CONTINUING PROFESSIONAL DEVELOPMENT

Shock: types, classifications and explorations of their physiological effects

Aims and outcomes
This article provides a comprehensive review of the classifications and types of shock that can occur following trauma, surgery, organ damage/disease (neurogenic, hypovolaemic, cardiogenic) or during/following treatment (septic, anaphylaxis) and the stages (compensatory, progressive and irreversible) of shock. It provides an overview of the physiological changes that occur in the cell, at organ level, to oxygen consumption and demand, and coagulation.

It considers the effects of shock on the brain, heart, lungs, kidneys, liver and gastrointestinal tract (GIT) and considers influencing factors such as age, pain, hypothermia, and previous medical history. Nurse’s need knowledge of the processes of shock to help explain incidents that occur in clinical practice and their rationale. After reading this article, and undertaking the activities, you should be able to:

- Understand the classifications and types of shock;
- Identify the three stages of shock: the compensatory mechanisms involved; the physiological changes that occur in the cell and at organ level; and irreversible;
- Identify the influencing factors that can enhance the progression of shock.

Introduction
The pathophysiological mechanisms of shock are complex and often occur in hospital. Nurses are responsible for assessing and observing for states of shock by intermittent monitoring of heart rate, respiratory pattern and rate, colour, temperature, fluid balance, urine output and blood pressure. Any changes in these parameters may alert the nurse to the deterioration in a patient’s condition, and the onset of shock. It is important with regard to nursing interventions that the stages and types of shock are understood, to ensure that appropriate medical and nursing interventions are carried out, a failure to do so could be fatal.

Types and classifications of shock
Shock is a condition where the cardiovascular system fails to perfuse the body tissues adequately, bringing about a widespread disruption of cellular metabolism, resulting in functional disturbances at organ/tissue level. The causes of shock are generally any factor which affects - blood volume, blood pressure or cardiac function.

The blood pressure can be used as a basis for defining shock and are explained in two forms (Table I):

- Hypotensive shock – further subdivided into: (i) low cardiac-output shock characterised clinically by cold skin (ii) high cardiac-output shock characterised by warm skin Normotensive or hypertensive shock - blood pressure is compensated.

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

CONTINUING PROFESSIONAL DEVELOPMENT

TIME OUT 1
Discuss with a group of your colleagues and outline why you undertake the BP so often in clinical practice and what you are doing it for?

What do you understand so far regarding the terms compensation and homeostasis?

ditional classification is by categorising shock according to the primary defect that produced it. With this system there are five forms of shock: anaphylactic, septic, neurogenic, cardiogenic, and hypovolaemic.

(B) Anaphylactic shock

Anaphylaxis occurs when a sensitised person is exposed to an antigen, to which he or she is allergic. The antigen enters the body and combines with immunoglobulin E (IgE) antibodies on the surface of the mast cells and basophils (Khun 1990). Mast cells and basophils are primarily found in the lungs, small intestines, skin and connective tissue. An antigen-antibody reaction occurs which induces the release of histamine and prostaglandins into the blood. This causes:

- Selective vasodilatation (systemic circulation and the heart) and vasoconstriction (pulmonary bed, hepatic and other large veins)
- Increased capillary permeability (causing movement of circulating fluids into the interstitial space causing a relative hypovolaemia)
- A reduced cardiac output and low arterial pressure
- A fall in cellular perfusion – metabolic demands are not met, resulting in acidosis, coagulopathies, and capillary pooling (O’Neill 1990).

The implications of this for the patient are: bronchospasm; oedema formation in the glottis and pharynx; oedema in the lungs and in subcutaneous tissue; and changes in cardiac function, for instance reduced contractility and dysrhythmias (Hollingsworth and Giansiracusa 1992).

(B) Septic shock

Septic shock is caused by an overwhelming infection and may be the result of a suppressed immune system, a massive burn injury, or anything else that can introduce an infecting organism into a compromised victim. The most common organism is a gram-negative enteric bacillus such as Escherichia coli, Pseudomonas, or gram-positive Staphylococcus aureus (Bone 1991) (Table 2). These organisms enter the vascular system release endotoxins (waste products), which cause an interstitial fluid leak, increased vascular permeability, and vasodilatation, leading to shock (Hoffman & Natanson 1993).

The result of septic shock is tachycardia and a high cardiac output (Figure 1). In this state the patient may feel warm, have a high temperature, a low circulating volume owing to venous pooling, increased capillary permeability and third-space fluid shift (Bone 1991). Cardiac output is maintained at a normal / high level by the increasing tachycardia, but if volume loss is not corrected, hypovolaemia will persist, cardiac output will decrease and the skin will become cool. As in all other types of shock, the primary problem is tissue hypoperfusion, consequently, nutrients and oxygen fail to be delivered to cells.

