

Shock: A Review of Pathophysiology and Management. Part I

L. I. G. WORTHLEY

Department of Critical Care Medicine, Flinders Medical Centre, Adelaide, SOUTH AUSTRALIA

ABSTRACT

Objective: *To review pathophysiology and management of hypovolaemic, cardiogenic and septic shock in a two-part presentation.*

Data sources: *Articles and published peer-review abstracts and a review of studies reported from 1994 to 1998 and identified through a MEDLINE search of the English language literature on septic shock, cardiogenic shock and hypovolaemic shock.*

Summary of review: *Shock is a clinical syndrome characterised by hypotension (i.e. a systolic blood pressure less than 90 mmHg or a mean arterial pressure less than 60 mmHg or reduced by greater than 30%, for at least 30 minutes), oliguria (i.e. a urine output less than 20 mL/hr or 0.3 ml/kg/hr for 2 consecutive hours), and poor peripheral perfusion (e.g. cool and clammy skin which demonstrates poor capillary refill). Hypovolaemic and cardiogenic shock are associated with disorders that cause an underlying haemodynamic defect of a low intravascular volume and a reduction in myocardial contractility, respectively.*

The understanding and management of hypovolaemic shock has changed very little over the past 50 years with treatment requiring management of the causative lesion (i.e. surgical correction of blood loss) and replacement of the intravascular volume by infusing blood and/or 0.9% sodium containing colloid or crystalloid fluids. Due to recent developments in percutaneous coronary revascularisation techniques, management of cardiogenic shock in some centers has changed. Emergency cardiac catheterisation with urgent myocardial reperfusion (using percutaneous transluminal coronary angioplasty or coronary artery stenting in selected cases) and use of glycoprotein IIb/IIIa antagonists while supporting the circulation using an intra-aortic balloon pump, has been reported to reduce mortality of cardiogenic shock in acute myocardial infarction. Large randomised, controlled multicentre trials are awaited.

Conclusions: *Hypovolaemic shock requires urgent management of the underlying defect and replacement of the intravascular volume loss. Recent studies in management of cardiogenic shock using urgent revascularisation and intra-aortic balloon counterpulsation in patients with acute myocardial infarction have shown a reduction in mortality in selected cases. (Critical Care and Resuscitation 2000; 2: 55-65)*

Key Words: Shock, hypovolaemic shock, cardiogenic shock, intra-aortic balloon pump, acute myocardial infarction

Shock, or cardiovascular collapse, is a clinical condition diagnosed in the presence of:

- hypotension (i.e. a systolic blood pressure less than 90 mmHg or a mean arterial pressure [MAP] less

Correspondence to: Dr. L. I. G. Worthley, Department of Critical Care Medicine, Flinders Medical Centre, Bedford Park, South Australia 5042

than 60 mmHg or reduced by greater than 30%, for at least 30 minutes),

- oliguria (i.e. a urine output less than 20 ml/hr or 0.3 ml/kg/hr for 2 consecutive hours), and
- poor peripheral perfusion (e.g. skin is cool and clammy and demonstrates poor capillary refill). With cardiogenic or septic shock the skin often exhibits a cyanotic mottling, which often occurs first over the knees.

Shock is classified as, hypovolaemic, cardiogenic, obstructive or distributive, and has been defined as a pathophysiological state in which there is an inadequate supply or inappropriate use of metabolic substrate (particularly oxygen) by peripheral tissues.¹

Hypovolaemic and cardiogenic shock will be discussed in this section. Distributive shock characterises those conditions in which an abnormal distribution of the peripheral circulation occurs with one cause being septic shock, which will be discussed in the next section. Obstructive shock describes shock associated with vascular obstructive defects including pulmonary embolism, pericardial tamponade, atrial myxoma, tension pneumothorax, hydrothorax or haemothorax and even ascites.² Treatment of these disorders centers upon the relief the obstructive defect. These conditions will not be discussed.

HYPOVOLAEMIC SHOCK

Hypovolaemic shock is caused by a loss of intravascular fluid which is usually whole blood or plasma.

Whole blood loss: blood loss from an open wound is an obvious cause for hypovolaemic shock. However, blood loss may be concealed in the abdominal or thoracic spaces (e.g. haemothorax, lacerated liver, spleen or kidney, ectopic pregnancy, gastrointestinal haemorrhage), in retroperitoneal tissues (with ruptured aorta or coagulation abnormality) or in tissues surrounding bony fractures (e.g. blood loss associated with an adult fracture of the humerus ranges from 500-1000 mL, tibia and fibula 750-1200 mL, femur 1000-1500 mL, and pelvis 1500-2500 mL).

