Pathophysiology of shock

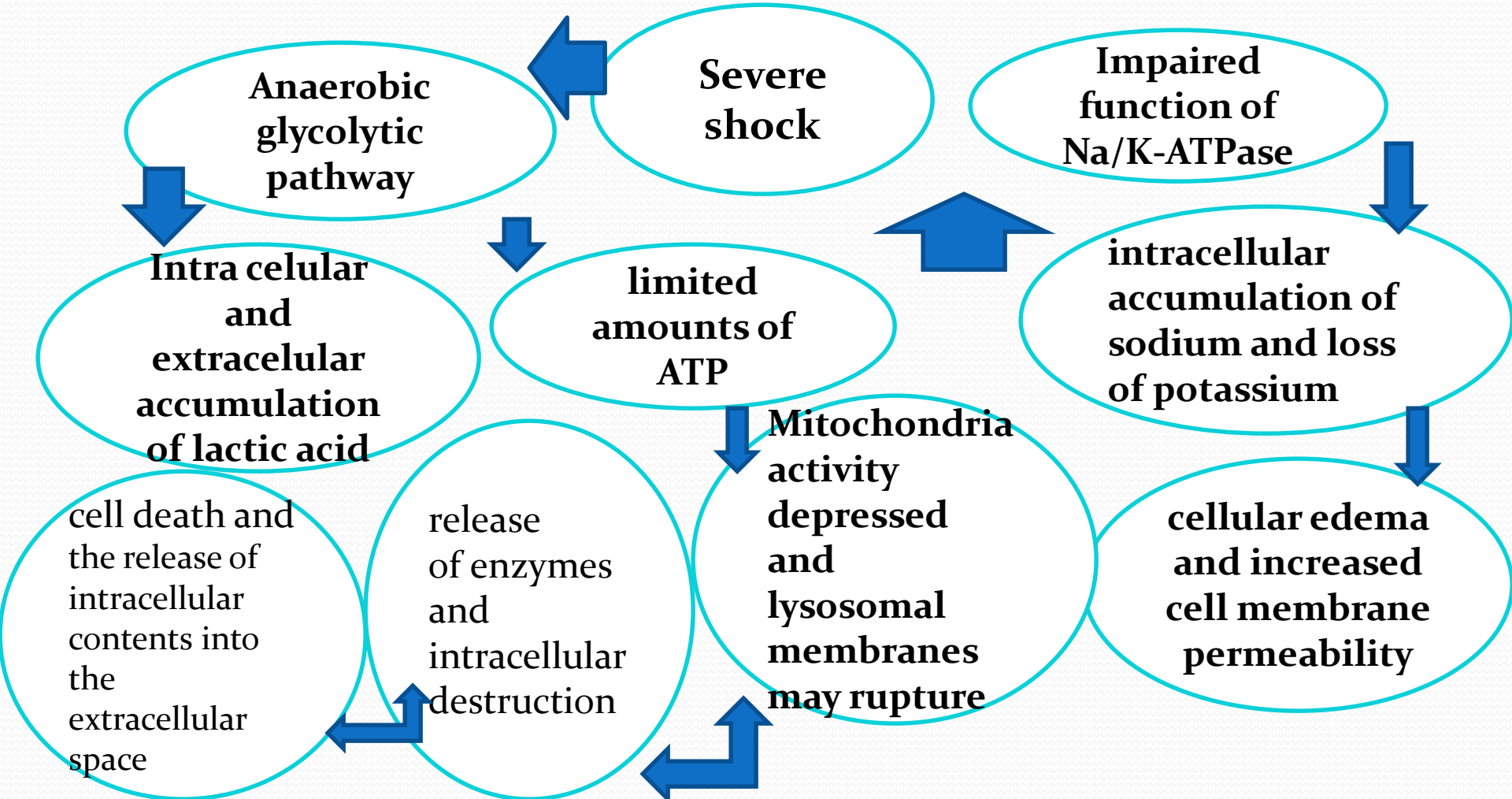
Definition of shock

Hypoperfusion of organs
and tissues, which in turn results in insufficient supply
of oxygen and nutrients for cellular function.

Shock:

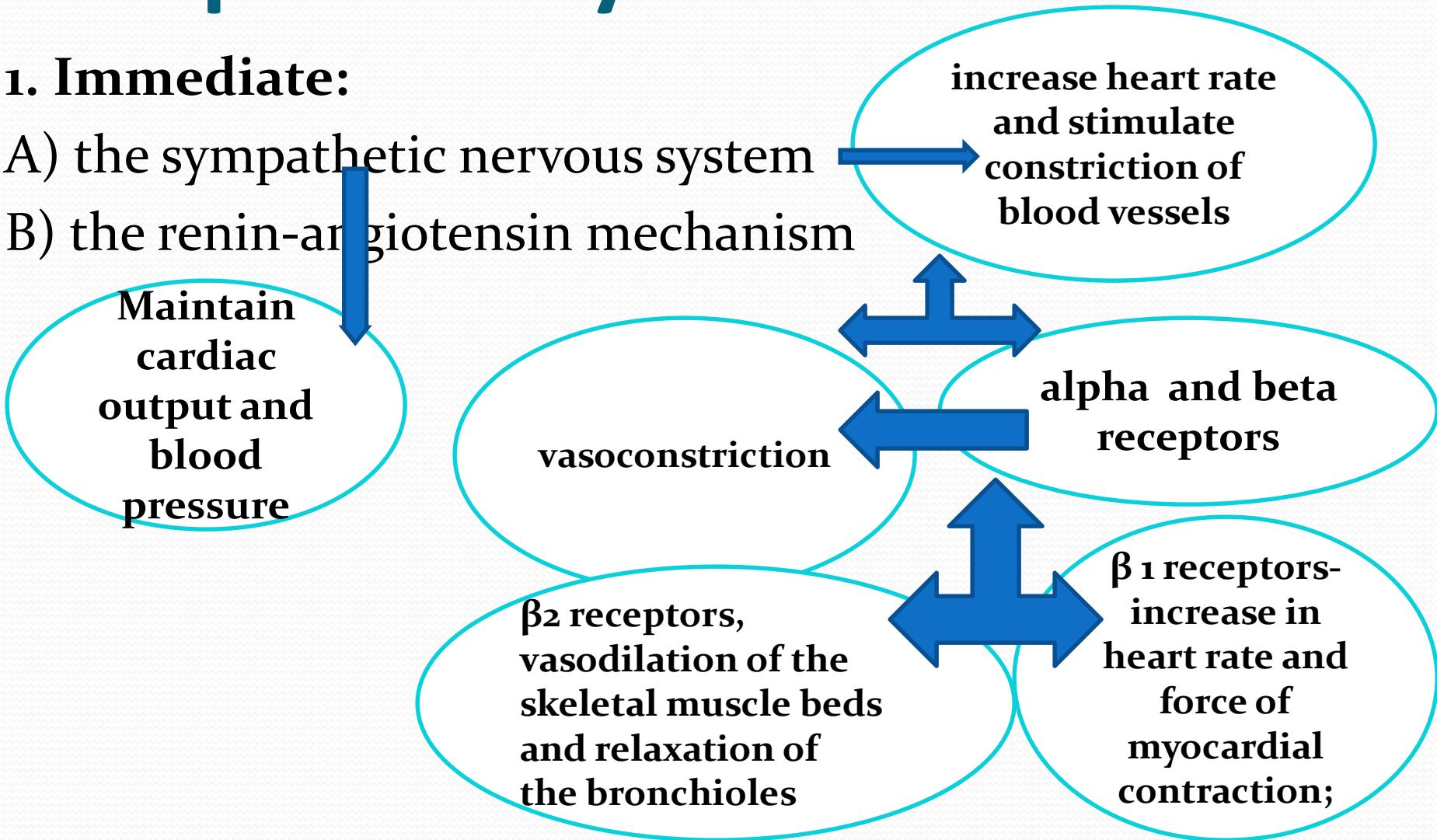
- An inadequate delivery of oxygen and substrates ⟶
- create the cellular injury, induces the production and release of inflammatory mediators ⟶
- compromise perfusion through functional and structural changes within the microvascular circulation ⟶ impaired perfusion ⟶ cellular
- injury ⟶ maldistribution of blood flow ⟶ compromising cell perfusion .