(B) Neurogenic shock

Neurogenic shock may be the result of a severe brain stem injury at the level of the medulla, an injury to the spinal cord, or spinal anaesthesia. Neurogenic shock causes changes to the smooth muscle tension in the walls of the circulatory vessels, through the actions of the nervous system, and an imbalance between parasympathetic and sympathetic stimulation occurs. There is a loss of sympathetic tone, causing peripheral vasodilatation, resulting in severe hypotension (Figure 1). There is decreased vascular tone and systemic vascular resistance (M), inadequate cardiac output, reduced tissue perfusion, and impaired cellular metabolism. Neurogenic shock may mask signs and symptoms of other types of shock. If neurogenic shock is present, there should be a heightened suspicion for an undetected source of haemorrhage.

(B) Cardiogenic shock

Cardiogenic shock occurs when the heart, due to impaired myocardial performance, cannot produce an adequate cardiac output to sustain the metabolic requirements of body tissues. Myocardial infarction is the most common cause of cardiogenic shock, as the area infarcted becomes dysfunctional, and depending on the size of the infarction, stroke volume and cardiac output may decrease with a concurrent increase in left ventricular end-diastolic pressure (Anderson 1982). Compensatory mechanisms are stimulated by the decrease in blood pressure, and catecholamines are released. This causes an increase in heart rate, contractility, blood pressure, and M to maintain arterial pressure.

The compensatory mechanisms improve blood flow for a time, but more oxygen is required by the already ischaemic cardiac muscle to pump blood into the constricted systemic circulation, consequently increasing cardiac workload. The heart becomes more ischaemic, and cardiac failure worsens. The result is to jeopardise potentially viable tissue, and worsen left ventricular function. As cardiac output continues to decline, blood pressure and tissue perfusion decrease which results in cardiogenic shock and ends with the patient’s death (Figure 1). Cardiogenic shock is often complicated by arrhythmias or pulmonary oedema.
Shock is a very complex syndrome as the problem not only concerns the amount of blood volume, but also delivery in terms of blood flow to organs and cells of the body. There are a number of variables that affect the course of shock, such as the age and the general state of health of the person before the shock insult (see later).

The first stage of shock is known as compensated shock, the body's compensatory mechanisms are able to maintain cardiovascular dynamics and stabilise the circulation, in the face of whatever defect is causing the shock. The compensatory mechanisms involved are:

**(B) Compensated shock**

The primary compensatory mechanism is mediated through the sympathetic nervous system and the adrenal glands. This sympathoadrenal response is initiated by the decrease in arterial pressure that stimulates the baroreceptors located in the aortic arch and carotid sinuses (Marieb 1998).

**(C) Sympathetic nervous system**

Baroreceptors respond to any decrease in arterial blood pressure, whether it is due to haemorrhage, peripheral blood pooling, or a decrease in myocardial contractility. A decrease in circulation causes the pressure exerted by the blood in the artery (blood pressure) to reduce. This decreases the rate of firing of both the carotid sinus and the aortic arch baroreceptors, and supplies sensory information to the cardiovascular centre that regulates blood pressure in the medulla of the brain. This regulating reflex increases:

- Sympathetic nervous system discharge which will increase heart rate
- Myocardial contractility and peripheral resistance by vasoconstriction of blood vessels
- Total peripheral vascular resistance
- Arterial blood pressure
- Myocardial after-load.

This increase in resistance caused by baroreceptor control is not uniform throughout the body’s organ systems. Some systems are given preference as to the amount of blood flow they will receive. Varying the distribution of cardiac output, with some organs being well perfused and others being hypoperfused. This difference in distribution of cardiac output is due to the distribution of alpha (to gut, skin) and beta (heart, lungs) adrenergic receptors in the body (Table 3). These receptors respond to catecholamines (adrenaline and noradrenaline) liberated from postganglionic sympathetic nerve endings and

**(A) The stages of shock**

Shock from what ever the initial cause, the end result is always the same, that is the tissues fail to receive oxygen and nutrients and to rid themselves of waste products. It is inadequate tissue and cell perfusion which causes widespread disruption to cellular metabolism.

Shock is a very complex syndrome as the problem not only concerns the amount of blood volume, but also delivery in terms of blood flow to organs and cells of the body. There are a number of variables that affect the course of shock, such as the age and the general state of health of the person before the shock insult (see later).