Plasma loss: intravascular volume depletion may occur with any condition that leads to excessive extracellular fluid loss with or without loss of plasma protein. For example, pancreatitis, peritonitis, burns, crush syndrome and anaphylaxis tend to have a high plasma protein loss, whereas, vomiting, diarrhoea, excessive nasogastric, fistula or enterostomy losses, sodium losing nephropathy and diuretic therapy are usually associated with low plasma protein losses.

Physiological responses to intravascular volume loss

Neural or immediate response

With a reduction in blood volume, a neural or immediate response occurs within minutes. The right atrial and left atrial pressures fall, activating low pressure receptors in the atria and walls of the pulmonary arteries, great veins and ventricles. With further intravascular blood loss, the reduction in venous return causes a decrease in cardiac output and blood pressure, activating high pressure stretch receptors in the aortic arch and carotid sinus. Severe hypotension (e.g. MAP of 50 mmHg or less) activates chemoreceptor receptors of the carotid and aortic bodies; and at a MAP of 40 mmHg or less, a central nervous system ischaemic response occurs. These signals are transmitted to the vasomotor centre in the medulla and pons, which sends efferent impulses via the sympathetic and vagus nerves to increase the heart rate, myocardial contractility and peripheral arteriolar and venous tone. The baroreceptor mechanisms act for a few hours only because continued stimulation leads to adaptation, causing the baroreceptors to reset to a new value in less than 2 days.³

The parasympathetic response normally causes a reduction in vagal tone and increase in heart rate, although a vasovagal response may occur in 7% of hypovolaemic patients causing a relative (and uncharacteristic) bradycardia.^{4,5}

In severe hypotension, the circulating levels of adrenaline may be increased up to 1000 pg/mL (from adrenal gland catecholamine release) and circulating levels of noradrenaline may be increased to 2000 pg/mL (largely from sympathetic synaptic cleft spill).⁶ β -endorphins are released from the anterior pituitary, reducing the patients' pain perception, and may play a role in causing the decompensated phase of hypotension, with a reduction in the sympathetic vasoconstrictor response and direct venodilation. β -endorphin release usually begins after one-quarter of the blood volume (i.e. 1250 mL/70 kg) has been lost.^{7,8} Depression in myocardial contractility does not occur unless the patient has a severe reduction in coronary oxygen delivery (i.e. is profoundly hypotensive or anaemic). The early changes from a normal haemodynamic status (figure 1) are shown in figure 2.

Intrinsic or intermediate response

An intrinsic or intermediate response occurs over a period of hours. The reduced capillary pressure provides a movement of fluid from the interstitium to the vascular compartment at a rate which can exceed 1 litre in the first hour.⁹ Protein (mainly albumin) then moves from

the interstitium to the plasma and, in the adult, up to a total of 2 L of fluid in 24-48 h may move from the interstitial and intracellular compartments to the intravascular compartment, to replace the intra-vascular volume lost.¹⁰ Blood volume may also be replaced in part by the osmotic effect of the elevation of blood glucose during shock,¹¹ increasing the vascular compartment in an adult by approximately 17 mL for each 1 mmol/L increase in blood glucose.

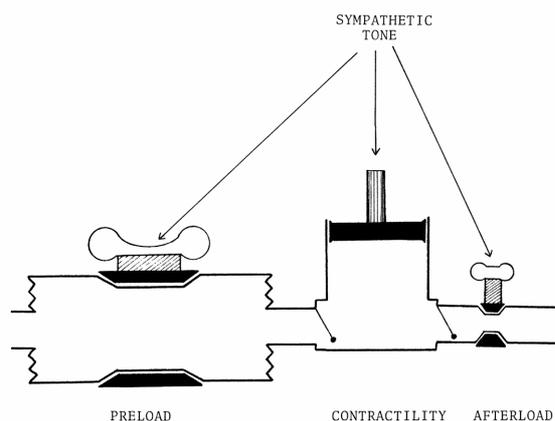


Figure 1. A mechanical model of the circulation where the preload, contractility and afterload are represented as separate elements and the influence of the sympathetic system on each is also shown.

