

## Shock

### SHOCK

Shock is a systemic state of low tissue perfusion which is inadequate for normal cellular respiration. With insufficient delivery of oxygen and glucose, cells switch from aerobic to anaerobic metabolism. If perfusion is not restored in a timely fashion, cell death ensues.

### Pathophysiology

#### Cellular

As perfusion to the tissues is reduced, cells are deprived of oxygen and must switch from aerobic to anaerobic metabolism.

The product of anaerobic respiration is lactic acid. When enough tissue is under-perfused, the accumulation of lactic acid in the blood produces a systemic metabolic acidosis.

As glucose within cells is exhausted, anaerobic respiration ceases and there is failure of sodium/potassium pumps in the cell membrane and intracellular organelles. Intracellular lysosomes release autodigestive enzymes and cell lysis ensues. Intracellular contents, including potassium are released into the blood stream.

#### Microvascular

As tissue ischemia progresses, these changes result in activation of the immune and coagulation systems. Hypoxia and acidosis activate complement and prime neutrophils, resulting in the generation of oxygen free radicals and cytokine release. These mechanisms lead to injury of the capillary endothelial cells. These, in turn, further activate the immune and coagulation systems. Damaged endothelium loses its integrity and becomes 'leaky'. Spaces between endothelial cells allow fluid to leak out and tissue oedema ensues, exacerbating cellular hypoxia.

#### Systemic

#### Cardiovascular

As preload and afterload decrease, there is a compensatory baroreceptor response resulting in increased sympathetic activity and release of catecholamines into the circulation. This results in tachycardia and systemic vasoconstriction (except in sepsis)

#### Respiratory

The metabolic acidosis and increased sympathetic response result in an increased respiratory rate and minute ventilation to increase the excretion of carbon dioxide (and so produce a compensatory respiratory alkalosis).

## Renal

Decreased perfusion pressure in the kidney leads to reduced filtration at the glomerulus and a decreased urine output. The renin–angiotensin–aldosterone axis is stimulated, resulting in further vasoconstriction and increased sodium and water reabsorption by the kidney.

## Endocrine

As well as activation of the adrenal and renin–angiotensin systems, vasopressin (antidiuretic hormone) is released from the hypothalamus in response to decreased preload and results in vasoconstriction and resorption of water in the renal collecting system. Cortisol is also released from the adrenal cortex contributing to the sodium and water resorption and sensitizing the cells to catecholamines.

Table 2.1 Cardiovascular and metabolic characteristics of shock.

	Hypovolaemia	Cardiogenic	Obstructive	Distributive
Cardiac output	Low	Low	Low	High
Vascular resistance	High	High	High	Low
Venous pressure	Low	High	High	Low
Mixed venous saturation	Low	Low	Low	High
Base deficit	High	High	High	High

## *Ischemia–reperfusion syndrome*

During the period of systemic hypoperfusion, cellular and organ damage progresses due to the direct effects of tissue hypoxia and local activation of inflammation. Further injury occurs once normal circulation is restored to these tissues. The acid and potassium load that has built up can lead to direct myocardial depression, vascular dilatation and further hypotension. The cellular and humoral elements activated by the hypoxia (complement, neutrophils, microvascular thrombi) are flushed back into the circulation where they cause further endothelial injury to organs such as the lungs and the kidneys.

This leads to acute lung injury, acute renal injury, multiple organ failure and death. Reperfusion injury can currently only be attenuated by reducing the extent and duration of tissue hypoperfusion.

### Classification of shock

All types of shock are characterized by systemic tissue hypoperfusion and different states may coexist within the same patient.

#### *Hypovolemic shock*

Hypovolemic shock is due to a reduced circulating volume. Hypovolemia may be due to hemorrhagic or non-hemorrhagic causes. Non-hemorrhagic causes include poor fluid intake (dehydration), excessive fluid loss due to vomiting, diarrhea,

urinary loss (e.g. diabetes), evaporation, or 'third-spacing' where fluid is lost into the gastrointestinal tract and interstitial spaces, as for example in bowel obstruction or pancreatitis.

