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# Understanding hypovolaemic, cardiogenic and septic shock

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## Summary

Shock is a complex physiological syndrome. If it is not detected and treated promptly, it can lead to death. This article reviews and summarises the latest findings, treatment and nursing and medical interventions for three of the most common forms of shock, namely, hypovolaemic, cardiogenic and septic shock.

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## Keywords

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## Aims and intended learning outcomes

The aim of this article is to provide nurses with a comprehensive overview of three of the most common types of shock, namely, hypovolaemic, cardiogenic and septic shock. Reading this article will assist nursing staff to become more confident in the identification and care of this group of patients. After reading this article you should be able to:

- ▶ Define shock.
- ▶ Describe the stages of shock, the major causes and the clinical manifestations of hypovolaemic, cardiogenic and septic shock.

- ▶ Discuss the treatment options available for the various types of shock described.
- ▶ Outline the nursing measures required to manage these patients.

## Time out 1

The clinical condition of shock is one of complex physiology. To understand the chain of events surrounding the shocked state, review the basics of cardiac and vascular anatomy and physiology before reading on.



## Introduction

Shock describes a life-threatening condition resulting from an imbalance between oxygen supply and demand, and is characterised by hypoxia and inadequate cellular function that lead to organ failure and potentially death (Kleinpell 2007).

Caused as a result of direct injury, or an underlying medical condition, shock can be life-threatening (Hand 2001, Chavez and Brewer 2002, Bench 2004). Shock occurs when the circulatory system is no longer able to complete one of its essential functions, such as providing oxygen and nutrients to the cells of the body or removing subsequent waste (Chavez and Brewer 2002). This results in inadequate tissue perfusion (Cottingham 2006, Justice and Baldisseri 2006, Medline Plus 2007), which triggers a cascade of events.

Shock has many causes, including sepsis, cardiac pump failure, hypovolaemia and anaphylaxis (Hand 2001, Justice and Baldisseri

2006), and is classified into various types. The three basic types of shock are hypovolaemic, cardiogenic and distributive (Hand 2001). Distributive shock can be further classified as neurogenic, anaphylactic and septic shock. The purpose of this article is to identify and discuss three of the most common types of shock, in addition to reviewing the causes, treatment modalities and nursing considerations.

## Stages of shock

Research into shock has resulted in the classification of four distinct stages of shock (Chavez and Brewer 2002, Kleinpell 2007). These are: initial, compensatory, progressive and refractory (Kleinpell 2007).

**Initial** In the initial stage of shock, the body experiences a reduced cardiac output (Box 1). During this stage, nurses should be aware that the cells switch from aerobic to anaerobic metabolism, which can lead to lactic acidosis, that is, excess acid resulting from a build up of lactic acid in the blood and lowering of the pH. Although clinical signs and symptoms may be

subtle at this time, cellular damage can occur. A serum lactate level will provide an accurate assessment of acidosis because septic patients typically convert to anaerobic metabolism as a result of hypoperfusion. If the underlying cause of shock is not treated at this time, the patient will progress to stage two.

**Compensatory** This stage is characterised by the body's attempt to regain homeostasis and improve tissue perfusion. Here, the sympathetic nervous system is stimulated resulting in catecholamine release (Box 1) (Chavez and Brewer 2002). This neurohormonal response causes increased cardiac contractility, vasoconstriction and a shunting of blood to the vital organs. The adrenal/renal system releases aldosterone, which promotes water conservation in an effort to maintain intravascular volume.

**Progressive** In this third stage of shock, the body has lost its compensatory mechanisms, which have sustained tissue perfusion to this point. This decrease in perfusion results in metabolic acidosis, electrolyte imbalance and respiratory acidosis. The clinical symptoms will be such that there should be no doubt as to the severity of the patient's

### BOX 1

#### Glossary

Term	Definition
Afterload	A resistance that the left ventricle must work against to pump blood through the aorta.
Arterial blood gases	A blood sample taken from an artery, that when analysed enables evaluation of gaseous exchange in the lungs by measuring the partial pressure of gases dissolved in arterial blood.
Cardiac output	The amount of blood ejected from the left ventricle per minute. Usual cardiac output is 4-8L/min.
Catecholamine	Naturally occurring chemicals that stimulate the nervous system, constrict peripheral blood vessels, increase heart rate and dilate the bronchi.
Central venous pressure (CVP)	CVP reflects the amount of blood returning to the heart and the ability of the heart to pump the blood into the arterial system. It is a good approximation of right atrial pressure.
Colloids	A large molecule, such as albumin, that does not cross the capillary membrane in solution.
Crystalloids	A solute, such as sodium or glucose, that crosses the capillary membrane in solution, for example, sodium chloride 0.9% solution
Inotrope	A drug that affects the contraction of cardiac muscle.
Intra-aortic balloon pump	A balloon-type device inserted into the aorta, with the goal of being able to reduce the workload of the left ventricle and improving coronary perfusion.
Mean arterial pressure	The average arterial pressure during a single cardiac cycle.
Peripheral oedema	The accumulation of fluids in the interstitial tissues of those areas affected by gravity, such as the legs, feet and hands. Any oedema is an abnormal condition.
Pulmonary oedema	Fluid accumulation in the lungs due to failure of the heart to remove fluid from the lung circulation or following direct injury to the lungs. It leads to impaired gas exchange and may cause respiratory failure.
Preload	A stretching force exerted on the ventricle muscle by the blood it contains at the end of diastole.
ScvO <sub>2</sub>	The oxygen saturation of venous blood as it returns to the heart, measured at the superior vena cava.
SpO <sub>2</sub>	The oxygen saturation of peripheral blood, which can reflect respiratory status.
Thrombolysis	Dissolution or destruction of a thrombus.
Vasopressors	Drugs that stimulate cardiac contraction of the muscular tissues of the capillaries and arteries.

