Team Medicine

Shock

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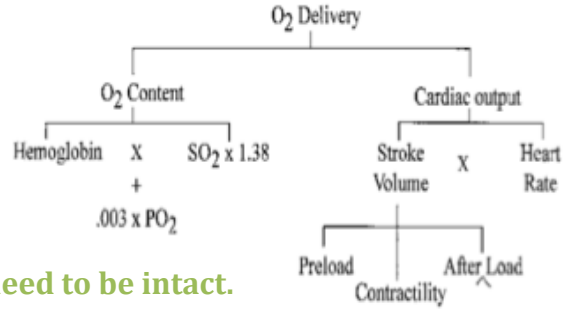
■ Important notes ■ Notes & doctor's notes

431 Teams
medical students

Definition:

- Reduction of effective tissue perfusion leading to cellular and circulatory dysfunction. It is a medical emergency, if unrecognized or inadequately treated will result in high mortality
- **Shock is the term used to describe acute circulatory failure with inadequate or inappropriately distributed tissue perfusion resulting in generalized cellular hypoxia and/or an inability of the cells to utilize oxygen. Shock is not a disease itself it's a reflection of a disease.**

The aim of perfusion is to achieve adequate Cellular Oxygenation and this requires:



1) Red Cell Oxygenation:
   a. Oxygen delivery to alveoli:
      - Adequate F_iO_2[1]
      - Patent airways **(from the mouth up to the terminal bronchi)**
      - Adequate ventilation **(need healthy lung)**

      Rib cage, spine bones, muscles and nerves need to be intact.
   b. Oxygen exchange with blood:
      - Adequate oxygen diffusion into blood

      We need to know about diffusion of oxygen from the alveoli to the blood. Sometimes it cannot happen if alveoli are filled with fluids as in pneumonic consolidation or if they are fibrosed. So, they must have good perfusion capacity.
      - Adequate RBC capacity to bind O_2 affected by:
        - **pH**
        - **Temperature**

RBCs can be diseased (e.g.: 1- inherited diseases: sickle cell. 2- acquired disease: fever)

2) Red Cell Delivery To Tissues
   a. 1. Adequate perfusion:
      - Blood volume
      - Cardiac output
        - Heart rate
        - Stroke volume
      - Conductance
        - Arterial resistance **(closed or constricted)**
        - Venous capacitance
   b. Adequate Hgb :
      - Adequate Hgb levels
      - Adequate RBC capacity to bind O2: - PH - Temperature

[1] Fraction of inhaled oxygen is an assumed percentage of oxygen concentration participating in gas exchange in the alveoli.

Consequences of Shock

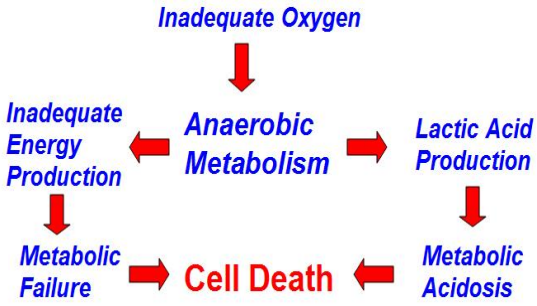
Inadequate tissue perfusion:

- Poor cellular oxygenation
- Shift from aerobic to anaerobic metabolism

Anaerobic Metabolism

- Occurs without oxygen

Oxidative phosphorylation can't occur without oxygen
Glycolysis can occur without oxygen
Cellular death leads to tissue and organ death
Can occur even after return of perfusion
⇒ Organ dysfunction or death



➢ **(The Doctor preferred the term dysfunction not death)**
The mechanism of anaerobic metabolism causes a sudden increase in waste products called lactic acid that further injures the cells, which causes further cell death in a cyclical fashion.

Maintaining Perfusion Requires:

- Adequate volume
- Normal cardiac function
- Normal vessels

Markers of Hypoperfusion

- ↑ Serum Lactate
- Metabolic acidosis
- Hypotension

Signs and symptoms:

- Restlessness, anxiety, combativeness (Earliest signs of shock)
- Brain→ Decreased mental status
- Kidney → Oliguria
- Peripheral Circulation → Hypotension & Cold clammy skin
- Lung → Hypoxemia
- Liver → Increased liver enzymes **(Can't be detected clinically)**

Shock Syndromes:

- Hypovolemic Shock
  - Blood volume problem
- Cardiogenic Shock
  - Blood pump problem
- Obstructive Shock
  - Filling Problem
- Distributive Shock
  - Blood vessels problem

