



Understanding shock



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Understanding shock

NS54 Collins T (2000) Understanding shock. *Nursing Standard*. 14, 49, 35-39.
Date of acceptance: July 4 2000.

Aims and intended learning outcomes

The aim of this article is to provide an overview of shock, focusing on the five different types of shock, treatment for each and the nursing care of a patient suffering from shock. Every nurse might potentially encounter a patient in shock, so it is important that nurses have an awareness of its signs and symptoms and can distinguish between the different types of shock. After reading this article you should be able to:

- Understand the pathophysiology of shock and how this becomes a life-threatening situation.
- Identify the five different types of shock and the differences between them.
- Identify the signs and symptoms of a patient suffering from shock.
- Understand the issues in providing nursing care to a patient suffering from shock.

Introduction

Shock is a potentially life-threatening condition that requires immediate attention. It is a complex physiological phenomenon that involves many organs of the body and it must be reversed or death might occur. As such, nurses should always be alert for shock in patients and should understand the physiological processes related to shock.

The five types of shock are: hypovolaemic, cardiogenic, anaphylactic, septic and neurogenic. An understanding of the differences means that aggressive treatment can immediately be given to the patient. The management of the patient in shock requires skills in patient assessment, monitoring of vital signs, an understanding of the pathophysiology of shock and the administration of intravenous fluids and drugs.

Pathophysiology

Shock is an acute state in which tissue perfusion is inadequate to maintain the supply of oxygen and nutrients necessary for normal cell function. (Alexander *et al* 1994), which results in widespread hypoxia. The main cause is circulatory failure, which produces a fall in blood pressure and cardiac output. This leads to a lack of blood perfusion of the key organs (brain, lungs etc), which is ultimately life threatening. There are three reasons tissue perfusion might become inadequate:

- A decreased circulating blood volume.
- A failure of the heart to pump effectively.
- A massive increase in peripheral vasodilation.

TIME OUT 1

Before reading further, reflect on a patient with shock you have treated and relate the stages of shock to this patient. Identify what actions were taken to stop the next stage of shock from occurring. Also recall what signs and symptoms were occurring and compare this to the pathophysiology of shock.



Stages of shock

The patient's symptoms will vary depending on the type of shock they have.

Initial stages When cells are deprived of oxygen, the mitochondria can no longer continue to produce adenosine triphosphate (ATP), which is essential for the generation of energy in the cells. Due to reduced intracellular oxygen, the cell membranes become damaged. Without oxygen, the cells start to perform

in brief

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Summary

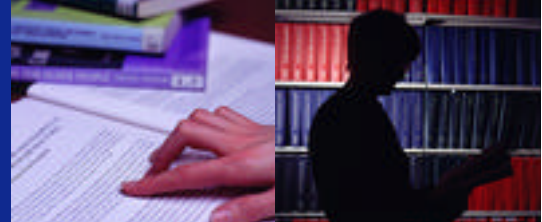
Shock is a serious condition that involves many organs of the body and must be treated immediately to ensure that the patient recovers. Timothy Collins provides a guide to shock, including the different types, the signs and symptoms and appropriate nursing care.

Keywords

- Intensive care
- Shock

These key words are based on the subject headings from the British Nursing Index. This article has been subject to double-blind review.

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Box 1. Estimated blood loss in trauma fractures

Pelvis	3,000ml
Femur	1,000ml
Tibia	650ml
Abdominal injury	2,000ml
Thoracic injury	2,000ml

anaerobic metabolism, in which the production of lactic and pyruvic acids causes the body to develop a systemic metabolic acidosis. Anaerobic metabolism is far less efficient than the normal aerobic metabolism, which uses oxygen. Eventually, lactic acid builds up as a waste product of anaerobic metabolism. This is harmful to the cells and needs to be removed by the blood and subsequently broken down in the liver; however, this process also requires oxygen.

Compensatory stage In this stage, the body tries to intervene with physiological adaptations to attempt to overcome the shock. In an effort to compensate for the metabolic acidosis due to anaerobic metabolism, hyperventilation occurs (Adomat 1992). Hypotension is detected early by the aortic and carotid sinus baroreceptors, which control blood pressure. This stimulates the release of adrenaline and noradrenaline, which cause vasoconstriction, leading to an increase in both blood pressure and heart rate (Clancy and McVicar 1996). The adrenaline and noradrenaline cause vasoconstriction of the skin, kidneys, gastrointestinal tract and other organs and concentrate the blood supply to the heart and brain, as the brain is more susceptible to damage than the other organs. As general blood flow is reduced, the kidneys are especially sensitive; urine output is reduced and oliguria might well develop.