condition. The nurse will be able to observe severe hypotension, pallor, tachycardia and irregular rhythm, peripheral oedema, cool and clammy extremities and an altered level of consciousness. During this stage the blood pH decreases as the lactic acid production increases (Kleinpell 2007).

**Refractory** At this late stage, irreversible cellular and organ damage occur. Shock becomes unresponsive to treatment and death is likely (Hand 2001) (Table 1).

diverse origins, as opposed to a few defined and specific causes. It is characterised by an inadequate intravascular volume caused by significant blood and/or fluid loss (Hand 2001, Chavez and Brewer 2002, Bench 2004). This intravascular depletion can be caused by sustained vomiting, diarrhoea or severe dehydration (Hand 2001, Diehl-Oplinger and Kaminski 2004), as well as burns, traumatic injury and surgery (Diehl-Oplinger and Kaminski 2004). Simple blood loss is often the most common source of hypovolaemic shock (Medline Plus 2007), which can occur from bleeding either inside or outside the body. Internal fluid collection, such as ascites and peritonitis, may also cause hypovolaemic shock (Hand 2001).

**Clinical manifestations of hypovolaemic shock**

Hypovolaemic shock has many clinical manifestations (Box 2), which coincide with the stages of shock as defined earlier. With a fluid loss of less than 750ml, the body may enter a compensated state (Bench 2004), and changes to vital signs may be subtle and difficult to detect. At this point, the body essentially maintains homeostasis and the patient may be asymptomatic. For this reason, nurses should pay close attention to even the subtlest change in vital signs, while using their nursing judgement to summon help for the patient as appropriate. As fluid loss increases to more than 750ml, cardiac output begins to fall (Hand 2001, Chavez and Brewer 2002), and changes in vital signs occur. As a result of the falling cardiac output, the sympathetic chain of the autonomic nervous system is initiated with the ‘fight or flight’ response (Hand 2001), and catecholamines are released. Vasoconstriction occurs as the nervous system endeavours to manoeuvre the blood away

**Time out 2**

Bill, a 52-year-old patient, returns from vascular surgery at 2pm. He is in no pain and breathing normally with an oxygen saturation (SpO<sub>2</sub>) of 98% on 40% oxygen (Box 1). His vital signs are stable, with a slightly elevated heart rate of 99 beats per minute (bpm). He is receiving sodium chloride 0.9% at 100ml/hr and urine output has been greater than 50ml/hr for the past three hours. By 4pm, Bill's vital signs are as follows: heart rate 137bpm, blood pressure 89/50mmHg, temperature 36.9°C, SpO<sub>2</sub> 92% on 55% oxygen. His urine output was 30ml for the past hour. His abdomen is firm and his skin looks pale. He is showing no electrocardiogram (ECG) changes, other than tachycardia. Given this clinical condition, which type of shock is Bill most likely to be experiencing? Provide a possible suggestion for the cause of shock. A suggested answer is provided on page 55.

**Hypovolaemic shock**

Hypovolaemic shock is different from cardiogenic and septic shock in that it has many varied and

**TABLE 1**

**The stages of shock**

Initial stage	Compensatory stage	Progressive stage	Refractory stage
<ul style="list-style-type: none"> <li>▶ Body switches from aerobic to anaerobic metabolism</li> <li>▶ Elevated lactic acid level</li> <li>▶ Subtle changes in clinical signs</li> </ul>	<ul style="list-style-type: none"> <li>▶ Sympathetic nervous system stimulated</li> <li>▶ ↑ catecholamine release</li> <li>▶ ↑ cardiac contractility</li> <li style="text-align: center;">↓</li> <li>▶ Neurohormonal response: vasoconstriction and blood shunted to vital organs</li> <li style="text-align: center;">↓</li> <li>▶ Aldosterone released</li> <li>▶ ↓ urine output (&lt;30ml/hr)</li> <li style="text-align: center;">↓</li> <li>▶ ↑ Heart rate</li> <li style="text-align: center;">↓</li> <li>▶ ↑ Glucose levels</li> </ul>	<ul style="list-style-type: none"> <li>▶ Electrolyte imbalance</li> <li>▶ Metabolic acidosis</li> <li>▶ Respiratory acidosis</li> <li>▶ Peripheral oedema</li> <li>▶ Irregular tachyarrhythmias</li> <li>▶ Hypotension</li> <li>▶ Pallor</li> <li>▶ Cool and clammy skin</li> <li>▶ Altered level of consciousness</li> </ul>	<ul style="list-style-type: none"> <li>▶ Irreversible cellular and organ damage</li> <li>▶ Impending death</li> </ul>