Cellular Responses:

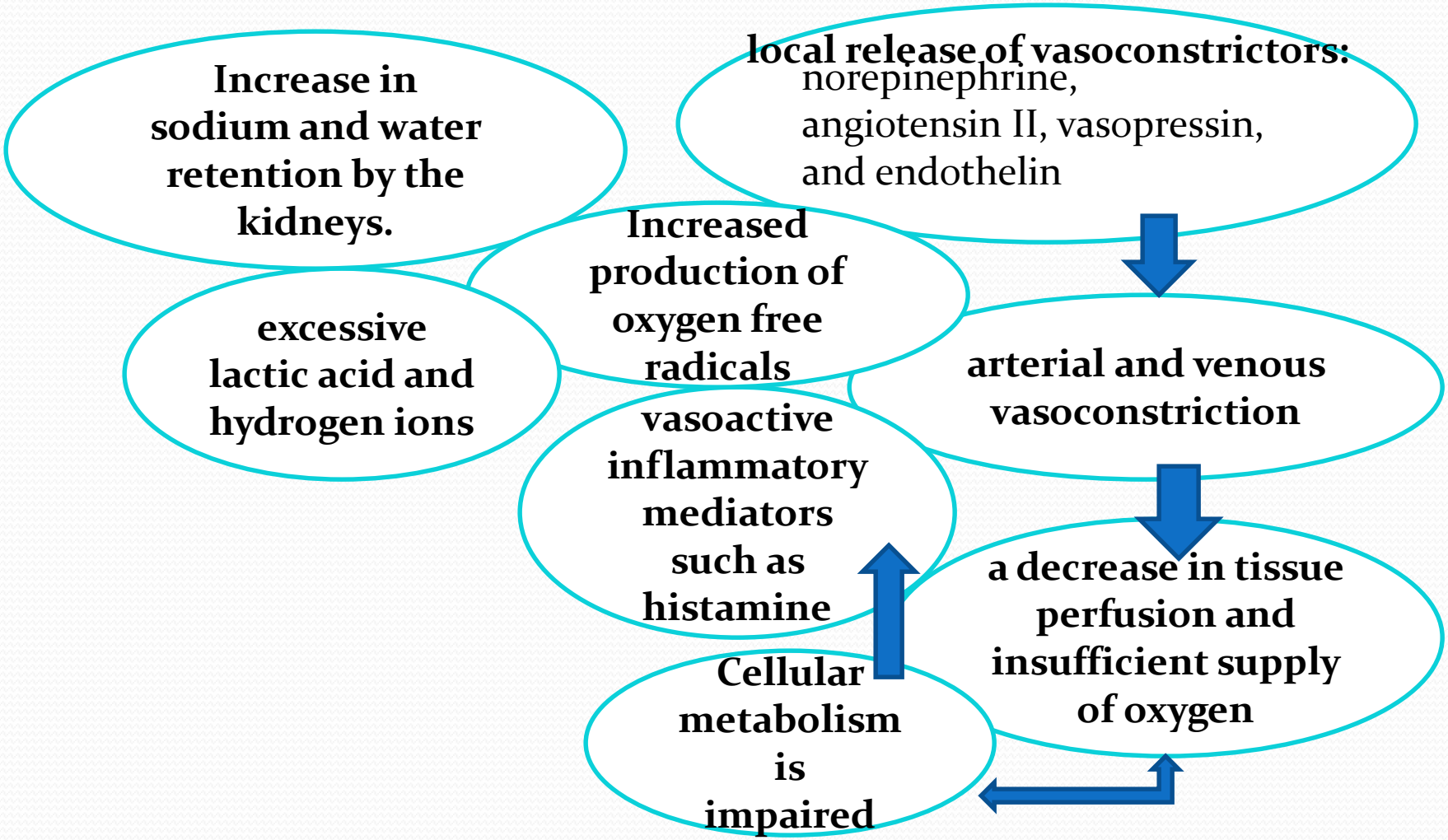


Compensatory Mechanisms:

- **1. Immediate:**
- A) the sympathetic nervous system
- B) the renin-angiotensin mechanism



renin-angiotensin mechanism:



Types of Shock:

CHART 20–1 Classification of Circulatory Shock

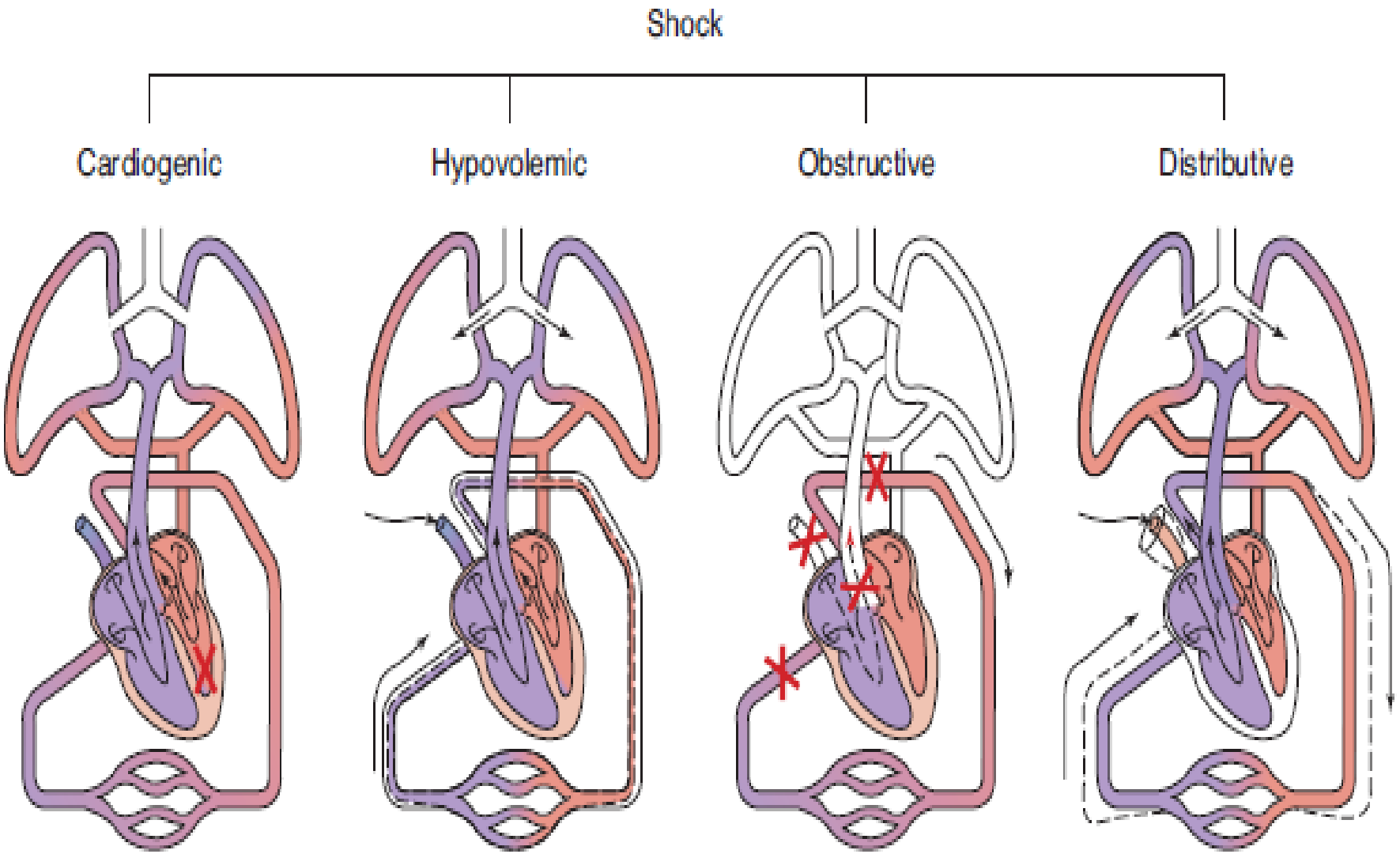
Cardiogenic
Myocardial damage (myocardial infarction, contusion)
Sustained arrhythmias
Acute valve damage, ventricular septal defect
Cardiac surgery