Progressive stage This is where the compensatory mechanisms that the body has implemented to try and combat shock start to fail. If the originating problem, such as blood loss, has not been corrected, the compensatory mechanisms that the body uses to intervene in shock will eventually fail. Due to the decreased perfusion of the cells, sodium collects inside the cells, while potassium leaks out. As anaerobic metabolism continues, this will increase the body's metabolic acidosis, causing the arteriolar and precapillary sphincters to constrict such that blood is trapped in the capillaries (Alexander *et al* 1994). Due to this, the hydrostatic pressure will increase and, combined with histamine release, this will lead to leakage of fluid and protein into the surrounding tissues (Alexander *et al* 1994). As this fluid is lost, the blood concentration and viscosity increase, causing sludging of the microcirculation. The prolonged vasoconstriction will also cause the vital organs to be compromised due to reduced perfusion of the organs.

Refractory stage At this stage, the vital organs have failed and the shock can no longer be reversed. Brain damage and cell death have occurred. Death will occur within a few hours.

Classification of shock

The five types of shock produce a variety of signs and symptoms and the treatment varies for each type.

Hypovolaemic shock This occurs when circulating blood volume is reduced, which causes a reduction in

cardiac output and results in a low perfusion state. Hypovolaemia normally results from a haemorrhage, which can be either internal, such as bleeding from the spleen, or external, for example, from trauma. Other causes include loss of fluid from extracellular compartments caused by severe vomiting and diarrhoea, bowel obstruction, pancreatitis, peritonitis, burns and an excessive diuresis normally related to inappropriate diuretic therapy.

Hypovolaemic is the most common form of shock. Sometimes the cause of fluid loss can be clearly identified, as with severe arterial bleed, but sometimes the cause of the fluid loss is not obvious. For example, an average man could accumulate one litre or 20 per cent of the blood's circulatory volume in the gastrointestinal tract during a paralytic ileus and this might not be immediately identifiable (Alexander *et al* 1994). Patients who have sustained trauma are extremely likely to have hypovolaemia, as large blood losses occur around fracture sites; for example, three litres of blood can be lost with pelvic injuries (Skinner *et al* 1990) (Box 1).

In early hypovolaemic shock, the patient's blood volume can be reduced by as much as 10-15 per cent before symptoms occur. This is primarily due to delays in the body recognising the blood loss and implementing the compensatory stage of shock that has already been discussed. It is only when the compensatory mechanisms fail that the classic symptoms of shock occur and it is imperative that the nurse identifies that the patient is in shock as early as possible to optimise the patient's outcome (Box 2).

If blood loss continues, the patient will develop a tachycardia as a result of the catecholamines adrenaline and noradrenaline that have been released to try to increase the cardiac output. The pulse will be noticeably rapid, weak and thready due to the low blood flow in the body, despite an increased heart rate. Generally, this is the earliest sign of shock, but heart rate can also increase due to other unrelated factors, such as pain or stress, so the presence of a tachycardia alone is not diagnostic of shock (Flavell 1994).

Vasoconstriction also occurs because of the increased secretion of catecholamines (adrenaline and noradrenaline) due to the body concentrating the blood flow to the essential organs of the brain and heart. The skin will look cold and clammy and the feet and fingers will be particularly affected. The nurse should assess for vasoconstriction by testing the capillary refill. This is done by applying pressure to the patient's fingernail and waiting for the colour to return to the nail bed. If it takes more than two seconds for the colour to resume, the patient has poor capillary refill, which indicates vasoconstriction.

Hypotension is normally a later sign of hypovolaemia that appears only after the patient has lost 30 per cent or more of his or her blood volume (Flavell 1994). There will also be a narrowing of the pulse pressure,



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which is the difference between the systolic and diastolic blood pressures. This is due to decreased cardiac stroke volume and increased peripheral resistance (Alexander *et al* 1994). A decrease in urine output will occur: urine production provides a direct measure of blood flow and perfusion to the kidneys, as vasoconstriction of the renal artery in shock causes the blood to be directed away from the kidneys to the heart and brain. Initially, the respiratory rate and depth increases in response to the cellular hypoxia, but as the shock progresses, the respiratory rate decreases and metabolic acidosis increases (Flavell 1994). A change in mental status indicates decreased cerebral perfusion and oxygenation and the patient might appear confused, restless or unresponsive.