(Springhouse 2004)

**BOX 2****Clinical manifestations of hypovolaemic shock**

- ▶ Altered or decreased level of consciousness
- ▶ Anxiety and restlessness
- ▶ Decreased urine output
- ▶ Delayed capillary refill
- ▶ Increased heart rate
- ▶ Increased respiratory rate
- ▶ Pale, cool and clammy skin
- ▶ Systolic blood pressure <90mmHg or 40mmHg below baseline

from the non-vital organs of the gut and extremities, and towards the central core. As a result, the patient's extremities may be cool and clammy and the patient may exhibit signs of anxiety (Hand 2001). Other compensatory mechanisms are initiated by the renal system (Bench 2004) and its release of renin. A cascade of events ensues with the production and release of the angiotensin-angiotensin II-aldosterone flow. This sequence promotes vasoconstriction and the reabsorption of sodium and water in an attempt to increase blood volume. Both of these mechanisms may increase blood pressure. Other clinical indicators that are noticeable in patients with hypovolaemic shock include increased respirations and decreased urine output (Chavez and Brewer 2002).

If the hypovolaemia remains undetected or untreated, the patient's clinical condition will worsen, the patient will become unstable and his or her blood pressure will drop significantly. As a result of tachycardia, cardiac arrhythmias and/or chest pain may occur due to the inability of the coronary arteries to fill adequately during diastole (American College of Surgeons Committee on Trauma: Shock 1997). Respirations will increase as the body tries to rid itself of accumulating lactic acid. Eventually an altered level of consciousness ensues because of lack of tissue perfusion (Diehl-Oplinger and Kaminski 2004).

After approximately a 40% fluid loss, the situation can become life threatening. Multi-organ damage and cellular necrosis can occur with impending death likely (American College of Surgeons Committee on Trauma: Shock 1997).

**Treatment** An essential aspect of treatment for patients with hypovolaemic shock is to restore fluid volume and blood pressure. This is usually achieved with intravenous fluids and vasopressors (Box 1).

Oxygen should be administered to counteract the respiratory effects of shock (Hand 2001). In some cases, it may be enough to administer oxygen by facial mask, but if shock progresses there may be a need for endotracheal intubation

and mechanical ventilation. Whenever possible the head of the patient's bed should be elevated to promote comfort and adequate respirations. This is also a preventive measure against pneumonia, especially if the patient is ventilated (Craven 2006). Nevertheless, if the patient's condition is such that hypotension is pronounced, this may not always be possible. Nurses should use critical thinking skills and draw on their clinical knowledge to determine the appropriate position for the patient.

Hypovolaemic shock requires immediate fluid resuscitation (Diehl-Oplinger and Kaminski 2004), in conjunction with treating the underlying cause if the patient is to survive.

There has been much debate in recent years as to whether colloids or crystalloids (Box 1) are the best choice for fluid resuscitation (Bench 2004). There seems to be growing consensus that crystalloids are the superior choice (Diehl-Oplinger and Kaminski 2004) because they mimic intracellular fluid more closely. Bench (2004) and Kirschman (2004) claim that colloids have been associated with multiple issues such as infections and reactions to the properties of the fluid(s). Common practice dictates that crystalloids are used for fluid volume loss of less than 1,500ml (Hand 2001) while whole blood is used if the volume loss is greater, or if the sole cause of the hypovolaemia is blood loss (Chavez and Brewer 2002, Bench 2004, Diehl-Oplinger and Kaminski 2004). Crystalloids such as lactated Ringer's solution or sodium chloride 0.9% can address both hypovolaemia and electrolyte imbalances (Chavez and Brewer 2002). Treatment goals include raising mean arterial pressure (MAP) (Box 1) to 70mmHg by infusing 1,000-2,000ml of warmed crystalloid (Cottingham 2006). Additional fluids are administered based on the patient's clinical condition.

If colloids are used, they are used in smaller quantities than crystalloids (Diehl-Oplinger and Kaminski 2004) because they are a hypertonic solution. Blood transfusions may also be required clinically. However, regardless of the type of fluid infused, caution is necessary in patients with pre-existing heart failure, so as not to promote pulmonary oedema (Box 1). As with all large fluid infusions, blood samples should be drawn and tested at regular intervals to check haemoglobin levels, haematocrit, electrolytes and other tests as prescribed by the consulting physician.

It may also be necessary to induce vasoconstriction with inotropic and vasopressor medication (Box 1). The most common pharmacologic agents used in the critically ill shock patient are adrenaline (epinephrine), noradrenaline (norepinephrine) (Chavez and Brewer 2002) and dobutamine (Cottingham 2006). The goal of treatment is to increase cardiac output and MAP. Nurses should be aware that

inotropic drugs or vasopressors should not be started until the patient has an adequate fluid volume or fluid volume has been replaced.