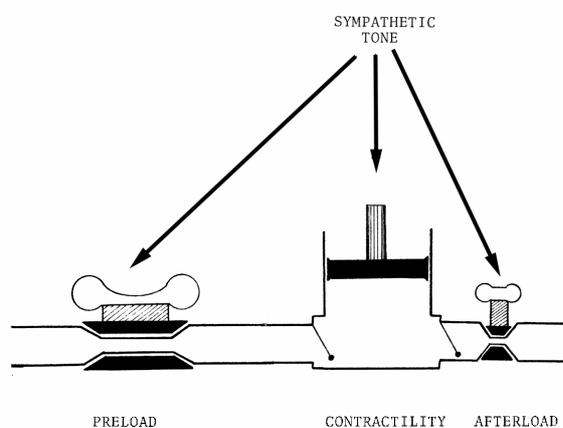


Figure 2. The haemodynamic characteristics of the early phase of hypovolaemic shock with a reduction in intravascular volume (preload), increase in peripheral resistance (afterload) and an increase in the sympathetic tone.

Humoral or delayed response

A humoral or delayed response occurs within days with antidiuretic hormone, aldosterone and renin secretion all being activated to increase renal retention of fluid and increase the intravascular volume.

Clinical features

The clinical features of hypovolaemic shock include pallor, tachycardia (although up to 7% have a relative bradycardia^{4,5}), hypotension, dyspnoea, diaphoresis, faint heart sounds (or an infantile 'tic-tic' cadence due to a similar pitch of both first and second heart sounds), agitation, and poor urine output.

Right-heart catheterisation will usually reveal a low central venous pressure (CVP), pulmonary artery occlusion pressure (PAoP), cardiac output and mixed venous oxygen content. During spontaneous ventilation, pulsus paradoxus may occur whereas during mechanical ventilation the systolic blood pressure only transiently increases during the inspiratory phase followed by a rapid decrease (with a systolic pressure variation of greater than 10 mmHg being suggested as a method to diagnose hypovolaemia in a mechanically ventilated patient with normal pulmonary compliance¹²).

In a 70 kg male the reduction in intravascular volume may be classified as:

Class 1: reduction by 500-750 mL (i.e. 10-15% blood volume), which is usually associated with no clinical features,

Class 2: reduction by 750-1500 mL (i.e. 15-30% blood volume), which is usually associated with venous and arterial constriction and postural hypotension,

Class 3: reduction by 1500-2000 mL (i.e. 30-40% blood volume), which is usually associated with hypotension and tachycardia, and all physiological defense mechanisms are usually fully operative, and

Class 4: reduction by greater than 2000 mL (i.e. 40% blood volume or more), where the patient is usually in severe shock. If the loss is greater than 2000 mL in an adult (i.e. > 40% of blood volume), 50% of patients will probably die, if nothing is done.

While studies have shown that a reduction of blood volume by 20% reduces the MAP by 15% and cardiac output by 41%,¹³ individual responses are remarkably variable and a reduction in plasma volume by as much as 25% may occur without arterial hypotension.¹⁴ The presence of cardiovascular disease, autonomic neuropathy or anaemia, or prior treatment with β -adrenergic blockers or calcium-channel blockers may worsen the cardio-vascular response to blood loss.

Investigations

Hypovolaemia is commonly inferred indirectly from measurements of arterial pressure, heart rate, urinary output and haematocrit. However, for the critically ill patient, alteration of these measurements may not indicate blood loss¹⁵ and cardiac monitoring with right-

heart catheterisation and estimation of CVP, PAoP and cardiac output may be required. Normal blood volume is approximately 75 mL/kg for males and 70 mL/kg for females, although during resuscitation from haemorrhage, trauma or sepsis, patients may do better with 500 mL blood volume in excess of these normal values, to compensate for maldistributions such as pooling of blood in the splanchnic area.¹⁵

Treatment

Operative control of blood loss is the major consideration in patients who have continuing haemorrhage. In one study, an improvement in outcome was reported in hypotensive patients with penetrating torso injuries, when aggressive fluid resuscitation was delayed until operative intervention had occurred,¹⁶ suggesting that with uncontrolled haemorrhage temporary or definitive haemostasis (even in the presence of hypotension) should be performed first, followed by intravascular fluid replacement.¹⁷ Nonetheless, in the severely hypotensive trauma patient in whom haemostasis will be delayed, initial administration of intravenous fluids will still be required.¹⁸

While passive leg raising is sometimes used as a method to increase central blood volume during resuscitation, only 100-150 mL are transferred to the intravascular space by this method.¹⁹ Intravenous fluids including blood, colloid and saline solutions are administered until blood pressure and peripheral perfusion are satisfactory or until the PAoP is between 12-18 mmHg.^{20,21} Replacement of blood loss in an adult with colloid or crystalloid solutions (e.g. fresh frozen plasma, 5% albumin, polygeline, 0.9% saline) will cause a reduction in the haemoglobin by approximately 1 g/100mL per 500 ml of colloid or crystalloid solution remaining in the vascular compartment.