It is the responsibility of the nurse that the development of shock must be prevented. This includes early interpretation of observational and measurable data to recognise its early development. For easy understanding and recognition of shock it can be divided into three stages: compensated, progressive or non-complicated and irreversible. These stages are not distinct and should be regarded as a continuum.

**(B) Hypovolaemic shock**

Hypovolaemic shock is the most common type of shock. It is the state that results from hypovolaemia, and shock occurs due to a decrease in the circulating fluid volume so large that the body's metabolic needs cannot be met. The decline in blood volume produced by continued bleeding, plasma loss, water or fluid shifts (Edwards 1998), decreases venous return and cardiac output. The decrease in intravascular volume, primarily affects tissue perfusion.

The degree of shock depends on the amount of blood lost, the rate at which it was lost, the age and general physical condition of the patient, and the patient's ability to activate compensatory mechanisms. Numerous compensatory mechanisms, to increase venous tone, are activated when the circulating volume and venous return is decreased (Meyers & Hickey 1988). As a result, venous capacity is decreased to match the smaller blood volume, and adequate transport of oxygen and nutrients is maintained.

If the fluid loss exceeds the ability of homeostatic mechanisms to compensate for the loss, the central venous pressure (CVP), diastolic filling pressure, stroke volume, and systemic arterial blood pressure will fall. As the severity of shock increases, blood is pooled in the capillary and venous beds, with further impairment of the effective vascular volume available for oxygen transport and tissue perfusion. To identify the different types of shock in a patient see Figure 1.

Patients in shock will not uncommonly have components of more than one of the forms of shock. For example, patients in cardiogenic shock may also be hypovolaemic due to loss of fluid into the tissues as a result of high venous pressures or increased capillary permeability. Hypovolaemia is also frequently a complication of septic shock, and in late stages of hypovolaemic shock patients’ usually have some degree of cardiac failure and vasomotor collapse complicating their shock picture.

**TIME OUT 2**

Before reading any further, identify the clinical picture of a patient in compensatory shock.
from the adrenal medulla.

(D) Noradrenaline and Adrenaline

Stimulation of the adrenal glands occurs early in shock, mainly due to stress, and activates the sympathetic nervous system, which causes a release of catecholamines (noradrenaline and adrenaline). Circulating concentrations of Adrenaline during shock can increase within 5-60 minutes of injury. Adrenaline increases arteriolar resistance, which helps to support perfusion pressure in the face of a relatively low cardiac output. This is done at the expense of certain organs and tissues which will be hypoperfused (GIT, skin, skeletal muscles) and may cause problems when and if the shock continues.

In addition, adrenaline:
- Increases circulating glucose concentrations by inhibiting insulin secretion, consequently raising blood glucose level
- Stimulates pancreatic glucagon release and gluconeogenesis
- Stimulates beta receptors in the heart, increasing myocardial contractility (Inotropic effect), heart rate (Chronotropic effect) which improves cardiac output, and increases blood pressure.

Noradrenaline concentrations are released more slowly from sympathetic nerve endings and have a strong alpha stimulating effect. Activation of alpha receptors on the cell membranes of vascular smooth-muscle cells causes intense vasoconstriction. About 64 per cent of all blood in the circulatory system is in the systemic veins beyond the capillaries and acts as a blood reservoir for the circulation. Even after a loss of 20-25 per cent of the body's total circulating volume, the circulatory system can function almost normally, due to the action of noradrenaline.

The functioning of venous constriction is dependent on vascular compliance. This is the increase in volume a vessel is able to accommodate for a given increase in pressure (Marieb 1998). The compliance determines a vessel's response to pressure changes. If for example the patients veins are stiff from long term illness such as hypertension this response may fail to function during a shock state.

(C) Renal autoregulation

The kidneys play a complex role in restoring extracellular fluid volume and increasing systemic blood pressure. This system is stimulated principally when there is a decrease in blood pressure. This elaborate set of interlinked processes involves the renin-angiotensin-aldosterone system.

A decrease in kidney perfusion activates the renin-angiotensin-aldosterone mechanism. Renin is released by the kidneys and converted in the blood via the angiotensinogenes to angiotensin I. Once in the lungs, angiotensin I is converted to angiotensin II. Angiotensin II has two primary actions, it stimulates the:
1. Release of noradrenaline causing vasoconstriction of the peripheral vasculature;
2. Release of aldosterone.

