SHOCK

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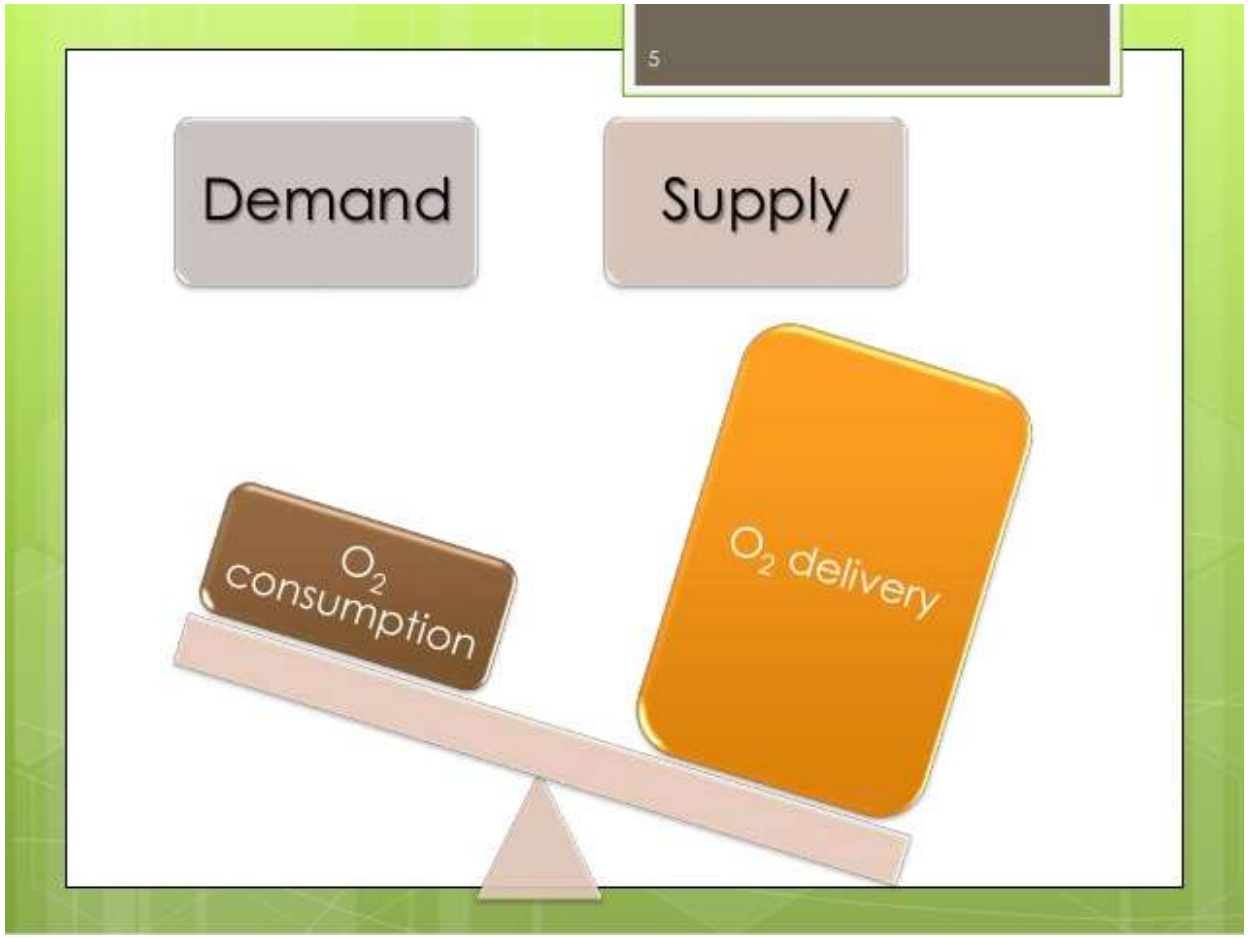
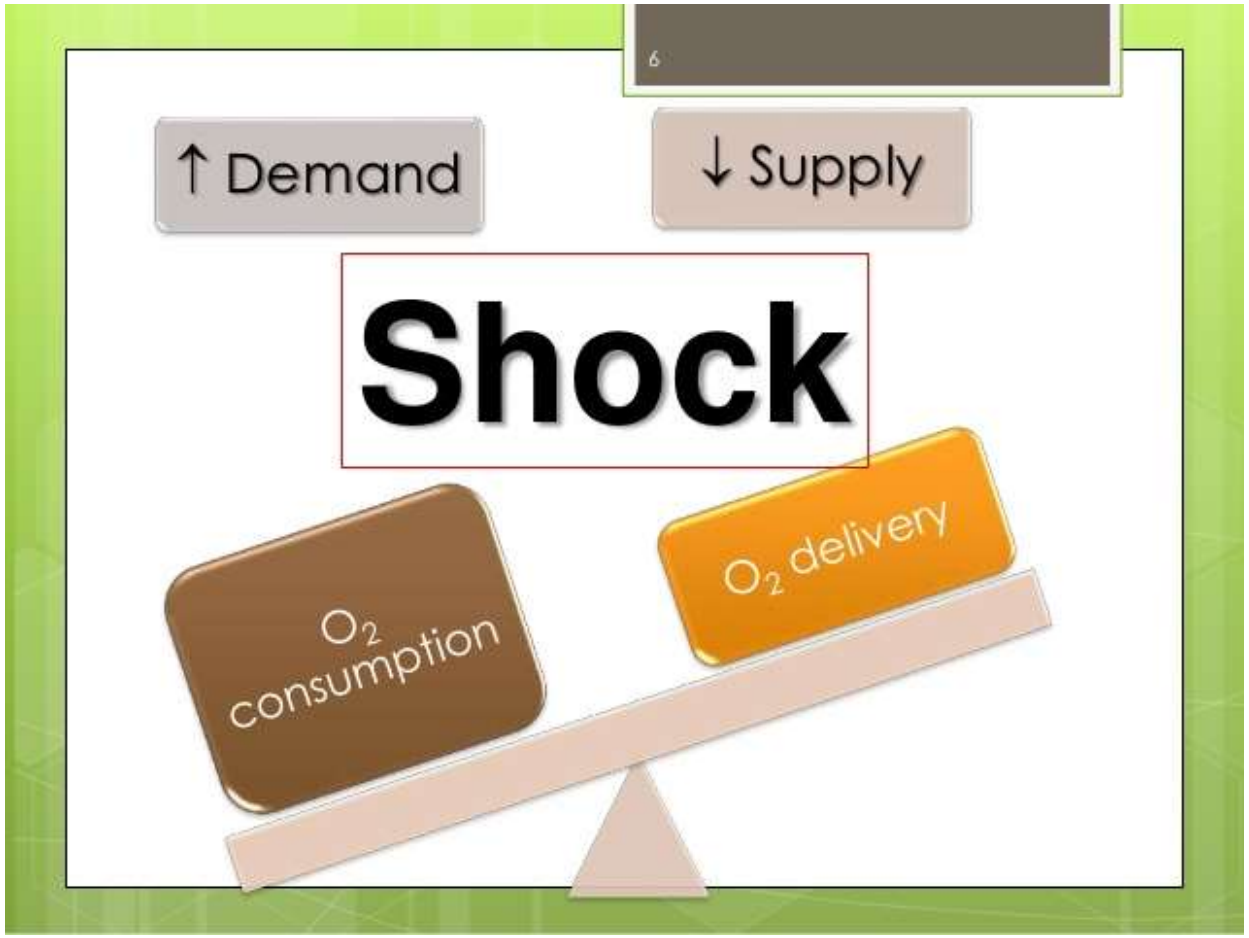
Definition

