CARDIOVASCULAR PHYSIOLOGY

SHOCK

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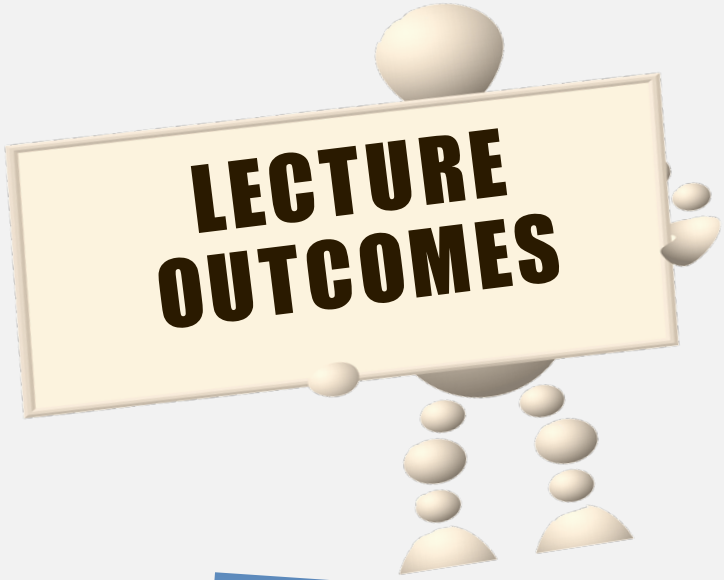


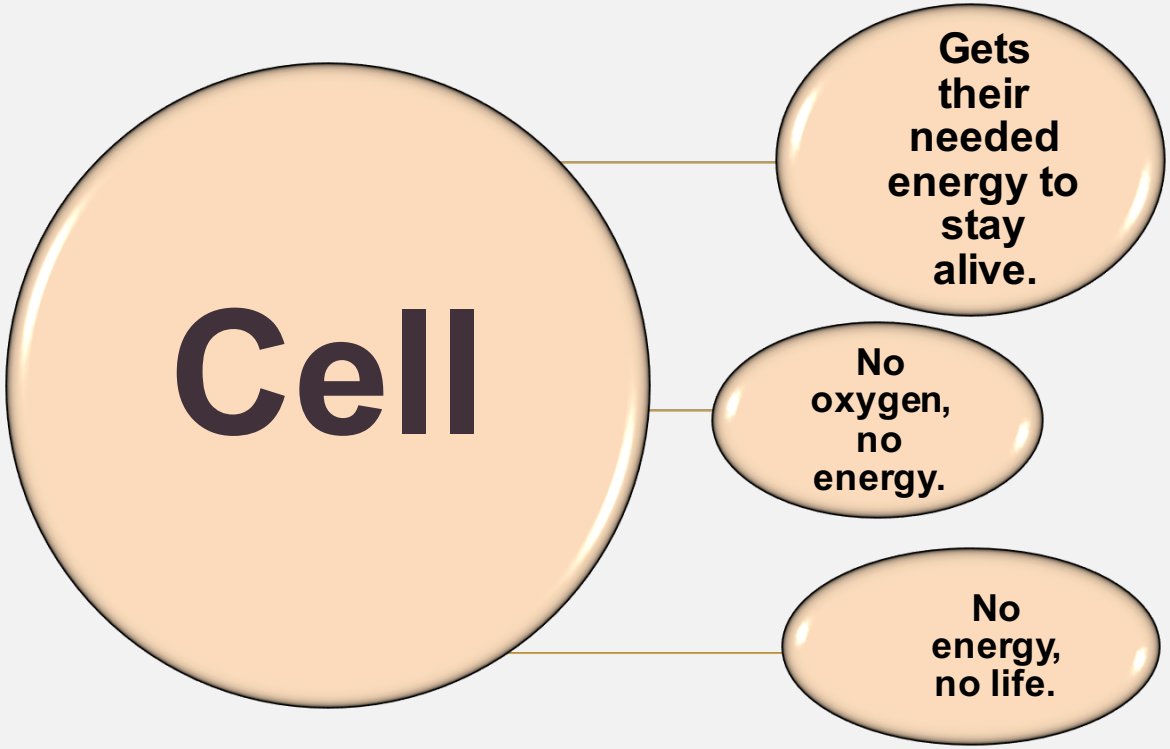
Types & causes of shock.