The vasoconstriction will directly increase the blood pressure by increasing the systemic vascular resistance (SVR), to maintain blood pressure in the face of acute blood loss. Aldosterone causes increased sodium reabsorption in the renal tubules, resulting in the reduction in urine output observed in the early stages of shock. Water reabsorption follows sodium and there is a subsequent increase in intravascular volume, resulting in increased venous return to the heart, cardiac output and blood pressure, thus providing a longer term compensation for blood loss.

(C) Arterial chemoreceptors

Chemoreceptors are further specialised areas within the aortic and carotid arteries which are sensitive to concentrations of oxygen, carbon dioxide, and hydrogen ions (pH) in the blood (McCance & Huether 1997). They transmit impulses to the medulla of the brain which regulates blood pressure. A decrease in arterial oxygen concentration or pH causes vasoconstriction and a reflexive increase in blood pressure (Nisasi & Keyes 1994). Whereas an increase in carbon dioxide causes vasodilatation and a decrease in blood pressure. These blood pressure changes are carried out by smooth muscle layers in the vessels. If hypoxia occurs due to shock then patients are at risk of having an increased blood pressure, and this can be compounded by heart conditions or varicose veins, which can further increase their resistance and thus blood pressure.

(C) Osmoreceptors

To maintain fluid and electrolyte balance, water and electrolytes are in constant motion, between intracellular (about 25 L) and extracellular compartments (divided into interstitial fluid – 12 L, and plasma volume - 3 L) (Edwards 1998). If concentrations of sodium (the major cation in extracellular fluid) are increased, as in the case when there is a loss of extracellular water (Bove 1994), osmoreceptors in the hypothalamus are stimulated. Osmoreceptors are highly specialised hypothalamic neurons, which continually monitor the solute concentration (and thus water concentration) of the blood. When solutes threaten to become too concentrated, as in conditions that cause an increased sodium concentration (excessive sweating, inad-
equate fluid intake, burns), the osmoreceptors transmit excitatory impulses to the hypothalamic neurons and release anti-diuretic hormone (ADH).

ADH is a chemical substance that, once liberated into the blood targets kidney tubules and, inhibits or prevents urine formation. The tubule cells respond by increasing water absorption at the renal collecting duct and returning it to the circulation. As a result less urine is produced and blood volume increases, thereby improving venous return to the heart, cardiac output, and blood pressure. Urinary output will decrease and the sense of thirst will be aroused (Bove 1994).

Generally, the clinical picture of a patient in the compensatory stage of shock is:

- Tachycardia, narrowing pulse pressure, increase in temperature and blood glucose level due to the effect of catecholamines
- Pale skin colour and cool to cold skin due to the redistribution of blood away from the skin
- Decrease in urine output, due to selective vasoconstriction of the renal bed and the actions of ADH and aldosterone
- Absent bowel sounds
- An increase in blood pressure and rate and depth of respiration
- Mental state alterations ranging from restlessness to coma
- Complaining of thirst.

These protective mechanisms, observed in the compensatory stage of shock can maintain circulation and blood pressure (Time out !). These mechanisms will eventually cease to function and circulatory failure will ensue. If the metabolic acidosis, circulatory failure or volume is not corrected or treatment instigated, progressive shock will occur in a short space of time.

(B) Progressive or uncompensated shock

Once shock has developed, the course it takes is complex. Why some shock patients take a progressively downward course despite best efforts at treatment is not fully understood. Certainly the prognosis in some forms of shock particularly hypovolaemic shock is excellent if treated in the early compensatory stage. Once shock has progressed into the second stage the outcome is no longer as predictable. As shock progresses, there are deleterious changes which occur to: oxygen supply and demand; the cells to energy production and to the cellular membrane; to blood coagulation; and specific organs.

(C) Oxygen supply and demand

An imbalance between oxygen supply and tissue demand is fundamental to the nature of shock. Oxygen supply and demand is maintained in balance as long as supplies of oxygen to the body and carbon dioxide is eliminated through ventilation, perfusion, diffusion and cell metabolism. Under normal circumstances, whole body oxygen consumption (VO2) is maintained over a wide range of oxygen delivery (DO2) by varying oxygen extraction (Skowronski 1999). For example as VO2 increases so does DO2. When VO2 / DO2 increases, and if DO2 falls below a critical level this gives rise to an oxygen debt, and hypoxia occurs. The events following shock places increased demands on these processes and when overwhelmed the victim of shock is at risk of pulmonary complications, leading to a supply-demand deficit and hypoxia.

