السلام عليكم ورحمة الله وبركاته

Cardiovascular Physiology

Shock

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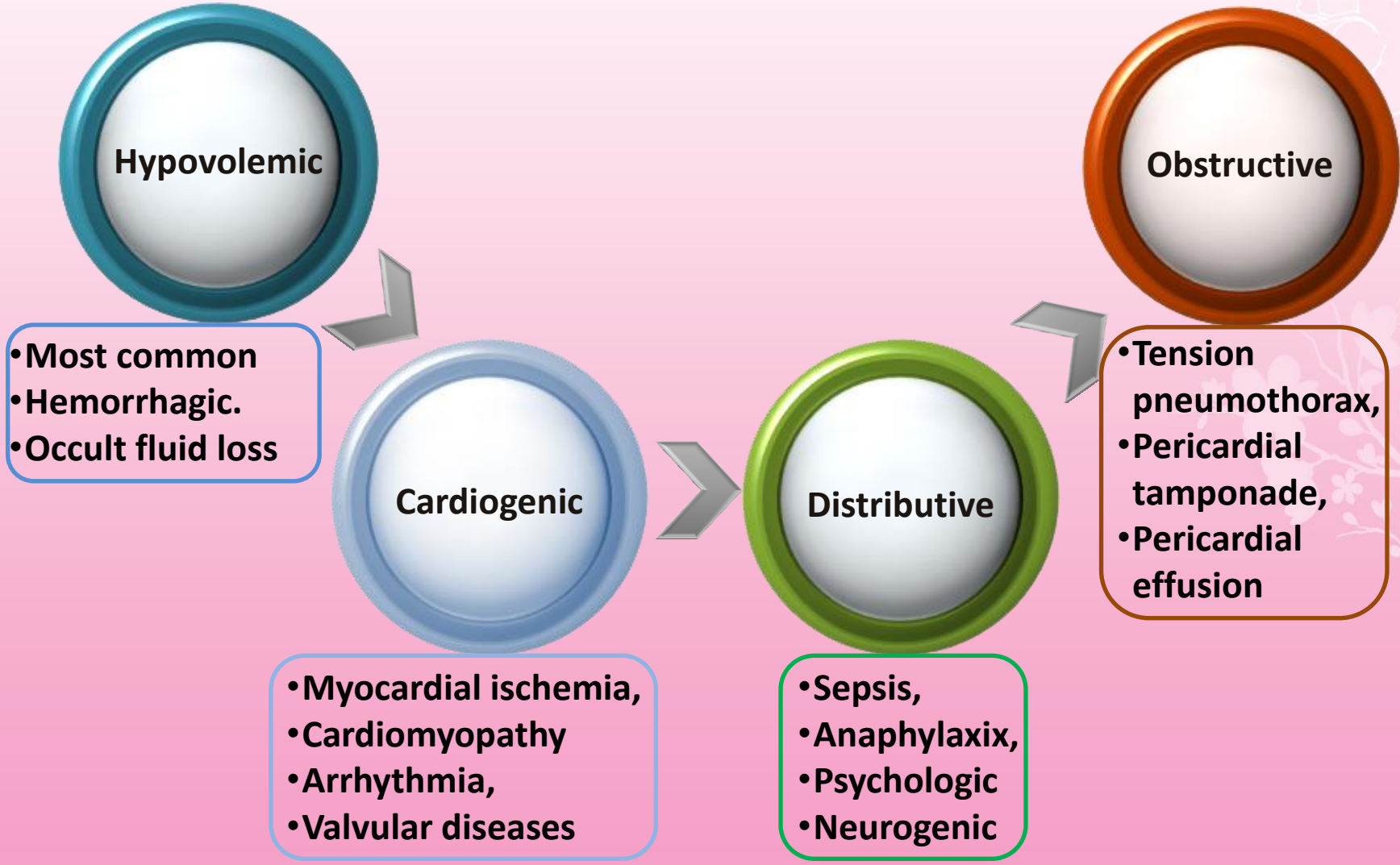
Learning Objectives

- Define Circulatory Shock
- Types of circulatory Shock
- Causes & Clinical Presentation of different types of Shock
- Pathophysiology of Shock
- Metabolic Changes & Cellular Response To Shock
- Compensatory Mechanisms of Shock
- Causes of irreversible stage of Shock