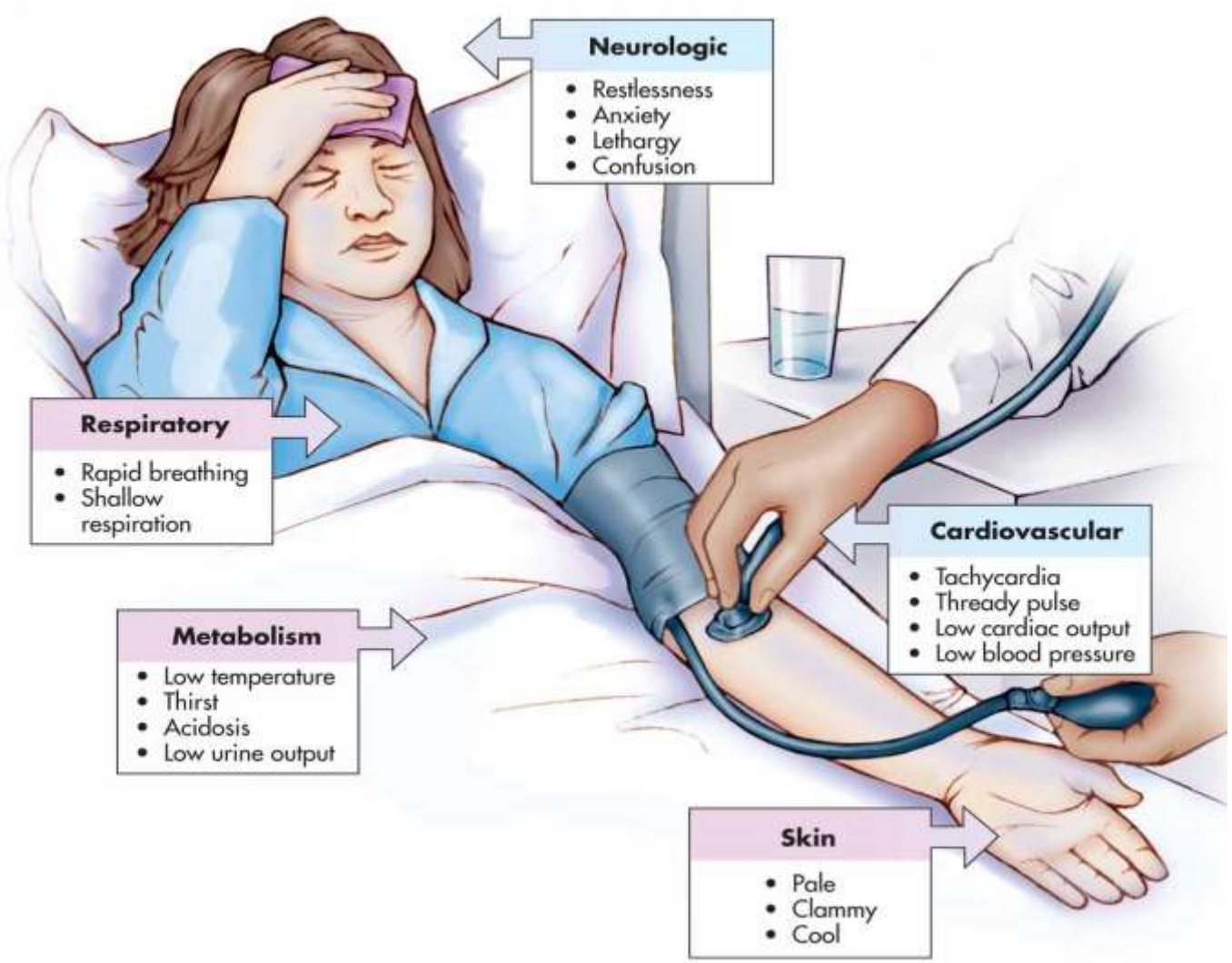
Sever and generalizes reduction of tissue perfusion by O_2 and nutrients due to failure of microcirculation leading to reversible then irreversible tissue injury.