Define circulatory shock.

Body compensatory mechanisms during reversible phases of hemorrhagic shock.

Mechanisms responsible for the irreversible phase of hemorrhagic shock.

BASIC UNIT OF LIFE



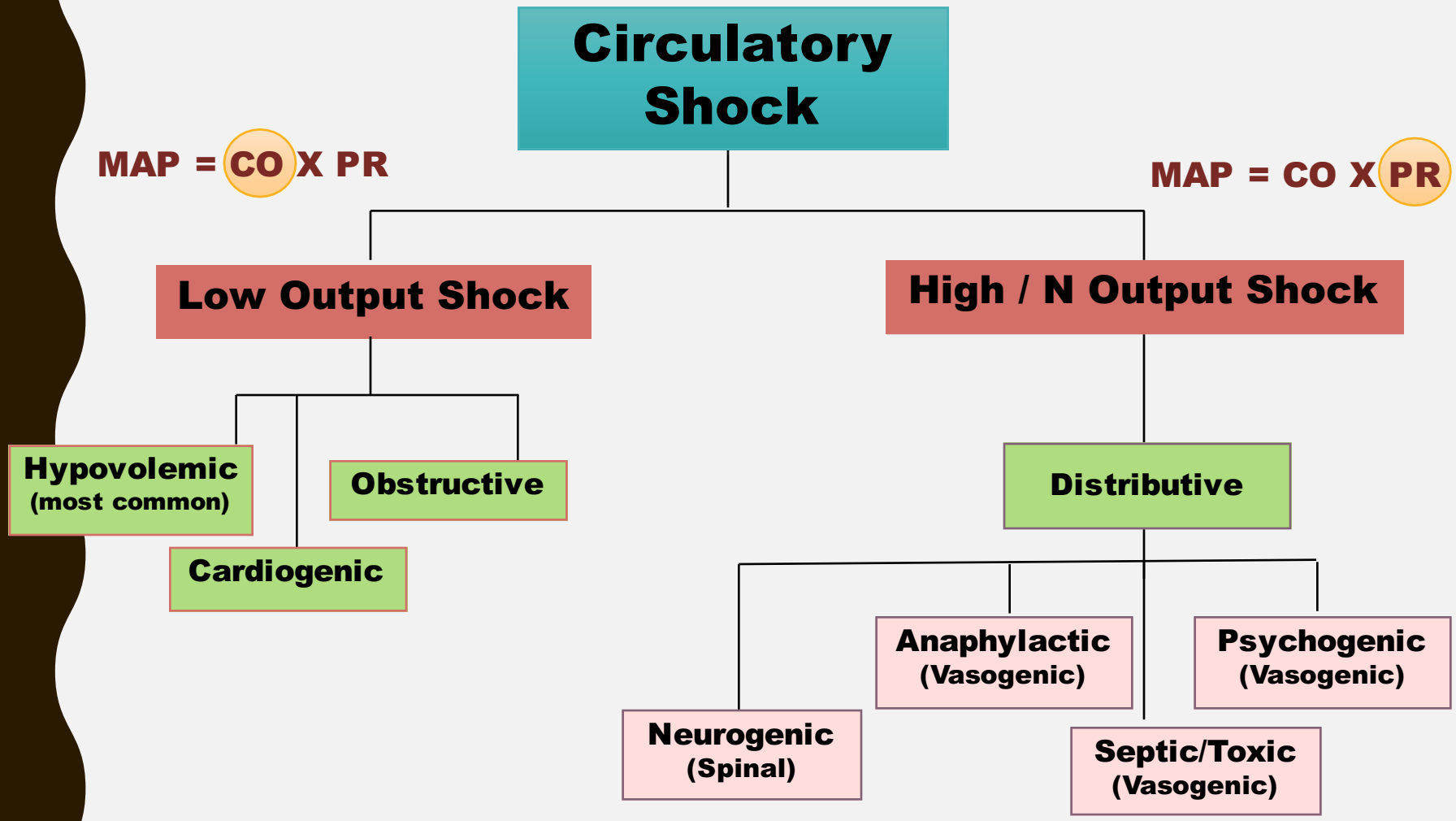
WHAT IS SHOCK ?