(C) Cellular changes

Cellular shock can be caused by any of the types of shock mentioned above, and is generally the end result of all types of shock. However, before shock occurs at the level of the cell, specific vascular changes begin as described for the different types of shock above. Yet, all shock states interfere with tissue perfusion, oxygen transport and the synthesis of adenosine triphosphate (ATP), all of which lead to a reduction in the availability of energy, nutrients and ultimately hypoxia, causing serious cell damage (Figure 2).

(D) Cellular energy production

Nutrients, such as glucose and fatty acids, as well as oxygen, enter the cell across the cell membrane. Shock results in an inadequate flow of nutrients and oxygen to the cell. This causes a reduction in adenosine-triphosphate (ATP) concentrations in the mitochondria of the cell, which fall within 15 minutes following hypoxia (Gosling 1999). Mitochondria activity is diminished due to a lack of oxygen for glycolysis. The end product of anaerobic metabolism by the cell is lactic acid, which rapidly builds up in the blood eventually lowering the pH. This gives rise to a decrease in the energy available for cell work, consequently leading to metabolic acidosis, tissue and organ dysfunction.

Poor blood flow also impairs the normal removal of carbon dioxide, which is converted to carbonic acid, further lowering blood pH. Consequently, the results of anaerobic metabolism are the production of lactic acid and a reduction in the energy available for cell work. Lactic acidosis also reduces myocardial contractility, arteriolar responsiveness to further adrenergic and noradrenaline release, potentiating vasomotor collapse and stimulates the intravascular clotting mechanism. However, acidemia has the beneficial effect of shifting the oxyhaemoglobin dissociation curve to the right, thereby facilitating the release of oxygen from haemoglobin (Marieb 1998).

Eventually, a large number of cytotoxic, vasodilator, vasoactive and other substances are
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released from the cell into the circulation, resulting in progressive vasodilatation, myocardial depression, increased capillary permeability, and eventually intravascular coagulation. These substances include histamine, serotonin, kinins, lysosomal enzymes, and endogenous mediators (Huddleston 1992). If cellular acidaemia becomes extreme, cellular dysfunction becomes intemperate and if permitted to continue may finally become irreversible.

**Table 1 - A classification of shock states according to blood pressure response**

<table>
<thead>
<tr>
<th>Type of shock</th>
<th>Initial presentation</th>
<th>Result</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypotensive shock</td>
<td>Cold skin: low cardiac output and high peripheral resistance</td>
<td>Low CVP (caused by large loss of circulating volume).</td>
<td>Responds well to IV fluid replacement</td>
</tr>
<tr>
<td></td>
<td></td>
<td>High CVP (caused by cardiac failure)</td>
<td>Responds poorly to IV fluids may respond to vasodilators, which decrease cardiac work</td>
</tr>
<tr>
<td></td>
<td>Vasodilatation (caused by gram-negative or gram-positive sepsis)</td>
<td></td>
<td>Treatment difficult due to the development of arteriovenous shunts</td>
</tr>
<tr>
<td></td>
<td>Warm skin: high cardiac output</td>
<td>Vasodilatation (caused by spinal anaesthesia or drugs)</td>
<td>Responds well to vasopressor drugs and fluid administration</td>
</tr>
</tbody>
</table>

**Table 2 - Micro-organisms associated with septic shock**

<table>
<thead>
<tr>
<th>Type of bacteria</th>
<th>Micro-organisms</th>
<th>Occurrence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gram-negative</td>
<td>Escherichia coli, Klebsiella, Enterobacter, Pseudomonas aeruginosa, Serratia, Proteus, Bacteriodes fragilis (anaerobe)</td>
<td>50 per cent of all cases</td>
</tr>
<tr>
<td>Gram-positive</td>
<td>Staphylococcus aureus, Pneumococcus, Alpha and beta-haemolytic streptococci</td>
<td>10 per cent of all cases</td>
</tr>
<tr>
<td>Fungi</td>
<td>Candida</td>
<td>2 per cent of all cases</td>
</tr>
</tbody>
</table>

If cellular acidaemia due to hypoxia becomes extreme, the hypoxic cell swells, becomes distorted and finally ruptures. This is because the high intracellular potassium and low intracellular sodium and calcium concentration are maintained by active transport systems: the sodium/potassium adenosine triphosphatase (ATPase)-dependent pump, and the ATP-dependent calcium transport pump. The shortage of ATP changes the normal ionic gradients across the cell membrane, with a rapid efflux of potassium from the cell, and movement of sodium and chloride into the cell (Gosling 1999).