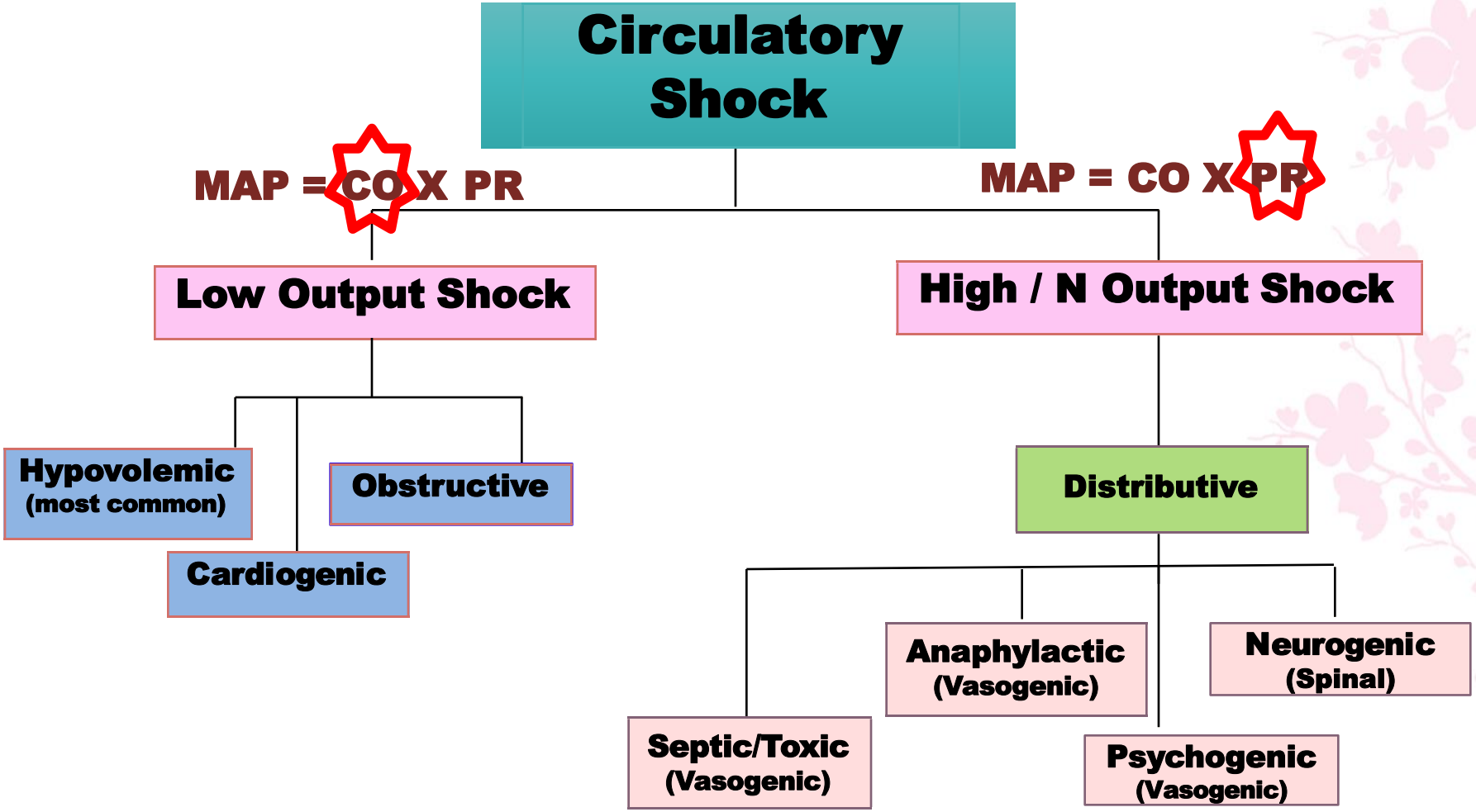
What Is Shock?

- A normal cardiac pump, circulatory system and/or volume are important to maintain blood flow to tissues.
- Shock is profound hemodyamic and metabolic disturbance characterized by failure of the circulatory system to deliver oxygen & to maintain adequate perfusion of vital organs relative to metabolic requirement.
- It is defined as Circulatory Shock.

Types Of Circulatory Shock



Types Of Circulatory Shock



Shock Syndromes

(1) Hypovolemic Shock
Blood VOLUME problem

(2) Cardiogenic Shock
Blood PUMP problem

(3) Obstructive shock.
Blood FLOW problem

(4) Distributive Shock
Blood VESSEL problem

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 of Hypovolemic Shock

- Internal fluid loss:
  - Increased capillary membrane permeability
  - Decreased plasma colloidal osmotic pressure
- External fluid loss:
  - Hemorrhage (commonest)
  - Plasma loss as in extensive burns
  - Severe vomiting, excess diarrhea, excess sweating, or massive diuresis

Clinical Presentation of Hypovolemic Shock

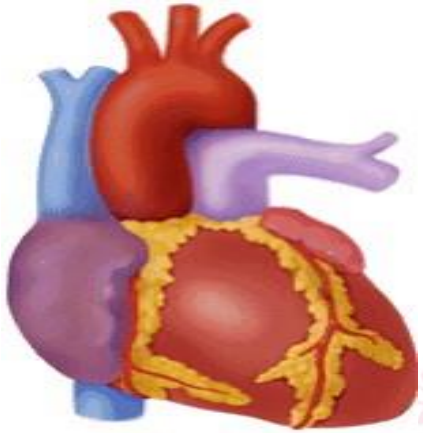
- Tachycardia… Compensation for ↓ MAP sensed by Baroreceptors.
- Tachypnea (rapid respiration)… Compensation for hypoxia sensed by Chemoreceptors.
- Rapid, weak, & thready pulse… (? 140/min).
- Hypotension… (? ≤ 85/40 mmHg)
- Cold, pale skin… due to hypo-perfusion.
- Intense thirst.
- Oliguria (low urine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

