Emergency Medicine

Cardiogenic shock

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Introduction

Cardiogenic shock is a syndrome characterized by a low cardiac output, elevated left ventricular filling pressure (usually greater than 18 mm Hg) and arterial hypotension (commonly less than 80 mm Hg systolic). The commonest cause is extensive left ventricular damage resulting from myocardial infarction and this has a poor prognosis. Other causes, which may be surgically correctable and so have a better prognosis, include rupture of the interventricular septum and papillary muscle dysfunction (Table I). Prompt and accurate haemodynamic assessment of a patient with shock is important as this will characterize the type of shock and provide valuable information on the most appropriate treatment. Inotropic agents, vasodilators and diuretics are the main drugs used in the treatment of cardiogenic shock but they have both potentially beneficial and deleterious effects on myocardial and target organ function.

Pathogenesis

In acute myocardial infarction, the severity of left ventricular dysfunction is related principally to the extent of myocardial damage (Alonso *et al.*, 1973), presumably because a critical mass of contractile tissue is necessary to sustain pump function of the heart. The syndrome of cardiogenic shock appears to be a direct consequence of massive myocardial destruction, involving at least 45% of the left ventricle (Caulfield *et al.*, 1972).

Scheidt and co-workers (1970), observed that cardiogenic shock frequently occurred hours or sometimes days after the onset of symptoms and suggested that this delay reflected progressive myocardial damage. Early intervention in acute myocardial infarction has been shown to limit myocardial damage and so prevent cardiogenic shock (Pantridge, 1970). This approach forms the basis for the use of throm-

bolytic agents and beta blockade in recent acute myocardial infarction.

An imbalance between oxygen supply and demand in the ischaemic zone of the myocardium is currently believed to be the chief pathogenetic mechanism in infarct extension (Hillis & Braunwald, 1977). Therapies to limit infarct size and to support the haemodynamic state in cardiogenic shock affect one or more of the three principal determinants of myocardial oxygen consumption which are: (1) heart rate, (2) left ventricular contractility, and (3) left ventricular wall tension – determined largely by ventricular radius, systemic blood pressure and filling pressure.

The pathophysiological response to acute infarction is mediated chiefly by the sympathoadrenal response. This tends to increase heart rate, contractility and blood pressure, all of which alter unfavourably the balance between oxygen supply and demand with further deterioration in left ventricular function. Coronary perfusion falls as blood pressure decreases. The resulting low cardiac output produces pulmonary oedema which jeopardizes further the precarious metabolic balance in the myocardium. This vicious circle leads to extension of the infarct and further left ventricular damage.

The major determinant of prognosis after myocardial infarction and in cardiogenic shock is left ventricular function (Dwyer *et al.*, 1983) and this can be assessed clinically (Peel *et al.*, 1962; Killip & Kimball, 1967), or non-invasively with radionuclide ventriculography (Schulze *et al.*, 1977) or echocardiography (Nador *et al.*, 1984).

Clinical features

The clinical signs of cardiogenic shock reflect the low cardiac output and poor tissue and organ perfusion and depend chiefly on the severity of the left ventricular impairment and the presence of other cardiac pathology.

Co-existing confusion makes history taking difficult

but if the patient is able to give a history it may help to exclude other causes of shock, producing a similar clinical picture (Table II). Some patients may be comatose. In most patients the hands and feet will be cold and clammy with a high venous tone. There will be a tachycardia with a low systolic blood pressure (commonly less than 80 mm Hg) and oliguria or anuria. The character of the carotid arterial upstroke may be of help in distinguishing causes of cardiogenic shock due to left ventricular outflow tract obstruction or aortic regurgitation. The jugular venous pressure is commonly raised in severe right ventricular infarction or with causes of pulmonary hypertension. A left ventricular aneurysm may be detected by a dyskinetic apex beat. Evidence of cardiac tamponade should also be sought clinically. Auscultation may point to serious valvular or septal disorders but often the low cardiac output makes this difficult.

Management

Haemodynamic measurements

The differential diagnosis of cardiogenic shock is presented in Table II.

