 to 70% even in tertiary care centres.[2] In 75% of cases, shock follows acute myocardial infarction; hospital mortality rates average 65% with shock versus 10% without. Whereas most deaths occur in elderly patients with acute on-chronic heart failure, young patients with remediable problems also die unless supported by mechanical blood pumps. In this setting the surgeon and, increasingly, surgeon-led shock teams play an important role in structural cardiac repair and rescue from shock with circulatory support devices.

Cardiogenic shock

Cardiogenic shock is a complex degenerating spiral of multi-organ dysfunction triggered when the heart is unable to provide adequate pressure and flow. The 5–10% incidence of shock in myocardial infarction patients translates to as many as 50,000 and 65,000 cases annually in the US and Europe. Without effective intervention, including primary percutaneous revascularization, mortality routinely exceeds 50%.[2] In 80% of patients, loss of around 40% of functional myocardium leads to left ventricular failure, either acutely or following repeated ischaemic events. The remaining 20% develop shock through ventricular free wall rupture, septal defect, mitral regurgitation or right ventricular failure. The average left ventricular ejection fraction (LVEF) at onset is 30%. Acute right ventricular failure occurs in half of inferior myocardial infarctions or secondary to mitral regurgitation with pulmonary hypertension. Randomized clinical trials employ similar definitions.[3] Values for decreased cardiac index range from <1.8 L/min/m^2 to <2.2 L/min/m^2. The cut-off point for systolic blood pressure is <90 mmHg, though shock can also exist in those with systolic pressure >100 mmHg when inotropic support or an intra-aortic balloon pump (IABP) elevates mean pressure. Hypoperfusion manifests as cool, clammy extremities, poor capillary refill, disorientation, confusion or loss of consciousness with urine output <30 ml/h. Elevated left atrial pressure causes pulmonary congestion and dyspnoea, particularly in association with acute mitral regurgitation.

Shock may be acute, transient and responsive to percutaneous coronary revascularization, inotropes and vasopressors.[3] These patients have predominant myocardial stunning (or myocarditis). In contrast, those with refractory shock do not respond to medical therapy and, without circulatory support, progress inexorably towards death. They usually have extensive anterior infarction, pre-existing left ventricular dysfunction or stuttering progression of infarction through failure to re-perfuse. Patients with ventricular septal defect, free mitral regurgitation or cardiac tamponade, and those with endocarditis, valvular disruption and intra-cardiac fistula fall into this category. Others differentiate between profound shock (blood pressure <75 mmHg despite inotropes or IABP) and non-profound shock (systemic pressure >75 mmHg in response to treatment).[4] Relative mortalities of profound and non-profound shock are 71% versus 22%.

Stephen Westaby *MS. PhD FRCS FECTS FESC FACC FICA is a Consultant Cardiac Surgeon, Oxford University Hospitals NHS Trust, Professor of Bioengineering, Institute of Life Sciences, University of Swansea, and Medical Director, Calon Cardio Technology, UK. Conflicts of interest: none declared.*