Cardiogenic Shock

Low CO due to:

MAP = CO X PR

- **Failure of myocardial pump, despite adequate ventricular filling pressure.**
- **Is associated with loss of > 40% of LV myocardial function.**
- **Mortality rate is high, 60-90%.**
- **Causes:**



- **Decreased Contractility Myocardial Infarction.. (Most common), myocarditis, cardiomypothy, congestive heart failure, post resuscitation syndrome following cardiac arrest.**
- **Sustained Arrhythmia – Heart block, ventricular tachycardia, supraventricular tachycardia, atrial fibrillation etc.)**
- **Mechanical Dysfunction – Acute valvular dysfunction, e.g. papillary muscle rupture post-MI, severe aortic stenosis, rupture of ventricular aneurysms etc.**
- **Cardiotoxicity (B blocker and calcium channel blocker overdose)**

Clinical Presentation of Cardiogenic Shock

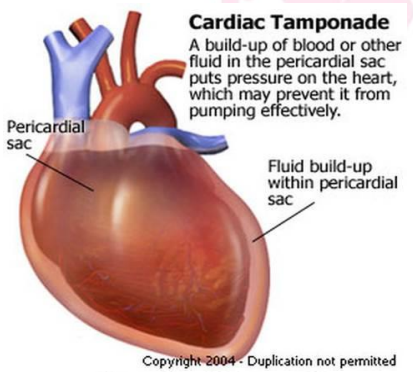
- Similar signs & symptoms to that of hypovolemic shock.
- May not show typical tachycardic response: if patient on Beta blockers, in heart block, or nodal tissue ischemia
- Mean arterial pressure below 70 mmHg compromises coronary perfusion
- Congestion of lungs & viscera: (Chest XR)
  - Interstitial pulmonary oedema.
  - Alveolar edema.
  - Cardiomegaly.

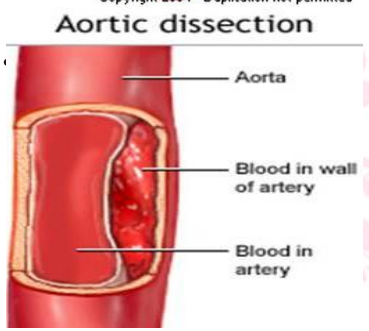
Obstructive Shock

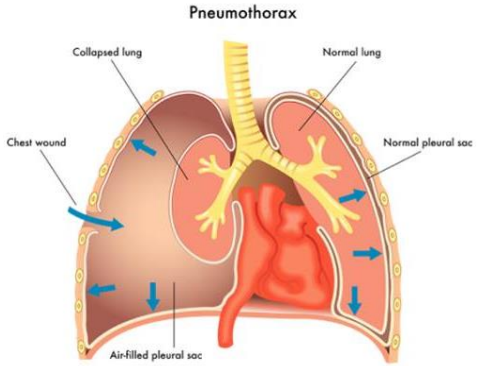
MAP = CO X PR

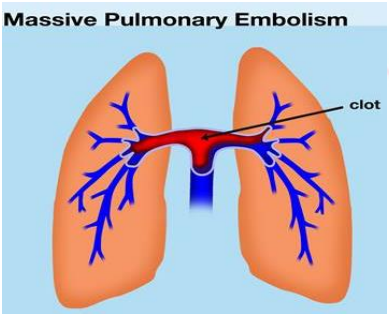
CO is reduced by obstruction to the flow of the blood, but the heart pumping capacity is well.

- **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.



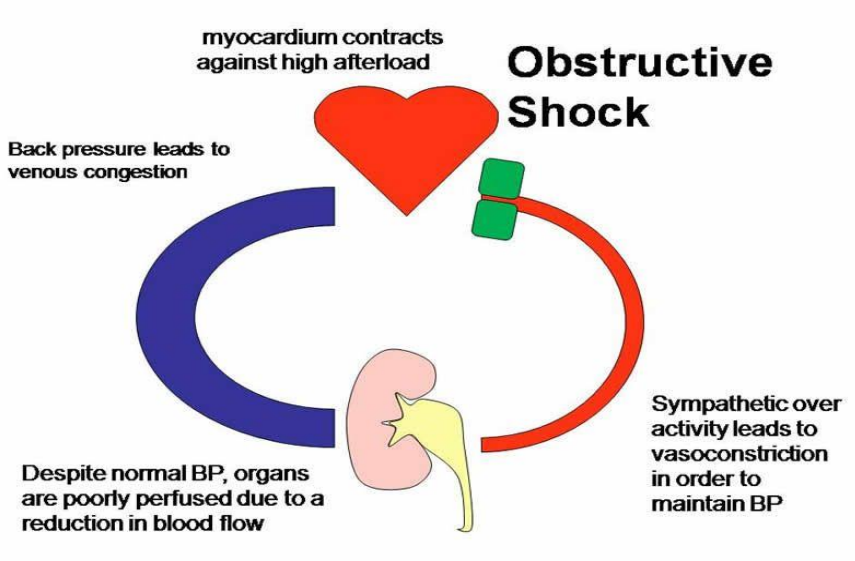






Clinical Presentation

- Jugular venous distension
- Distant heart sound in cardiac tamponade.
- Tracheal deviation & decreased or absent unilateral breath sounds in tension pneumothorax
- Chest pain, dyspnea and hemoptysis in pulmonary embolism