An accurate haemodynamic assessment is invaluable in diagnosing the type and severity of circulatory failure, selecting the optimum medical treatment and assessing the response to therapy. Right and left ventricular filling pressures and cardiac output can be measured with a flow-directed, balloon-tipped thermodilution catheter (Swan *et al.*, 1970). Direct arterial catheterization allows an accurate assessment of blood pressure which may be otherwise inaccurate or unrecordable when measured by a cuff sphygmomanometer in a patient who has peripheral vasoconstriction. For peripheral artery cannulation the radial artery is preferred as the hand usually has collateral circulation from the ulnar artery which should be tested before catheterization using Allen's test. Infection and ischaemia are the main but rare complica-

Table I Causes of cardiogenic shock

(1)	Left ventricular infarction.
(2)	After cardiopulmonary bypass and cardiac surgery.
(3)	Acute mechanical disorders (septal or free wall rupture, mitral or aortic regurgitation, left ventricular aneurysm).
(4)	Tachyarrhythmias or bradyarrhythmias.
(5)	Left ventricular outflow tract obstruction (aortic stenosis, hypertrophic obstructive cardiomyopathy).
(6)	Left ventricular inflow tract obstruction (mitral stenosis, left atrial myxoma, right ventricular infarction).
(7)	Chronic heart muscle disease (end-stage cardiomyopathy).

Table II Differential diagnosis of cardiogenic shock

(1)	Hypovolaemia.
(2)	Obstructive shock (cardiac tamponade).
(3)	Massive pulmonary embolism.
(4)	Distributive shock (septicaemic, haemorrhagic, neurogenic, endocrine failure, toxic, anaphylactic shock).

tions. Catheters should be flushed continuously with heparinized saline. The catheter can also be used for arterial blood gas analysis. The arterial and venous catheters should be clearly labelled so that drugs like diazepam and barbiturates are not given into an artery.

In patients with heart disease, measurement of only the central venous pressure may provide misleading information as it is a poor guide to left ventricular function. Very often a patient may have been given diuretics or opiates before reaching the intensive care unit. These drugs reduce the left ventricular end-diastolic pressure by reducing the preload and although the patient may still have a third heart sound and clinical and radiographic features of pulmonary oedema, the left ventricular end-diastolic pressure may be within normal levels. On the other hand, a proportion of patients without clinical evidence of left

Figure 1 Portable AP chest X-ray showing the tip of a deflated Swan-Ganz catheter in the right pulmonary artery in a patient with resolving pulmonary oedema.

ventricular failure may have wedge pressures exceeding 18 mm Hg

Catheterization of the right heart using a balloon-tipped, flow-directed, Swan-Ganz catheter allows measurement of the pulmonary occluded (wedge) pressure and an estimate of left atrial pressure (left ventricular preload or end-diastolic pressure). It is indicated in all patients with severe circulatory failure. Cardiac output can also be measured if the catheter has a thermistor.

Swan-Ganz catheters can be inserted at the bedside and the tip position ascertained by pressure measurements. It is, however, easier to position the catheter using X-ray guidance and this is important in patients with cardiogenic shock who do not tolerate lying flat for long periods. Problems in inserting and maintaining Swan-Ganz catheters and interpretation of the pressure recordings have been well reviewed Wiedemann *et al.*, 1984a,b). The catheter can be inserted in several sites for venous access to the pulmonary artery (Figures 1 and 2) but the subclavian and internal jugular veins are the most widely used. Pneumothorax is a well recognized complication with subclavian vein puncture.

The criteria for a true wedge position include a mean pulmonary wedge pressure lower than or equal to the pulmonary artery diastolic pressure and a characteristic left atrial pressure waveform (Figure 3). The main complications of pulmonary artery catheterization are infection, pulmonary artery rupture, pulmonary infarction, balloon rupture, knotting of the catheter, complications near the insertion site (pneumothorax, arterial puncture and air embolism) and arrhythmias which are usually transient (Civetta, 1983).

Figure 2 Portable AP chest X-ray showing the tip of a deflated Swan-Ganz catheter in the 'wedge' position. N.B. The exact tip position is confirmed by the pressure reading.

Figure 3 Normal pressure trace (mm Hg) from a Swan-Ganz catheter withdrawn from the wedge position (PAW) (top left) to the pulmonary artery (PA), to the right ventricle (RV) to the right atrium (RA) showing characteristically normal waveforms.