Recently, some have proposed the use of hypertonic saline or hypertonic and hyperoncotic solutions as a resuscitation fluid for the treatment of haemorrhagic and hypovolaemic shock, particularly in burns patients and trauma patients who sustain simultaneous head trauma with high intracranial pressures.²² While these solutions may have specific indications, they should not be used as the sole resuscitation fluid in patients with hypovolaemic shock.²³ Furthermore, with uncontrolled haemorrhage, hypertonic saline, in comparison with 0.9% saline, may increase mortality.²⁴

Lower body positive pressure apparatus (e.g. inflatable trousers or military anti-shock trouser - MAST) have been recommended in the management of traumatic shock, despite the lack of data supporting their efficacy.²⁵ If they are to be used they should only be

used during patient transport, to splint and control haemorrhage for pelvic and lower limb fractures, to tamponade haemorrhage in soft tissue, and to stabilize and maintain the upper torso circulation, when intravenous therapy cannot be administered or when volume replacement is inadequate.²⁶

Treatment of hypovolaemia with catecholamine infusions are only used in patients in whom cardiac arrest is imminent to divert flow from the splanchnic circulation to serve the cerebral and coronary circulations, as it can cause left ventricular outflow obstruction.²⁷

CARDIOGENIC SHOCK

Cardiogenic shock may occur with any disease that causes direct myocardial damage or otherwise inhibits the cardiac contractile mechanism.²⁸ Right-heart catheterisation will reveal a high CVP, PAoP (greater than 18 mmHg), and peripheral resistance; and a low cardiac output (cardiac index less than 2.2 L/min/m²) and mixed venous oxygen content.^{29,30} A model characterising the haemodynamic effects of cardiogenic shock is shown in figure 3.

Causes

The common causes of cardiogenic shock are listed in Table 1. Anaesthetic agents may reduce cardiac contractility by many mechanisms (e.g. calcium-channel blockade, inhibiting the sarcoplasmic reticulum calcium release, increasing the binding of calcium by the sarcolemma³¹), all of which reduce the amount of calcium available for contractile activation.

If cardiogenic shock is caused by myocardial infarction (in the absence of a ventricular septal defect, ruptured papillary muscle, left ventricular outflow tract obstruction,³² cardiac tamponade, pulmonary embolism, cardiac arrhythmia or right ventricular infarction with hypovolaemia) there is a greater than 40% functional loss of the left ventricle.³³ This occurs in 7% - 10% of patients with acute myocardial infarction and has a mortality rate of 60% - 80%. The myocardial abnormality is characterised by both systolic and diastolic dysfunction.^{28,34} The ischaemic myocardial injury may be reversible (e.g. myocardial stunning or hibernating myocardium may be present which may recover completely with restoration of myocardial blood flow) or irreversible (leading to myocardial cell necrosis or apoptosis).³⁵

Myocardial 'stunning'

Reperfusion of ischaemic myocardium within 6 hr of a coronary artery thrombosis, does not lead to immedi-

Table 1. Causes of cardiogenic shock*Direct myocardial damage*

Myocardial infarction
 Cardiomyopathy
 Cardiac bypass
 Cardiac trauma
 Myocarditis

Inhibition of the contraction mechanism

Drug toxicity
 antiarrhythmics, local anaesthetics
 antihistamines
 tricyclic antidepressants
 β -adrenergic blockers
 calcium-channel inhibitors

Anaphylaxis
 Septicaemia
 Pancreatitis
 Biliary peritonitis
 Endocrine causes
 Addisonian crisis
 pituitary apoplexy
 myxoedema