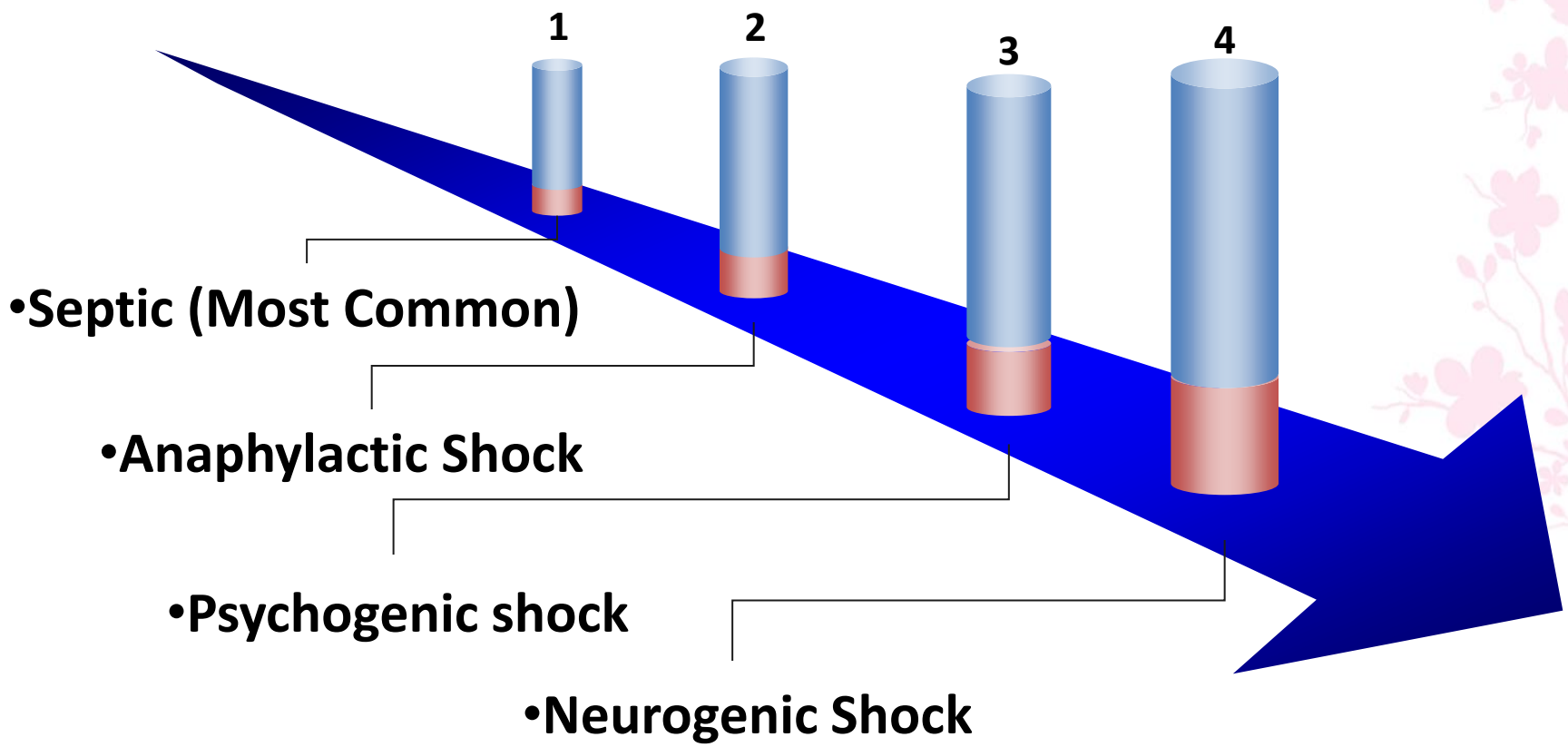
Distributive Shock/ Vasogenic (low resistance shock)

CO is Normal or Elevated

MAP = CO X PR

- Shock is due to inadequate perfusion of tissues through maldistribution of blood flow.
- Intravascular volume is maldistributed because of alterations in blood vessels, i.e. loss of vascular resistance.
- Cardiac pump & blood volume are normal but blood is not reaching the tissues (there is peripheral vasodilation due to loss of vessel tone).