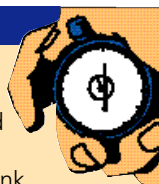
Treatment for the shock consists of treating the cause. For example, if the patient is bleeding, the cause must be found and treatment given to stop the bleeding, which might require emergency surgery. All patients in the acute phase of shock should be given 100 per cent oxygen via non-rebreathe masks. However, COPD patients should be closely monitored for reduced respirations and O₂ saturations. Regular blood gases should also be taken. Oxygen saturations and blood gases need to be monitored for both oxygenation and for metabolic acidosis. Ideally, oxygen saturation should be greater than 95 per cent, but this will not always be possible, particularly for patients with hypovolaemic shock. Two large bore cannulae should be inserted for the rapid administration of fluids; the ideal replacement fluid is blood, as colloid and crystalloid fluids will increase the circulating volume, but cannot transport oxygen and dilute the blood further.

When patients are in shock it can be difficult to obtain IV access. Normally the anti-cubital fossa veins in the arms are the easiest to cannulate in shock. Other options may be an IV cut down, where the skin is surgically cut to expose the vein and then it is cannulated or a central line may be inserted.

Vital signs should be monitored regularly, including temperature, because hypovolaemia does predispose to hypothermia. It might not be possible to obtain a blood pressure reading, but if a peripheral pulse can be palpated it is safe to assume that the patient has a blood pressure of at least 80mmHg (Buckley 1992). The patient's level of consciousness should be regularly assessed using the Glasgow Coma score (Ellis and Cavenagh 1992) and any deterioration in consciousness should be reported immediately.

TIME OUT 2

Refer to Box 2. Imagine that you have a patient who has class 2 hypovolaemic shock and has lost 15-30 per cent blood volume following a trauma. Think about the ways you might prevent any further blood loss.



Cardiogenic shock This is characterised as left ventricular failure of the heart itself, which results in reduced tissue perfusion and impaired cellular activity (De Jong 1997). This pump failure, which produces a low cardiac output state, normally follows a myocardial infarction. This type of shock can also occur following heart failure, cardiomyopathy, trauma or myocarditis (De Jong 1997). Due to the low cardiac output state, there is a reduction in arterial blood pressure. The baroreceptors then release both noradrenaline and adrenaline, hormones that cause an increase in the contractility of the heart and also cause vasoconstriction within the body. Due to the damage to the heart muscle, the increase in heart rate will probably do little to increase cardiac output. This tachycardia can also be detrimental to the heart, as it is being placed under more pressure and requires a greater oxygen supply to the heart muscle that is already ischaemic due to the shock.

As with hypovolaemic shock, the falling cardiac output and blood pressure cause poor perfusion of blood to the tissues and eventually the cells are unable to maintain cellular homeostasis. The resultant hypoxia causes widespread vasodilation due to lactic acidosis, resulting in a further drop in blood pressure that the compensatory mechanisms are unable to prevent. Pulmonary oedema will eventually occur when the pulmonary circulation and the right side of the heart also begin to fail. Despite recent advances in haemodynamic monitoring and drug therapy, the mortality rate remains high, with 80 per cent of patients dying from cardiogenic shock (Alexander *et al* 1994).

A patient in cardiogenic shock will have a fast, weak pulse and a systolic blood pressure below 90mmHg. His or her skin will be cold, clammy and cyanotic. The patient will have a urine output below 30ml/hour and may be confused as a result of poor cerebral perfusion. Treatment involves supporting the heart and reducing the patient's anxiety and pain. The patient should be given 100 per cent oxygen via face mask and frequent observation of vital signs is necessary, including blood gases, O₂ saturations, blood pressure, frequency and depth of respirations, pulse, heart rhythm and temperature. Diamorphine is given for pain and the patient might require inotropic support to increase the contractility of the heart, thus increasing its cardiac output.

TIME OUT 3

Compare hypovolaemic and cardiogenic shock and make a list of the differences in treatment. How do you think nursing care might differ for each?



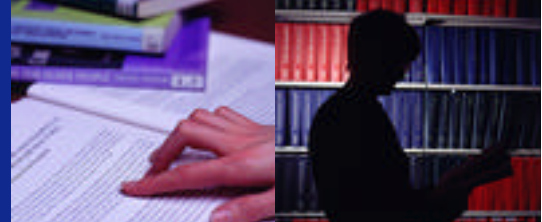
Anaphylactic shock This type of shock is a severe allergic reaction in which an antigen-antibody reaction occurs. There are many causes, including