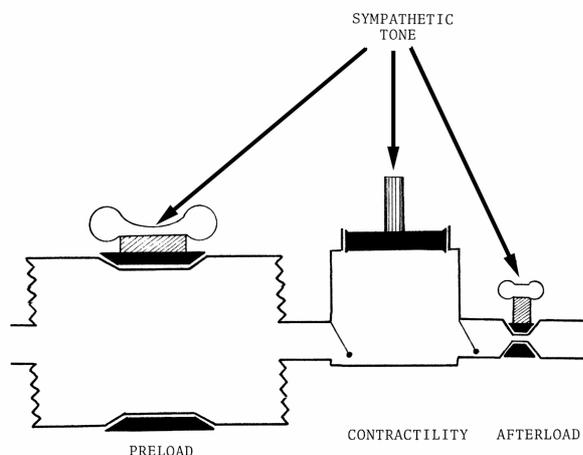


Figure 3. A model characterising the haemodynamic changes found in cardiogenic shock, with an increase in venous pressure, reduced contractility and an increase in sympathetic tone increasing the peripheral resistance.

ate and full recovery in regional myocardial function. Instead, the return of contractility in tissue salvaged by reflow is often delayed for hours, days or even weeks, a phenomenon which has been termed 'stunned' myocardium.^{36,37} Although the stunned myocardium is dysfunctional, it does maintain a latent capacity to contract and, unlike infarcted myocardium, is responsive to positive inotropic stimulation.³⁸ When 'stunning'

contributes to life-threatening cardiac failure, myocardial contractility can be enhanced by pharmacological or mechanical support.³⁸ Myocardial 'stunning' can also occur following cardiopulmonary bypass.³⁹

The aetiology of this disorder may be due to:

1. *Free oxygen radicals.* During reperfusion, molecular oxygen becomes converted to oxygen metabolites known as free radicals, which have one or more unpaired electrons. These toxic substances may cause reperfusion arrhythmias and tissue injury,⁴⁰ although direct evidence of oxygen free radicals causing injury to the human heart, has not been found.^{41,42}

2. *Intracellular calcium abnormality.* Reperfusion causes a 10-fold increase in myocardial cell uptake of calcium, decreasing the ability of mitochondria to manufacture ATP.⁴³ One mechanism that increases intracellular calcium is via activation of the sarcolemmal Na^+/H^+ exchanger (NHE-1, i.e. one of the four described NHE isoforms) with intracellular acidosis. The Na^+/H^+ exchangers regulate cell volume (e.g. activation of NHE-1 has been reported with hyperosmolality and cell shrinkage) as well as intracellular pH.⁴⁴ When myocardial ischaemia is followed by reperfusion (i.e. when extracellular pH is increased but intracellular pH is still low) Na^+/H^+ exchange increases cytosolic Na^+ which in turn increases intracellular Ca^{+2} by altering $\text{Na}^+/\text{Ca}^{+2}$ exchange (particularly when the activity of the sarcolemmal Na^+ pump is critically reduced⁴⁵). The $\text{Na}^+/\text{Ca}^{+2}$ exchange usually moves Ca^{+2} out of the cell during diastole, although it can move Ca^{+2} in either direction across the cell membrane, depending upon the electrochemical Na^+ gradient (i.e. when the gradient decreases or reverses for any reason, the intracellular Ca^{+2} will increase).⁴⁶ Reperfusion with hypertonic saline has been reported to reduce myocardial stunning via a $\text{Na}^+/\text{Ca}^{+2}$ exchange mechanism in the experimental model.⁴⁶

Inhibitors of NHE-1 (e.g. amiloride, HOE-694, HOE-642) have been used during myocardial ischaemia and reperfusion in experimental models to successfully reduce reperfusion injury (e.g. arrhythmias, stunning, cell necrosis) and may become clinically useful before, and during, thrombolysis or percutaneous transluminal coronary angioplasty (PTCA) for myocardial infarction and during coronary artery bypass grafting (CABG).⁴⁷ However, one large, prospective randomised, placebo controlled trial in non-ST segment elevated acute coronary syndrome patients undergoing high-risk coronary artery bypass surgery or PTCA, cariporide (HOE-642) did not alter the 36 day mortality (although at higher doses of 120 mg 8-hourly i.v. for 2 - 7 days, it reduced the incidence of Q-wave myocardial infarction

in patients undergoing CABG).⁴⁸ Isoprenaline also inhibits the Na⁺/H⁺ exchanger via a β_2 -adrenergic receptor mechanism.