Etiologies of Distributive Shock

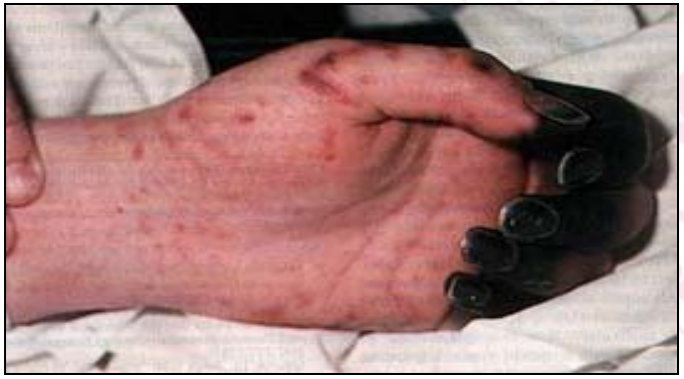


Septic Shock

Causes:

Bacterial endotoxins release inflammatory mediators that trigger endothelial injury, increase capillary permeability & peripheral vasodilatation (hyperdynamic response)

E.g:-



- Peritonitis.
- Generalized bodily infection.
- Generalized gangrenous infection.
- Infection spreading into the blood from the kidney or urinary tract.

Clinical Manifestations

- Patient is flushed & warm due to his hyperdynamic state
- Increased heart rate
- Tachypnea
- Massive vasodilation

Anaphylactic Shock

- A type of distributive shock that results from massive & generalized systemic allergic reaction to an antigen (IgE- mediated hypersensitivity).
- Basophils and mast cells releases histamine which triggers peripheral vasodilation & ↑ capillary permeability.
- It can lead to low output distributive shock.
- This hypersensitive reaction is LIFE THREATENING

Allergy to insect parts and molds

Allergy to foods and additives

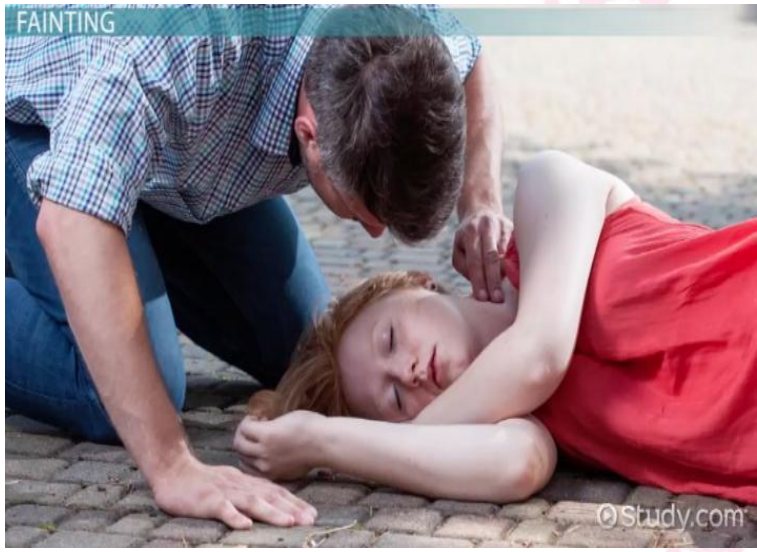
Clinical Presentation

- Circulatory collapse
  - Tachycardia, vasodilation, hypotension
- Cutaneous manifestations
  - Urticaria, erythema, pruritis, angioedema
- Respiratory compromise
  - Stridor, wheezing, bronchorrhea, respiratory distress

Psychogenic SHOCK

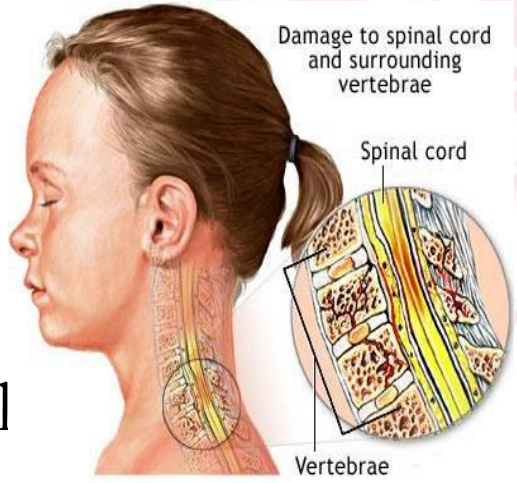
- Simple fainting (syncope.)
- Caused by stress, pain, fright or emotional crisis.
- ↓ HR.
- Sudden temporary, generalized dilation of blood vessels
- Brain becomes hypoperfused.

  → Loss of consciousness.