### Cardiogenic shock

Cardiogenic shock results in a decline in cardiac output and tissue hypoxia, despite adequate fluid volume. It remains the most serious complication of acute myocardial infarction (MI) (Sanborn and Feldman 2004), often resulting in death (Ducas and Grech 2003, Mann and Nolan 2006). Cardiogenic shock occurs in 5-10% of MI patients (Ducas and Grech 2003, Sanborn and Feldman 2004, Mann and Nolan 2006), and has a mortality rate of more than 50% (Sanborn and Feldman 2004). If revascularisation of the myocardium does not occur promptly, the outcome is usually fatal (Mann and Nolan 2006).

The causes of cardiogenic shock are limited, unlike that of hypovolaemic shock. It typically occurs following an MI and more commonly in ST-segment elevation MIs (STEMIs) (Holmes 2003) as a result of left ventricular failure (Ducas and Grech 2003, Sanborn and Feldman 2004, Bouki *et al* 2005). However, other causes include acute, severe mitral regurgitation and ventricular septal rupture. Mann and Nolan (2006) add cardiac tamponade as a further source. In some rare cases medications have been known to cause this condition, specifically metoprolol and clopidogrel (Mann and Nolan 2006). McLuckie (2003) also asserts that the older female patient is more at risk of developing cardiogenic shock, as are patients with diabetes and those with a history of previous MI.

#### Clinical manifestations of cardiogenic shock

The clinical manifestations of cardiogenic shock can be similar to that of hypovolaemic shock, although in cardiogenic shock the patient's condition can deteriorate more quickly (Bench 2004). Essentially, the damaged left ventricle is unable to pump effectively (Chavez and Brewer 2002), and thus cardiac output is reduced. Characteristically, the cardiac output will be reduced to less than 2.2L/min (usual cardiac output is 4-8L/min). In an effort to compensate, the remaining non-ischaemic myocardium becomes hypercontractile (Ducas and Grech 2003). This action raises the oxygen demands of the heart, which further increases the workload. As blood pressure falls, catecholamines are released which cause vasoconstriction. In contrast to hypovolaemic shock, vasoconstriction can be detrimental in patients with cardiogenic shock, in

that it forces the already damaged myocardium to work even harder (McLuckie 2003, Bench 2004). The failing myocardium cannot work as efficiently as usual, and so may not be able to clear blood quickly enough, which results in a build up of blood in the atrium. In turn, this can cause congestion in the lungs leading to pulmonary oedema and a compromised respiratory system, necessitating supplementary oxygen and potential mechanical ventilation.

Further symptoms include increased central venous pressure (CVP) (Box 1), chest pain as a result of the decreased coronary artery perfusion and low urine output (Bench 2004). The patient may also experience anxiety, as a result of pain, and feelings of doom and general demise (Hand 2001). The role of the nurse in this situation is to reduce the patient's pain and anxiety, thereby decreasing myocardial workload.

**Treatment** Treatment options for cardiogenic shock are varied. An essential focus is on revascularisation of the damaged myocardium, and improving cardiac contractility and blood pressure (Bench 2004). Nevertheless, Ducas and Grech (2003) and Mann and Nolan (2006) maintain that respiratory function and oxygen delivery should be the first consideration. Oxygen is necessary to combat the effects of cardiac ischaemia and associated chest pain. Additional attention should be given to the management of pulmonary oedema, which can be addressed with diuretics. Evaluation of arterial blood gases (ABGs) (Box 1) and cardiac monitoring are important nursing responsibilities.

Reperfusion of the myocardium can occur as a result of thrombolysis (Box 1), or mechanical revascularisation by means of invasive procedures such as percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG) (Mann and Nolan 2006). Thrombolysis involves the injection of pharmacologic agents such as streptokinase, urokinase or tissue plasminogen activator (TPA), which act to dissolve a clot in the affected coronary artery (American Heart Association 2007). However, one of the keys to their success is that they are used within a few hours of the initial insult and before cardiogenic shock sets in. Once cardiogenic shock develops, thrombolysis may have little effect on the patient's clinical condition.

Mechanical reperfusion can be accomplished through PCI or CABG, and in patients under 75 years of age, the American College of Cardiology/American Heart Association have listed these procedures as Class I recommendations (Mann and Nolan 2006). A report from the National Registry of Myocardial Infarction reported improved patient survival when PCI, such as coronary angioplasty, was used for cardiogenic shock in this age group (Babaev *et al* 2005). Survival

rates from PCI and CABG are similar at 55.6% and 57.4% respectively (Sleeper *et al* 2005).

### Time out 3

List the most frequently used drugs to treat patients with cardiogenic shock. Discuss the dose range and the effects that treatment with these medications will have.

The use of inotropic agents and vasopressors in cardiogenic shock is widespread (Table 2), although they are usually viewed as a supportive measure rather than a curative intervention (Justice and Baldisseri 2006). When administered in the intensive care unit (ICU) usual medications include dopamine, dobutamine and noradrenaline (norepinephrine). Dopamine, however, has been associated with increased mortality (Bench 2004), and so should be used with caution. Vasodilators such as sodium nitroprusside and glycerin trinitrate (GTN) may also be used to reduce left ventricular afterload (Box 1). As with any medications that can potentially affect blood pressure, frequent monitoring of the patient's vital signs is imperative (Bench 2004). Fluids may also be necessary in cardiogenic shock, but should be administered with extreme caution, especially in the presence of pulmonary oedema (Chavez and Brewer 2002, Ducas and Grech 2003).