- Any condition in which the circulatory system is unable to provide adequate circulation & tissue perfusion, resulting in failure to deliver oxygen to the tissues & vital body organs relative to its metabolic requirement.
- Defined as **Circulatory Shock**.
- Results in organ dysfunction & cellular damage.
- If not quickly corrected, it may lead to irreversible shock & death.

Types of Shock

- Hypovolemic – most common
  - Hemorrhagic, occult fluid loss
- Cardiogenic
  - Ischemia, arrhythmia, valvular, myocardial depression
- Distributive
  - Anaphylaxis, sepsis, neurogenic
- Obstructive
  - Tension pneumo, pericardial tamponade, PE

TYPES OF CIRCULATORY SHOCK



HYPOVOLEMIC SHOCK

- **Low CO due to:**

MAP = CO X PR

  - Inadequate blood/plasma volume (loss of 15-25% / 1-2 L).
  - Reduced venous return (preload.).

- **Causes:**

  - Blood loss/ Hemorrhage: (commonest)
    - Internal or external.
  - Fluid/plasma loss: Vomiting, diarrhea, burn, excess sweating, dehydration, trauma.

CARDIOGENIC SHOCK

- **Low CO due to:** **MAP = CO X PR**
  - Failure of myocardial pump, despite adequate ventricular filling pressure.
- **Causes:**
  - Myocardial Infarction.. (Most common.)
  - Myocarditis.
  - Cardiomyopathy.
  - Cardiac tamponade.
  - Acute valvular dysfunction, e.g. rupture of papillary muscle post MI.
  - Congestive heart failure.
  - Sustained Arrhythmias, e.g. heart block, ventricular tachycardia.
  - Pulmonary embolism.
- Is associated with loss of > 40% of LV myocardial function.
- Mortality rate is high, 60-90%.

OBSTRUCTIVE SHOCK

- **CO is reduced by vascular obstruction:**
  - **Obstruction of venous return:**
    - e.g. Vena Cava Syndrome (usually neoplasms).
  - **Compression of the heart:**
    - e.g. hemorrhagic pericarditis → cardiac tamponade.
  - **Obstruction of the outflow of the heart:**
    - Aortic dissection.
    - Massive pulmonary embolism.
    - Pneumothorax.

MAP = CO X PR

DISTRIBUTIVE SHOCK:
HIGH/ NORMAL OUTPUT

- CO is normal or elevated.
- Distribution is inappropriate.
- Shock is due to loss of vascular resistance.

MAP = CO X PR

DISTRIBUTIVE SHOCK:
HIGH/ NORMAL OUTPUT

- **Septic/ Toxic/ Endotoxic Shock:** **MAP = CO X PR**
  - Bacterial endotoxin triggers peripheral vasodilatation & endothelial injury.
  - Hyperdynamic state.