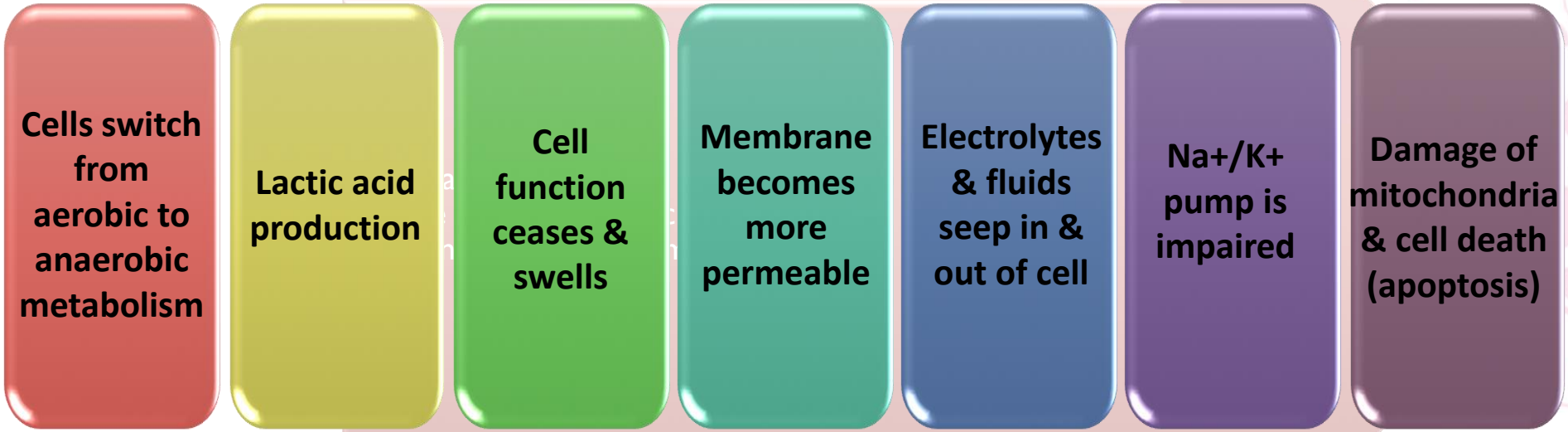
Neurogenic (Spinal) Shock

- Loss or drop in vasomotor tone (generalized peripheral vasodilation especially in the veins).
- CO is severely reduced as vascular capacity increases with venous blood pooling in peripheral veins (↓venous return) (Behaves like hypovolemic shock).
- Neurogenic is the rarest form of shock! ***Caused by***:-
  - Spinal cord injury (above C7)
  - Spinal anesthesia.
  - Deep general anesthesia.
  - Brain damage.
  - Prolonged brain ischemia that cause total inactivation of the vasomotor neurons.



Pathophysiology of shock

- Impaired tissue perfusion occurs when an imbalance develops between cellular oxygen supply and demand.



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^+& Cl^-).

→Lysosomes, nuclear membranes & mitochondrial breakdown.

Metabolic Changes & Cellular Response To Shock…Cont.

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…Cont.

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

5. **Cerebral ischemia**→ depression of VMC → vasodilation + ↓ HR
   (vasomotor center .. Sympathetic)
   Further ↓ 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.**

Stages of shock

- ***Reversible stage (Nonprogressive, Compensated Shock)***
  - In which compensatory mechanism (neurohormonal activation) & appropriate treatment help restoration of blood pressure & blood loss.
  - Defense mechanisms are successful in maintaining perfusion.

- ***Progressive***
  - Defense mechanisms begin to fall.
  - Multi-organ failure.

- ***Irreversible stage (Decompensated Shock)***
  - There is complete failure of compensatory mechanisms.
  - Series of positive feedback mechanisms take place leading to further deterioration & tissue hypoxia.
  - This stage is reached and patient may die, when blood loss is excess and not immediately replaced and proper treatment is delayed.

Compensatory Mechanisms

1- ↓ BP stimulates baroreceptors reflex → sympathetic stimulation.

2 - Acidosis stimulates chemoreceptors reflex → sympathetic stimulation.

3- Sympathetic stimulation →vasoconstriction & tachycardia. This increases TPR and hence ABP.

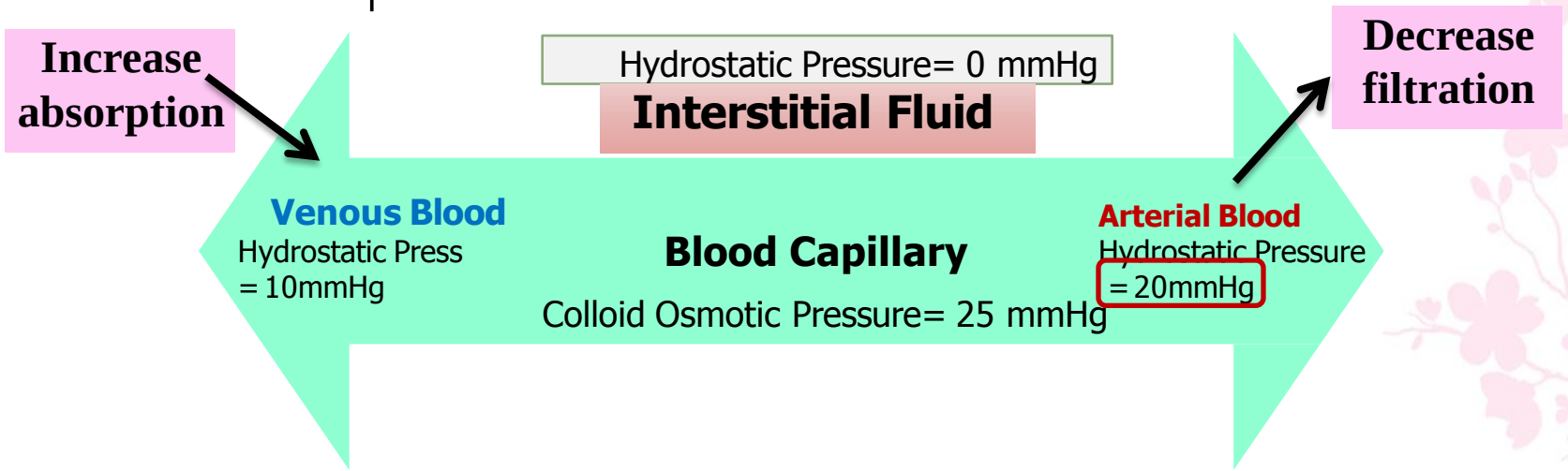
4- Tachypnea: Caused by activation of chemoreceptor reflex and sympathetic overactivity.