CLINICAL PICTURE OF SHOCKED PATIENTS

- Atrial blood pressure <60mmHg
- Low COP
- Tachcardia
- Urine output < 20ml/hour
- Anxiety, confusion, pallor, sweating.



AIM OF TREATMENT

- Treat cause
- Replacement of any fluid lost from circulation.
- Maintenance of diastolic blood pressure and perfusion to vital organs.

PRECAUSTIONS

- Avoid sedatives
- Avoid alcohol
- Avoid over heating
- Avoid head-down position (better raise the foot of the bed 15-30cm)

TYPES OF SHOCKS

1. PRIMARY OR NEUROGENIC
2. SECONDARY OR HYPOVOLEMIC
3. CARDIOGENIC
4. SEPTIC
5. ANAPHYLACTIC

Neurogenic shock

Causes:

1. Spinal anesthesia or spinal trauma→ decreased sympathetic activity → **V.D.** → drop of BP.
2. Pain or anxiety due to release of mediators (kinin and histamine) → **V.D.**

Treatment:

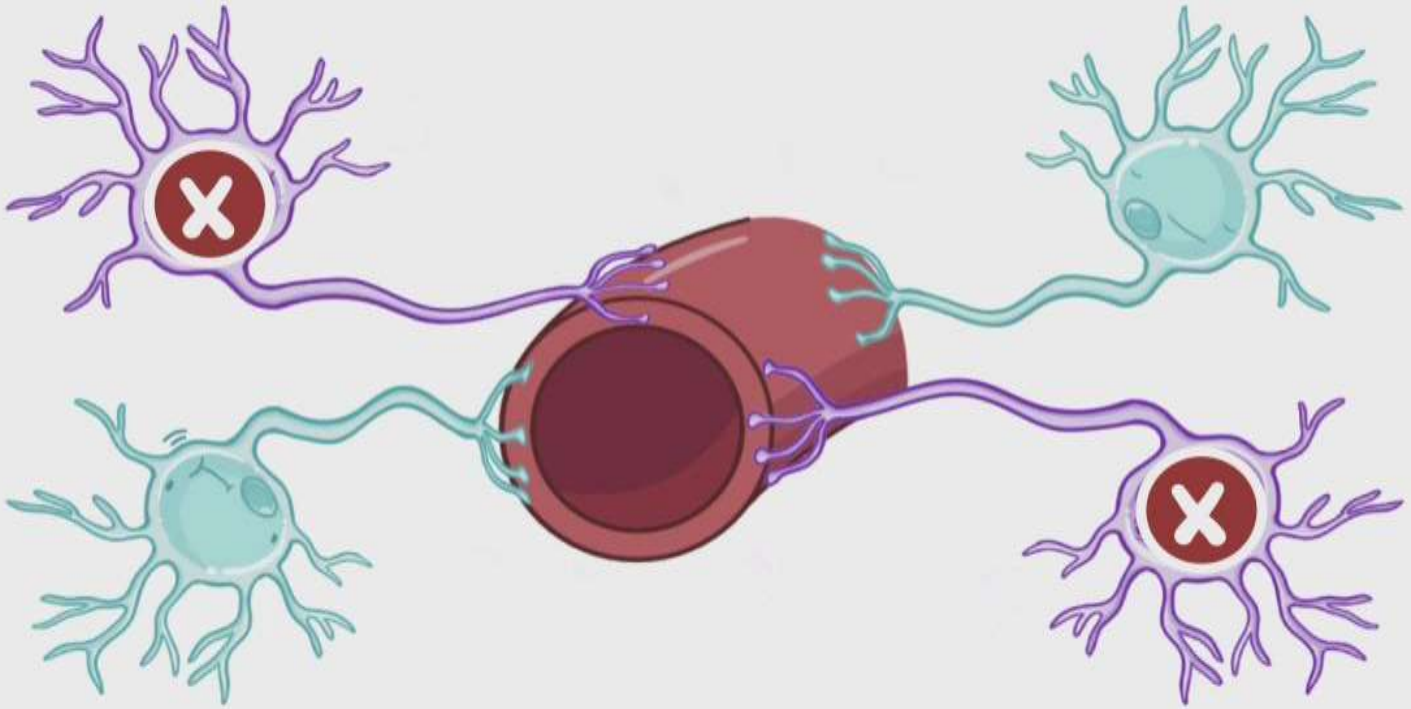
1. Position of the patient: recumbent with elevation of the lower limbs to prevent pooling of blood into the lower half of the body and to attain good perfusion of vital organs.
2. Vasopressor Sympathomimetics as:

- Ephedrine [25mg i.v.] ***or*** Dopamine by i.v. infusion [10 up to 50 μg/kg/min i.v infusion].

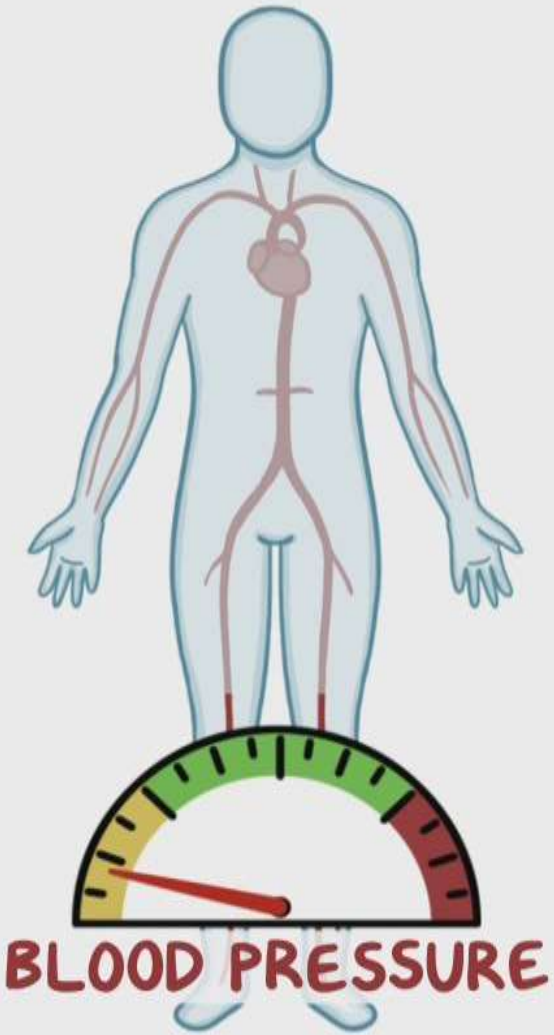
3. In case of severe pain, morphine can be given [from 5 mg up to 15mg i.v.]

NEUROGENIC SHOCK

LOSS of VASCULAR SYMPATHETIC TONE



UNOPPOSED PARASYMPATHETIC RESPONSE



Pharmacological actions of dopamine:

- **Slow rate of infusion [2- 5µg/kg/min]:** dopamine stimulates D_1 receptors in renal, splanchnic, coronary and cerebral circulation → VD in the renal vasculature. D_1 receptor activation increases renal blood flow and urine output.
- **Moderate rate of infusion [5-10µg/kg/min]**: dopamine stimulates β_1-adrenoceptors → positive inotropic and chronotropic effects → ↑↑ cardiac output.
- **High rate of infusion of dopamine [>10µg/kg/min]** stimulates α_1-adrenoceptors → VC → ↑↑ BP.