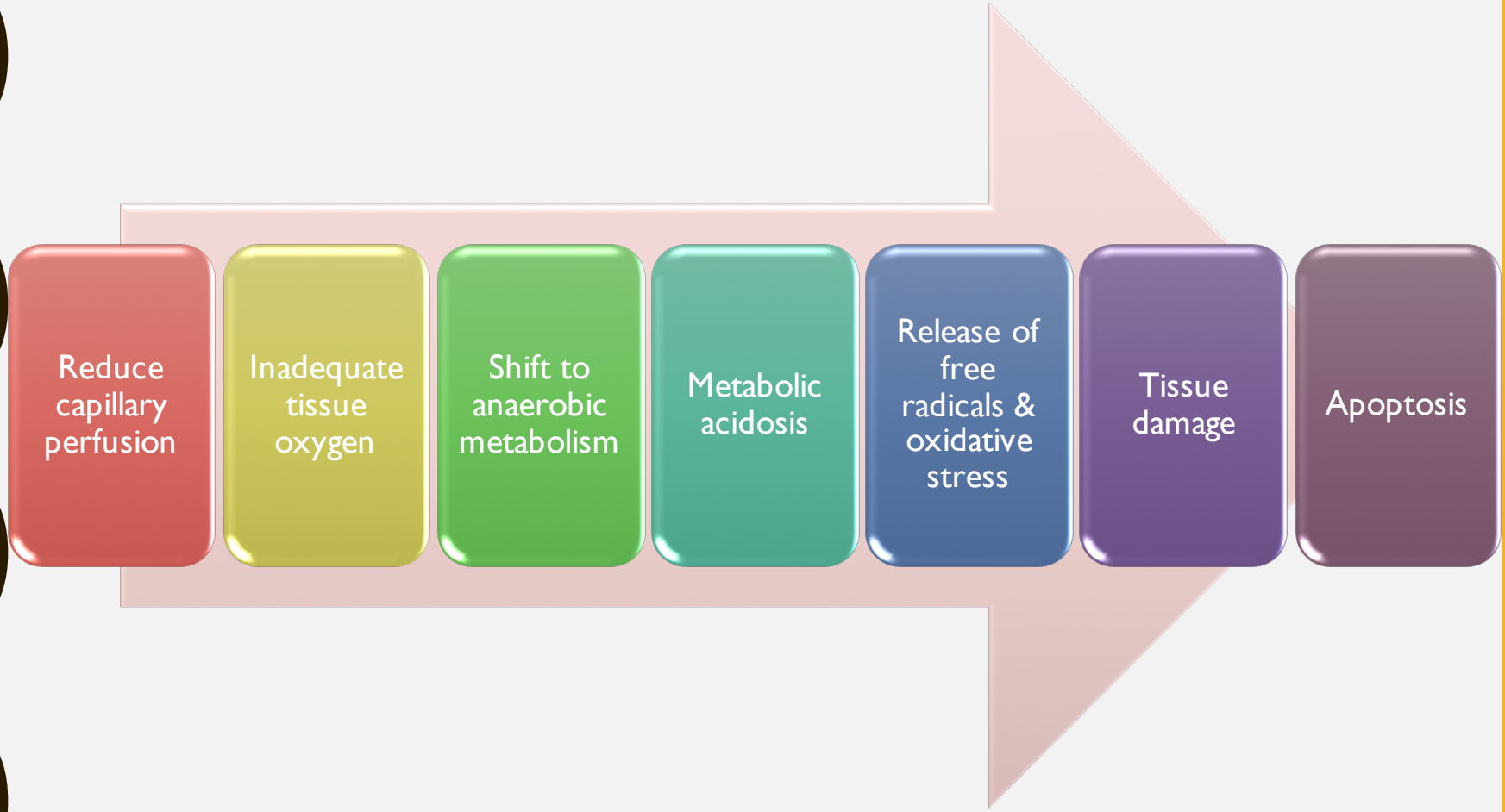

- **Anaphylactic shock:**
  - Massive & generalized allergic reaction.
  - IgE- mediated hypersensitivity.
  - Histamine triggers peripheral vasodilation & ↑ capillary permeability.
  - Can lead to low output distributive shock.


- **Psychogenic shock:**
  - Simple fainting (syncope.)
  - Caused by stress, pain, or fright.
  - ↓ HR & vessels dilate.
  - Brain becomes hypoperfused.
  - Loss of consciousness.

DISTRIBUTIVE SHOCK:
HIGH/ NORMAL OUTPUT

- **Neurogenic/ Spinal Shock (venous pooling):**
  - Loss or drop in vasomotor (vascular) tone/ spinal cord injury.
  - Generalized peripheral vasodilation.
  - Blood volume remains normal.
  - CO is severely reduced as blood is pooled in peripheral veins.. (Capacity of blood ↑, & venous return ↓.)
  - Behaves like hypovolemic shock.

PATHOPHYSIOLOGY OF SHOCK



METABOLIC CHANGES & CELLULAR RESPONSE TO SHOCK

1. **Reduce capillary perfusion:**

→ Spasm of pre/post capillary sphincters.

→ hypoxic tissue damage, (oxidative stress.)

→ anaerobic metabolism (anaerobic glycolysis.)

→ lactic acid production.

→ **metabolic acidosis (intracellular acidosis)**.

→ **Failure of Na+/K+ pump** (↑ [Na^+] & [C^{2+}]).

→ **Lysosomes, nuclear membranes & mitochondrial breakdown**.

METABOLIC CHANGES & CELLULAR RESPONSE TO SHOCK

2. **After 3 - 5 hrs of shock:**

→ precapillary sphincters dilate, venules are still constricted.

→ blood stagnation in capillaries.

→ hypoxia continue + fluid leaves to extra vascular compartment.

→ further reduction in circulating blood volume.

3. **Granulocytes accumulation at injured vessels:**

→ **free radicals release.**

→ further tissue damage.