5- Release of vasoconstrictor factors/hormones as

- Catecholamines
- Vasopressin →vasoconstriction, increase BP & acts on renal tubules to restore fluid volume & thirst stimulation.
- Glucocorticoids to ↑ blood sugar to meet increased metabolic needs.
- Renin-angiotensin-aldosterone → angiotension II → potent vasoconstriction & releases aldosterone adrenal cortex → Na^+ & water retention (↑ intervascular volume).

Compensatory Mechanisms…Cont.

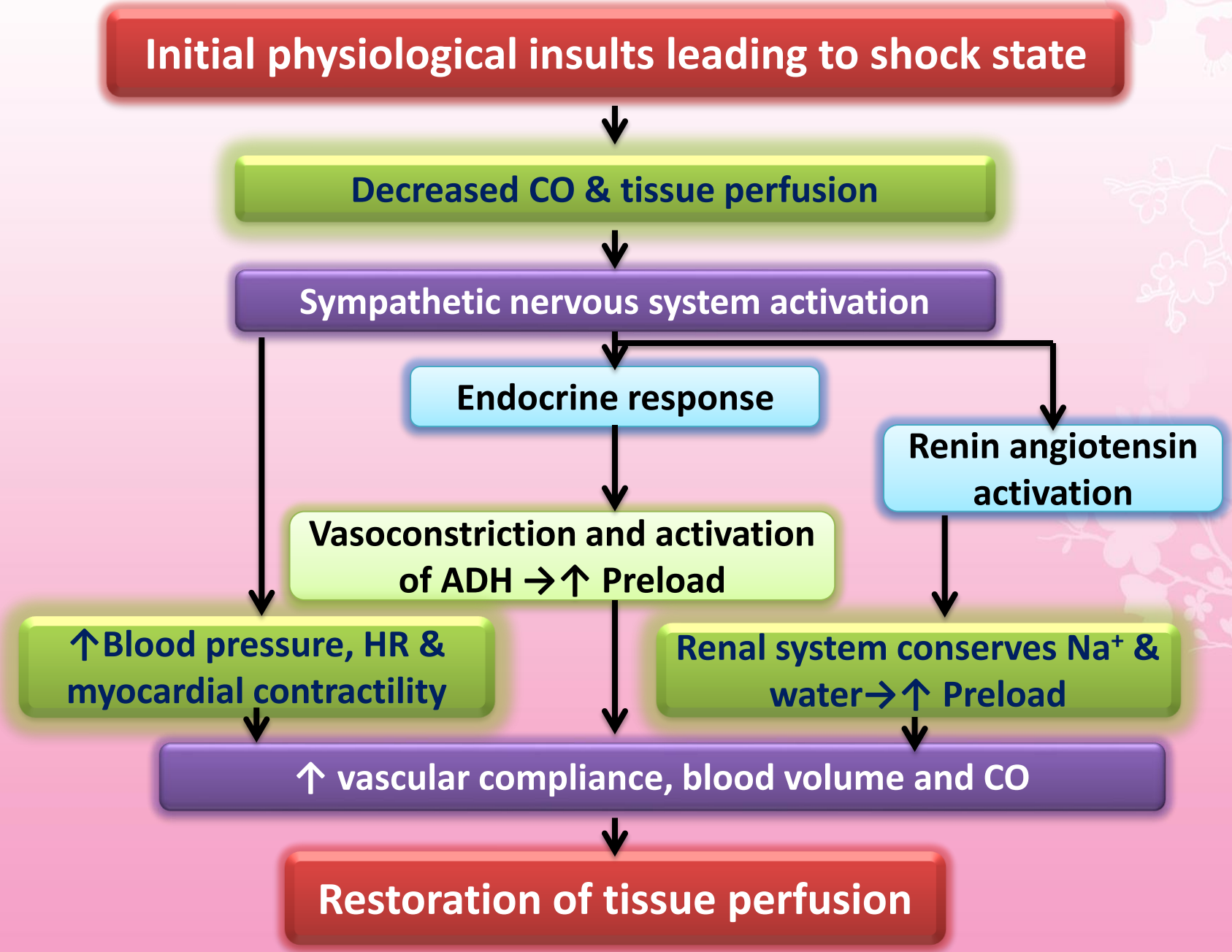
6- Increased movement of interstitial fluid into capillaries (capillary fluid shift) as a result of decreased capillary hydrostatic pressure while oncotic pressure is constant→↑blood volume & BP.