In clinical practice, however, the stunned myocardium often responds well to sympathomimetic agents or calcium infusions, indicating that a lack of calcium rather than excess intracellular calcium for the intracellular contractile apparatus may be important in some cases.⁴³

3. Intracellular oedema⁴⁰

Hibernating myocardium

With chronic myocardial ischaemia, contractile dysfunction of the myocardium can exist without myocardial necrosis, due to a chronic down-regulation of contractility as an adaptive response, reducing myocardial oxygen demand to match the levels of the limited oxygen supply.⁵⁰ This phenomenon has been termed 'myocardial hibernation', and describes myocardial tissue that remains viable and will improve its contractile function if reperfused.^{5,51,52}

While myocardial hibernation and myocardial stunning are different pathophysiologically, the two may coexist and may be responsible for a large element of cardiac dysfunction in the patient with cardiogenic shock.

Clinical features

The clinical features of cardiogenic shock include, poor peripheral tissue perfusion (manifest by oliguria, drowsiness or agitation, peripheral cyanosis), tachycardia, hypotension, dyspnoea, diaphoresis and faint or infantile heart sounds due to a similar pitch of both first and second heart sounds.

Investigations

Electrocardiogram, chest X-ray, echocardiography (to demonstrate the defect and assess the regional and global ventricular function, and presence of a mechanical defect including ventricular septal defect, papillary muscle or free wall rupture rupture and tamponade), laboratory tests (including, blood gases, arterial lactate, plasma creatine phosphokinase, troponin, electrolytes, and creatinine), right heart catheter (to measure cardiac output, central venous, pulmonary artery and wedge pressures and mixed venous blood) and urinary catheter to measure hourly urine output, may all be required.⁵³

Treatment

Treatment of cardiogenic shock consists of methods to improve myocardial oxygenation as well as methods to improve peripheral tissue perfusion.

Improving myocardial oxygenation

This may be achieved by reducing myocardial oxygen demand (e.g. decreasing afterload and pulse rate), and increasing coronary perfusion (e.g. thrombolytic therapy, coronary bypass surgery, coronary angioplasty or coronary artery stenting) and ensuring adequate coronary perfusion pressures (e.g., MAP between 60-80 mmHg⁵⁴).

1. *Reducing myocardial oxygen demand.* While the use of β -adrenergic blocking agents to reduce myocardial oxygen demand in acute myocardial infarction has been associated with a reduction in mortality,⁵⁵ these agents have not been shown to benefit patients with cardiogenic shock.

2. *Increasing coronary perfusion.* Thrombolytic therapy reduces the likelihood of subsequent development of shock in patients with acute myocardial infarction,⁵⁶ although studies of cardiogenic shock due acute myocardial infarction have not shown that thrombolytic therapy consistently reduce mortality^{56,57} (probably due to a lower rate of reperfusion compared with patients who have acute myocardial infarction and normal blood pressure).

Urgent coronary bypass surgery has also not been shown to reduce mortality in patients with cardiogenic shock probably due to logistic and time problems in mobilising the surgical team, and the high surgical morbidity and mortality rates associated with operating on patients who are in shock. Nevertheless, with mechanical support using intra-aortic balloon counterpulsation, both thrombolytic therapy⁵⁸ and coronary bypass surgery⁵⁹ may be associated with a reduction in mortality in patients with cardiogenic shock.

Direct PTCA can achieve TIMI grade 3 flow in 80% - 90% of patients with susceptible coronary artery lesions with acute myocardial infarction.⁶⁰ As it can be performed rapidly, is more convenient, and has been associated with more favourable mortality and morbidity results,^{61,62} immediate PTCA should be considered in all patients with acute myocardial infarction who have sustained hypotension and tachycardia,⁶³ or in whom thrombolytic therapy is contra-indicated.

In one prospective randomised trial in patients with acute myocardial infarction and cardiogenic shock (e.g. systolic blood pressure < 90 mmHg for at least 30 minutes within 36 hours of the infarction), while intraaortic balloon counterpulsation and emergency revascularisation (e.g. angioplasty or CABG) did not reduce overall mortality at 30 days, it did produce an overall survival benefit after 6 months.⁶⁴

Coronary artery stenting has also been reported to

improve outcome in patients with cardiogenic shock, although it is usually performed when failed or suboptimal results with PTCA have occurred.^{65,66} The addition of antiplatelet agents (e.g. aspirin, ticlopidine, clopidogrel and abciximab)⁶⁷ and IABP^{68,69} also play an important role in maintaining coronary flow in patients in shock.

If the contractile mechanism is inhibited by drug toxicity, then treatment usually requires methods to improve peripheral tissue perfusion (e.g. inotropic agents, IABP, etc.) while specific treatment for the underlying disorder is underway.