In the most severely ill patients an intra-aortic balloon pump (IABP) may be used (Box 1). The IABP is another Class I recommendation (Chavez and Brewer 2002, Ducas and Grech 2003, Sanborn and Feldman 2004, Mann and Nolan 2006). This invasive balloon-attached catheter is inserted via the femoral artery, and sits in the descending thoracic aorta (Bouki *et al* 2005). The IABP is attached to an external machine which aids balloon inflation and deflation at exact moments in the

cardiac cycle. Inflation occurs during diastole, with deflation following during systole. The goal of the IABP is to increase coronary artery perfusion during diastole and reduce systemic afterload during systole. The IABP is often used in conjunction with pharmacological agents and other interventions.

The ventricular assist device (VAD) is a final treatment option that may be used in the cardiothoracic ICU as a 'bridge to transplantation' (Cleveland Clinic Foundation 2004). This device is used in a last-stage effort to save the patient's life, when the damage from cardiogenic shock is so severe that only a cardiac transplant will prevent death. The VAD is a mechanical pump that is attached to the patient's heart and is situated outside the body (Figure 1). It is used to circulate blood and assist the failing heart.

### Septic shock

In North America and Europe more than 750,000 individuals develop sepsis each year (Institute for Healthcare Improvement (IHI) 2005a). If septic shock develops, the mortality rate is estimated to be approximately 40-50% (Jindal *et al* 2000, Oppert *et al* 2005). Septic shock, the result of an overwhelming infection (Box 3), leads to hypotension, altered coagulation, inflammation, impaired circulation at a cellular level, anaerobic metabolism, changes in mental status and multi-organ failure (Kleinpell 2003a, 2003b, Rivers *et al* 2005). In septic shock, 'there is a complex interaction between pathologic vasodilation, relative and absolute hypovolaemia... direct myocardial depression' (Beale *et al* 2004). Although recognising the early signs of septic shock may be difficult, the nurse's role is pivotal in identifying these changes and facilitating immediate medical treatment (Bridges and Dukes 2005).

TABLE 2

Medications used to treat patients in cardiogenic shock			
Drug	Class	Dose	Effect
Dobutamine	Inotrope	2-40mcg/kg/min	Increase cardiac contractility and cardiac output.
Dopamine	Inotrope	5-20mcg/kg/min	Increase contractility and vasoconstriction.
Milrinone	Phosphodiesterase inhibitor	0.375-0.75mcg/kg/min (reduce dose in renal failure)	Increase cardiac contractility and dilate vascular smooth muscle.
Noradrenaline	Catecholamine	2-30mcg/min	Vasoconstriction. Increases peripheral vascular resistance.
Nitroglycerin	Vasodilator	Start at 5mcg/min Maximum dose 200mcg/min	Decreases preload and myocardial oxygen demand. Improves coronary artery blood flow.
Sodium nitroprusside	Vasodilator	0.5-6mcg/kg/min. Maximum dose is 10mcg/kg/min for <10 minutes	Reduces afterload in decreased cardiac output states.

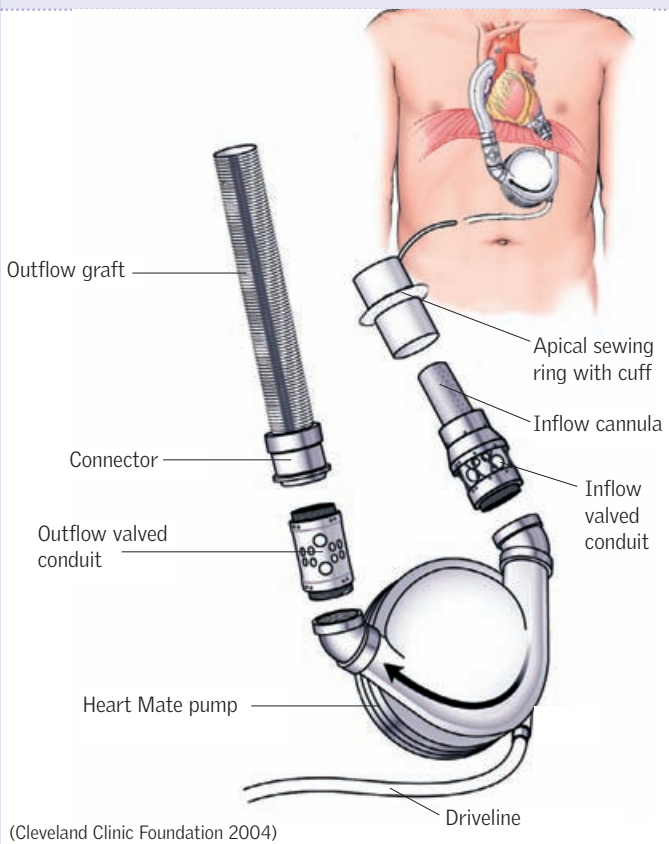
(Adapted from Sasada and Smith 2003, Lynn McHale-Wiegand and Carlson 2005)

Guidelines, which closely mirror those traditionally used for other diseases such as acute MI, stroke and trauma, have now been developed for the early diagnosis and treatment of sepsis (Rivers *et al* 2005). The Surviving Sepsis Campaign, an international initiative, recommends a 24-hour sepsis pathway, with the therapeutic goal of improving survival of these patients and decreasing mortality (IHI 2005b).