Hypovolemic shock

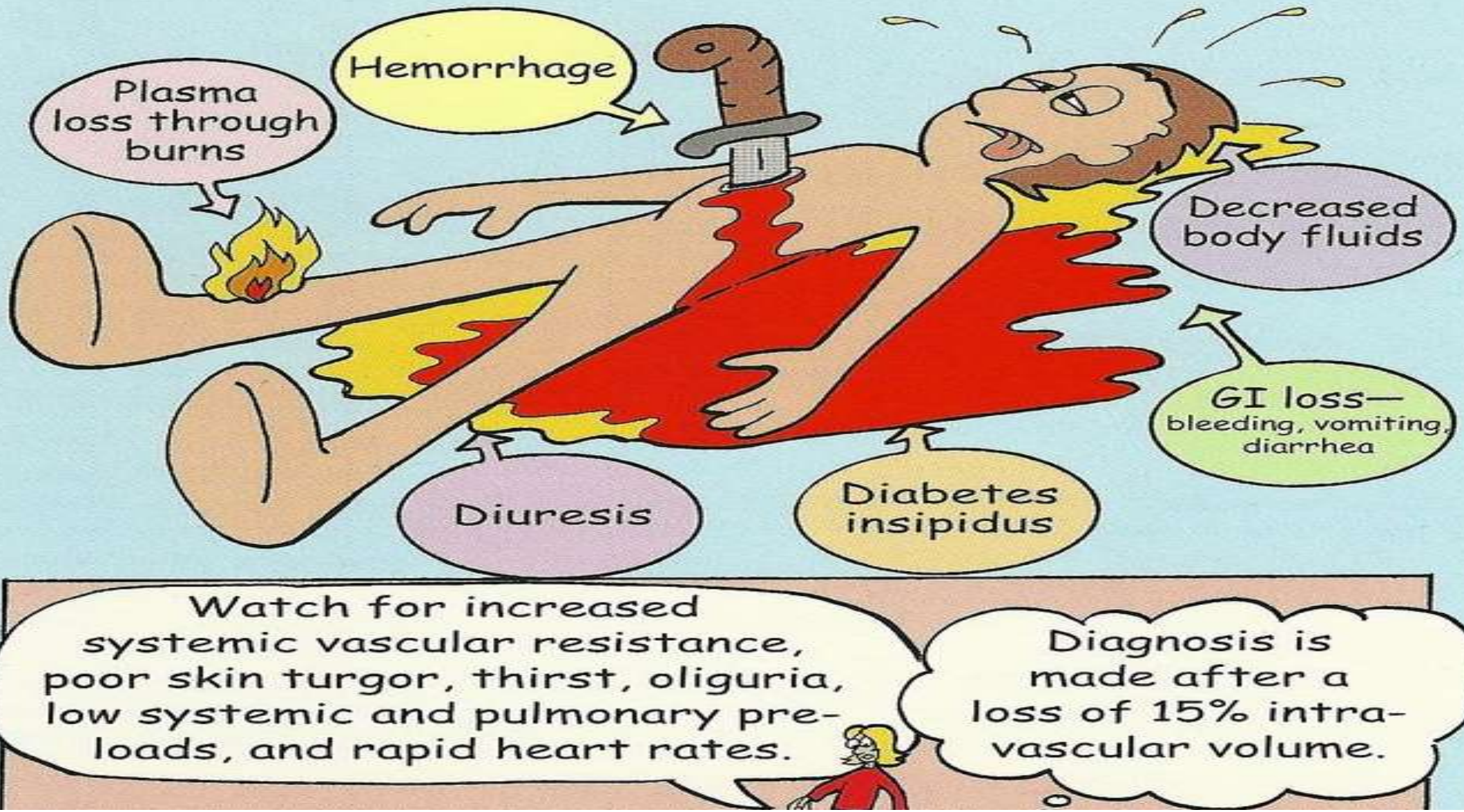
Causes:

- Rapid loss of large volume of blood [hemorrhage].
- Loss of plasma as in burn.
- Loss of fluids as in severe vomiting and diarrhea.

Treatment:

1. Volume replacement [blood, plasma or fluids].
2. Dopamine 2-5ug/kg/min → vasodilatation of renal blood vessels] → protect from renal hypo-perfusion and renal failure.
   - ❖ The rate of dopamine infusion can be increased according to patient hemodynamic state. But correction of hypovolemia must be done before dopamine infusion.