METABOLIC CHANGES & CELLULAR RESPONSE TO SHOCK

4. **Damage in GIT mucosa** → allows bacteria into circulation.

5. **Cerebral ischemia** → depression of VMC → vasodilation + ↓ HR
   (vasomotor center .. sympathetic)
   → further decrease in blood pressure.

6. **Myocardial ischemia** → depressed contractility + myocardial damage
   → more shock & acidosis.

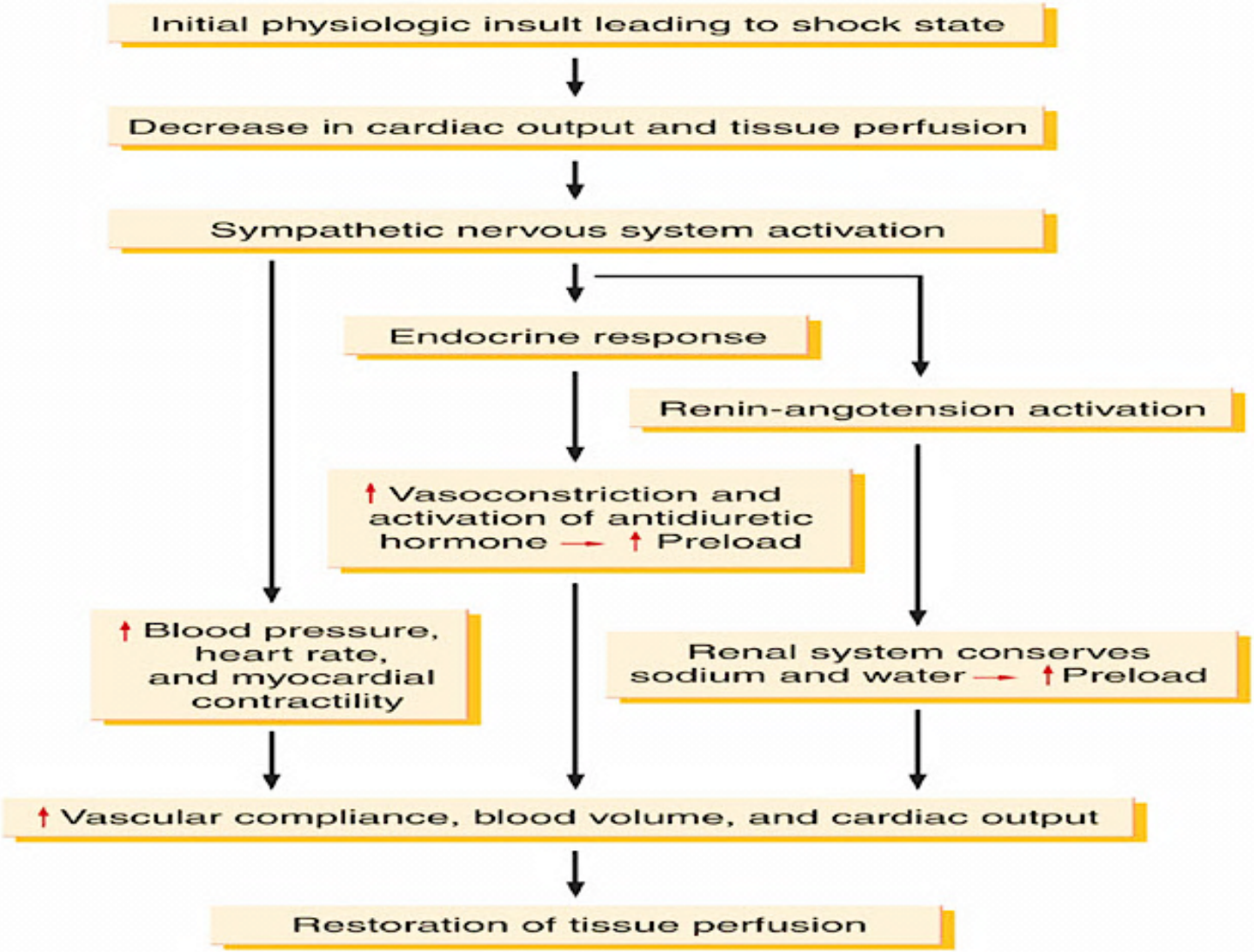
7. **Respiratory distress syndrome** occurs, due to damage of capillary endothelial cells & alveolar epithelial cells, with release of cytokines.