Hypovolemic
Loss of whole blood
Loss of plasma
Loss of extracellular fluid

Obstructive
Inability of the heart to fill properly (cardiac tamponade)
Obstruction to outflow from the heart (pulmonary embolus, cardiac myxoma, pneumothorax, or dissecting aneurysm)

Distributive
Loss of sympathetic vasomotor tone (neurogenic shock)
Presence of vasodilating substances in the blood (anaphylactic shock)
Presence of inflammatory mediators (septic shock)

Types of Shock:



Cardiogenic Shock:

Cardiogenic shock occurs when the heart fails to pump blood sufficiently to meet the body's demands

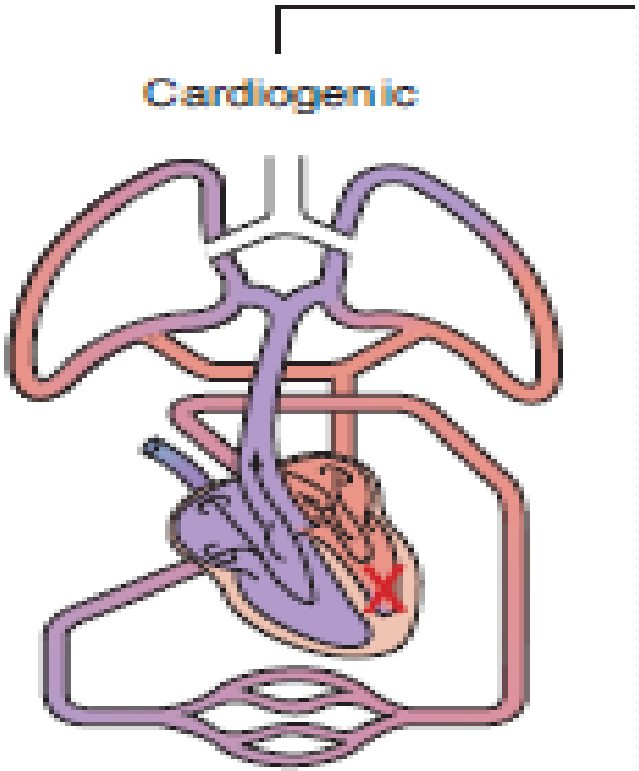
Clinically, it is defined as decreased cardiac output, hypotension, hypoperfusion, and indications of tissue hypoxia despite an adequate intravascular volume

Causes of cardiogenic shock:

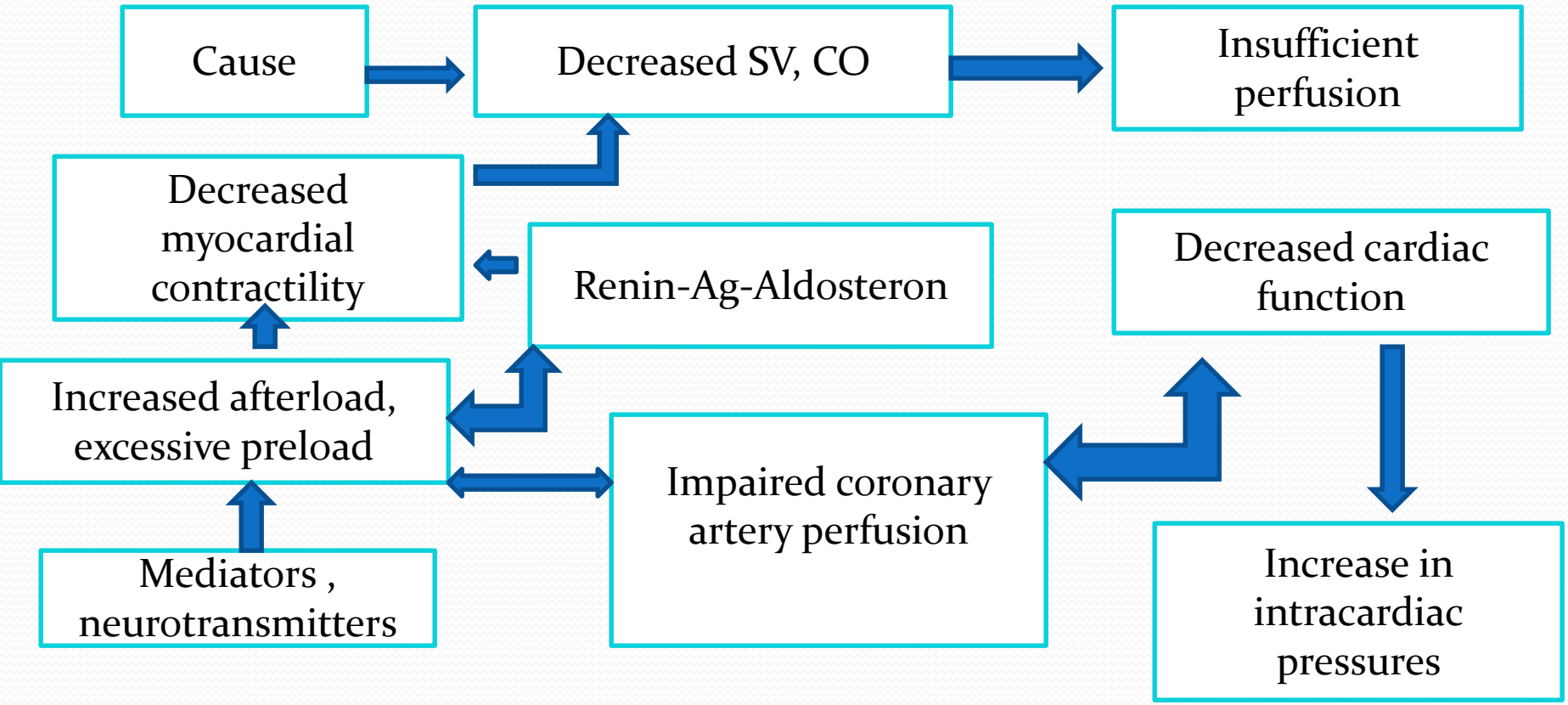
- myocardial infarction,
- myocardial contusion,
- acute mitral valve regurgitation
- papillary muscle rupture,
- sustained arrhythmias,
- severe dilated cardiomyopathy,
- cardiac surgery.