Improving tissue perfusion

Perfusion of peripheral tissues can be increased without increasing myocardial oxygen requirements or reducing coronary blood flow when preload and afterload are optimised with fluid and vasoactive agents respectively. While agents that increase myocardial contractility can also increase tissue perfusion, they also increase myocardial oxygen requirements.

1. *Preload optimisation*: the intravascular volume is usually increased until the PAoP is 18 mmHg (to maximise preload and minimise risk of hydrostatic pulmonary oedema).

2. *Contractility*: inotropic agents generally increase myocardial oxygen requirements by their chronotropic rather than inotropic action;^{70,71} therefore in myocardial infarction, if an inotropic agent is deemed necessary, dobutamine (usually at 5 - 20 µg/kg/min) is believed to be the agent of choice, because its chronotropic effect is minimal.⁷² If hypotension remains refractory then adrenaline or noradrenaline at 2 - 20 µg/min (usually in association with intra-aortic balloon counterpulsation) may be used, titrated carefully to maximise coronary perfusion pressure with the least possible increase in myocardial oxygen demand.³⁵ Other inotropic agents (e.g. milrinone, theophylline, digoxin, glucagon) have long half-lives and are of no added benefit (and may even increase mortality) in patients with cardiogenic shock.

If cardiogenic shock is induced by agents that inhibit the contractile mechanism without inhibiting myocardial oxygenation (e.g. drug toxicity caused by local anaesthetics, tricyclics, β-adrenergic blockers, calcium-channel blockers or class I antiarrhythmics), myocardial oxygen requirements are usually not jeopardized, and inotropic agents such as isoprenaline, adrenaline, dopamine may be used to advantage.

Endocrine disorders (e.g. Addisonian crisis, myxoedema, pituitary apoplexy) require replacement therapy with hydrocortisone and/or tri-iodothyronine

before the cardiac contractile mechanism responds normally to an increase in intravascular volume and inotropic agents.

3. *Afterload optimisation*: vasodilators may be used to reduce the MAP to 60-80 mmHg. In the presence of a MAP of 60 mmHg or less, balloon counterpulsation can be effective.

Intra-aortic balloon counterpulsation⁷³

Intra-aortic balloon counterpulsation or intra-aortic balloon pumping (IABP) involves the percutaneous insertion of a balloon device into the descending aorta. The balloon is inflated during diastole and deflated during systole to reduce systolic afterload and increase diastolic perfusion pressure, thereby augmenting cardiac output and coronary blood flow.

The balloon catheter is usually inserted into the aorta via a femoral artery, so that the tip is positioned just below the level of the left subclavian artery (Figure 4). This is achieved by preparing and draping the patient and laying the balloon on the patient's chest and abdomen 1 cm below the angle of Louis and noting the level where the balloon would exit the femoral artery. The device is then inserted percutaneously under local anaesthesia using a Seldinger technique.⁷⁴ Heparin anticoagulation is optional (e.g. not included in the presence of a coagulopathy or recent surgery, but is often used if the balloon compromises the circulation to the lower limb).

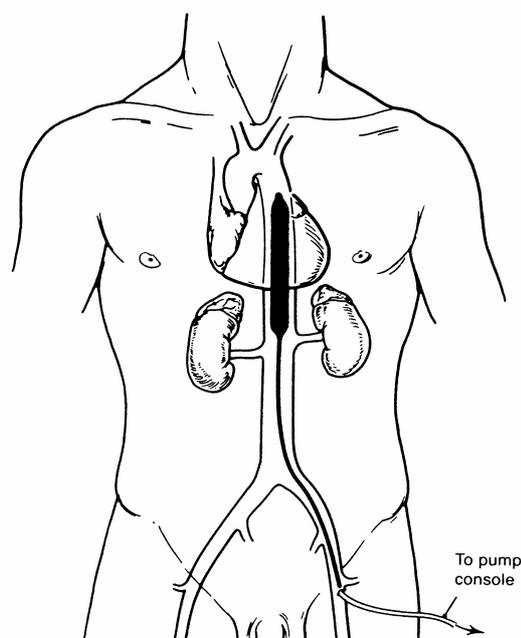


Figure 4. Positioning of the intra-aortic balloon in the descending thoracic aorta distal to the left subclavian artery.