7- Increased 2,3 DPG concentration in RBCs: important to help Hb deliver more O_2 to the tissues (shift O_2 dissociation curve to the right)

8- Restoration of circulatory plasma volume, plasma proteins and RBCs mass.

Summary of Compensatory Mechanisms



Causes of irreversible stage of Shock

1. Cardiac depression.

The drop in APB leads to drop in coronary flow → (-) heart → drop CO

2. Vasomotor failure.

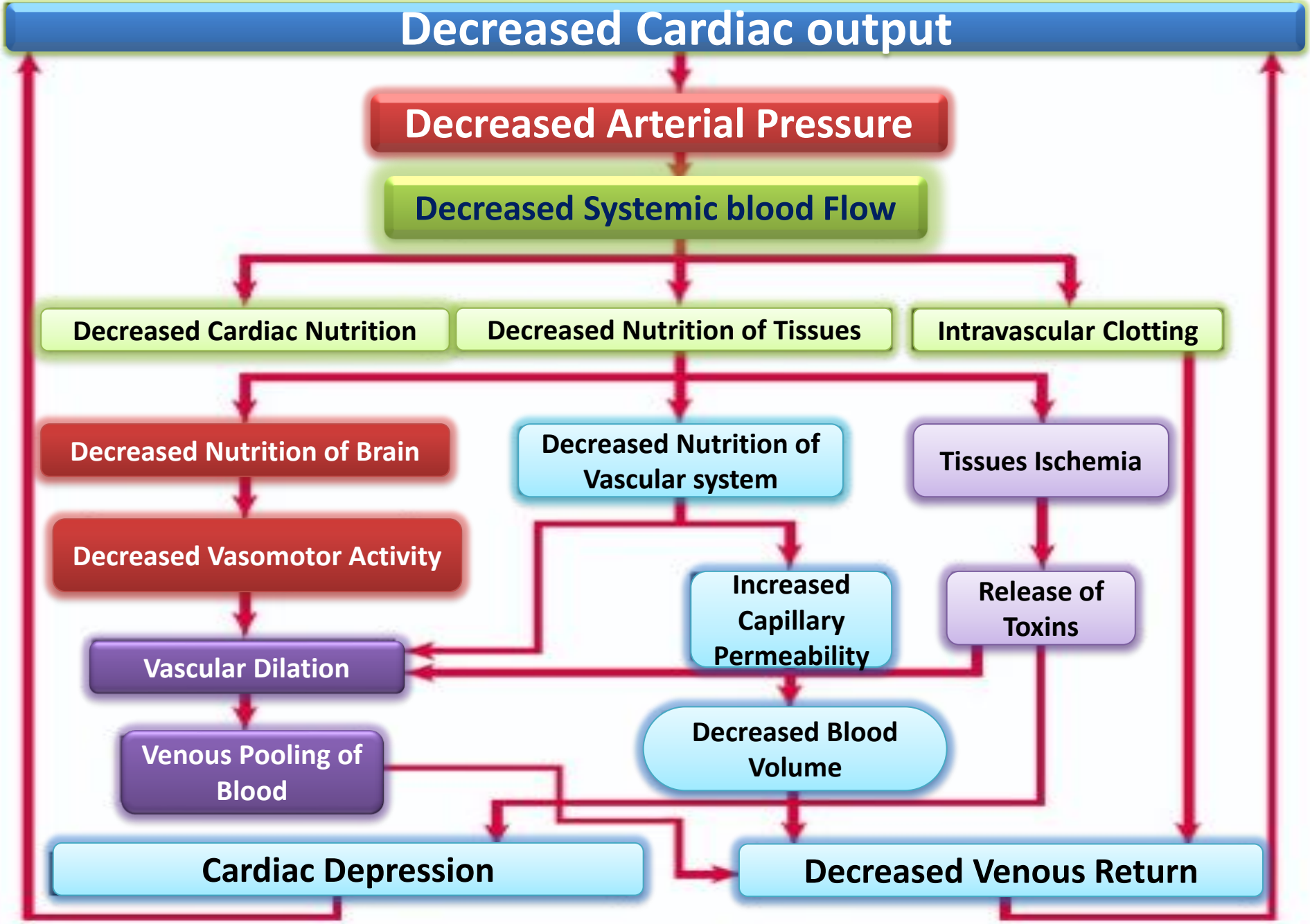
Results from depression of vasomotor center → the heart becomes depressed and CO drops.

3. Release of toxins & endotoxin: →Cardiac depression.

4. Blockage of Very Small Vessels—"Sludged Blood."

5. Increased capillary permeability due to capillary hypoxia.

6. Generalized cellular deterioration: ↓ in ATP, lysosomes rupture, ↓Na^+ and K^+ pump, cell swelling, depressed cellular metabolism of nutrients.



Different types of "positive feedback" that can lead to progression of shock


THANK YOU