Hypovolemia is probably the most common form of shock, and to some degree is a component of all other forms of shock. Absolute or relative hypovolemia must be excluded or treated in the management of the shocked state, regardless of cause.

### *Cardiogenic shock*

Cardiogenic shock is due to primary failure of the heart to pump blood to the tissues. Causes of cardiogenic shock include myocardial infarction, cardiac dysrhythmias, valvular heart disease, blunt myocardial injury and cardiomyopathy. Cardiac insufficiency may also be due to myocardial depression due to endogenous factors (e.g. bacterial and humoral agents released in sepsis) or exogenous factors, such as pharmaceutical agents or drug abuse. Evidence of venous hypertension with pulmonary or systemic oedema may coexist with the classical signs of shock.

### *Obstructive shock*

In obstructive shock there is a reduction in preload due to mechanical obstruction of cardiac filling. Common causes of obstructive shock include cardiac tamponade, tension pneumothorax, massive pulmonary embolus or air embolus. In each case, there is reduced filling of the left and/or right sides of the heart leading to reduced preload and a fall in cardiac output.

### *Distributive shock*

Distributive shock describes the pattern of cardiovascular responses characterizing a variety of conditions, including septic shock, anaphylaxis and spinal cord injury. Inadequate organ perfusion is accompanied by vascular dilatation with hypotension, low systemic vascular resistance, inadequate afterload and a resulting abnormally high cardiac output.

In anaphylaxis, vasodilatation is due to histamine release, while in high spinal cord injury there is failure of sympathetic outflow and adequate vascular tone (neurogenic shock). The cause in sepsis is less clear but is related to the release of bacterial products (endotoxin) and the activation of cellular and humoral components of the immune system. There is maldistribution of blood flow at a microvascular level with arteriovenous shunting and dysfunction of cellular utilization of oxygen.

### *Endocrine shock*

Endocrine shock may present as a combination of hypovolemic, cardiogenic or distributive shock. Causes of endocrine shock include hypo- and hyperthyroidism and adrenal insufficiency.

Hypothyroidism causes a shock state similar to that of neurogenic shock due to disordered vascular and cardiac responsiveness to circulating catecholamines. Cardiac output falls due to bradycardia & cardiomyopathy.

Thyrotoxicosis may cause a high-output cardiac failure.

Adrenal insufficiency leads to shock due to hypovolemia and a poor response to circulating and exogenous catecholamines.

Adrenal insufficiency may be due to pre-existing Addison's disease or be a relative insufficiency due to a pathological disease state, such as systemic sepsis.

## Consequences

### *Un-resuscitatable shock*

Patients who are in profound shock for a prolonged period of time become 'un-resuscitatable'. Cell death follows from cellular ischemia and the ability of the body to compensate is lost.

There is myocardial depression and loss of responsiveness to fluid or inotropic therapy. Peripherally there is loss of the ability to maintain systemic vascular resistance and further hypotension ensues. The peripheries no longer respond appropriately to vasopressor agents. Death is the inevitable result.

This stage of shock is the combined result of the severity of the insult and delayed, inadequate or inappropriate resuscitation in the earlier stages of shock.

### *Multiple organ failure*

As techniques of resuscitation have improved, more and more patients are surviving shock. Where intervention is timely and the period of shock is limited, patients may make a rapid, uncomplicated recovery. However, the result of prolonged systemic ischemia and reperfusion injury is end-organ damage and multiple organ failure.

Multiple organ failure is defined as two or more failed organ system

Management is supporting of organ systems with ventilation, cardiovascular support and hemofiltration/dialysis until there is recovery of organ function. Multiple organ failure currently carries a mortality of 60 per cent; thus, prevention is vital by early aggressive identification and reversal of shock.

## RESUSCITATION

Once 'airway' and 'breathing' are assessed and controlled, attention is directed to cardiovascular resuscitation.