### Time out 4

John is a 72-year-old patient admitted to the intensive care unit with a change in level of consciousness, blood pressure of 86/46 mmHg, respiratory rate of 36, urine output of 15ml/hr for the past three hours, a rectal temperature of 38.6 °C, and a heart rate of 140 beats per minute. The urinalysis completed in the accident and emergency department was positive for a urinary tract infection. The patient has already received two litres of sodium chloride 0.9%, with no response in blood pressure. What two vasopressors are most likely to be prescribed by the consultant on the unit? A suggested answer is on page 55.

**FIGURE 1**  
**Ventricular assist device**



### BOX 3

#### Sources of infection in septic shock

- Blood:** bacteraemia.
- Bone:** osteomyelitis.
- Cardiovascular:** endocarditis and pericarditis.
- Central nervous system:** meningitis.
- Intra-abdominal:** diverticulitis, appendicitis and perforated or ischaemic bowel.
- Invasive catheters:** central venous or peripheral cannula.
- Pulmonary:** community acquired or healthcare-associated pneumonia.
- Soft tissue:** cellulitis, skin and wound infections and necrotising fasciitis.
- Surgical wounds:** incision and deep infection.
- Urinary tract:** urinary tract and kidney infections.

**Treatment** A classic sign of septic shock is the patient's development of both absolute and relative hypovolaemia (Hollenberg 2001, Beale *et al* 2004), and as such, fluid resuscitation is a primary element in the treatment plan. Absolute hypovolaemia can be the result of fluid loss due to vomiting, diarrhoea, sweating or oedema. Relative hypovolaemia occurs as a result of vasodilation and peripheral blood pooling (Vincent and Gerlach 2004). The type of fluid to be used during resuscitation is still under debate (Vincent and Gerlach 2004, Bridges and Dukes 2005); however, it is recommended that a minimum of 20ml/kg of crystalloid (or colloid equivalent) is administered initially to patients who are hypotensive as a result of sepsis-related hypovolaemia (IHI 2005c). If fluid resuscitation efforts are unsuccessful at maintaining a MAP of 60-65mmHg or if fluid resuscitation is in progress and hypotension remains life-threatening, then vasopressor therapy should be initiated (Bridges and Dukes 2005, IHI 2005c, Robson and Newell 2005). Indicators of adequate fluid resuscitation and tissue perfusion include a urine output greater than 0.5ml/kg/hr, a decrease in serum lactate level, improved level of consciousness and a CVP ranging between 8-12mmHg or 12-15mmHg for patients receiving mechanical ventilation (Bridges and Dukes 2005, Shapiro *et al* 2006).

Commonly used vasopressor therapy includes dopamine, noradrenaline (norepinephrine), adrenaline (epinephrine) and phenylephrine (Bridges and Dukes 2005). The Surviving Sepsis Campaign guidelines recommend dopamine and noradrenaline (norepinephrine) as the first-line choice in septic shock (Bridges and Dukes 2005, IHI 2005d, Levy *et al* 2005). An arterial catheter is typically used for continuous and accurate

blood pressure monitoring (Beale *et al* 2004). Careful consideration is required when implementing vasopressor treatment to ensure adequate fluid volume resuscitation occurs; otherwise its use may be harmful and result in a further decrease in organ perfusion (Bridges and Dukes 2005).

Another resuscitation goal includes maintaining a central venous oxygen saturation (ScvO<sub>2</sub>) greater than 70% (Box 1) (Robson and Newell 2005, Shapiro *et al* 2006). If the ScvO<sub>2</sub> is less than 70% and the haematocrit is less than 30%, then a blood transfusion can be considered. If the haematocrit is greater than 30%, dobutamine may be used (Shapiro *et al* 2006).

Once a diagnosis of sepsis has been determined, antibiotic therapy should be administered in a timely fashion – within minutes rather than hours. However, controversy exists regarding the timeframe in which antibiotic therapy should be initiated (Kumar *et al* 2006). The choice of antibiotic is dependent on the pathogen, drug tolerance and other underlying diseases. It is recommended that broad-spectrum antibiotics are administered within three hours for patients seen in the accident and emergency department and within one hour for ward and ICU patients (IHI 2005e).