Cardiogenic Shock

- Clinically is defined as
- de creased CO, hypotension, hypoperfusion, indications of tissue hypoxia



Pathophysiology:



Clinical Features of Cardiogenic Shock

- Lips , nail beds, and skin-cyanotic ⟵ stagnation of blood flow and increased extraction of oxygen from Hb
- Urine output decreases ⟵ lower renal perfusion pressures and the increased release of aldosterone.
- Elevated preload ⟵ CVP and PCWP
- Neurologic changes (alteration in cognition or consciousness) ⟵ low cardiac output and poor cerebral perfusion

CARDIOGENIC SHOCK

Cardiogenic shock is the inability of the heart to maintain cardiac output necessary to meet body needs. Extra strain on the heart causes decreased tissue perfusion.

Causes

- Systolic dysfunction
- Diastolic dysfunction
- Arrhythmias
- Structural problems

Clinical Symptoms

- Tachycardia
- Anxiety and delirium
- Increased preload
- Pulmonary congestion
- Decreased cardiac output
- Dusky skin color
- Decreased blood pressure
- Narrow pulse pressure
- Oliguria
- Dyspnea

Hypovolemic Shock

It is characterized by diminished blood volume such that there is inadequate filling of the vascular compartment

Causes of hypovolemic shock:

- Acute loss of 15-20% of the circulating blood volume
- External loss of whole blood (hemorrrhage), plasma (severe burns), or extracellular fluid (severe dehydration or loss of gastrointestinal fluids with vomiting or diarrhea
- Internal hemorrhage
- Third –space losses

Acute bleeding or other conditions leading to decrease in blood volume

Compensatory mechanisms

Mechanisms to maintain cardiovascular function

Mechanisms to maintain blood volume

Heart

Increased heart rate and cardiac contractility

Blood vessels

Vasoconstriction of vessels in skin and nonvital organs

Hypothalamus

Stimulation of thirst

Posterior pituitary

Stimulation of ADH release

Kidney

Sodium and water retention

Decreased urine output

Liver

Constriction of veins and sinusoids with mobilization of blood stored in liver

Renin-angiotensin-aldosterone mechanism

Adrenal cortex

Release of aldosterone

Clinical features:

- Depend on severity and are related to:
- 1. Low peripheral blood flow
- 2. Excessive sympathetic stimulation
- - thirst
- -increased heart rate
- -cool and clammy skin
- -decreased arterial blood pressure
- -decreased urine output
- -changes in mentation

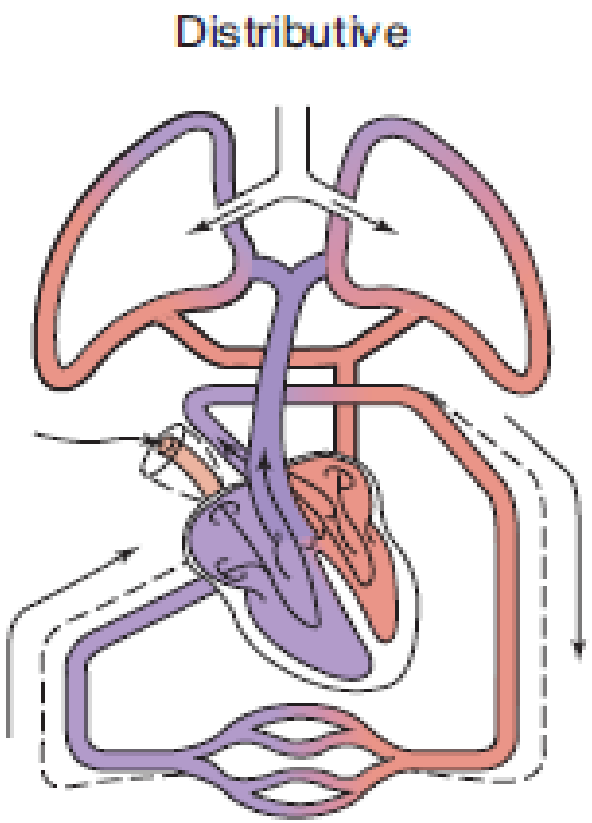
Laboratory tests:

- Hb level is normal
- Ht increases
- Low pH
- Lactic acid increases
- Metabolic acidosis
- Coagulopathy
- Hypothermia
- Circulatory failure

Pathophysiology of clinical features

- Increased heart rate
- Pulse becomes weak and thready ⟸ vasoconstriction and reduced filling of the vascular compartment;
- Thirst ⟸ decreased blood volume and increased serum osmolality;
- Arterial blood pressure decreases;
- Respiration becomes rapid and deep ⟹ compensates metabolic acidosis
- Decreased venous return to the heart and a decreased CVP
- Peripheral veins may collapse
- Cool and mottled skin sympathetic stimulation
- Urine output decreases
- Restlessness, agitation, and apprehension

Distributive Shock



-Distributive shock or vasodilatory shock is characterized by loss of blood vessel tone;

-Enlargement of the vascular compartment;

-displacement of the vascular volume away from the heart;

-central circulation

- ***-Normovolemic shock !***

Distributive Shock

- **Causes:**
- 1. a decrease in the sympathetic control of vasomotor tone
- 2. release of excessive vasodilator substances
- 3. prolonged and severe hypotension due to hemorrhage *(irreversible or late-phase hemorrhagic shock);*