8. **Multiple organ failure & death.**

COMPENSATORY MECHANISMS

- ↓ BP stimulates **baroreceptors reflex** → sympathetic stimulation.
- Acidosis stimulates **chemoreceptors reflex** → sympathetic stimulation.
- **Renin-Angiotensin mechanism stimulation:**
  - Angiotensin II & III: powerful vasoconstrictors.
  - Aldosterone: Na+ retention.
- **ADH (vasopressin) stimulation:**
  - Water retention, vasoconstriction & thirst stimulation.
- **Plasma proteins synthesis**.
- **Fluid- shift mechanism**.

Compensatory Mechanisms

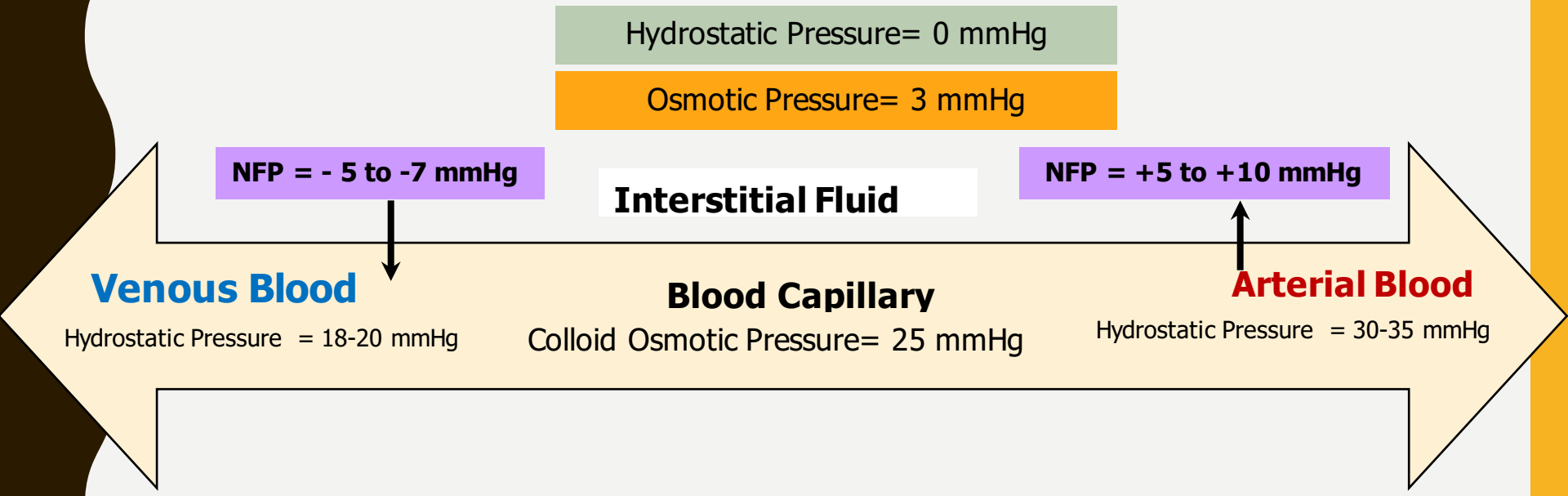


FLUID- SHIFT MECHANISM IN SHOCK

- In shock, the hydrostatic pressure decreases & oncotic pressure is constant, as a result:
  - The fluid exchange from the capillary to the extracellular space decreases.
  - The fluid return from the extracellular space to the capillary increases.

