Shock Dr. Abbas Alhasani

* Shock is the most common and therefore the most important cause of death among surgical patients.
* Death that is caused by shock could be one of the followings:
1. Rapid death, in cases of profound shock.
2. Delayed death, as in:
* Consequences of organ ischaemia.
* Ischaemia-Reperfusion injury.

# Shock:

Insufficient delivery of O2 and Glucose 🡪 Anaerobic Metabolism 🡪 Cell Death.

## Pathophysiology

### Cellular

* Anaerobic metabolism 🡪 Lactic acid (instead of CO2 in aerobic metabolism) 🡪 Lactic Acidosis (Metabolic acidosis).
* Intracellular glucose is exhausted 🡪 failure of active transport across the membranes, this in turn causes:
1. Intracellular lysosome release 🡪 Auto-digestion.
2. Potassium release to the blood stream (normally K+ is mainly intracellular).

### Microvascular

Tissue ischaemia 🡪 Hypoxia and Acidosis 🡪 Activation of immune and coagulation systems 🡪 Injury of the capillary endothelial cells 🡪 More activation of immune and coagulation systems.

# Systemic impact of shock

### Cardiovascular

Decrease pre and after load 🡪 Baroreceptor response 🡪 Sympathetic activity and Catecholamine (adrenaline and Noradrenaline) 🡪 Tachycardia and systemic vasoconstriction (except septic shock).

### Respiratory

Metabolic acidosis and increase sympathetic response 🡪 increase respiratory rate and minute ventilation 🡪 compensatory respiratory alkalosis.

### Renal

* Decrease renal perfusion 🡪 decrease filtration at glomerulus 🡪 decrease urine output.
* Renin Angiotensin Aldosterone axis stimulation 🡪 more vasoconstriction and increase Sodium and water reabsorption.

### Endocrine

* Adrenal gland medulla 🡪 adrenaline and noradrenaline.
* Renin Angiotensin system.
* Vasopressin (ADH) from hypothalamus in response to decrease preload 🡪 vasoconstriction and increase water reabsorption from the collecting duct in the kidney.
* Cortisol from adrenal cortex 🡪 sodium and water reabsorption and sensitizing cells to catecholamines.

# Ischaemia-reperfusion syndrome

* Systemic hypoperfusion 🡪
* Direct effect of tissue hypoxia.
* Local activation of inflammation.
* Cellular and organ damage progresses.
* Further injury occurs once normal circulation is restored to these tissues.
* The acid and potassium load that has built up 🡪 direct myocardial depression, vascular dilatation and further hypotension.
* The cellular and humeral elements (complements, neutrophils, microvascular thrombi) 🡪 more endothelial injury to organs such as lungs (acute lung injury), kidneys (acute renal injury), multiple organ failure and death.

To attenuate the reperfusion injury:

1. Reduce the extent of tissue hypoxia.
2. Reduce the duration of tissue hypoxia.

# Classification of shock

* Depending on the initiating mechanism of shock.
* Different states may coexist within the same patient.
1. Hypovolaemic shock
2. Cardiogenic shock
3. Obstructive shock
4. Distributive shock
5. Endocrine shock

## Hypovolaemic shock

This is caused by a reduced circulating volume, may be the followings:

1. Haemorrhagic hypovolaemia (bleeding)
2. Non-haemorrhagic hypovolaemia:
* Poor fluid intake (dehydration)
* Excessive fluid loss:
* Vomiting
* Diarrhea
* Urinary loss (Diabetes)
* Evaporation
* "Third-spacing" loss (fluid is lost into the gastrointestinal tract and interstitial spaces e.g. bowel obstruction, pancreatitis)
* Hypovolaemia is the most common form of shock.
* Hypovolaemia is to some degree a component of all other forms of shock.

## Cardiogenic shock

Cardiogenic shock is due to primary failure of the heart to pump blood to the tissues.

### Causes:

1. Myocardial infarction (M.I.)
2. Cardiac dysrhythmias.
3. Valvular heart disease.
4. Blunt myocardial injury.
5. Cardiomyopathy.
6. Myocardial depression:
* Endogenous factors e.g. bacterial and humoral agents result from sepsis.
* Exogenous factors e.g. pharmaceutical agents or drug abuse.

Evidence of venous hypertension with pulmonary or systemic oedema may coexist with the classic signs of shock.

## Obstructive shock

In obstructive shock there is a reduction in preload because of mechanical obstruction of cardiac filling 🡪 fall in cardiac output.