One of the manifestations of septic shock can be an alteration in coagulation (Kleinpell 2003a, 2003b). This occurs as a result of an inflammatory response, stimulation of the coagulation cascade and a reduction in protein C and antithrombin III. These events produce an enhanced state of coagulation, sepsis-associated coagulopathy and even death (Kleinpell 2003a). Drotrecogin alfa (activated) is an adjunctive therapy used to treat patients with this type of enhanced state of coagulation (Kleinpell 2003a, Robson and Newell 2005). The Protein C Worldwide Evaluation in Severe Sepsis (PROWESS) trial indicted that the use of drotrecogin alfa (activated) or recombinant activated protein C improves survival in septic patients, and is recommended for septic patients at high risk of death (Kleinpell 2003a). Recombinant activated protein C can increase the risk of bleeding and is contraindicated in some patients (Robson and Newell 2005, IHI 2005f). As a consequence, specific nursing considerations are necessary when administering this drug (Box 4).

Corticosteroid therapy is an additional treatment option. The anti-inflammatory effect of glucocorticoids has meant that they have been used for decades in the treatment of septic patients (Keh and Sprung 2004), yet high-dose corticosteroid therapy has not been shown to improve patient outcomes (Oppert *et al* 2005).

Glucocorticoids administered in high dosages, for example, 2-8g methylprednisolone, may even be detrimental (Oppert *et al* 2005). More recently

the use of low-dose glucocorticoids has been added to treatment plans (Keh and Sprung 2004). Adrenal function tests can be used to steer the decision regarding the use of corticosteroid therapy if adrenal insufficiency is suspected (Keh and Sprung 2004).

## Nursing considerations

Given the clinical complexity and potentially devastating consequences of shock, it is essential that the nurse remains diligent in the care of these patients. Understanding the clinical signs that the patient demonstrates during each stage of shock will assist nurses with patient assessments and in carrying out the treatment plan.

As with every clinical situation, the basics of nursing and medical attention should be paramount. Oxygenation and respiratory function are always a priority, whether patients are able to maintain their own airway or mechanical ventilation is required. Proper positioning to promote respiratory function is essential and ABGs should be monitored as necessary. Circulatory function should be addressed with a combination of fluids and/or medications, which may be reliant on the type of shock involved.

As noted earlier in this article, clinical signs during the initial stage of shock may be cryptic, so the astute nurse should identify patients at risk, and monitor vital signs carefully, including body temperature, haemodynamic function, urine output, level of consciousness and laboratory values. Monitoring lactic acid levels is of primary importance, as in the initial stage the body is

### BOX 4

#### Nursing considerations when administering drotrecogin alfa (activated)

- ▶ Administer medication through a dedicated intravenous (IV) catheter.
- ▶ Administer continuously at a rate of 24mcg/kg per hour for 96 hours or according to a specific hospital policy.
- ▶ Ensure a bedside risk assessment is completed to avoid administration to high-risk patients such as those with active or recent internal bleeding, recent haemorrhagic stroke, trauma with increased risk of bleeding or the presence of an epidural catheter.
- ▶ Administer with the use of an IV infusion pump.
- ▶ Discontinue infusion two hours before any procedure that may carry with it a risk of bleeding.
- ▶ Restart infusion one hour after an uncomplicated minor procedure or 12 hours after a major procedure or surgery.

(Kleinpell 2003a)



converting from aerobic to anaerobic metabolism, and this laboratory value may be one of the first signs of impending shock.

The established plan of care should focus on preventing the progression of shock. In the event that this goal cannot be accomplished and the patient advances to the compensatory stage or further, a transfer to the ICU is warranted.

The ICU nurse should focus on the maintenance of homeostasis through the use of fluid resuscitation, vasopressors and antibiotics, depending on the type of shock. In addition to continued diligence in vital sign monitoring and regular physical assessments, a central catheter should be inserted to facilitate rapid infusion of fluids and/or medications. Evaluation of a patient's fluid status should be monitored regularly by measuring the CVP. An indwelling urinary catheter should be inserted to assist in maintaining accurate fluid balance. If the patient is in cardiogenic shock and IABP monitoring is necessary, close observation of the patient's limb is important, as ischaemia can be a complication of these devices. IABPs can reduce blood flow to the leg or thrombus

formation can occur around the catheter. As more invasive devices are used to monitor and treat the patient in shock, strict adherence to aseptic technique is vital to prevent infection.

The nurse should also remain attentive to basic nursing measures, such as frequent and thorough mouth care, pressure area care and repositioning, pain control and emotional support. Emotional support is vital for the patient, but it is also extremely important for nurses to recognise the turmoil that family members may be experiencing.

## Conclusion

Shock is a complex clinical syndrome that, if not detected and treated promptly, can lead to death. Despite the many advances in medical and nursing care in recent years, the mortality rate remains high for patients who develop shock, and especially for those patients who develop cardiogenic or septic shock. For these reasons it is essential for nursing staff to realise that patients may present in shock, yet provide little indication of this in terms of changes in vital signs or other outward deterioration, at least in the initial stages. It is essential that nurses caring for these patients understand the clinical manifestations

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of shock and how each type of shock differs from the other(s). Nurses should also be familiar with treatment options and best practices for this patient group. Dealing with the patient in shock is a challenge for critical care practitioners, yet basic assessment skills and a good knowledge of pathophysiology can assist the nurse to provide the best care possible for these patients **NS**

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### Suggested answers to time out activities

#### Time Out 2

Bill is apyrexial, which combined with very recent surgery and a firm abdomen would rule out septic shock at this point. He experiences no ECG changes and is not in any pain, cardiac or otherwise, which would mean that cardiogenic shock is unlikely. The most likely cause given these clinical circumstances is hypovolaemic shock as a result of the firm abdomen, increased heart rate and decreased blood pressure and

urine output. These clinical indicators are suggestive of internal haemorrhage. A possible cause may be a bleeding blood vessel which occurred during surgery.