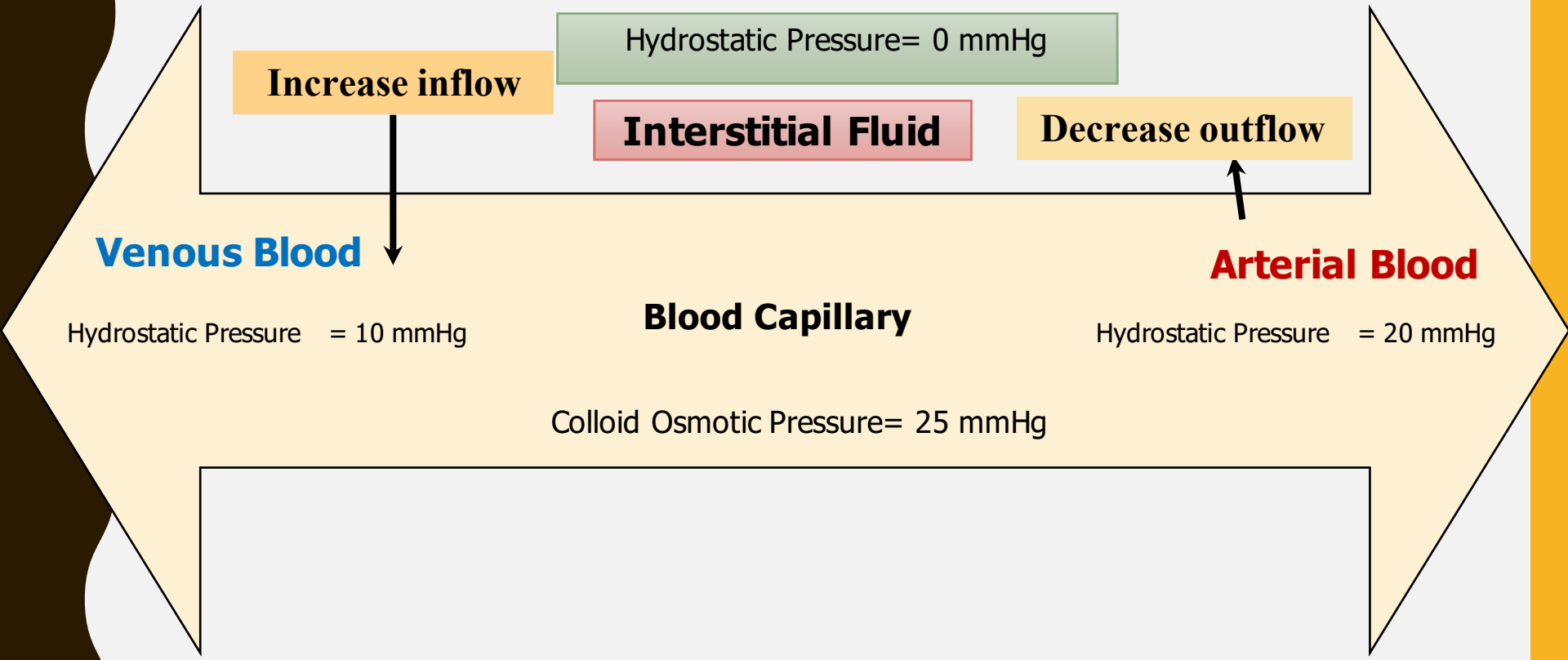
That will increase the blood volume & will increase BP helping to compensate shock situations.

IN NORMAL MICROCIRCULATION



- **At arterial end:**
  - Water moves **out** of the capillary with a NFP of +5 to +10 mmHg.
  - Hydrostatic pressure dominates at the arterial end & net fluid flows out of the circulation.
- **At venous end:**
  - Water moves into the capillary with a NFP of -5 to -7 mmHg.
  - Oncotic pressure dominates at the venous end & net fluid will flow into the bloodstream

