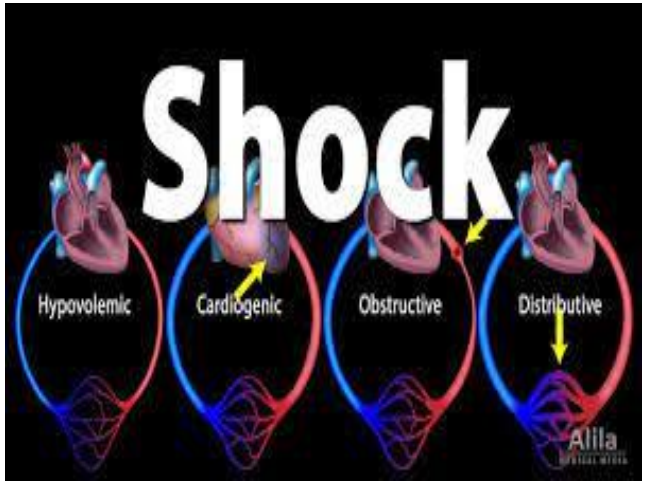
shock in adults



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- Shock is a life-threatening condition of circulatory failure that most commonly presents with hypotension.
- it is important that the clinician rapidly identify the etiology so that appropriate therapy can be administered to prevent MOF and death.

Mechanisms of shock

- Cellular hypoxia occurs as a result of:
  - ✓ reduced tissue perfusion/oxygen delivery
  - ✓increased oxygen consumption or
  - ✓inadequate oxygen utilization

- Cellular hypoxia, causes cell membrane ion pump dysfunction, intracellular edema, leakage of intracellular contents into the extracellular space, and inadequate regulation of intracellular PH.

Physiology

- The major physiologic determinants of tissue perfusion (and BP) are (CO) and (SVR):

BP = CO X SVR

- CO is the product of heart rate (HR) and stroke volume (SV):

CO = HR X SV

- The stroke volume is determined by:

✓**Preload**

✓**Myocardial contractility**

✓ **Afterload**

- SVR is governed by:

✓**Vessel length**

✓ **Blood viscosity**

✓ **Vessel diameter (vessel tone)**

- biologic processes that change any one of these physiologic parameters can result in hypotension and shock.

Stages of shock

- Pre-shock
- Shock
- End-organ dysfunction

Pre-shock

- It is characterized by compensatory responses to diminished tissue perfusion .
- tachycardia, a modest change in systemic blood pressure (increase or decrease), or mild to moderate hyper lactatemia, may be the only clinical signs of early shock .

Shock

- the compensatory mechanisms, signs and symptoms of organ dysfunction:
- symptomatic tachycardia
- dyspnea
- Restlessness
- Diaphoresis
- metabolic acidosis
- Hypotension
- Oliguria
- cool, clammy skin.

End-organ dysfunction

- Progressive shock leads to irreversible organ damage, multi organ failure (MOF), death.
- anuria and acute renal failure develop, acidemia further depresses CO, hypotension becomes severe, often related to vasoplegia, hyper lactatemia often worsens, and restlessness evolves into obtundation and coma.
- Death is common in this phase of shock.

CLASSIFICATION

- Shock is defined as a state of cellular and tissue hypoxia due to reduced oxygen delivery and/or increased oxygen consumption or inadequate oxygen utilization.
- This most commonly occurs when there is circulatory failure manifest as hypotension.