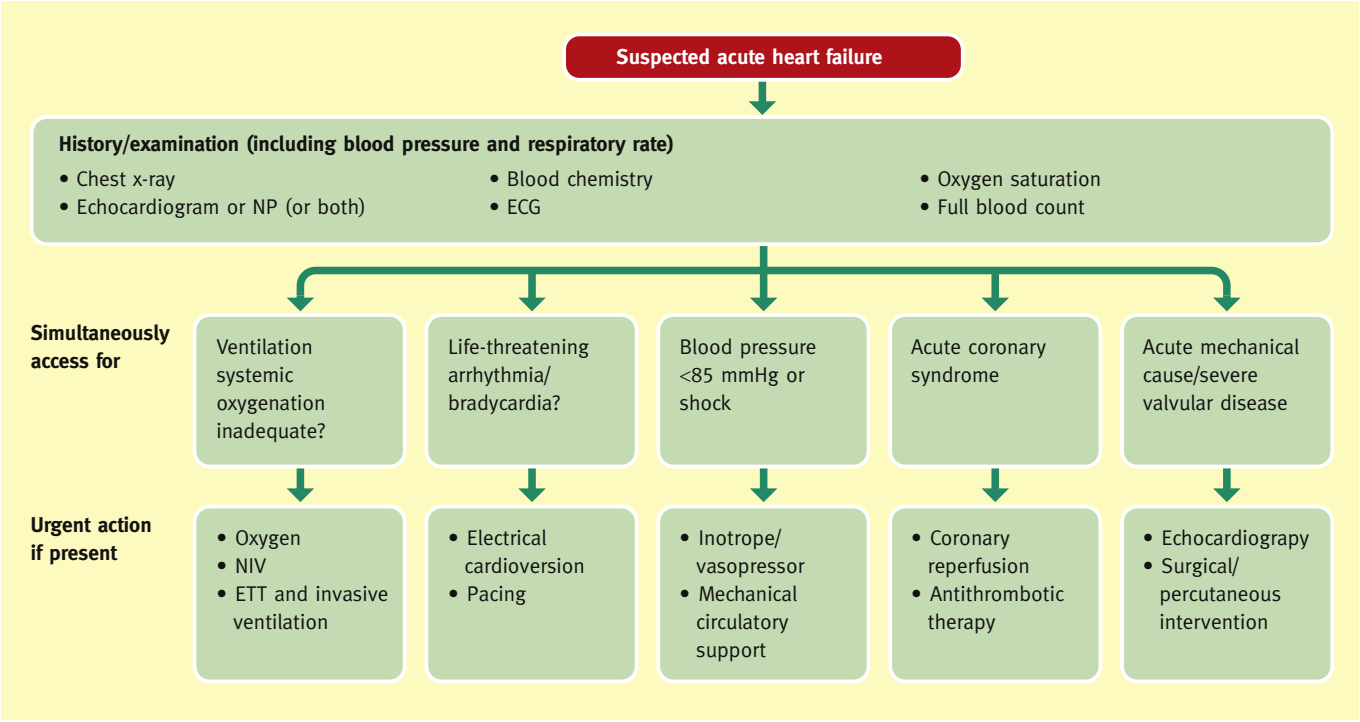

Figure 1 Adapted with permission from the European Society of Cardiology. From McMurray JJV, Adamopoulos S, Anker SD, Auricchio A, Bohm M, Dickstein K et al. ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2012. *Eur Heart J* 2012;**33**:1787–1847. ECG, electrocardiogram; ETT, endotracheal tube; NIV, non-invasive ventilation; NP, natriuretic peptide.

When mean arterial pressure is <55 mmHg, serum lactate >11 mmol/L, base deficit >12 mmol/L and SVO_2 <65% despite treatment, recovery is unlikely without circulatory support.

Cardiogenic shock is not simply an issue of low pressure and flow. As coronary perfusion falls function of the injured ventricle is further impaired. This causes pulmonary congestion and hypoxia affecting the kidneys, liver and gut. The inflammatory response to myocardial infarction and cytokines from ischaemic tissue triggers biochemical failure and worsening impairment of ventricular function.[5] With these self-perpetuating mechanisms only rapid restoration of cardiac index and coronary perfusion can arrest the vicious cycle. High levels of nitric oxide synthase expression after release of inflammatory mediators cause inappropriate vasodilation. This negates reflex vasoconstriction in response to hypotension. Nitric oxide has a biphasic effect on myocardial contractility. Low levels are positively inotropic. High levels negate inotropic responsiveness through suppression of mitochondrial respiration. Infarction patients who die from shock have high plasma concentrations of the inflammatory cytokine interleukin 6.[6] This also exerts a negative inotropic effect predisposing the patient to multi-organ failure. Whereas peripheral vasoconstriction is the reflex response to hypotension, 20% develop low systemic resistance through inappropriate vasodilatation.[6] Depressed contractility with vasodilation causes severe hypotension, hypoperfusion, lactic acidosis and profound shock.

Limitations of medical management

Most patients with myocardial ischaemia will have undergone primary coronary angioplasty and been given treatment with heparin and anti-platelet agents. The intuitive response to worsening hypotension is to employ high-dose intropes and vasopressors but these have damaging effects. Although stunned myocardium is partially responsive to adrenergic therapy, elevation of stroke work, wall tension and myocardial oxygen consumption depletes energy reserves. This predisposes to endocardial necrosis and impaired diastolic function with overall negative effects on myocardial recovery.

Concomitant right ventricular failure adds to the complexity of medical management. This occurs after right coronary occlusion or secondary to worsening of left ventricular failure with pulmonary hypertension. A right ventricle without pre-existing hypertrophy cannot generate pulmonary artery pressures exceeding 50–60 mmHg. If right atrial filling pressure is low (<15 mmHg), right ventricular ejection fraction will not be adequate. Positive pressure ventilation further impairs ejection and a fall in pulmonary artery pressure reflects worsening right ventricular failure. Management is based on optimization of volume status, reduction in afterload by use of selective pulmonary artery dilators, and inotropic support for both ventricles.[2] Inhaled nitric oxide reduces pulmonary vascular resistance without systemic vasodilatation. The combination of dobutamine with nitric oxide increases cardiac output while reducing pulmonary vascular resistance. The selective phosphodiesterase III inhibitor, milrinone, is an inodilator that decreases pulmonary vascular resistance but its use is limited by the systemic vasodilatory effect. Levosimendan has global vasodilatory and anti-ischaemic properties, and works through sensitization of cardiac troponin C to the effects of intra-cellular calcium. This increases contractility without an increase in myocardial oxygen consumption. The pulmonary vasodilatory effects of levosimendan lower pulmonary vascular resistance and increase