### Causes:

1. Cardiac tamponade.
2. Tension pneumothorax
3. Massive pulmonary embolus.
4. Air embolus.

## Distributive shock

A shock, in which an inadequate organ perfusion is accompanied by:

* Vascular dilatation with hypotension.
* Low systemic vascular resistance.
* Inadequate afterload 🡪 abnormally high cardiac output.

There is maldistribution of blood flow at microvascular level with arterio-venous shunting and dysfunction of the cellular utilization of oxygen.

### Causes:

1. Septic shock: release of bacterial endotoxins the activation of cellular and humoral components of immune system.
2. Anaphylaxis: vasodilation is caused by histamine release.
3. Neurogenic shock, spinal cord injury: failure of sympathetic out flow and adequate vascular tone.

In the late phases of septic shock there is hypovolaemia from fluid loss into the interstitial spaces and there may be concomitant myocardial depression, which complicates the clinical picture.

## Endocrine shock

Endocrine shock may present as a combination of Hypovolaemic, cardiogenic and distributive shock.

### Causes:

1. Hypothyroidism: disordered vascular and cardiac responsiveness to circulating catecholamines 🡪 fall in cardiac output (similar to neurogenic shock)
2. Hyperthyroidism: may cause a high-output cardiac failure.
3. Adrenal insufficiency: either caused by adrenal pathology (Addison's disease) or poor response to catecholamines such as systemic sepsis.

# Severity of shock

* Compensated shock vs. Decompensated shock
* Mild, moderate and severe shock

## Compensated shock

Cardiovascular and endocrine responses 🡪 reduce flow to non-essential organs (skin, muscle and gastrointestinal tract) 🡪 preserve preload and flow to kidneys, lungs and brain.

* Clinically, tachycardia and cool peripheries, but normal vital signs and urine output.
* There is occult metabolic acidosis and activation of humoral and cellular elements within the underperfused organs.
* This state will lead to multiple organ failure and death if prolonged (ischaemia-reperfusion effect)
* If a patient has this state uncorrected for 12 hours, then he is liable for:
1. A significant high mortality rate.
2. High infection rate.
3. High incidence of multiple organ failure.

## 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

* Patient may exhibit mild anxiety.
* Tachycardia and tachypnea.
* Mild reduction of urine output.
* Normal blood pressure.
* Cool and sweaty peripheries (except in septic distributive shock).
* Prolonged capillary refill (except in septic distributive shock).

## Moderate shock

* Patient becomes drowsy and mildly confused.
* Further tachycardia.
* Renal output dips below 0.5 ml kg-1 h-1 (normally 1-2 ml kg-1 h-1 in adults) due to decrease renal perfusion and failure of renal compensatory mechanism.

## Severe shock

* Patient is unconscious.
* Profound tachycardia and laboured respiration.
* Hypotension.
* Urine output falls to zero.

# Pitfalls

The classic cardiovascular responses are not seen in every patient.

## Capillary refill

Its not a specific marker of shock.

* A patient with a 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 on beta blockers.
* Patients who have implanted pacemakers are unable to mount a tachycardia.
* 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 shock state.

## Blood pressure

Hypotension is one of the last signs of shock.

* Children and fit young adults are able to maintain blood pressure until the final stage of shock (i.e. profound shock but normal blood pressure).
* Elderly patients who are already hypertensive may present with a 'normal blood pressure' but be hypovolaemic and hypotensive relative to their usual blood pressure.
* Beta blockers or other medications may prevent a tachycardic response.

# Consequences

## Unresuscitatable shock

Patients who are in profound shock for a prolonged period of time become 'unresuscitatable'.

Prolonged profound shock 🡪 cellular ischaemia 🡪 cell death 🡪 loss of the body ability for compensation.

* Myocardial depression.
* Loss of responsiveness to fluid or inotropic therapy.
* Peripherally, there is loss of the ability to maintain systemic vascular resistance and further hypotension ensues.
* Death is the inevitable result.

## Multiple organ failure

When intervention is timely and the period of shock is limited, patients may make a rapid, uncomplicated recovery.

Multiple organ failure is defined as two or more failed organ systems.

Multiple organ failure currently carries a mortality rate of 60%.

No specific treatment for multiple organ failure, so the management is by supporting organ systems.

* Lung 🡪 acute respiratory distress syndrome (need ventilation)
* Kidney 🡪 acute renal insufficiency (need haemofiltaration/dialysis)
* Liver 🡪 acute liver insufficiency
* Clotting 🡪 coagulopathy
* Cardiac 🡪 cardiovascular failure (needs cardiovascular support)