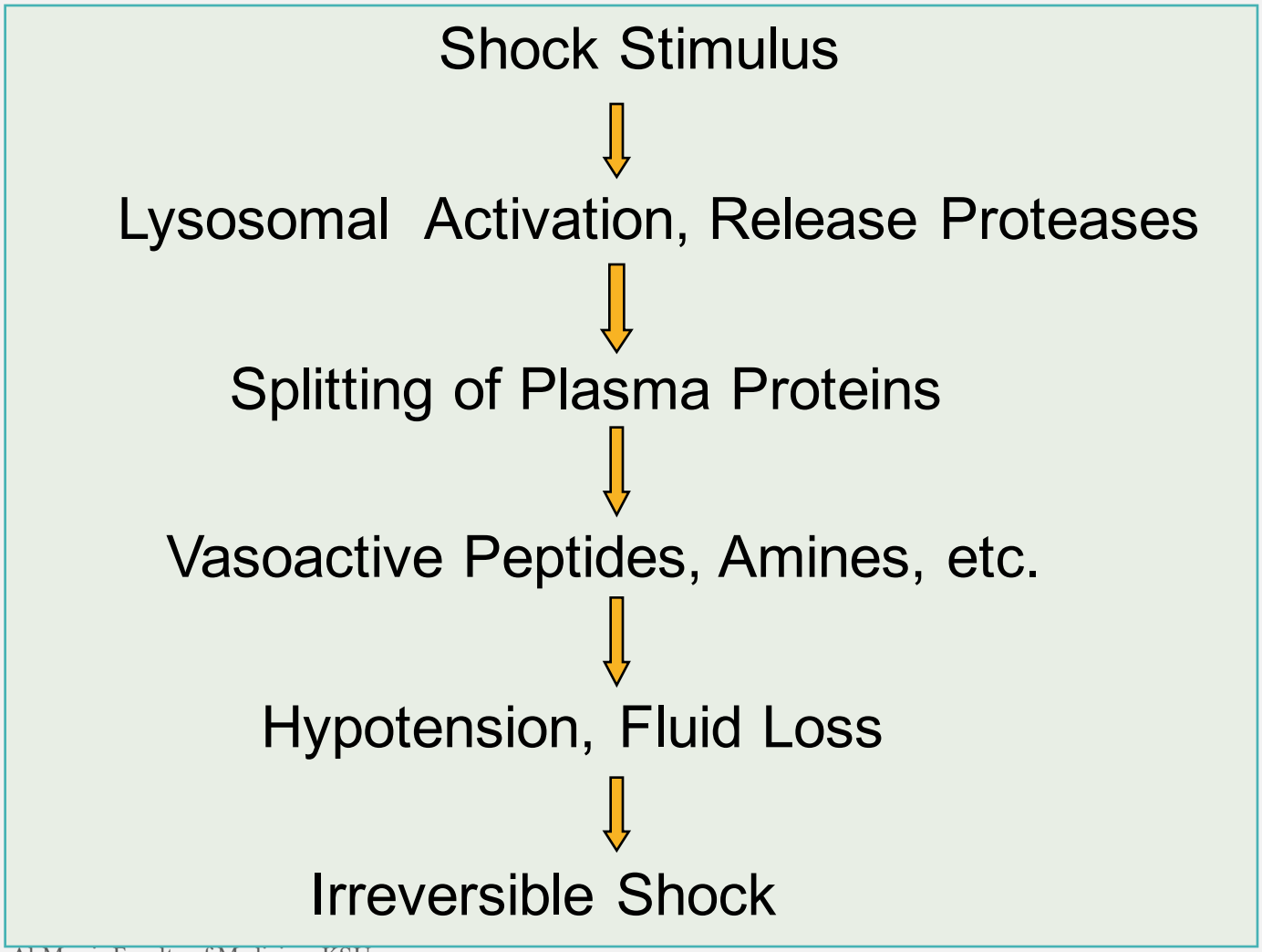
Fluid – Shift Mechanism In Shock



STAGES OF SHOCK

- **Reversible shock: (Compensated)**
  - Changes can be reversed by compensatory mechanism (neurohormonal activation) or by treatment.
  - Defense mechanisms are successful in maintaining perfusion.
  - Non-progressive.
- **Progressive:**
  - Defense mechanisms begin to fall.
  - Multi-organ failure.
- **Irreversible shock:**
  - Complete failure of compensatory mechanisms.
  - Can lead to death.

POSSIBLE MECHANISM IN DEVELOPMENT IRREVERSIBLE SHOCK



SIGNS/SYMPTOMS:
HYPOVOLEMIC SHOCK

- Hypotension… (? ≤ 85/40 mmHg)
- Tachycardia… Compensation for ↓ MAP sensed by Baroreceptors.
- Rapid, weak, & thready pulse… (? 140/min).
- Intense thirst.
- Tachypnea (rapid respiration)… Compensation for hypoxia sensed by Chemoreceptors.
- Restlessness… due to hypo-perfusion.
- Cold, pale skin… due to hypo-perfusion.
- Oliguria (low urine output)/ Anuria (no urine output).
- Blood test: Lactic acidosis.

SIGNS/SYMPTOMS:
CARDIOGENIC SHOCK

- Similar signs & symptoms to that of hypovolemic shock.
- Congestion of lungs & viscera: (CXR)
  - Interstitial pulmonary oedema.
  - Alveolar edema.
  - Cardiomegaly.

SIGNS/SYMPTOMS:
SEPTIC SHOCK

- Patient flushed & warm due to his hyperdynamic state.


Thank You