#### Time Out 4

Dopamine and noradrenaline (norepinephrine) are the two vasopressors most likely to be prescribed by the consultant. Dopamine is usually administered because of its ability to increase mean arterial pressure (MAP).

Dopamine does cause an increase in heart rate, which may indicate a need to add a second type of vasopressor therapy. Noradrenaline (norepinephrine) is the second most likely choice of vasopressor. Noradrenaline also increases the MAP as a result of vasoconstriction but does not have an effect on the heart rate.

### Time out 5

Now that you have completed the article you might like to write a practice profile. Guidelines to help you are on page 60.

(Activated) by a Standard Policy. [www.ihl.org/IHI/Topics/CriticalCare/Sepsis/Changes/IndividualChanges/AdministerDrotrecoginAlfaActivate.dbyaStandardPolicy.htm](http://www.ihl.org/IHI/Topics/CriticalCare/Sepsis/Changes/IndividualChanges/AdministerDrotrecoginAlfaActivate.dbyaStandardPolicy.htm) (Last accessed: July 27 2007)

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# Shock

TEST YOUR KNOWLEDGE AND WIN A £50 BOOK TOKEN

## HOW TO USE THIS ASSESSMENT

This self-assessment questionnaire (SAQ) will help you to test your knowledge. Each week you will find **ten multiple-choice questions which are broadly linked to the learning zone article.**

Note: There is only one correct answer for each question.

### Ways to use this assessment

- ▶ You could test your subject knowledge by attempting the questions before reading the article, and then go back over them to see if you would answer any differently.
- ▶ You might like to read the article to update yourself before attempting the questions.

The answers will be published in *Nursing Standard* two weeks after the article appears.

### Prize draw

Each week there is a draw for correct entries. Send your answers on a postcard to: Nursing Standard, The Heights, 59-65 Lowlands Road, Harrow, Middlesex HA1 3AW, or via email to: zena.latcham@rcnpublishing.co.uk

Ensure you include your name and address and the SAQ number. This is SAQ No 406. Entries must be received by 10am on Tuesday September 4 2007.

When you have completed your self-assessment, cut out this page and add it to your professional portfolio. You can record the amount of time it has taken you. Space has been provided for comments and additional reading. You might like to consider writing a practice profile, see page 60.

### 8. A central nervous system source of infection in septic shock is:

- a) Osteomyelitis
- b) Meningitis
- c) Appendicitis
- d) Urinary tract infection

### 9. An infusion of drotrecogin alfa (activated) should be restarted how many hours after surgery?

- a) One
- b) Four
- c) Six
- d) 12

### 10. What percentage of patients who have had a myocardial infarction experience cardiogenic shock?

- a) 1-5
- b) 5-10
- c) 15-25
- d) 30-50

*This self-assessment questionnaire was compiled by Lisa Berry*

### 1. A cause of shock is:

- a) Sepsis
- b) Anaphylaxis
- c) Cardiac pump failure
- d) All of the above

### 2. Hypotension is a manifestation of which stage of shock?

- a) Initial
- b) Compensatory
- c) Progressive
- d) Refractory

### 3. In hypovolaemic shock, systolic blood pressure is likely to be:

- a) <90mmHg
- b) 110mmHg
- c) 120mmHg
- d) 140mmHg

### 4. For fluid volume loss of less than 1,500ml in hypovolaemic shock, what fluid resuscitation measure should be used?

- a) Colloids
- b) Crystalloids
- c) Plasma
- d) Hypertonic

### 5. In patients with cardiogenic shock, cardiac output is usually reduced to:

- a) 3.2L/min
- b) 4.2L/min
- c) 4-8L/min
- d) <2.2L/min

### 6. A drug used to treat patients with cardiogenic shock is:

- a) Dobutamine
- b) Broad-spectrum antibiotics
- c) Drotrecogin alfa (activated)
- d) Corticosteroids

### 7. What nursing intervention is of primary importance in the initial stage of shock?

- a) Monitoring lactic acid levels
- b) Insertion of an intra-aortic balloon pump
- c) Central venous pressure measurement
- d) Insertion of an indwelling urinary catheter

## Report back

This activity has taken me \_\_\_\_ hours to complete.

Other comments:

Now that I have read this article and completed this assessment, I think my knowledge is:

- Excellent
- Good
- Satisfactory
- Unsatisfactory
- Poor

As a result of this I intend to:

### Answers

Answers to SAQ no. 404

- 1. a 2. b 3. b 4. d 5. d
- 6. b 7. a 8. c 9. d 10. b