Box 2. Four classes of hypovolaemic shock

Blood loss	Observations
Class 1: (Up to 15 per cent blood volume)	Usually few clinical signs Compensatory mechanisms are activated to cope with blood loss
Class 2: (15-30 per cent blood volume)	Rapid, thready pulse; cool skin Urine output reduced Confusion Change in all observations, except for blood pressure, which remains stable
Class 3: (30-40 per cent blood volume)	A dramatic deterioration in all observations Severe tachycardia and hypotension
Class 4: (More than 40 per cent blood volume)	Immediate threat to patient's life Drastic action and surgery required

(American College of Surgeons 1993)

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drugs, such as antibiotics; transfusion of incorrectly cross-matched blood; foods such as nuts, eggs or fruits; insect bites; and even food additives, such as preservatives. One of these factors causes the body to be hypersensitive to the antigen that the body is allergic to. The body then goes into circulatory collapse because of degranulation of the mast cells, which are found within the loose connective tissues, bronchi and gastro-intestinal mucosa. The mast cells then release large amounts of histamine and bradykinin that have a massive vasodilatory effect on the body (Henderson 1998). The patient might well develop laryngeal oedema, bronchospasm, and/or itchy urticaria skin rashes.

This is a medical emergency, as anaphylaxis causes a sudden increase in vascular permeability, leading to oedema, which can then develop to obstruct the airway. The treatment for anaphylaxis is prompt administration of adrenaline. Adrenaline causes an increase in blood pressure by producing vasoconstriction effects and it inhibits the mediators released by the immune response. It also causes the smooth muscle to relax, leading to bronchodilation of the airways (Henderson 1998). Hydrocortisone and chlorpheniramine are given, as well as oxygen. Regular monitoring of blood pressure, pulse, frequency and depth of respirations, and O₂ saturations should take place. IV colloid therapy is started to increase circulatory volume.

Septic shock This can arise from a variety of overwhelming bacterial toxins. The bacteria that most commonly cause septic shock are Gram-negative bacteria; however, Gram-positive organisms, such as streptococci, can also be responsible (Adam and Osborne 1997). The bacteria invade phagocytes, causing damage or even death to the cell (Adam and Osborne 1997).

This results in the release of histamine and proteolytic enzymes, leading to vasodilation and increased capillary permeability (Adomat 1992). This causes a maldistribution of blood flow, leading to a mismatch between the supply of and demand for oxygen within the microvasculature, which has been greatly increased due to massive vasodilation (Alexander *et al* 1994). This initial vasodilation induced by bacterial toxins can cause the patient to be flushed and warm, as the body allows more blood to the peripheral circulation (Clancy and McVicar 1996). This causes hypotension, which might cause a reduction of the glomerular filtration rate, possibly resulting in acute renal failure and oliguria. Symptoms of septic shock include restlessness and anxiety, tachycardia and hypotension, pyrexia, thirst and respiratory failure. Treatment focuses on increasing the circulatory volume with fluids and antibiotics to try to reduce the infection and the invading endotoxins. Oxygen therapy will be required and admission to intensive care for mechanical ventilation and cardiac support with inotropes is often necessary.

TIME OUT 4

Identify which patients you consider to be most at risk from developing septic shock. Are there any predisposing factors, such as age, medical condition, nutritional status etc?



Neurogenic shock This type of shock is related to the nervous system homeostasis. Neurogenic shock occurs as the result of the loss of sympathetic nerve activity from the brain's vasomotor centre due to disease, a drug or traumatic injury (Acadam and Osborne 1997). The loss of sympathetic impulses causes massive vasodilation of the arterioles and venules as the vasoconstrictor tone has been interrupted, resulting in a significant decrease in peripheral vascular resistance. This then leads to a reduction in venous return to the heart, resulting in a decrease in cardiac output, with hypotension rapidly following. This type of shock can be caused by disease or injury of the brain stem or spinal cord, emotional trauma, or the effects of depressive drugs (usually anaesthetic agents). Treatment involves intravenous fluid therapy to increase the circulatory volume, oxygen therapy and circulatory support, which might involve the use of vasoconstrictor inotropes that compensate for the loss of sympathetic nerve activity (Clancy and McVicar 1996).

TIME OUT 5

What type of shock is caused by failure to produce adequate cardiac output? List the ways to improve cardiac output. What is caused by a loss of sympathetic nervous activity and what type of shock results? Write a list of the main causes of loss of sympathetic nervous activity.



