

Shock

It 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 timely fashion, cell death occurs.

Classification of shock

- Hypovolemic
- Cardiogenic
- Obstructive
- Distributive
- Endocrine

Hypovolemic shock

It is caused by reduced circulating volume. It may be due to haemorrhagic or non hemorrhagic causes. Non hemorrhagic causes include poor fluid intake, and excessive fluid loss due to vomiting, diarrhoea, urinary loss as in diabetes, evaporation and third space loss. This type of shock also includes traumatic shock and burn shock.

Cardiogenic shock

It is due to primary failure of the heart to pump blood to the tissues. Causes include MI, arrhythmia, valvular heart disease, blunt myocardial injury and cardiomyopathy.

Obstructive shock

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

Distributive shock

It describes the pattern of cardiovascular responses to characterizing a variety of conditions including septic shock, anaphylactic shock, spinal cord injury, vasovagal shock and psychogenic shock. Inadequate organ perfusion is accompanied by vascular dilatation with hypotension, low systemic vascular resistance, inadequate after load and a resulting abnormally high cardiac output.

Endocrine shock

It may present as a combination of hypovolemic, cardiogenic, and distributive shock. Causes of endocrine shock include hypo and hyperthyroidism and adrenal insufficiency. Hypothyroidism causes a shock state similar to that of neurogenic shock as a result of disordered vascular and cardiac responsiveness to circulating catecholamines. Cardiac output falls because of bradycardia in low inotropy.

Thyrotoxicosis may cause a high output cardiac failure. Adrenal insufficiency cause shock as a result of hypovolemia and a poor response to circulating and exogenous catecholamine.

Causes of shock

Hypovolaemic

- Blood loss (ruptured abdominal aortic aneurysm, upper GI bleed, multiple fractures, etc.).
- Plasma loss (burns, pancreatitis).
- Extracellular fluid losses (vomiting, diarrhoea, intestinal fistula).

Cardiogenic

- Myocardial infarction.
- Dysrhythmias (AF, VT, AFlutter).
- Pulmonary embolus.
- Cardiac tamponade.
- Valvular heart disease

Septic

Gram –ve or, less often, Gram +ve infections.

Anaphylactic/distributive

Release of vasoactive substances when a sensitized individual is exposed to the appropriate antigen.

Notes on terms used

Resistance arterioles are the small-calibre vessels, 0.02—0.05 mm in diameter, containing abundant smooth muscle in their walls, the tone of which is controlled by local humoral factors and the sympathetic nerve fibres. The calibre of these small vessels gives rise to the peripheral vascular resistance, controlling blood pressure and blood flow through the capillary beds. The larger arteries merely serve to supply the arterioles with blood.

Capacitance veins comprise the entire venous network from the postcapillary venules to the large-calibre veins in limbs, abdomen and thorax and which normally contain 70 per cent of the circulating blood volume. Although thin walled with relatively little smooth muscle, sympathetic nerve stimulation contracts them, reducing their diameter and emptying the blood into the arterial side of the circulation.

A colloidal solution is one in which the majority of solute particles has a molecular weight greater than 30 000. The term includes all plasma solutions, including human plasma protein fraction (HPPF), dextrans, gelatin (e.g. Haemacel) and hydroxyethyl starch. Blood is not usually included in this term.

Minute volume ventilation is the volume of air (or oxygen) which enters the patient's lungs in 1 (each) minute, and is the product of respiratory rate and tidal volume.

Hyperventilation occurs when the patient is 'overbreathing' due to pain, anxiety or shock, such that the arterial carbon dioxide tension (PaCO₂) is lowered from the normal 40 mmHg (5.5 kPa).

Aspects of the pathophysiology of haemorrhage and shock

Low cardiac output is an early feature in shock, except for warm septic shock and neurogenic shock. Vasoconstriction occurs in an attempt to maintain perfusion pressures to the vital organs, such as the brain, liver and kidneys, as well as the heart muscle itself. Venoconstriction pushes more blood into the dynamic circulation whilst tachycardia helps to maintain a falling cardiac output. The minute ventilation rises 1.5—2 times and the respiratory rate 2—3 times maintaining oxygenation (except in cardiogenic shock with pulmonary oedema). The renal blood flow is reduced with consequent reduction in glomerular filtration and urine output. The renin—angiotensin mechanism is activated with further vasoconstriction and aldosterone release, causing salt and water retention. Release of antidiuretic hormone (ADH) decreases the volume and increases the concentration of urine. However, in early sepsis the patient, although hypovolaemic, may produce inappropriately large amounts of dilute urine.

As cardiac output falls, the hypotension and tachycardia cause poor perfusion of the coronary arteries, and this, in conjunction with hypoxia, metabolic acidosis and the release of specific cardiac depressants (endotoxaemia or pancreatitis), causes yet further cardiac depression and pump failure.

The cells become starved of oxygen, and anaerobic metabolism leads to lactic acidosis. Eventually, the cell membranes cannot pump sodium out of the cells; sodium enters the cells and potassium leaks out. Thus, the serum potassium is elevated. Calcium, however, leaks into the cells lowering the serum calcium. Furthermore, the intracellular lysosomes break down and release powerful enzymes causing further damage — 'the sick cell syndrome'. The platelets are activated in shock owing to the stagnation of blood in the capillaries. Blood sludging with red cell aggregation may progress to the formation of small clots and, indeed, to DIC. Several coagulation factors are consumed (platelets, fibrinogen, Factor V, Factor VIII, prothrombin), and troublesome bleeding may occur from needle puncture sites, wound edges and mucosal surfaces.