An increased sodium in the interior of cells result in water also entering the cell, driven by osmotic forces causing cellular swelling and distortion, which may interfere with organelle function (Buckman et al 1992) and lead to disintegration of the mitochondrial matrix. This is why by just delivering to patients with shock an adequate or higher oxygen concentration than normal does not always lead to prevention or recovery from cellular hypoxia, and aerobic respiration is not always fully restored (Gosling 1999) (Case study 2).

The influx of calcium into the cell during shock has a different cause than the initial membrane permeability change involving sodium and potassium. The mechanisms by which the calcium content of cells is regulated are defunctionalised because of a lack of ATP (Gosling and Alpar 1999). Intracellular calcium is an important signalling system responsible for activation of phospholipases and proteases, and its derangement results in membrane disruption. As a result, calcium accumulates in the mitochondria, causing structural derangements of the organelles, and may be the hallmark of irreversible cellular injury and eventually cell death (Buckman et al 1992).
The release of thromboplastic substances. Early in shock, changes in blood coagulation can be seen. Hypercoagulability occurs as a result of the release of lysosomal-enzyme damage to the cell (Guthric 1982).

(D) The role of lysosomes

Lysosomes are an important cell structure and are affected during shock. Lysosomes contain enzymes which function as a system for the breakdown of cell waste (Marieb 1998). The lysosomal membrane is ordinarily quite stable, but it becomes fragile when the cell is injured or deprived of oxygen. Lysosomal membrane instability is made worse by the lack of ATP and the cell starts to use its own structural phospholipids as a nutrient source. Eventually the lysosomal membrane becomes more permeable and may rupture, allowing the release of lysosomal enzymes resulting in self-digestion of the cell. The use of steroids in shock is thought to help stabilises the lysosomal membrane and prevent lysosomal-enzyme damage to the cell (Guthric 1982).

(D) Cellular fluid shifts

Any type of shock will automatically trigger an inflammatory response (Bove 1994). The normal body response will be to send nutrients, fluids, white blood cells, and clotting factors to the damaged site to repair tissue, prevent infection and if necessary stem blood loss (Huddleston 1992). Capillaries vasodilate and become more permeable to allow these factors to reach the site of injury, leading to localised swelling and lymphatic blockage.

The permeability causes movement of fluids, allowing water, electrolytes, and other particles (such as albumin) into the interstitial spaces, and is known as a third-space fluid shift (Bove 1994). When third-space fluid shift occurs, a patient can appear paradoxically ‘dry’ or hypovolaemic as fluid has moved into the intravascular spaces, yet may still have the same or excess amount of body water.

The vasodilation is caused by the release of cell mediators (such as histamine, kinins, complement) from the damaged endothelium, and causes a reduction in blood pressure, peripheral vascular resistance and an increase in heart rate, further compounding the appearance of a hypovolaemic state (Huddleston 1992). This relative (rather than true) hypovolaemic state stimulates baroreceptors, volume receptors, and osmoreceptors to reabsorb sodium and water to cause vasocostriction, in an effort to restore circulating volume and increase blood pressure.

(C) Coagulation defects

Early in shock, changes in blood coagulation can be seen. Hypercoagulability occurs as a result of the release of thromboplastic substances.

Systemic changes in coagulation also occur as a result of the effects of catecholamines, lysosomal enzymes, and acidosis. Platelet aggregation is increased in response to stress. This process of hypercoagulability may at first be compensatory if the cause of the shock is haemorrhage, but later it can produce drastic problems. The combination of hypercoagulation and stagnation of the blood in the capillaries may be responsible for microemboli developing during or following shock (Anderson 1982). These microemboli enhance tissue ischaemia and the progression of shock by decreasing the already poor blood flow through the capillaries to the tissues damaging organs, leading to multiple organ failure.

Late in shock, a state of hypocoagulability may develop owing to loss of clotting factors through: haemorrhage; replacement of lost volume by blood deficient in clotting factors, and/or crystalloid/ non crystalloid solutions causing haemodilution (Edwards 1998); and a decrease in the production of clotting factors due to poor tissue perfusion (Time out 2).

The progressive stage of shock is predominately marked by continuing hypoperfusion, cellular changes and hypoxia, leading to a reduction in blood pressure, and deteriorating organ function. How far the deterioration in organ function goes will vary from person to person, but organ function will largely determine the course and outcome. However, some organs bear the brunt of the body’s effort to compensate for a decrease in systemic pressure, and as a result these organs will suffer damage, and dysfunction will appear early in the shock syndrome. The point at which organ dysfunction becomes irreversible is not clear. The major organs effected, are the kidneys, liver, GIT, heart, lungs and brain.

