Circulatory Shock

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Circulatory Shock

- Circulatory Shock - An acute, system-wide loss in blood pressure that reduces blood flow to tissues
- Tissues depend on adequate blood flow reaching the microcirculation (network of capillaries)
    - Adequate blood flow depends on arterial blood pressure
- Normal BP is determined by:
    - Cardiac output
    - Plasma volume
    - Proper arteriolar constriction

Types of Shock

- **Primary** (**Neurogenic**) **Shock**
    - Rapid drop in blood pressure reduces blood flow to the brain (**syncope** - fainting)
    - Vasodilation **+** drop in cardiac output **=** decreased BP
        - Often in response to an emotional stimulus, brain damage, or CNS depression
        - Immediate onset → passes quickly → rapid recovery
        - Results in altered nervous system output to vessels and heart
- **Cardiogenic Shock** - **Malfunction i**n heart's pumping activity
- **Hypovolemic Shock** - Rapid loss of blood from circulatory system
- **Vascular Shock** - Widespread dilation of systemic arterioles
    - **Splanchnic circulation -** vessels of abdominal viscera are especially significant in vascular shock

Cardiogenic Shock

- Heart malfunction decreased cardiac output
- Most common cause - L. Ventricle failure from myocardial infarction
    - Shock results when >40% of the myocardium is lost due to MI
- Other causes
    - Thrombosis within heart or pulmonary circulation
    - Cardiac tamponade
    - Tension pneumothorax
    - Cardiac dysrhythmia

Hypovolemic Shock

- **Hypovolemia** - Inadequate circulating blood volume
- Usually the result of massive blood loss (hemorrhagic shock)
- Many causes
    - Massive burns (3rd degree)
    - Massive skin punctures
    - Internal rupture of major vessels
    - Dehydration

Vascular Shock

- Rapid, usually systemic vasodilation = BP, venous return, cardiac output
- **Septic Shock** (most common)- Caused by circulating vasodilators (often bacterial exotoxins)
- **Toxic Shock**- Circulating toxins are NOT bacterial in origin
- **Anaphylactic Shock**- Caused by the widespread release of endogenous vasodilators by the immune system (e.g. histamine)

Compensation in Shock

- **Baroreceptors**
    - Receptors that sense changes in pressure
    - Found predominantly in the aorta/common carotid artery
    - Send information **to the cardiovascular center** in the medulla oblongata
- **Cardiovascular reflexes** adjust to normal BP
    - **In extreme cases**:
        - **CNS Ischemic Response** → sympathetic stimuli rapidly raises BP
- **Kidney**
    - Signals body (via renin, angiotensin II) to retain water
    - Kidneys are VERY sensitive to shock- high O_2 demand, maintain water balance