In cardiogenic shock due to myocardial infarction, left ventricular compliance is decreased, the left ventricular end-diastolic pressure is usually equal to or greater than 18 mm Hg and the cardiac index below 1.8 l/min/m^2 . Prognosis is related to haemodynamic findings and worsens as the pulmonary wedge pressure increases (Forrester *et al.*, 1976).

In cardiac tamponade the filling pressures will be equal on both sides of the heart. Right ventricular infarction may be diagnosed with some certainty by electrocardiographic changes (Morgera *et al.*, 1984) and by pressure measurements (Dell'Italia *et al.*, 1984). The right ventricular filling pressure is raised and the patient will usually have a raised jugular venous and right atrial pressure which may be further elevated in the presence of tricuspid regurgitation. It is important to diagnose right ventricular infarction because diuretic treatment may be dangerous as an increased and not decreased right ventricular filling pressure is required to maintain cardiac output.

An elevated pulmonary wedge pressure with a normal left ventricular end-diastolic pressure may be due to mitral regurgitation or stenosis, or rarely to a left atrial myxoma or thrombus. Sector and M-mode echocardiography will usually be diagnostic.

Low right heart filling pressures with relatively normal left heart filling pressures are found in hypovolaemic, septic, neurogenic, toxic and anaphylactic shock.

General management of cardiogenic shock

Relief of pain and anxiety with adequate doses of opiates is an important part of the treatment. Hypotension, respiratory depression and nausea may result and so care is required in their use. Anti-emetics are usually given simultaneously.

Oxygenation is important and this may need to be

given by mechanical ventilation depending on the clinical status of the patient and the blood gases. Positive pressure ventilation may depress cardiac output (Jardin *et al.*, 1981). It decreases preload (Qvist *et al.*, 1975) and clears pulmonary oedema. Baseline haematology, biochemistry and arterial blood gases should be performed and any metabolic abnormalities corrected. Blood cultures and samples for bacteriology should be taken if appropriate.

Specific measures

Cardiac arrhythmias need specific treatment and electrical cardioversion is required if antiarrhythmic drugs are ineffective. Cardiac pacing is required for atrioventricular block with associated bradycardia and low cardiac output. Although atrial synchronized pacing improves haemodynamics significantly (Fowler *et al.*, 1984; Love *et al.*, 1984), long-term benefits are unclear.

In cardiogenic shock, there is a risk of damage to other organs. Shock lung is quite common and remains difficult to treat effectively. The subject has been recently reviewed (Stevens & Raffin, 1984a,b).

After the patient has been resuscitated as far as possible, surgically correctable causes of cardiogenic shock should be looked for clinically and also with echocardiography and measurements of oxygen saturation from the Swan-Ganz catheter. Technical advances in nuclear cardiac imaging and quantitation may, in the future, allow useful measurements of ejection fraction and left ventricular wall movement to be made at the bedside but these methods need further evaluation and are not widely available.

If haemodynamically important septal rupture, aneurysm, aortic or mitral regurgitation are suspected, coronary and left ventricular angiography may be necessary as early correction of these disorders may improve prognosis. The results of coronary revascularization alone, however, have been generally disappointing (Mundth, 1977).

The optimum filling pressure of the left ventricle after infarction is 14–18 mm Hg (Crexells *et al.*, 1973). Most patients are already at the top of their Starling curve and will need a lowering of their left ventricular filling pressure. Further increases in preload provide little or no haemodynamic improvement and may increase any existing pulmonary oedema. A small proportion of patients, however, may have pulmonary wedge pressures at the upper limit of normal (12 mm Hg) and others may have co-existing hypovolaemia. In these cases a fluid challenge such as low molecular weight dextran (50 ml increments) to increase the pulmonary wedge pressure to 20 mm Hg may improve the cardiac output (Rackley *et al.*, 1975; Russel *et al.*, 1976).

Intra-aortic balloon pumping has been reported to

be useful in the pre- and post-operative stabilization of patients but generally has a limited place in the treatment of a patient with cardiogenic shock (Scheidt *et al.*, 1973). Recently, however, it has been shown to be of some benefit (Weiss *et al.*, 1984). The aim of counterpulsation of blood is to improve coronary filling during diastole and to reduce left ventricular work by lowering aortic systolic pressure. The widely varying results for the efficacy of this treatment in cardiogenic shock are probably explained by the differing definitions of shock (Kuhn, 1978).