Table 3 - Adrenergic receptors

<table>
<thead>
<tr>
<th>Neurotransmitter</th>
<th>Receptor type</th>
<th>Major locations</th>
<th>Effects of binding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Noreadrenaline and adrenaline (released adrenal medulla)</td>
<td>Beta 1</td>
<td>Predominantly heart</td>
<td>Increased heart rate and strength; stimulates renin release by kidney.</td>
</tr>
<tr>
<td></td>
<td>Beta 2</td>
<td>Lungs</td>
<td>Dilatation of blood vessels and bronchioles</td>
</tr>
<tr>
<td></td>
<td>Alpha 1</td>
<td>Blood vessels serving the skin; all sympathetic target organs except heart</td>
<td>Constricts blood vessels and visceral organ sphincters</td>
</tr>
<tr>
<td></td>
<td>Alpha 2</td>
<td>GI tract</td>
<td>Constricts blood vessels serving the gut</td>
</tr>
</tbody>
</table>

TIME OUT 5

Look at the nursing management of shock in your clinical practice. Now relate that care to the physiological processes occurring in shock, and how the interventions might prevent further deterioration and complications.
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TIME OUT 6
Identify the variables that effect the course of shock. Consider the consequences of these variables on an individual suffering from shock.

(C) Kidneys

The kidneys are probably the most important and apparent organs affected early in shock. Renal blood flow is reduced early, as the total renal blood flow falls, the glomerular filtration rate (GFR) is reduced and renin will be released by the kidney (Marieb 1998). The GFR is preserved for a time, but oliguria nevertheless occurs, due to antidiuretic hormone (ADH) and aldosterone secretion.

The renal tubules are susceptible to ischaemic damage, and when there is a reduction in oxygen and energy the reabsorptive functions for sodium and water are lost (Buckman et al 1992), and the renal tubules undergo necrosis. Acute tubular necrosis of the kidney commonly occur in shock, and if severe may lead to acute renal failure, contributing, to late deaths following resuscitation.

(C) Liver

The liver is a highly complex organ having multiple functions. The liver plays a key role in carbohydrate and lipid metabolism, synthesises many plasma proteins, including albumin, protease inhibitors, transport proteins and coagulation factors (Marieb 1998) and is sensitive to ischaemia. Both hepatic arterial and portal venous blood flow are reduced in shock. Early in shock, the liver releases large amounts of glucose as the result of adrenaline-induced glycogenolysis and gluconeogenesis.

In decompensated shock, all liver functions, including bile and cholesterol formation, protein synthesis, gluconeogenesis, lactate metabolism, detoxification, glycogen stores are depleted, and the phagocytic activity of the Kupfer cells, are depressed (Buckman et al 1992).

However, the liver has considerable reserve capacity and removal of up to 90 per cent of liver function is compatible with life, since it has a remarkable ability to regenerate (Gosling 1999). In general the liver appears to cope well with shock, and liver failure is frequently of late onset and is more often due to multiple organ failure in association with pulmonary and renal function.

(C) Gastro-intestinal tract (GIT)

The gut suffers an early reduction of oxygen delivery in hypovolaemia and other forms of shock, as a result of the effects of vasopressin, angiotensin II, and catecholamines (Buckman et al 1992). This may be because the overall oxygen extraction ratio for the gut generally is only about 20 per cent, thus, it can accommodate major flow reductions before oxygen delivery becomes inadequate. There is however, a threshold whereby the reduction in blood flow effects food/gut motility, absorption and produces lactate in large amounts (Case study 3). The gut is the major source of lactic acidosis in haemorrhagic shock.

The gut contains bacterial and bacterial toxins together with potentially harmful secretions such as hydrochloric acid and enzymes. The mucosa of the gut forms an essential barrier between the intestine and the bloodstream to which nutrients must be delivered (Elia 1995). In shock it is proposed that the gut mucosa barrier loses its integrity and becomes permeable to bacterial and endotoxins from the intestinal lumen, resulting in damage / necrosis of the intestinal wall by digestive enzymes.

The increased permeability may then allow pathogens to enter the portal and systemic circulation, causing infection and multiple organ failure (Adam 1994). The translocation of bacteria into the portal and systemic circulation has been proposed as a compounding mechanism of acute shock and as a cause of septic syndromes occurring after resuscitation. In addition, there is mounting evidence that gastrointestinal failure not only closes an avenue for nutrition, but may initiate or perpetuate mechanisms which contribute to remote multiple organ failure and death (Huddleston 1992).