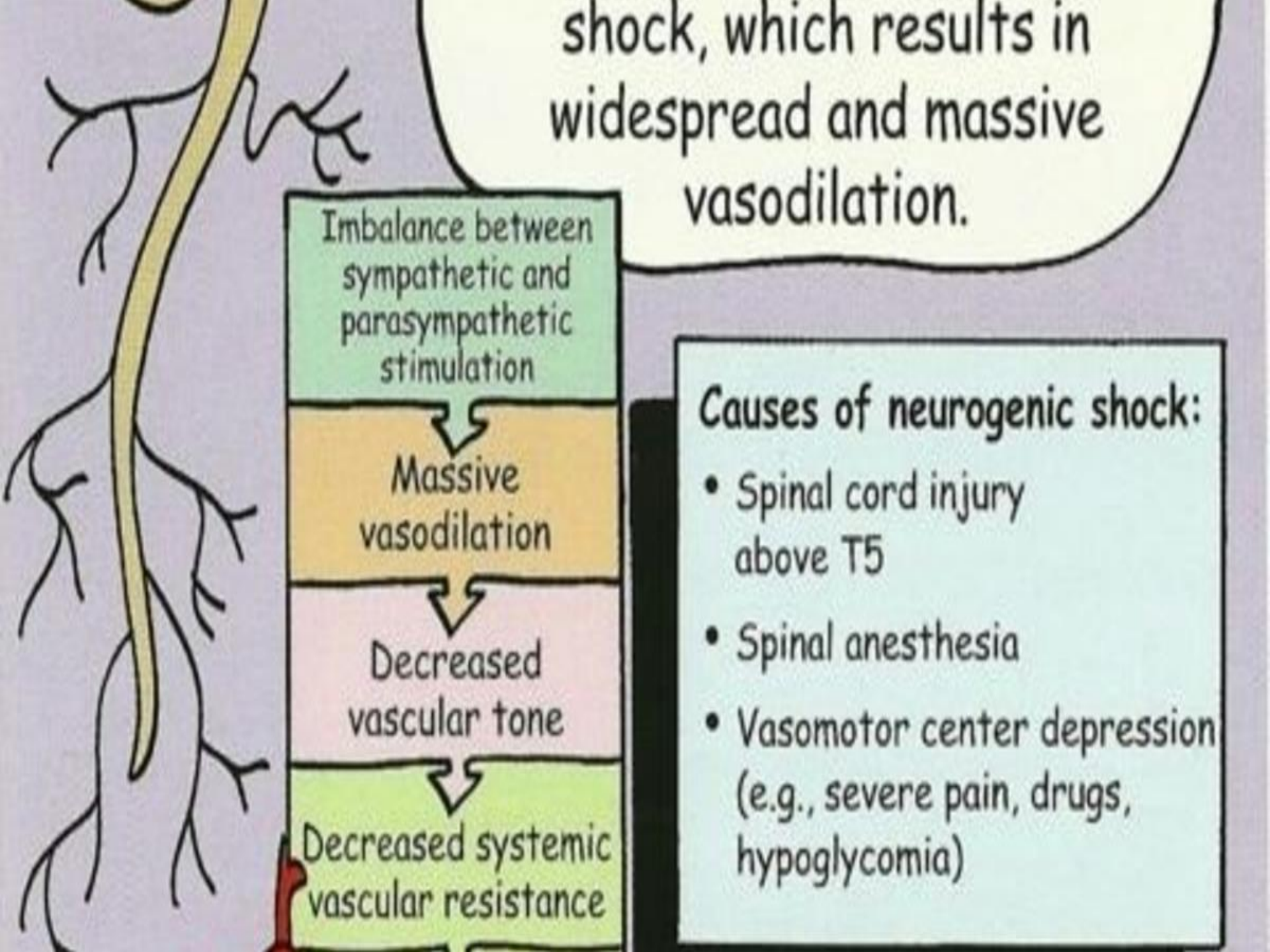
Neurogenic shock

Anaphylactic shock

Septic shock

Neurogenic shock

- Caused by decreased sympathetic control of blood vessel tone due to a defect in the vasomotor center in the brain stem or the sympathetic outflow to the blood vessels
- ***(spinal cord injury, brain injury, depressant action of drugs, general anesthesia, hypoxia, or lack of glucose);***


shock, which results in widespread and massive vasodilation.
Imbalance between sympathetic and parasympathetic stimulation
Massive vasodilation
Decreased vascular tone
Decreased systemic vascular resistance
Causes of neurogenic shock:
• Spinal cord injury above T5
• Spinal anesthesia
• Vasomotor center depression (e.g., severe pain, drugs, hypoglycomia)

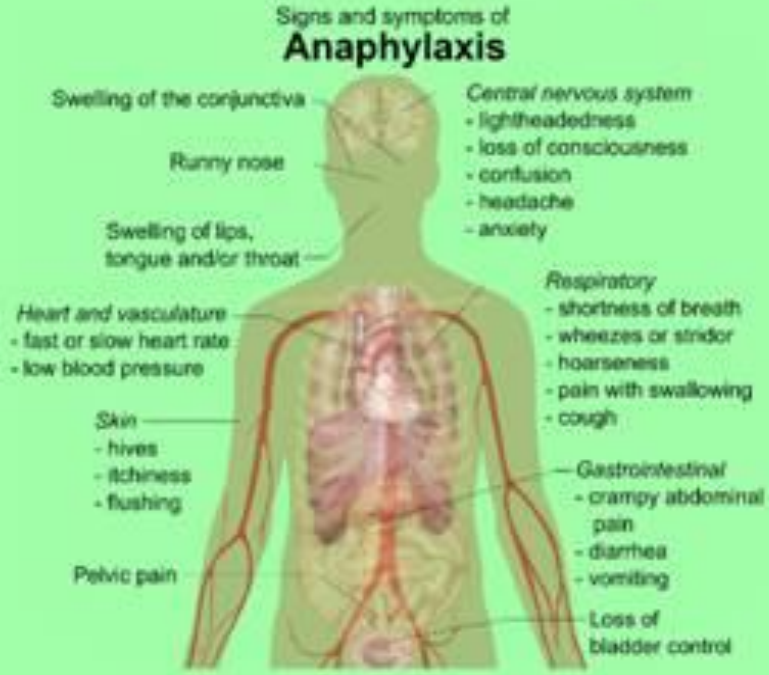
Anathylactic (Systemic) Reactions

A systemic life-threatening hypersensitivity reaction characterized by widespread edema, vascular shock, difficulty breathing

Systemic Anaphylaxis

- **Aetiology:**
- — injection of an antigen;
- —insect sting;
- — absorption across the epithelial surface skin or gastrointestinal mucousa

Anaphylactic Shock



Causes:
-Food
-Medication (antibiotics)
-Venom from animals (bugs, snakes, etc.)
-Allergic reactions

Treatment:
-EpiPen (Epinephrine)
-Histamine (pharmaceutical drug)
-911

How to Prevent it:
The most common cause of this kind of shock are allergic reactions. To counteract the severity of the reaction in the future, immunotherapy is the way to go. The person is gradually vaccinated with progressively larger doses of the allergen, small enough so that the body's immune system can counteract it on its own, conditioning the body.

Glogster by Nick Termini and Zack Assenmacher

Manifestations:

- Itching
- Hives
- Skin erythema
- Laryngeal edema
- Bronchospasm
- Respiratory distress
- Vomiting
- Abdominal cramps
- Diarreea
- Shock
- Die

Septic shock

The most common type of vasodilatory shock that is associated with severe infection and the systemic response to infection

Currently defined:

As suspected or proven infection, plus a systemic inflammatory response (*e.g.* fever, tachycardia, tachypnea, and elevated white blood cell count, altered mental state, and hyperglycemia in the absence of diabetes.

Severe sepsis-as sepsis with organ dysfunction(e.g. hypotension, hypoxemia, oliguria, metabolic acidosis, thrombocytopenia, or obtundation)

Septic shock- as severe sepsis with hypotension, despite fluid resuscitation

The Sepsis Continuum

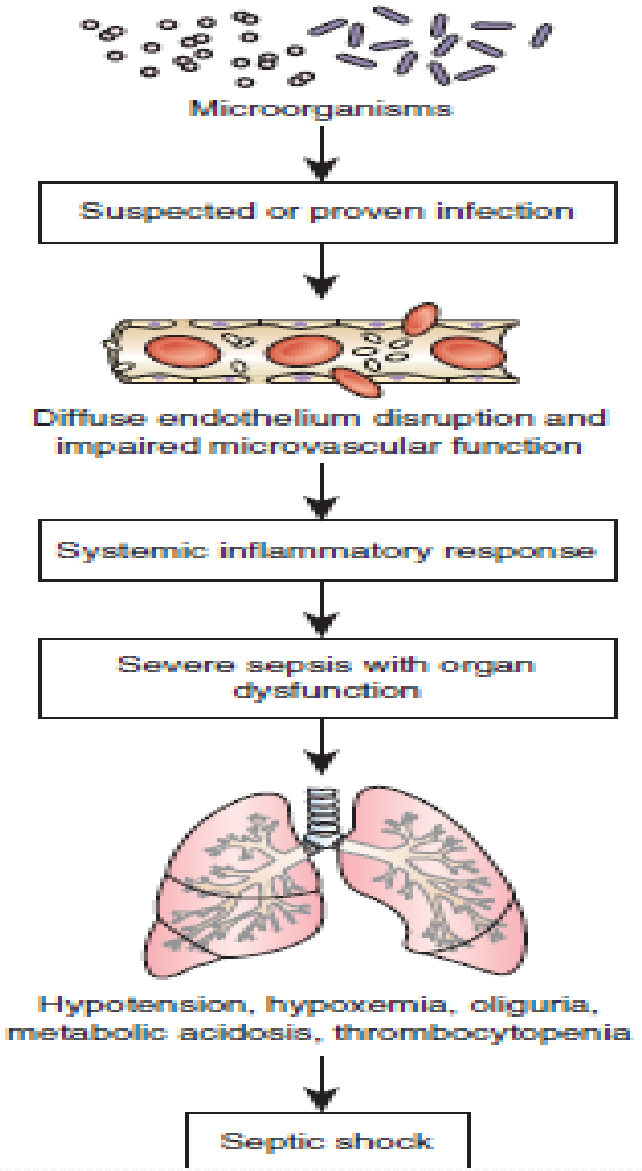
- A clinical response arising from a nonspecific insult, with ≥2 of the following:
  - T >38°C or <36°C
  - HR >90 beats/min
  - RR >20/min
  - WBC >12,000/mm^3 or <4,000/mm^3 or >10% bands