### Conduct of resuscitation

- 1- Resuscitation should not be delayed in order to definitively diagnose the source of the shocked state. However, the timing and nature of resuscitation will depend on the type of shock and the timing and severity of the insult. If there is initial doubt about the cause of shock, it is safer to assume the cause is hypovolemia and begin with fluid resuscitation, and then assess the response.
- 2- In patients who are actively bleeding (major trauma, aortic aneurysm rupture, gastrointestinal hemorrhage), it is counterproductive to institute high-volume fluid therapy without controlling the site of hemorrhage. Increasing blood pressure merely increases bleeding from the site while fluid therapy cools the patient and dilutes available coagulation factors. Thus, operative hemorrhage control should not be delayed and resuscitation should proceed in parallel with surgery.
- 3- Conversely, a patient with bowel obstruction and hypovolemic shock must be adequately resuscitated before undergoing surgery otherwise the additional surgical injury and hypovolemia induced during the procedure will exacerbate the inflammatory activation and increase the incidence and severity of end-organ insult.

### Fluid therapy

In all cases of shock, regardless of classification, hypovolemia and inadequate preload must be addressed before other therapy is instituted. Administration of inotropic or chronotropic agents to an empty heart will rapidly and permanently deplete the myocardium of oxygen stores and dramatically reduce diastolic filling and therefore coronary perfusion. Patients will enter the un-resuscitatable stage of shock as the myocardium becomes progressively more ischemic and unresponsive to resuscitative attempts.

First-line therapy, therefore, is intravenous access and administration of intravenous fluids. Access should be through short, wide-bore catheters that allow rapid infusion of fluids

as necessary. Long, narrow lines, such as central venous catheters, have too high a resistance to allow rapid infusion and are more appropriate for monitoring than fluid replacement therapy.

### *Type of fluids*

There is continuing debate over which resuscitation fluid is best for the management of shock. There is no ideal resuscitation fluid, and it is more important to understand how and when to administer it. In most studies of shock resuscitation there is no overt difference in response or outcome between crystalloid solutions (normal saline, Hartmann's solution, Ringer's lactate) or colloids (albumin or commercially available products).

### Monitoring for patients in shock

#### Minimum

- ECG
- Pulse oximetry
- Blood pressure
- Urine output

#### Additional modalities

- Central venous pressure (Invasive blood pressure)
- Cardiac output
- Base deficit and serum lactate

### Central venous pressure

There is no 'normal' central venous pressure (CVP) for a shocked patient, CVP measurements should be assessed dynamically as response to a fluid challenge. A fluid bolus (250– 500 mL) is infused rapidly over 5–10 minutes.

The normal CVP response is a rise of 2–5 cmH<sub>2</sub>O which gradually drifts back to the original level over 10–20 minutes. Patients with no change in their CVP are empty and require further fluid resuscitation. Patients with a large, sustained rise in CVP have high preload.

### Cardiac output

Measurement of cardiac output, systemic vascular resistance and preload can help distinguish the types of shock present (hypovolemia, distributive, cardiogenic), especially when they coexist.

Measurement of cardiac output is desirable in patients who do not respond as expected to first-line therapy, or who have evidence of cardiogenic shock or myocardial dysfunction.

### Base deficit and lactate

Lactic acid is generated by cells undergoing anaerobic respiration. The degree of lactic acidosis, as measured by serum lactate level and/or the base deficit, is sensitive for both diagnosis of shock and monitoring the response to therapy. Patients with a

base deficit over 6 mmol/L have a much higher morbidity and mortality than those with no metabolic acidosis. Furthermore, the duration of time in shock with an increased base deficit is important, even if all other vital signs have returned to normal.

### **End points of resuscitation**

Traditionally, patients have been resuscitated until they have a normal pulse, blood pressure and urine output.

However, these parameters are monitoring organ systems whose blood flow is preserved until the late stages of shock. A patient therefore may be resuscitated to restore central perfusion to the brain, lungs and kidneys and yet continue to underperfuse the gut and muscle beds. Thus, activation of inflammation and coagulation may be ongoing and lead to reperfusion injury when these organs are finally perfused, and ultimately multiple organ failure.

This state of normal vital signs and continued under-perfusion is termed 'occult hypoperfusion'. With current monitoring techniques, it is manifested only by a persistent lactic acidosis and low mixed venous oxygen saturation. The duration patients spend in this hypo-perfused state has a dramatic effect on outcome.

Patients with occult hypoperfusion for more than 12 hours have two to three times the mortality of patients with a limited duration of shock.

-THE END-