Septic (endotoxic) shock (Pathophysiology)

Hyperdynamic (warm) septic shock. This occurs in serious Gram-negative infections, for example from strangulated intestine, peritonitis, leaking oesophageal or intestinal anastomoses, or suppurative biliary conditions. At first, the patient has abnormal or increased cardiac output with tachycardia and a warm, dry skin, but the blood is shunted past the tissue cells, which become damaged by anaerobic metabolism (lactic acidosis). The capillary membranes start to leak and endotoxin is absorbed into the blood-stream, leading to a generalised systemic inflammatory state. The immediate and ready treatment of the cause, including the drainage of pus, is vital to the recovery of the patient at this stage (in strangulated hernia 'the danger is in the delay, not in the operation').

Hypovolaemic hypodynamic (cold) septic shock. This follows if severe sepsis or endotoxaemia is allowed to persist. Generalised capillary leakage

and other fluid losses lead to severe hypovolaemia with reduced cardiac output, tachycardia and vasoconstriction. The systemic infection induces cardiac depression, pulmonary hypertension, pulmonary oedema and hypoxia which, in turn, reduce cardiac output still further. The patient becomes cold, clammy, drowsy and tachypnoeic, but still can be converted to hyperdynamic (warm) shock by the administration of several litres of plasma or other colloidal solution. The similar use of crystalloid solutions may give rise to systemic and pulmonary oedema because of the larger volumes necessary.

Severity of shock

Compensated shock

As shock progresses the body's cardiovascular and endocrine compensatory responses reduce flow to non-essential organs to preserve preload and flow to the lungs and brain. In compensated shock there is adequate compensation to maintain the central blood volume and preserve flow to the kidneys, lungs and brain.

Apart from a tachycardia and cool peripheries (vasoconstriction, circulating catecholamines) there may be no other clinical signs of hypovolaemia.

However, this cardiovascular state is only maintained by reducing perfusion to the skin, muscle and gastrointestinal tract

There is a systemic metabolic acidosis and activation of humoral and cellular elements within the underperfused organs. Although clinically occult, this state will lead to multiple organ failure and death if prolonged because of the ischaemia-reperfusion effect. Patients with occult hypoperfusion (metabolic acidosis despite normal urine output and cardiorespiratory vital signs) for more than 12 hours have a significantly higher mortality rate, infection rate and incidence of multiple organ failure.

Decompensation

Further loss of circulating volume overloads the body's compensatory mechanisms and there is progressive renal, respiratory and cardiovascular decompensation. In general, loss of around 15% of the circulating blood volume is within normal compensatory mechanisms. Blood pressure is usually well maintained and only falls after 30–40% of the circulating volume has been lost.

Mild shock

Initially there is tachycardia, tachypnoea and a mild reduction in urine output and the patient may exhibit mild anxiety. Blood pressure is maintained although there is a decrease in pulse pressure.

The peripheries are cool and sweaty with prolonged capillary refill times (except in septic distributive shock).

Moderate shock

As shock progresses, renal compensatory mechanisms fail, renal perfusion falls and urine output dips below 0.5 ml kg⁻¹h⁻¹. There is further tachycardia and now the blood pressure starts to fall. Patients become drowsy and mildly confused.

Severe shock

In severe shock there is profound tachycardia and hypotension.

Urine output falls to zero and patients are unconscious with laboured respiration.

Clinical features of shock

Table Clinical features of shock

	Compensated	Mild	Moderate	Severe
Lactic acidosis	+	++	++	+++
Urine output	Normal	Normal	Reduced	Anuric
Level of consciousness	Normal	Mild anxiety	Drowsy	Comatose
Respiratory rate	Normal	Increased	Increased	Laboured
Pulse rate	Mild increase	Increased	Increased	Increased
Blood pressure	Normal	Normal	Mild hypotension	Severe hypotension

Notes (Atypical clinical features of shock)

The classic cardiovascular responses are not seen in every patient. It is important to recognise the limitations of the clinical examination and to recognise patients who are in shock despite the absence of classic signs.

Capillary refill

Most patients in hypovolaemic shock will have cool, pale peripheries with prolonged capillary refill times; however, the actual capillary refill time varies so much in adults that it is not a specific marker of whether a patient is shocked, and patients with short capillary refill times may be in the early stages of shock. In distributive (septic) shock the peripheries will be warm and capillary refill will be brisk despite profound shock.

Tachycardia

Tachycardia may not always accompany shock. Patients who are on β -blockers or who have implanted pacemakers are unable to mount a tachycardia. A pulse rate of 80 in a fit young adult who normally has a pulse rate of 50 is very abnormal. Furthermore, in some young patients with penetrating trauma, when there is haemorrhage but little tissue damage, there may be a paradoxical bradycardia rather than tachycardia accompanying the shocked state.

Blood pressure

It is important to recognise that hypotension is one of the last signs of shock. Children and fit young adults are able to maintain blood pressure until the final stages of shock by dramatic increases in stroke volume and peripheral vasoconstriction. These patients can be in profound shock with a normal blood pressure.

Elderly patients who are normally hypertensive may present with a 'normal' blood pressure for the general population but be hypovolaemic and hypotensive relative to their usual blood pressure. β -Blockers or other medications may prevent a tachycardic response. The diagnosis of shock may be difficult unless one is alert to these pitfalls.

To be continued.....

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