(C) Lungs

In the lungs, ventilation and perfusion are necessary for oxygenating haemoglobin and removing waste gases from the lungs. In shock the reduced pulmonary blood flow results in an imbalance between oxygen supply and tissue demands (Gosling 1999). These are compensated for by hyperventilation due to chemoreceptor stimulation and thus arterial partial pressure of oxygen is well maintained.

In advanced shock where there is a further reduction in the pulmonary circulation, ventilation and / or perfusion and gas exchange does not take place, resulting in progressive atelectasis, adult respiratory distress syndrome (ARDS), respiratory muscle fatigue from respiratory muscle hypoperfusion, and respiratory failure may result. Respiratory failure is the primary cause of death following successful initial resuscitation of shock. Pre-existing lung disease, chest trauma and cardiac failure may contribute.

(C) Heart

Early deaths from shock are usually associated with unsupportable reductions in cardiac function. The heart muscle relies on the delivery of oxygen and nutrients to its cells via the coronary arteries, and has a very high oxygen requirement (Marieb 1998). Thus, a major reduction in cardiac blood flow quickly renders the heart muscle ischaemic.

Blood flow during shock is relatively preserved due to homeostatic compensation,
even as blood flow to other organs suffer. Consequently, myocardial dysfunction only occurs if there is a reduction in coronary blood flow exceeding the limits of compensatory mechanisms. As blood supply to the coronary arteries continues to decrease, myocardial contractility and compliance are reduced, and the heart ceases to function adequately as a pump, causing a decrease in cardiac output (Gosling 1999). Failure of the circulatory pump intensifies the deficient oxygen delivery throughout the remainder of the body, as well as to the heart itself. Except in cardiogenic shock, major effects on myocardial function probably do not occur until the very late stages of shock. The additive effects of acidosis, low oxygen levels, result in a decrease in myocardial contractility and a further reduction in cardiac output. These effects may also produce dangerous cardiac arrhythmias.

**C. Brain**

Of all organs, the brain is both the most intricate and most susceptible to hypoxic injury (McCance and Huether 1997). The brain is primarily affected because it depends on glucose and oxygen to function. Although it is protected by the homeostatic vasoconstriction and by its own autoregulation, if the systolic blood pressure falls below 60mmHg, the capacity for autoregulation is exceeded.

Mental state abnormalities are associated with poor outcome, as respiratory alkalosis, hypoxaemia, electrolyte disturbances start to appear. If blood flow continues to deteriorate autoregulation can no longer maintain normal cerebral metabolism. Unconsciousness rapidly occurs. With severe degrees of reduction of blood flow, the brain becomes ischaemic, and irreversible brain injury occurs.

**B. Refractory (irreversible) shock**

This is the final stage of shock, and is where severe cellular and organ dysfunction leads to general decline and death. In this stage it may be possible to return arterial pressure to normal for a short while, but tissue and organ deterioration continue, and no amount of therapy will reverse the process (Guthrie 1981). So much tissue damage and necrosis has occurred, so many mediators and toxins have been released into the systemic circulation, acidosis is so profound that even a return of normal cardiac output and arterial pressure will not reverse the downward progression.

At this point there is an almost total depletion of ATP stores, which are very difficult to restore once they are gone. There is usually vasomotor failure due to central nervous system (CNS) ischaemia. The vasomotor centres become so depressed that no sympathetic activity occurs. The vascular bed is generally dilated owing to the CNS depression, acidosis, and toxins. Deterioration will continue and death will ensue.

**A. Other considerations**

There are a number of variables that affect the course of shock, such as the age; the general state of health of the person before the shock insult; hypothermia; and pain.
The elderly injured patient requires special attention in relation to shock, as they have:
- Slowed blood circulation
- Structural and functional changes in the skin
- An overall decrease in heat-producing conservation activities
- Decrease in shivering response (delayed onset and decreased effectiveness)
- Slowed metabolic rate
- Sedate lifestyle
- Decreased vasoconstrictor response
- Diminished or absent sweating
- Desynchronisation of circadian rhythm
- Undernutrition

A decreased perception of heat and cold. Therefore, are at more risk of developing late stage shock following injury quicker than a younger person. In addition, they are more likely to be suffering from dehydration and if prolonged can severely enhance hypovolaemic shock (Moodooinan 1991). Dehydration itself is a critical state, added to any other form of shock it can precipitate and progress very early to a critical state, added to any other form of shock. It is imperative as the occurrence of shock in these patients is more likely to occur, as they may be more susceptible to low flow states, micro-emboli and loss of limb.

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