Therapy Helps Treat Shock

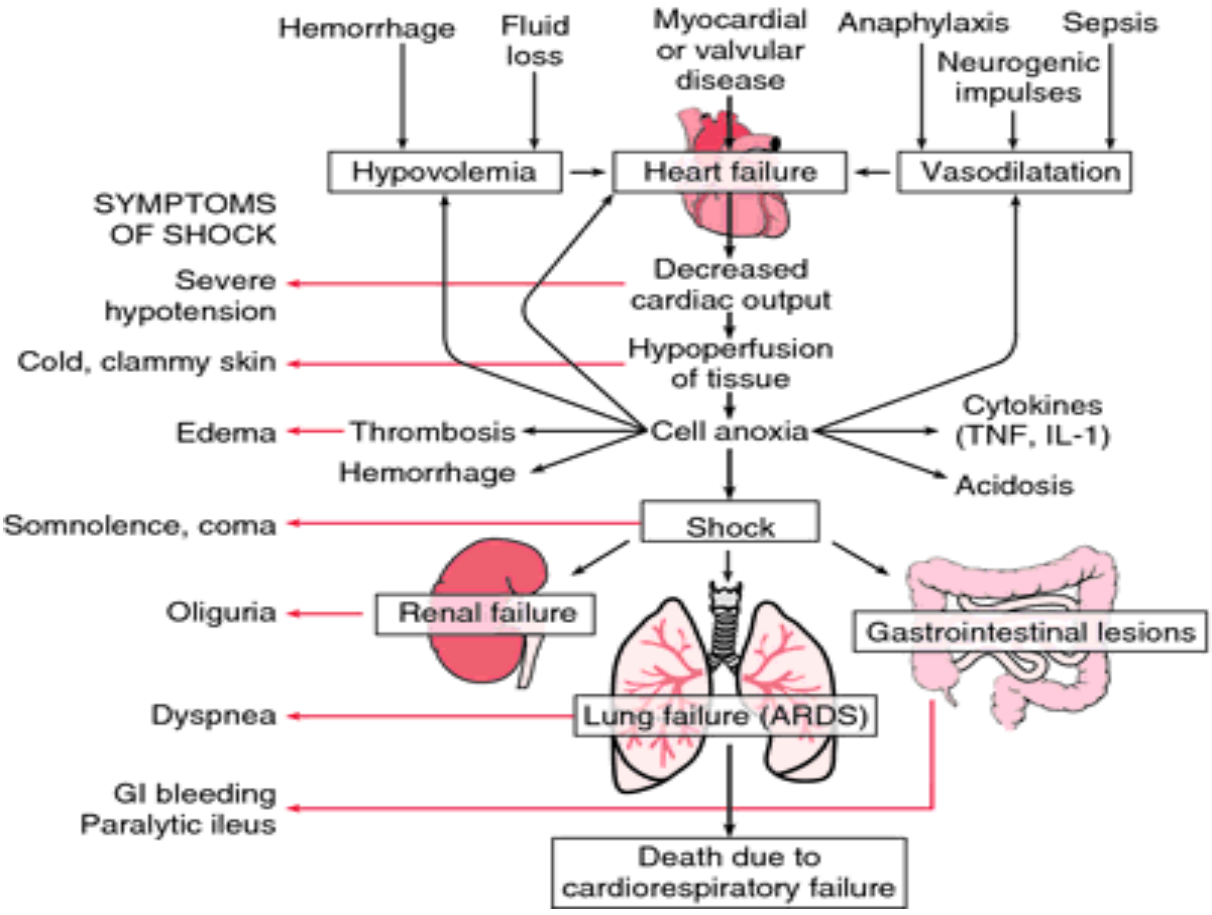
- **Primary concern -** Restoring blood flow to sensitive tissues
    - Transfusion - Compensate for loss in blood (hypovolemic shock)
    - Vasoconstriction Drugs - Increase blood flow (vascular shock)
    - Isoproterenol- increases cardiac output (cardiogenic shock)
- If possible, eliminate cause of shock (e.g. administering antibiotics for septic shock, surgery)
- Cope with secondary effects