HYPOVOLEMIC SHOCK



Cardiogenic shock

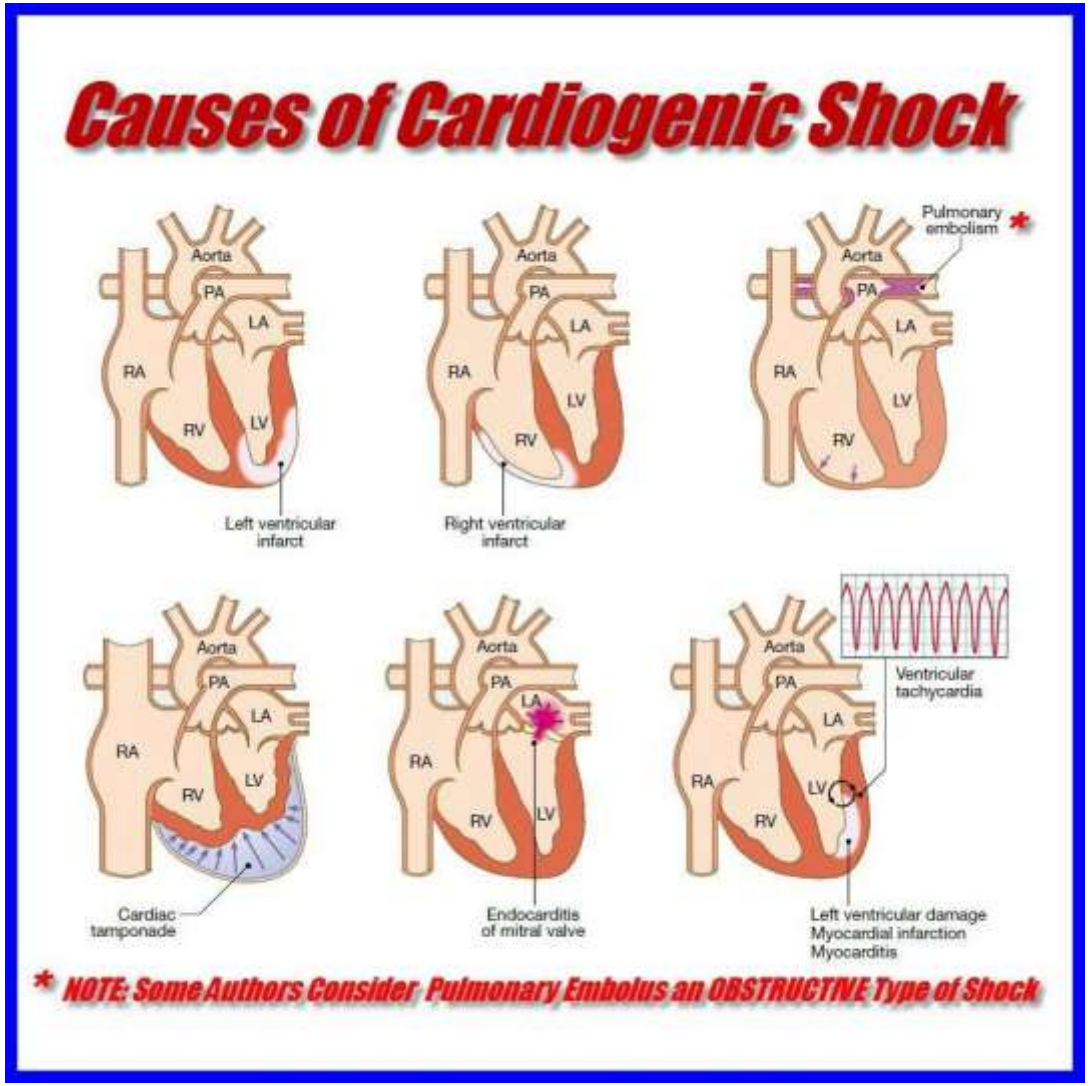
Causes:

1. Myocardial infarction
2. Massive pulmonary embolism
3. Myocarditis
4. Dysrhythmia

Treatment:

1- treatment of the cause

2- IV infusion of dobutamine.



- Dobutamine is a selective ***β_1 agonist*** increasing cardiac contractility with minimal increase in hear rate.
- The drug increases cardiac output and does not significantly elevates oxygen demands of the heart, a major advantage over other sympathomimetic drugs.
- It is given by IV infusion 2.5-10ug/kg min.
- It does not stimulate dopaminergic receptors.

Anaphylactic shock

Causes: Hypersensitivity reaction to an antigen for example [penicillin] → release of mediators [histamine, leukotrienes, PGs…] severe vasodilatation → shock

Treatment:

1. Adrenaline IM(0.5-1 mg repeated in 5-10min).
2. Antihistaminic IV (H1 blocker).
3. Hydrocortisone or prednisolone IV.

- Adrenaline which is life saving in anaphylaxis is a **Physiological antagonist to histamine (the main mediator in anaphylaxis)**

Physiological antagonism: (2 agonists + 2 Receptors ➔ 2 opposing actions).

Adrenaline ➔ bronchodilatation (β_2) & Histamine ➔ bronchoconstriction (H_1).

- Anti-histaminics act by reversible competitive antagonism to histamine on H1-receptors, producing:

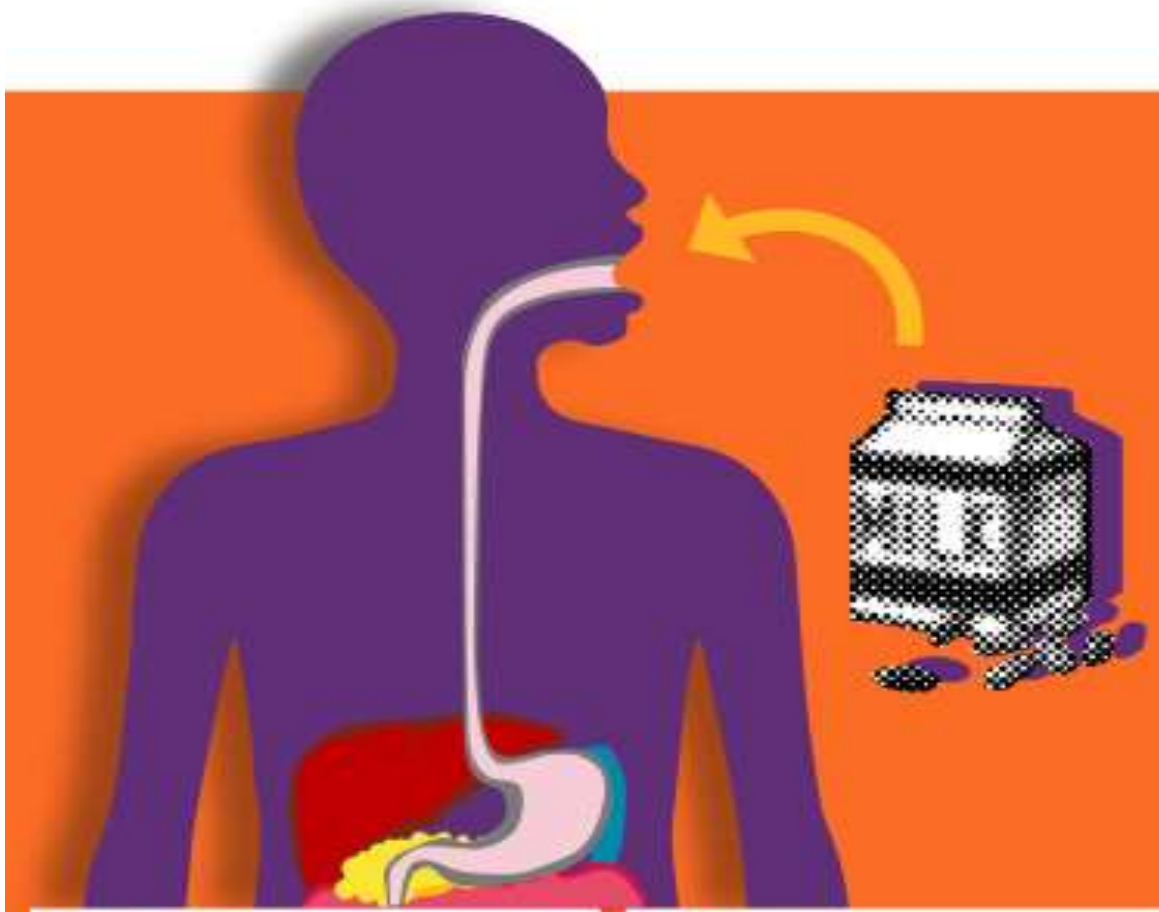
  - Antiallergic effect.

  - Complete antagonism of histamine-induced contraction on GIT and bronchi.

  - Partial antagonism of histamine on C.V.S.

- steroids can dramatically reduce the inflammatory response and to suppress immunity, through:

a. Indirect inhibition of phospholipase A_2 (due to the steroid-mediated elevation of lipocortin), thus blocks the release of arachidonic acid, the precursor of the inflammatory mediators prostaglandins and leukotrienes from membrane-bound phospholipids.
b. COX-2 synthesis in inflammatory cells is *reduced*, lowering the availability of prostaglandins.
c. Glucocorticoids stabilize mast cells and basophile membranes thus, interfering with mast cell degranulation resulting in decreased histamine release and capillary permeability.
d. Immunosuppressive by decrease Ab formation, Ag/ Ab reactions.
e. Salt and water retention so increase blood volume.



Allergic reaction

Anaphylaxis
affects the:

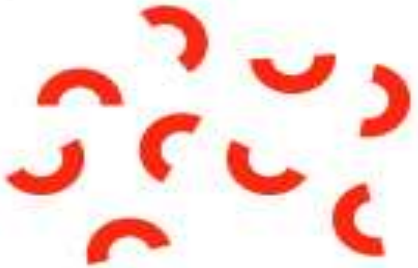
- Airway
- Breathing
- Consciousness

Mild/Moderate
symptoms

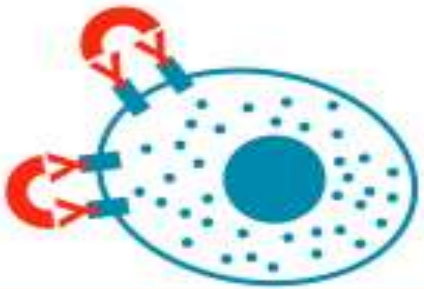
- Itching
- Swelling
- Nausea
- Vomiting
- Cramping