SIRS with a presumed or confirmed infectious process

Sepsis with organ failure

Refractory hypotension

Sepsis and septic shock



- 1. Cellular activation
- 2. Release of cytokines
- 3. Recruitment of neutrophils and monocytes
- 4. Involvement of neuroendocrine reflexes
- 5. Activation of compliment, coagulation , and fibrinolitic systems

Manifestations:

- Arterial hypotension;
- Warm and flushed skin
- Hypovolemia due to arterial ,venous dilatation and leakage of plasma into the interstitial spaces
- Abrupt changes in cognition or behavior due to reduced cerebral blood flow
- Fever,
- Increased leukocytes
- Metabolic acidosis

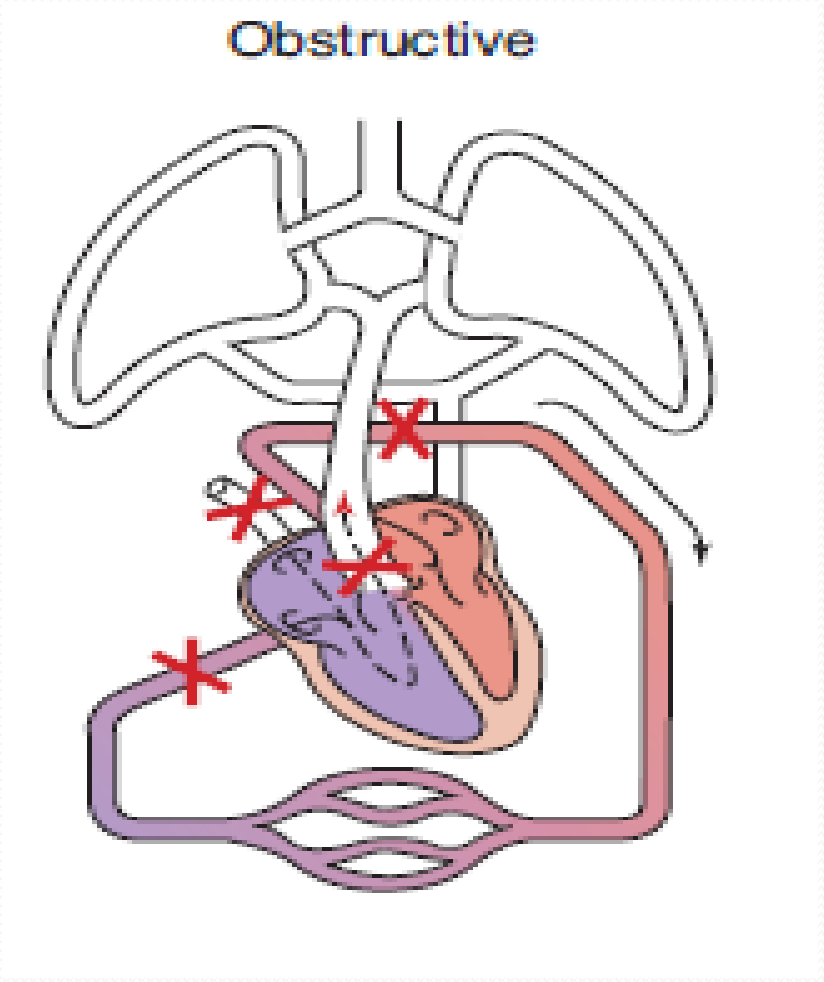
Obstructive shock

Circulatory shock that results from mechanical obstruction of the flow of blood through the central circulation (great veins, heart, or lungs)

Obstructive shock

- **Causes:**

- 1. dissecting aortic aneurysm
- 2. cardiac tamponade
- 3. pneumothorax
- 4. atrial myxoma
- 5. evisceration of abdominal contents into the thoracic cavity due to ruptured hemidiaphragm
- 6. **pulmonary embolism**



Obstructive shock

Pathogenesis

- Elevated right heart pressure due to impaired right ventricular function
- Pressures are increased despite impaired venous return to the heart

Manifestations

- Elevation of CVP
- Jugular vein distention

Complications of shock:

- 1. Acute Lung Injury/Acute Respiratory Distress Syndrome
- 2. Acute Renal Failure
- 3. Gastrointestinal Complications
- 4. Disseminated Intravascular Coagulation
- 5. Multiple Organ Dysfunction Syndrome