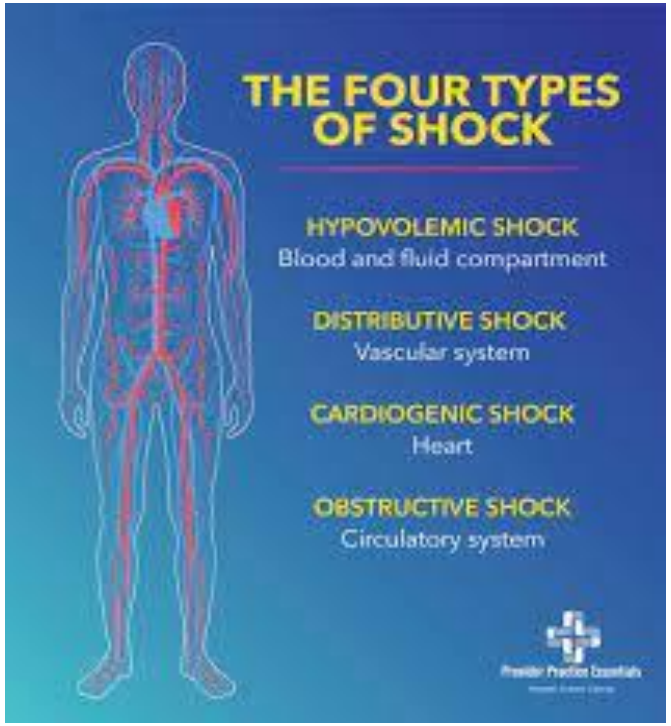
- four classes of shock :