Nursing care of a patient in shock

Assessment and treatment of the patient will depend on the type of shock, as signs and symptoms differ for each. The aim of the care is to re-establish perfusion of the vital organs and to prevent the shock from progressing. Regular observations are essential; the frequency depends on the patient's condition, but at a bare minimum they should be recorded half hourly. Ideally, the aim is to return the patient to normal homeostatic levels, but this is not always possible. The nurse should check for any fall in blood pressure and changes in heart rate, as well as the strength of the pulse, as a weak and thready pulse is indicative of shock. The patient should ideally be monitored for cardiac rate and rhythm, blood pressure and pulse oximetry. However, the nurse should not rely solely on diagnostic machinery, as physical observation can be



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important in identifying the state of the patient in shock. Anxiety, confusion and pain are all indicators that can be observed. The nurse should regularly assess the patient's conscious level and assessment should be used in conjunction with the Glasgow coma score, with any deterioration reported immediately. Coning is a rare complication in which brainstem herniation occurs. However, patients are likely to die from cardiac arrest or respiratory failure before this would occur.

The patient's respiratory rate should be measured, and the depth and pattern noted. High flow oxygen should be given to any patient in shock, so pulse oximetry and blood gases are taken to provide an assessment of the patient's respiratory status. If oxygen therapy is to continue for a long period, it is essential to use a humidifying circuit to moisten the oxygen with sterile water, as this will prevent damage to the bronchial mucosa and cilia and help to promote expectoration of secretions.

All patients should be given nil by mouth in case any further surgery is needed or there is any loss of consciousness which might put the patient at risk of aspirating stomach contents. Because of this, mouth care is essential, especially when oxygen therapy is in progress. Nurses should observe for tachypnoea and respiratory tiredness, as these indicate that the patient might require mechanical ventilation.

The patient's core and peripheral temperatures should be recorded to ensure there is no loss of peripheral circulation and an assessment of the patient's capillary refill is essential. Patients in shock should be kept warm, but should not be warmed too quickly, as this will cause peripheral vasodilation and could cause hypotension. A central venous catheter (CVP) should be inserted to measure the fluid status of the patient; this can also be used for administering large volumes of fluids and drugs. The CVP line should be measured hourly to monitor the circulating fluid level of the patient. Levels will be low in hypovolaemic shock and in cardiogenic shock. Strict asepsis must be maintained at all times to prevent infection.

Strict fluid balance is essential and any losses and inputs need to be recorded. If large amounts of fluids are to be administered, they should be warmed prior to transfusion, as this will reduce the incidence of hypothermia. A urinary catheter should be inserted and hourly urine measurements recorded: volumes of no less than 0.5ml/kg/hour should be reported to indicate hypo-perfusion of the kidneys. The nurse must ensure that if the patient is actively bleeding, blood has been cross-matched and intravenous fluids are being administered as quickly as possible, especially if the patient is bleeding excessively externally. The volume of blood lost should ideally be replaced. The patient's haemoglobin and haematocrit levels need to be taken to assess the degree of blood loss (Skinner *et al* 1990). Any patient in shock requires a minimum of two wide

bore cannulas for rapid intravenous fluid and drug administration. Drugs should be given intravenously to be absorbed quickly. However, this is not the case for anaphylactic shock, when adrenaline should be administered intramuscularly.


The patient should lie flat in the acute stage of shock and regular positional changes should be given to monitor pressure areas and to prevent fluid from accumulating in the lungs. Many patients in shock are admitted to intensive care, so the nurse must ensure that the appropriate equipment, personnel and patient notes accompany the patient on transfer to the intensive care unit. Relatives should always be kept informed of the patient's progress and should be allowed time to express their fears and anxieties.

It is essential when nursing a patient in shock to explain all the procedures to the patient and relatives. Time should be taken to keep relatives informed of the situation and it is imperative that the patient is reassured and is allowed to ask questions regarding his or her care.

TIME OUT 6

Consider the emotional, psychological and spiritual needs of the patient who has experienced or is experiencing shock. Think about the effects on the patient's family and/or carers and how they might respond to these needs.

**Conclusion**

Shock is a life-threatening condition arising from a failure of the body to maintain cellular homeostasis, resulting in cardiovascular collapse. Shock is a medical emergency and it is essential that nurses are able to identify and treat patients in shock. The nurse must be able to distinguish between the five types of shock and identify shock in its early stages so that the patient's outcome is optimised. The symptoms vary between the different types of shock; however, they will all eventually cause circulatory collapse if left untreated. Regular assessment and accurate monitoring of patients most at risk of shock is crucial and the nurse must ensure these skills are incorporated into his or her daily practice .

TIME OUT 7

Now that you have completed the article, you might like to think about writing a practice profile. Guidelines to help you write and submit a profile are outlined on page 41.

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