Hypovolemic Shock (the most common)	Cardiogenic Shock
Loss of Volume: 1. Blood loss Traumatic - Non-traumatic (Vaginal - GI - GU[2]) 2. Fluid Loss Dehydration – Burns – Diarrhea - Vomiting - Diuresis - Sweating 3. Third Space losses **(Shift from intravascular compartment to extravascular compartment)** • Pancreatitis • Peritonitis • Bowel Obstruction **Consequence Of Volume Loss:** • 15%[750ml]- compensatory mechanism maintains cardiac output • 15-30% [750-1500ml]-decreased BP & urine output • 30-40% [1500-2000ml] -profound shock along with severe acidosis • 40-50% - refractory stage **(organ failure may be irreversible)** **Management:** Goal: Restore circulating volume, tissue perfusion & correct cause • ABC[3] • Two large bore IV lines/central line • Fluids / Blood & Products → vasopressors **(after correcting hypovolemia)** ➢ Target arterial BP – SBP [4]≥ 90 mmHg - MAP[5] ≥65 mmHg • Bladder catheter • Arterial Cannulation **Pathophysiology:** **Hypovolemic shock is when fluid volume decreases in the circulating volume of blood. When that occurs, the preload to the heart is decreased, which causes a decrease in stroke volume, which will cause a decrease in the cardiac output. With reduced cardiac output there will be decreased cellular oxygen perfusion. When cells don't receive enough oxygen they infarct.**	**Pump failure** 1. Myopathic: • Myocardial infraction • Congestive Heart Failure • Cardiomyopathy 2. Arrhythmic: • Tachyarrhythmia • Bradyarrhythmia 3. Mechanical: • Valvular Failure • Hypertrophic Obstructive Cardiomyopathy **(HOCM)** History: • Chest pain, Palpitations, SOB • RHD, IHD Physical exam: • Signs of ventricular failure • Heart: Murmurs, S3, S4 ➢ **Treat [1]rate, then [2]rhythm, then [3]Blood Pressure** • Correct bradycardia or tachycardia • Correct irregular rhythms • Treat Blood Pressure ○ ↑Cardiac contractility (inotropes) ▪ Dobutamine, Dopamine **Pathophysiology:** **Anything that hinders the flow of blood out of the heart can cause cardiogenic shock. When blood flow out of the heart is decreased, there will also be a decrease in oxygen availability to the cells. This will cause the cells to switch over to anaerobic metabolism and the whole cycle of shock is started.**

[2] GI = Gastrointestinal, GU = Genitourinary
[3] ABC = Airway, Breathing, Circulation
[4] SBP = Systolic Blood Pressure
[5] MAP = Mean Arterial Pressure

Distributive Shock (Blood Vessels Problem)	Obstructive Shock
• Inadequate perfusion of tissues due to mal-distribution of blood flow. • Cardiac pump & blood volume are normal but blood is not reaching the tissues. **Types:** 1. Septic Shock (most common) 2. Anaphylactic Shock 3. Neurogenic/Vasogenic (spinal cord) 4. Endocrinologic **Septic Shock Management:** • **A**irway **B**reathing **C**irculation • Assist ventilation & Augment Oxygenation • Restore Tissue perfusion: ○ IV Fluids, Vasopressors • Identification & Eradication of septic foci • Specific Therapies (antibiotics) Septic shock, which is sepsis plus hypotension in spite of fluid resuscitation. In general terms, sepsis is the invasion of the body by bacteria that causes an immune response. The resulting fallout is that tissue perfusion is impaired and the cycle of shock is begun once again at the cellular level. The classic example of septic shock is TSS or toxic shock syndrome, which is an invasion of the body by a toxin producing gram-positive bacteria. **Neurogenic Shock Management:** • Patient supine; lower extremities elevated • Trendelenburg • Infuse isotonic crystalloid • Maintain body temperature The pathophysiology or how this occurs is that with sympathetic nervous system depression or loss it creates a vasodilatation of the extremities secondary to loss of muscle tone. This vasodilatation causes a decrease in cardiac preload and as explained before, this will ultimately cause the less than adequate oxygenation of cells, which will trigger shock. **Anaphylactic Shock Management:** • **A**irway **B**reathing **C**irculation • Antihistamines • Corticosteroids • Epinephrine • Isotonic fluid	**Causes:** ▪ Impaired cardiac filling Cardiac tamponade[6]. Constrictive pericarditis. Tension pneumothorax. ▪ Increased ventricular afterload ▫ Pulmonary embolism **Management:** • Control airway ▪ Intubation • Treat the underlying cause ▪ Tension Pneumothorax: Chest tube ▪ Pericardial Tamponade: Pericardiocentesis ▪ Pulmonary Embolism: Anticoagulation • Isotonic fluids

[6] An acute type of pericardial effusion in which fluid accumulates in the pericardium (the sac in which the heart is enclosed).

The pathophysiology of anaphylactic shock is that when the antibody-antigen response is triggered they in turn trigger the release of biochemical mediators. These mediators have a two-fold effect. First vasoconstriction of the airways occurs making it hard to breath. A decrease in available oxygen to the lungs causes a decrease in available oxygen to the rest of the body. Once the body's cells start to receive not enough oxygen the cellular death cycle will begin. Additionally vasodilatation of the peripheral vessels occurs. This causes a decrease in preload volume to the heart. The decreased preload to the heart causes a decreased stroke volume, which causes a decreased cardiac output. A decrease in cardiac out put ultimately causes a decrease in cellular oxygenation, which will also begin the cellular death cycle.	

Key Issues In Shock

- Recognize & treat during compensatory phase.
- Best indicator of resuscitation effectiveness is **level of consciousness**
- Tissue ischemic sensitivity
  - Heart, brain, lung: 4 to 6 minutes
  - GI tract, liver, kidney: 45 to 60 minutes
  - Muscle, skin: 2 to 3 hours
- Falling BP = **LATE** sign of shock.
- Blood Pressure is **NOT** same thing as perfusion.
- Pallor, tachycardia, slow capillary refill = hypoperfusion, until proven otherwise.

Summary

Shock:

Reduction of effective tissue perfusion leading to cellular and circulatory dysfunction. It is a medical emergency, if unrecognized or inadequately treated will result in high mortality.
Shock = Circulatory failure.

Shock Features:

- Restlessness, anxiety, combativeness.
- Brain→ Decreased mental status.
- Kidney → Oliguria.
- Peripheral Circulation → Hypotension & Cold clammy skin.
- Lung → Hypoxemia.
- Liver → Increased liver enzymes.

Shock leads to: Inadequate tissue perfusion:

- Poor cellular oxygenation
- Shift from aerobic to anaerobic metabolism