College of Medicine TUCOM

Dept. of Surgery

SHOCK

Shock is a systemic state of low tissue perfusion which is inadequate for normal tissue respiration.

Or inadequate perfusion to maintain normal organ function.

Reduced O2 & glucose delivery -----anaerobic metabolism-----cell death.

It's a clinical syndrome ***not*** a diagnosis!.

Pathophysiology

Cellular

Cells switch from aerobic (CO2) to **anaerobic** (lactic acidosis) metabolism, this leads to systemic metabolic **acidosis.**

Failure of Na/K pumps of cell membrane, release of lysosomal autodigestive enzymes & **cell lysis**, release of intracellular contents (k) into circulation.

Microvascular

Tissue hypoxia & acidosis activate the coagulation & immune systems(PMNs & complement) resulting in **O2 free radicals & cytokines** release, injury to capillary endothelial cells-----increased permeability --- tissue **oedema**--- exacerbating cellular hypoxia.

Systemic

CVS

Compensatory increased **sympathetic** activity & **catecholamines** release ---- tachycardia & systemic vasoconstriction.

Respiratory

Met. Acidosis & sympathetic overactivity leads to increased respiratory rate & minute ventilation.

Renal

Decreased GFR & UOP. Stimulation of the **rennin-angiotensin-aldosterone** system ----- further vasoconstriction & Na/H2O reabsorption.

Endocrine

ADH ----- vasoconstriction & water resorption at the collecting ducts. **Cortisol** ---- Na/H2O resorption & increased cell sensitivity to catecholamines.

Ischemia-reperfusion syndrome

Further injury occurs once normal circulation is restored to ischemic tissues.

Acid & k ---- direct **myocardial depression** & vascular dilatation, = further hypotension.

The activated humoral & cellular elements (complement, PMNs & thrombi) are flushed back to circulation----**further endothelial injury** to organs, e.g lung, kidney, MOF, & death.

Its injury minimized by reducing the extent & duration of tissue hypoperfusion.

Classification

Numerous ways, below depending on mechanism (different states may coexist in same patient):

1- ***Hypovolemic***
   Due to reduced circulating volume. It may be ***A***- Haemorrhagic (blood loss)
   Or ***B***- Non-haemorrhagic : poor fluid intake (dehydration), fluid loss in cases of vomiting, diarrhea, urinary loss(DM), evaporation, or "3rd spacing" loss (in GIT or interstitial spaces, e.g IO, & pancreatitis).
   It's the **most common** form of shock, & is a **component of nearly all** other forms, so it must be **initially excluded & treated in any** shocked state regardless of cause.
2- ***Cardiogenic***
   Primary failure of the heart to pump blood to the tissues. Causes 1-MI, 2-Dysrythmias, 3-VHDs, 4-CMP, 5-Depressing factors, endogenous (bacterial or humoral) & exogenous (drug abuse).
   Classical signs = venous hypertension + pulmonary or systemic oedema.
3- ***Obstructive***
   Reduced preload d.t. mechanical obstruction of cardiac filling = reduced COP. Causes 1- cardiac tamponade, 2- tension pneumothorax, 3- massive lung/air embolism.

4- Distributive

Vascular dilatation & hypotension, low SVR, inadequate afterload resulting in abnormally **high COP**. Examples a- septic (bacterial endotoxins)m b- anaphylactic (histamine), c- neurogenic, in SC injury (loss of sympathetic supply & its vasoconstriction)

5- Endocrine

May be a combination of other forms (1,3,4). Causes hypo/hyperthyroidism & adrenal insufficiency.

Compensation

Compensated shock

The CVS & endocrine **reduce flow to non-essential** organs (skin. Muscle, & GIT) to maintain central blood flow, & preserve perfusion to kidneys, lungs & brain.

Although, clinically occult, this state may lead to MOF & death if prolonged, due to ischemia-reperfusion syndrome.

Decompensated shock

In general, loss of about **15%** of circulating blood volume is within compensatory mechanism, but if more----- decompensated. Blood pressure usually maintained, until **(30-40)%** of circulating volume lost.

Table. Classification of Hemorrhage

Parameter / class	I	II	III	IV
Blood loss (ml)	<750	750–1500	1500–2000	>2000
Blood loss (%)	<15	15–30	30–40	>40
Heart rate (b/m)	<100	>100	>120	>140
Blood pressure	Normal	Orthostatic	Hypotension	Severe hypotension
CNS symptoms	Normal	Anxious	Confused	Obtunded

Table. Severity of shock.

	Compensated	Mild	Moderate	Severe
Lactic acidosis	+	++	++	+++
Urine output	Normal	Normal	Reduced	Anuric
Conscious level	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

Clinical features :

The main signs of shock are hypotension, rapid & shallow breathing; cold & clammy skin ; rapid, weak pulse (except in neurogenic shock , u may find bradycardia, & in the early stages of septic shock you may find warm hands in the presence of increased cardiac output) ; dizziness, fainting, or weakness, and oliguria.

Multiple organ failure MOF

Two or more failed organ systems. There is no specific treatment for MOF. It carries a mortality of 60 %; thus **prevention** is vital by early aggressive identification and reversal of shock.

Effects of organ failure

_ Lung: Acute respiratory distress syndrome
_ Kidney: Acute renal insufficiency
_ Clotting: Coagulopathy
_ Cardiac: Cardiovascular failure

Resuscitation

Immediate resuscitation to ensure a patent **A**irway **(A)** and adequate **B**reathing(**B**) and ventilation. Then **C**irculatory support**(C).**
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 hypovolaemia** and begin with fluid resuscitation, and then assess the response.
In patients who are actively bleeding, control the site of haemorrhage.

Resuscitation is complete when O2 debt is repaid, tissue acidosis is corrected, and aerobic metabolism restored.

Fluid therapy

In all cases of shock, regardless of classification, **hypovolaemia** and inadequate preload must be addressed before other therapy is instituted. 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. There is no overt difference in response or outcome between crystalloid solutions (normal saline, Hartmann's solution, Ringer's lactate) or colloids (e.g. albumin).
If blood is being lost, the ideal replacement fluid is blood, although crystalloid therapy may be required while awaiting blood products.

Vasopressor and inotropic support

They are **not indicated as first**-line therapy in hypovolaemia. Vasopressor agents (phenylephrine, noradrenaline & vasopressin) are indicated in **distributive** shock states (sepsis, neurogenic shock) where there is peripheral vasodilatation, and a low systemic vascular resistance, leading to hypotension despite a high cardiac output.
In **cardiogenic** shock, or where myocardial depression complicated a shock state**, inotropic** therapy (dobutamine) may be required to increase cardiac output and therefore oxygen delivery.

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

Good Luck