Drugs

Diuretics, inotropic agents and vasodilators are the three major classes of drugs used in the treatment of cardiogenic shock. There is no convincing evidence, however, that long term survival is improved or that left ventricular myocardium is preserved with the use of any of these agents (Rude, 1983; Sobel, 1984).

Diuretics

Diuretics must be used cautiously in cardiogenic shock as they may cause severe ototoxicity in the presence of renal failure. They are most useful in less severe cases of shock when the left ventricular filling pressure 20 mm Hg is less than Frusemide and ethacrynic acid are the loop diuretics most commonly used. Frusemide also reduces left ventricular end-diastolic pressure by reducing afterload and dilating the venous vascular bed. The consequent reduction in left ventricular end-diastolic volume is beneficial as left ventricular wall tension is also reduced thus decreasing myocardial oxygen consumption. Diuretics are dangerous in hypovolaemic shock and in right ventricular infarction as cardiac output may be further critically reduced. Therefore a serial assessment of left ventricular filling pressure is imperative in hypotensive patients being treated with these agents.

When diuretics are unhelpful in the treatment of shock, the remaining therapeutic approaches focus on enhancing myocardial contractility with inotropic agents and unloading the heart with vasodilator drugs.

Inotropic agents

The majority of patients with cardiogenic shock will need inotropic agents. A major drawback in their use is that they may, in certain doses, increase oxygen requirements by the failing left ventricle where the energy balance may be jeopardized further by the presence of pulmonary oedema.

Sympathomimetic inotropes may improve circulatory haemodynamics in cardiogenic shock in the short-term but long-term survival is rarely improved

(Sobel, 1984). Driving the heart out of failure is rarely, if ever, possible since the diseased myocardium is unable to respond for long periods to extra metabolic demands (Katz, 1973).

Digitalis glycosides

Digitalis has a very limited place in the current treatment of shock (Cohn *et al.*, 1969). It confers no benefit in acute infarction (Balcon *et al.*, 1968) and may increase infarct size (Varonkov *et al.*, 1977). It may, however, be of some use in patients with mild left ventricular failure as it may decrease filling pressure and increase cardiac output (Mason *et al.*, 1977). Its arrhythmogenic activity is a major cause for concern particularly in ischaemic myocardium (Morris *et al.*, 1969). The digitalis glycosides may also increase impedance to left ventricular ejection (Marcus, 1980) with loss of any inotropic gain. Nevertheless, it is useful in controlling supraventricular arrhythmias, particularly poorly controlled atrial fibrillation, where co-existing hypotension contra-indicates the use of verapamil or other anti-arrhythmic agents with similar negative inotropic activity (Mason *et al.*, 1977).

Dopamine

Dopamine, an endogenous catecholamine and the immediate precursor of noradrenaline has mainly beta-1 agonist activity. Its useful renal and coronary vasodilator properties are probably mediated by dopamine receptors. At low infusion rates of 2–5 µg/kg/min it produces increases in cardiac contractility, cardiac output and renal blood flow with negligible changes in heart rate (Kho *et al.*, 1980; Timmis *et al.*, 1981), although the peripheral resistance may decrease slightly. At higher doses (5–10 µg/kg/min) it increases the arterial pressure, peripheral resistance and heart rate but renal blood flow may fall (Mueller *et al.*, 1978). At even higher doses (greater than 20 µg/kg/min) it may provoke ventricular arrhythmias (Loeb *et al.*, 1974). It is particularly useful in patients with renal dysfunction as it also increases sodium excretion.

Dobutamine

Dobutamine, a synthetic catecholamine with predominantly beta-1 adrenoceptor agonist properties, has a variable effect on peripheral vascular resistance although it is usually reduced. It is less inotropic than dopamine but in contrast to dopamine, may produce a beneficial fall in pulmonary capillary wedge pressure (Leier *et al.*, 1978). Both dobutamine and dopamine are arrhythmogenic and dobutamine may be more so (Loeb *et al.*, 1974).