1 **Foods that can cause an allergic reaction**

2 **Food allergen**



3 **Mast Cell**



4 **Chemical release**

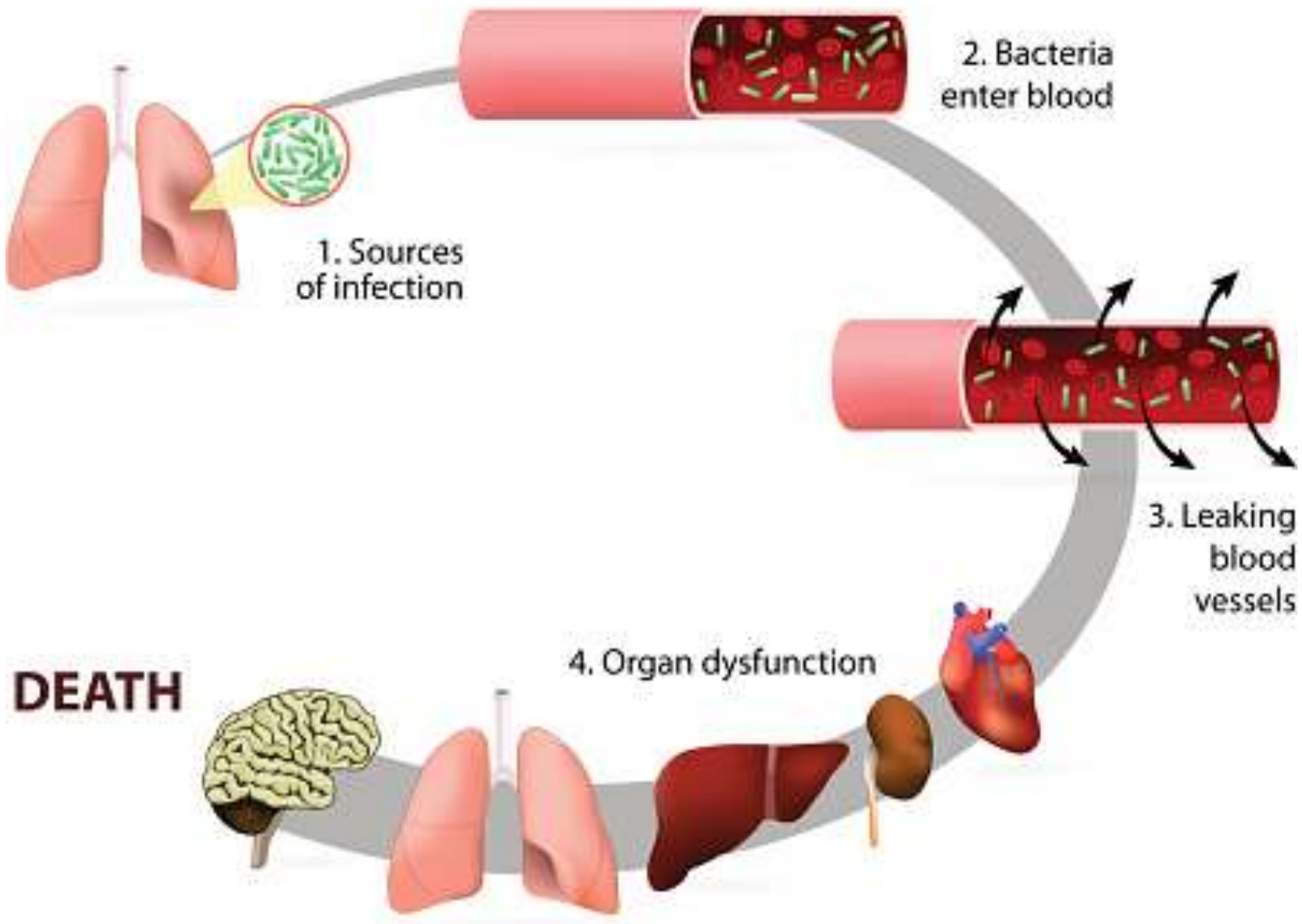
Septic shock

Caused by gram-negative and sometimes gram-positive bacterial infection → release of endotoxins that mediate vasodilatation

Treatment:

1. Full doses of bactericidal specific antibiotics until culture and sensitivity test are made.
2. Corticosteroids e.g. dexamethasone
3. Dopamine by IV infusion.
4. Monoclonal antibodies against bacterial endotoxins.

Sepsis


2. Bacteria enter blood
1. Sources of infection
3. Leaking blood vessels
4. Organ dysfunction
DEATH

- Corticosteroids restore cardiovascular homeostasis, terminate systemic and tissue inflammation, restore organ function, and prevent death in sepsis.
- Corticosteroids induce sodium retention via both mineralocorticoid and glucocorticoid receptors. Thereby, contribute to correct the hypovolemia that characterizes the early phase of sepsis.
- Corticosteroids cause rise in blood pressure,
  - ✓ by enhancing the vasoconstrictor action of adrenergic stimuli on small vessels via increasing the sensitivity to alpha agonist leading to increase in mean arterial pressure and systemic vascular resistance.
  - ✓ In addition, by favoring sodium and water accumulation in blood vessels' wall, corticosteroid will contribute to increase systemic vascular resistance.


Thank you