The aortic waveform is monitored through the central lumen of the balloon. The balloon is inflated in diastole, usually as soon as the aortic valve closes (often timed to inflate just after the dicrotic notch), and deflated just before the onset of systole (e.g. 20 - 40 mL of gas is removed from the balloon) causing the aortic pressure to fall just as systole begins (Figure 5). Generally a 30 mL balloon is used for adult females and a 40 mL balloon is used for adult males. The timing of inflation and deflation can be individually altered to maximise the effect and efficiency of the device. To avoid damage to the aortic intima, the balloon is set to inflate so that it does not completely occlude the aorta.

While the major benefits of IABP are believed to be an improvement in coronary perfusion (during diastole) and reduction in afterload (during systole), in patients with coronary insufficiency, studies have only confirmed a reduction in left ventricular afterload, with no substantial increase in coronary blood flow distal to stenotic coronary arteries.^{75,76,77} The reduction in afterload increases left ventricular stroke volume and reduces PAoP, left ventricular stroke work and myocardial oxygen demand.

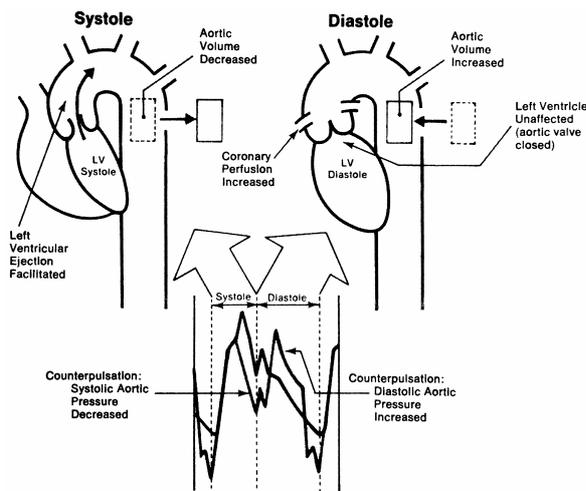


Figure 5. Haemodynamic changes with counterpulsation. Balloon inflation timed to the dicrotic notch and deflation timed to the onset of systole, reducing left ventricular afterload (facilitating left ventricular ejection) and increasing coronary perfusion pressure (Modified from Scheidt S, et al. Prog Cardiovasc Dis 1982;25:55-76)

The patient's balloon dependence is often tested daily by placing the balloon assist on standby, and observing the change in mean pulmonary artery pressure (MPAP) and MAP. If there is little change (e.g. no increase in MPAP or decrease in MAP) then the balloon augmentation (i.e. balloon inflation volume) is decreased and/or the pump frequency is reduced (e.g. the balloon pump is changed from augmenting every beat to alternate beats then every third beat over 4-8

hour periods) until the device is no longer needed. While the IABP is usually required for 4 - 8 days it has been used in some patients for up to 30 days.

Indications. Intra-aortic balloon counterpulsation is indicated in patients to provide circulatory support;

- until a surgical defect (e.g. ventricular septal defect, ruptured papillary muscle) has been corrected, or until the patient has received a cardiac transplant,⁷⁸
- in the cardiothoracic surgical patient when weaning from cardiopulmonary bypass has been difficult,
- in patients with refractory angina before coronary artery surgery is performed, and
- in patients with reversible cardiogenic shock (e.g. anaphylactic, local anaesthetic, quinidine or antihistamine toxicity⁷⁹).

While the use of these devices has not significantly increased survival in patients with ischaemia-induced cardiogenic shock in the absence of revascularisation procedures (e.g. PTCA or coronary artery stenting),^{80,81} in a review of patients with cardiogenic shock treated with thrombolytic therapy, early use of IABP was associated with a trend toward lower 30-day and 1-year all-cause mortality.⁸² The use of IABP has also been associated with a reduction in coronary artery reocclusion and cardiac events after angioplasty for acute myocardial infarction.^{68,69}

Contraindications. Intra-aortic balloon counterpulsation is usually contraindicated in patients who have severe aortic disease (e.g. dissecting aneurysm, bilateral aorto-iliac obstruction, recent aortic surgery, thoraco-abdominal aneurysm), or aortic regurgitation.

Complications. The complications associated with IABP include, insertion injuries, malposition of balloon, ischaemia of the leg in which the balloon is inserted, aortic embolism, aortic thrombus, aorto-iliac dissection, false femoral aneurysm, infection, thrombocytopenia, balloon rupture or leak with gas embolism and haemorrhage.

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