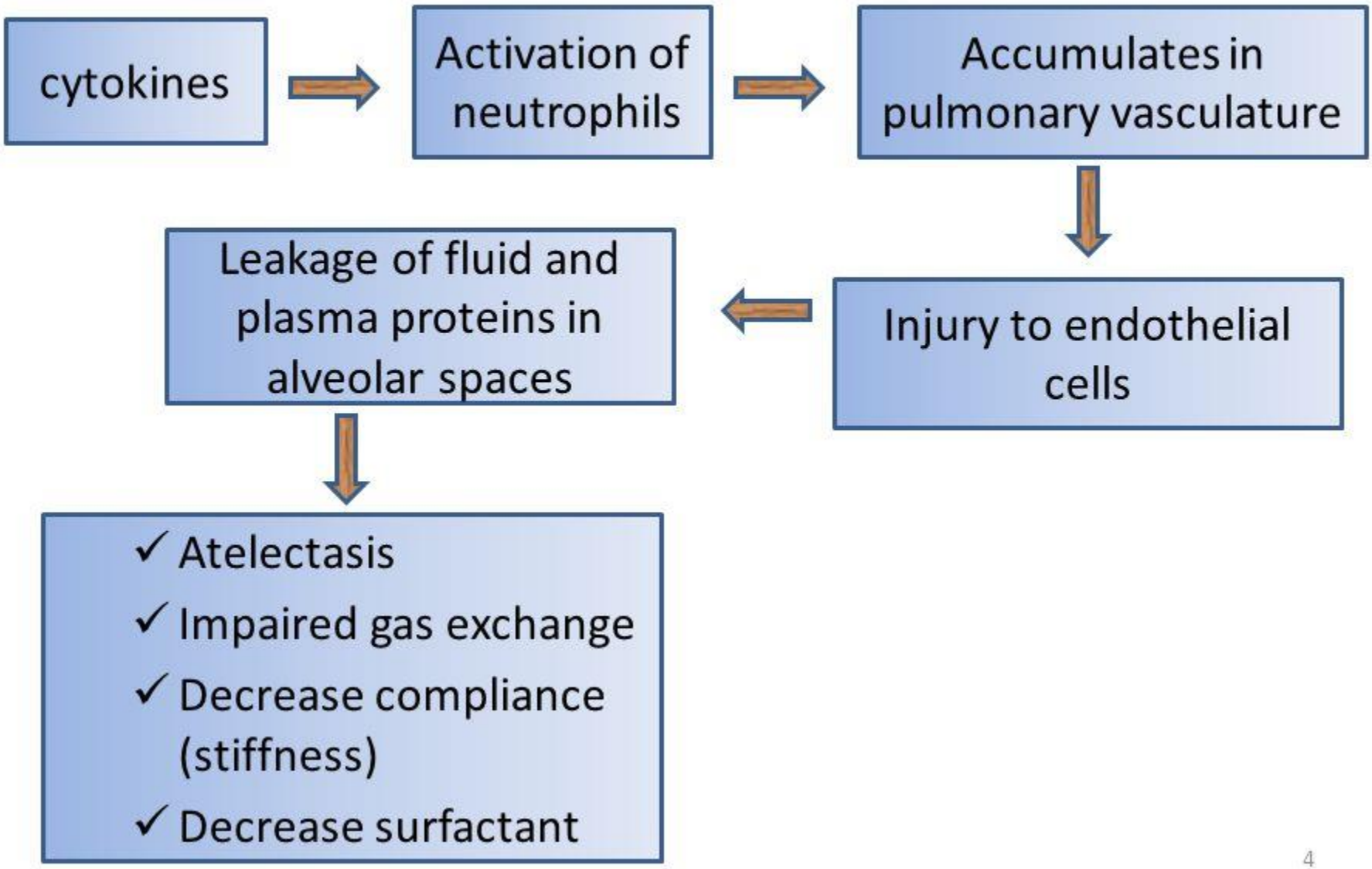
Acute Lung Injury/Acute Respiratory Distress

It is a potentially lethal form of pulmonary injury that may be either the cause or result of shock

Acute Lung Injury/Acute Respiratory Distress

- **Rapid onset of profound dyspneea** that occurs 12 to 48 hours
- **Respiratory rate and effort of breathing increases**;'
- **Profound hypoxemia** (impaired matching of ventilation and perfusion, greatly reduced diffusion of blood gases across the thickened alveolar membranes
-

Pathophysiology of (ARDS)



ARDS - PATHOGENESIS

Insult (direct or indirect)

↓

Activation of inflammatory cells & mediators

↓

Damage to alveolar capillary membrane

↓

Increased permeability of alveolar capillary membrane

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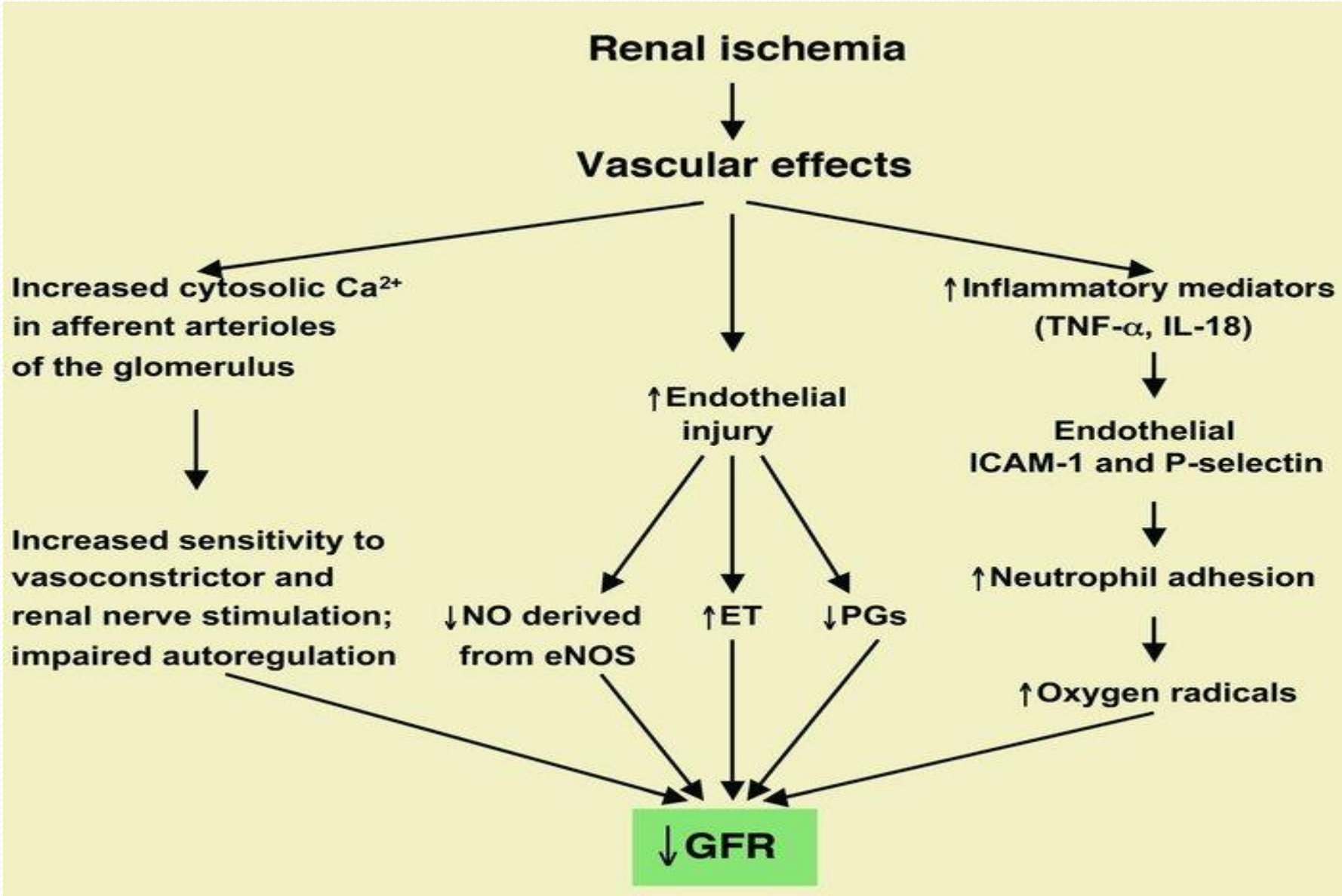
Influx of protein rich edema fluid and inflammatory cells into air spaces

Dysfunction of surfactant

Acute renal failure:

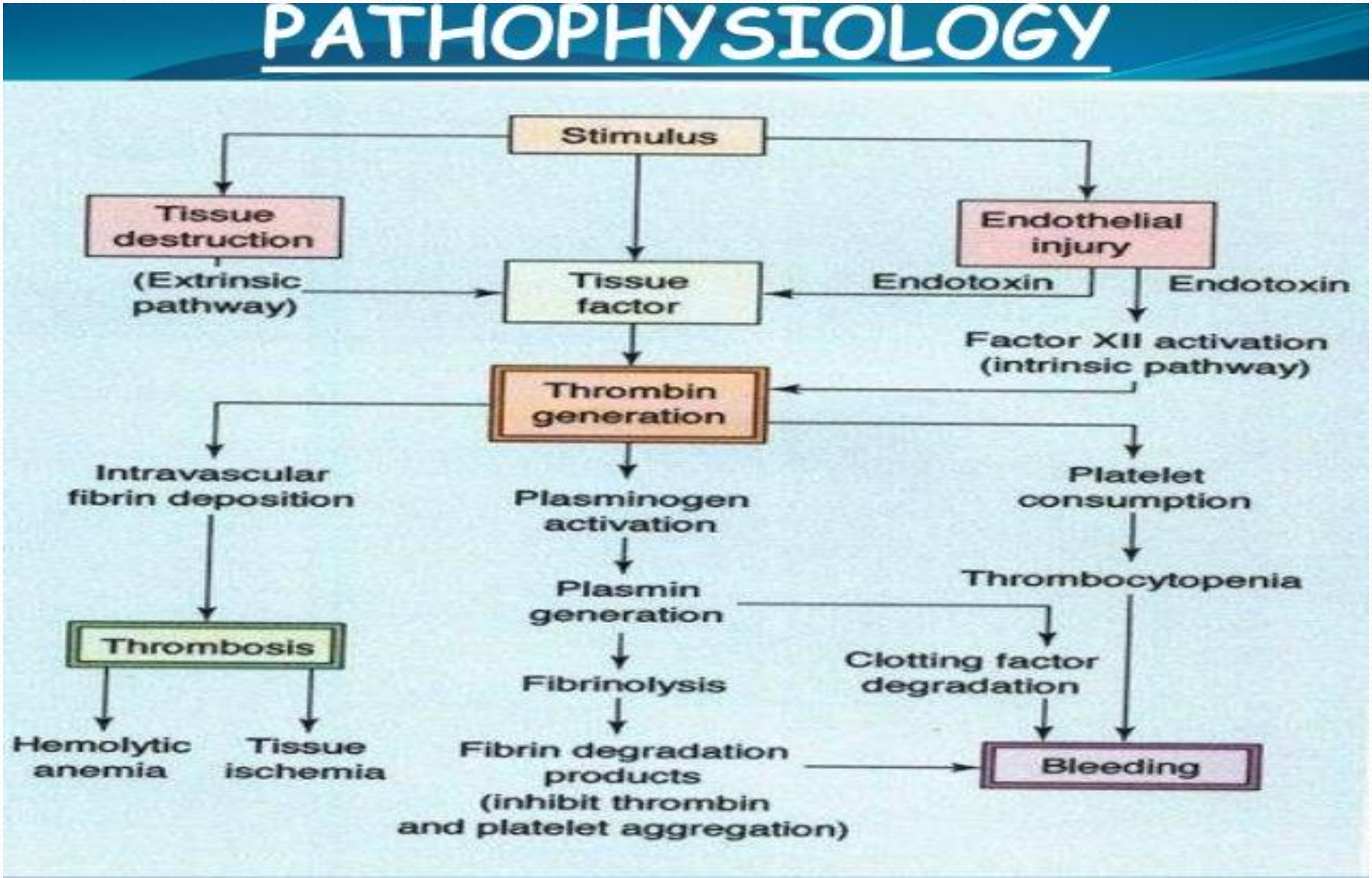


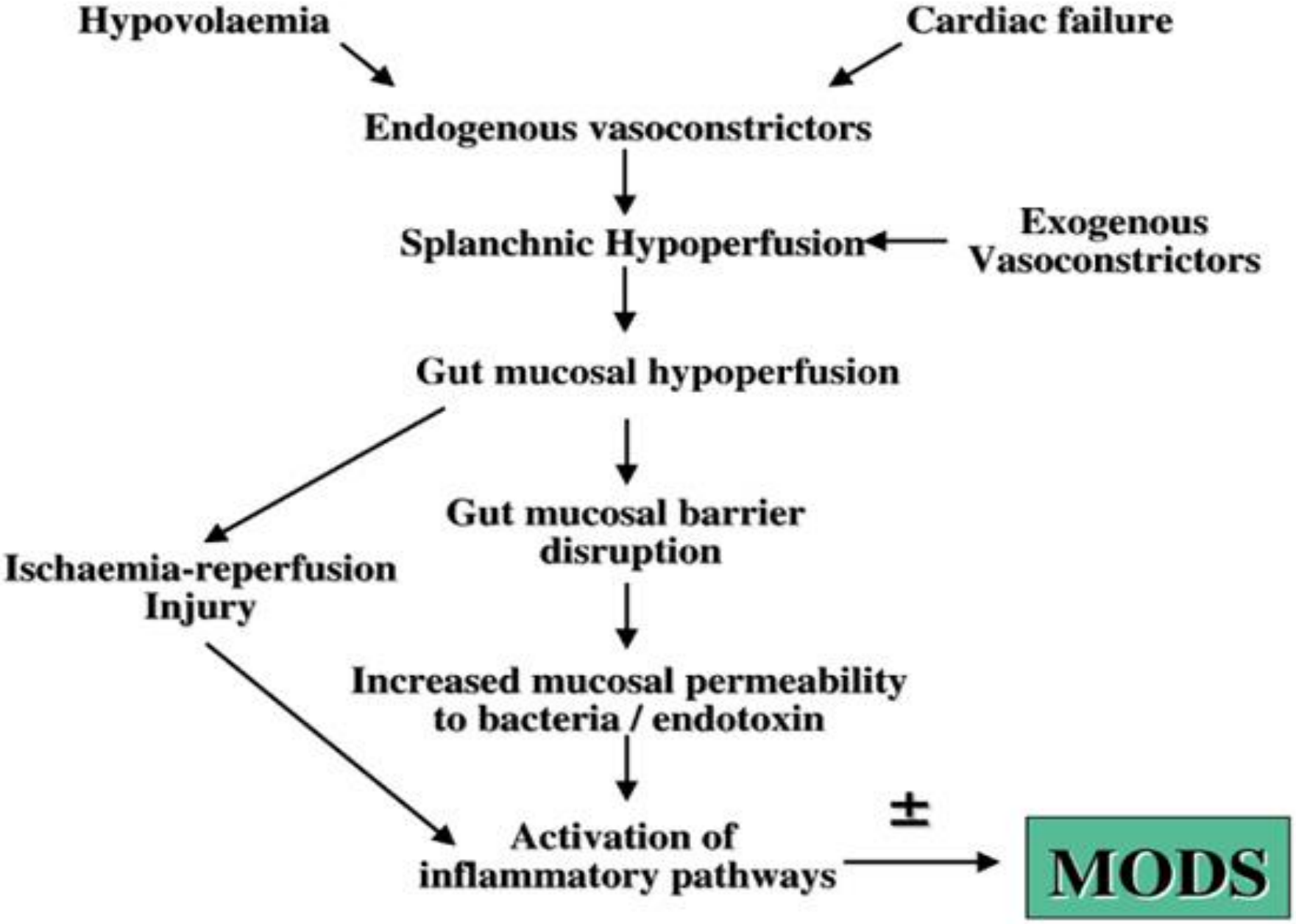
G.I Complication

- Constriction of vessels supplying GIT for redistribution of blood flow

  - Severe Decrease mucosal perfusion
    - GIT ulceration
    - Bleeding

Disseminated Intravascular Coagulation




Hypovolaemia
Cardiac failure
Endogenous vasoconstrictors
Splanchnic Hypoperfusion
Exogenous Vasoconstrictors
Gut mucosal hypoperfusion
Gut mucosal barrier disruption
Ischaemia-reperfusion Injury
Increased mucosal permeability to bacteria / endotoxin
±
Activation of inflammatory pathways
MODS