✓Distributive

✓Cardiogenic

✓Hypovolemic

✓Obstructive



Clinical manifestations

Hypotension

Tachycardia

Oliguria

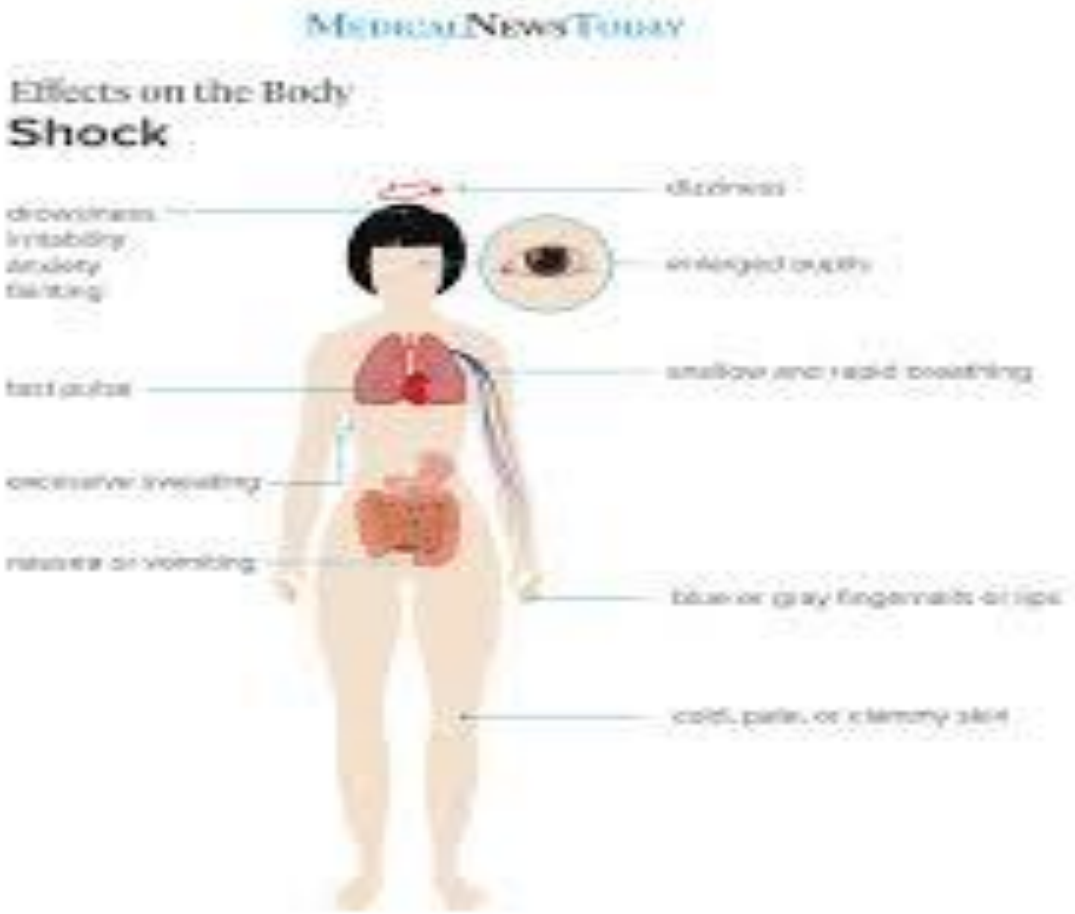
Abnormal mental status

Tachypnea

Cool, clammy, cyanotic skin

Metabolic acidosis

Hyperlactatemia



Hypotension

- may be :
  - ✓absolute (systolic blood pressure 40 mmHg)
  - ✓ orthostatic (>20 mmHg fall in SBP or >10 mmHg fall in DBP)
  - ✓profound (vasopressor-dependent)

Tachycardia

- Tachycardia is an early compensatory mechanism .
- It can be isolated or occur in association with hypotension.

Tachypnea

- Tachypnea is an early compensatory mechanism .

Oliguria

- Oliguria can be due to :
  - ✓shunting of renal blood flow
  - ✓direct injury to the kidney
  - ✓intravascular volume depletion

Mental status changes

- Altered sensorium is usually due to poor perfusion or metabolic encephalopathy.

Cool skin

- Cool, clammy skin is due to compensatory peripheral vasoconstriction that redirects blood centrally, to maintain vital organ perfusion .

Metabolic acidosis

- high anion gap metabolic acidosis should always raise the clinical suspicion for the presence of shock.

Hyperlactatemia

- the presence of an elevated serum lactate level has been associated with adverse outcomes.

Initial diagnostic evaluation

- A high clinical suspicion for the presence of shock is critical for diagnosis.
- A targeted history from pre hospital or hospital providers, the patient, their relatives, and/or the medical record should provide information on a patient's risk for shock, as well as the potential etiology.

- A thorough history
- assessment of sensorium, mucous membranes, lips and tongue, neck veins, lungs, heart, and abdomen, skin and joints
- Hypotension, oliguria, mental status changes, and cool, clammy skin
- A comprehensive assessment for the underlying etiology of shock should be performed after stabilization.

Laboratory evaluation

✓Serum lactate

✓Renal and liver function tests

✓ Cardiac enzymes and natriuretic peptides

✓ Complete blood count

✓Coagulation studies and D-dimer level

✓Blood gas analysis

Serum lactate level

- Elevated lactate levels in shock are reflective of poor tissue perfusion and are due to increased production from anaerobic metabolism, and decreased clearance by the liver, kidneys, and skeletal muscle.
- lactate levels can be serially measured to follow the response to therapies.

Renal and liver function tests

- Elevated BUN, creatine, and transaminases are usually due to shock-induced end-organ damage .
- but may also explain the etiology of shock (renal abscess, acute hepatitis, chronic cirrhosis).

Cardiac enzymes and natriuretic peptides

Elevated

- troponin-I or -T levels
- CPK
- brain natriuretic peptide
- N-terminal pro-BNP

may indicate cardiogenic shock.

CBC(diff)

- ⬆ hematocrit : hem concentration from hypovolemia
- Anemia in the setting of bleeding : hemorrhagic shock
- leukocytosis : septic shock, a stress response
- ⬇ white blood cell count and bandemia : sepsis

Coagulation studies and D-dimer level

- Evidence of DIC (⬆ FDP-⬆ D dimer-⬇ fibrinogen) can also be found in patients with severe shock.

VBG and ABG

- Hypoxemia :
    I. obstructive shock from pulmonary embolism
    II. cardiogenic shock from myocardial infarction
    III. septic shock from pneumonia, or ARDS

- Compensatory hypocapnia : with a metabolic acidosis
- Hypercapnia : with encephalopathy, brain injury, or increased dead space ventilation in patients with severe ARDS.

Imaging

- Chest radiography
- Other imaging directed at the etiology of shock