Any clear superiority of one of these agents in cardiogenic shock complicating acute infarction is

unclear but dobutamine has been advocated as the drug of first choice in patients with mild hypotension and in chronic low output states (Loeb *et al.*, 1977). Dopamine may be required if the blood pressure remains low in spite of higher doses of dobutamine (Rude, 1983).

Isoprenaline

This has both beta-1 and beta-2 agonism and no significant vasoconstrictor effects. It increases heart rate, myocardial contractility, cardiac output and decreases vascular resistance (Smith *et al.*, 1967). Because it increases myocardial oxygen consumption and is arrhythmogenic, it is potentially dangerous in recent infarction (Gunnar *et al.*, 1968). It may be useful in shock occurring after surgery.

Noradrenaline

This has beta-1 and alpha agonist action and increases arterial blood pressure by increasing myocardial contractility and peripheral vasoconstriction. It produces only minor changes in cardiac output. It may, like other sympathomimetic agents, damage the heart due to the ischaemia resulting from the increased afterload. In addition, it is arrhythmogenic and may also decrease renal blood flow. These disadvantages usually offset any advantage from an increased systolic blood pressure, although it may have a place in a severely hypotensive patient who has not responded to either (or both) dopamine or dobutamine. Nevertheless the prognosis remains extremely poor in this patient group.

Vasodilator drugs

The rationale for the use of vasodilators is that they decrease both afterload and preload, thereby reducing left ventricular work and myocardial oxygen consumption.

They are useful in heart failure but their hypotensive effects restrict their role in cardiogenic shock where tissue perfusion is already critically reduced (Chatterjee & Parmley, 1977). They are of greatest value in cardiogenic shock where forward failure is due to a surgically correctable problem like interventricular septal rupture or severe mitral regurgitation rather than primary muscle disease. They have been used in combination with dopamine and other inotropes to counteract sympathomimetic-induced peripheral vasoconstriction. Cardiac arrhythmias are not a problem with vasodilators as they have no direct cardiac effects.

Nitroprusside is of some value in acute myocardial infarction because its short half-life makes control of arteriolar and venous dilatation fairly easy. In acute

infarction it has been shown to decrease left ventricular filling pressure and increase cardiac output in patients with moderate or severe left ventricular failure (Chatterjee, 1976). Theoretically, nitroprusside, in common with agents with a similar mode of action (Hillis *et al.*, 1981), may increase myocardial ischaemia by coronary 'steal' and should not be given to patients in whom cardiogenic shock is due to ischaemic heart disease (Rude, 1983). Cyanide toxicity is a dose-related complication and can be a serious hazard during prolonged administration although hydroxocobalamin may provide some protection. It has also been reported to produce ventilation-perfusion mismatch (Bencowitz *et al.*, 1984).

The major effect of nitroglycerin is to reduce preload. It also reduces left atrial pressure in acute myocardial infarction (Borer *et al.*, 1975) and increases coronary flow directly. It produces no significant increases in cardiac output (Chatterjee, 1976) but is probably the vasodilator of choice in cardiogenic shock due to ischaemia.

Of other vasodilators isosorbide dinitrate is available for intravenous use but there is at present little information on its value in cardiogenic shock. Phenolamine is a weak alpha- and beta-adrenoceptor blocking agent but is a potent vasodilator. It is of greatest value in patients with severe left ventricular impairment complicating myocardial infarction.

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Future directions

Reduction in mortality from cardiogenic shock ultimately lies in the prevention of coronary disease. Limitation of infarct size may improve prognosis after infarction and this subject is currently of considerable interest (Kubler & Doorey, 1985). Various interventions including beta blockers (Peter *et al.*, 1978), intravenous nitroglycerin (Bussman *et al.*, 1981), hyaluronidase (Maroko *et al.*, 1977) and coronary thrombolysis (Taylor *et al.*, 1984; Mathey *et al.*, 1981; Kasper *et al.*, 1984) and surgical revascularization (DeWood *et al.*, 1979) have been tried but large controlled studies with adequate follow up are required to establish their efficacy.

Cardiogenic shock is a very difficult condition to treat and has a bad prognosis. An accurate haemodynamic assessment is important to distinguish other forms of shock where the management is different and the prognosis may be good.

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