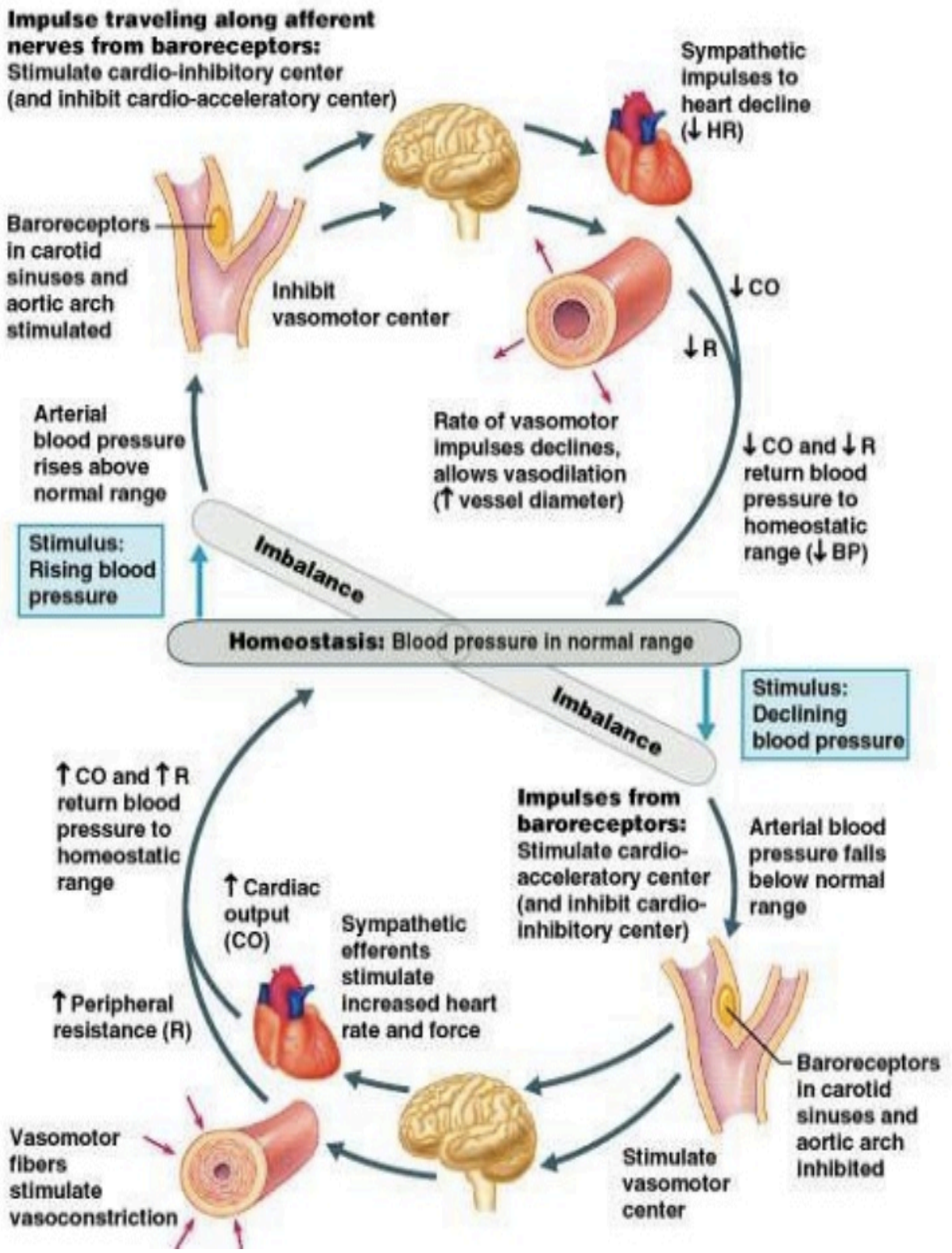
Systemic Effects of Shock

- Generalized muscular weakness
- Drop in body temperature
    - Metabolism slows → lack of nutrients
- Restlessness and confusion
- Lung Shock- highly permeable alveoli results in build up of fluid in lungs
- Anoxic damage to kidneys → acute renal failure
- Metabolic Acidosis**-** tissues switch to anaerobic respiration due to hypoxia
- GI track mucosae vulnerable to necrosis
- Heart and liver damage

Progression of Shock: 3 Stages

- **Non-Progressive Shock** - Therapy (internal or external) will usually correct the problem
- **Progressive Shock**
    - Tissue/organ damage will usually result
    - Positive feedback cycles cause progression of shock
    - Major cause- decline in functional capacity of circulatory system
    - May result in **metabolic acidosis**
- **Irreversible Shock** - Shock is acute and severe→ death will likely result even if normal BP is restored




Impulse traveling along afferent nerves from baroreceptors:
Stimulate cardio-inhibitory center
(and inhibit cardio-acceleratory center)
Sympathetic impulses to heart decline (↓ HR)
Baroreceptors in carotid sinuses and aortic arch stimulated
Inhibit vasomotor center
↓ CO
↓ R
Rate of vasomotor impulses declines, allows vasodilation (↑ vessel diameter)
↓ CO and ↓ R return blood pressure to homeostatic range (↓ BP)
Arterial blood pressure rises above normal range
Stimulus: Rising blood pressure
Imbalance
Homeostasis: Blood pressure in normal range
Imbalance
Stimulus: Declining blood pressure
↑ CO and ↑ R return blood pressure to homeostatic range
Impulses from baroreceptors:
Stimulate cardio-acceleratory center
(and inhibit cardio-inhibitory center)
Arterial blood pressure falls below normal range
↑ Cardiac output (CO)
Sympathetic efferents stimulate increased heart rate and force
↑ Peripheral resistance (R)
Baroreceptors in carotid sinuses and aortic arch inhibited
Vasomotor fibers stimulate vasoconstriction
Stimulate vasomotor center