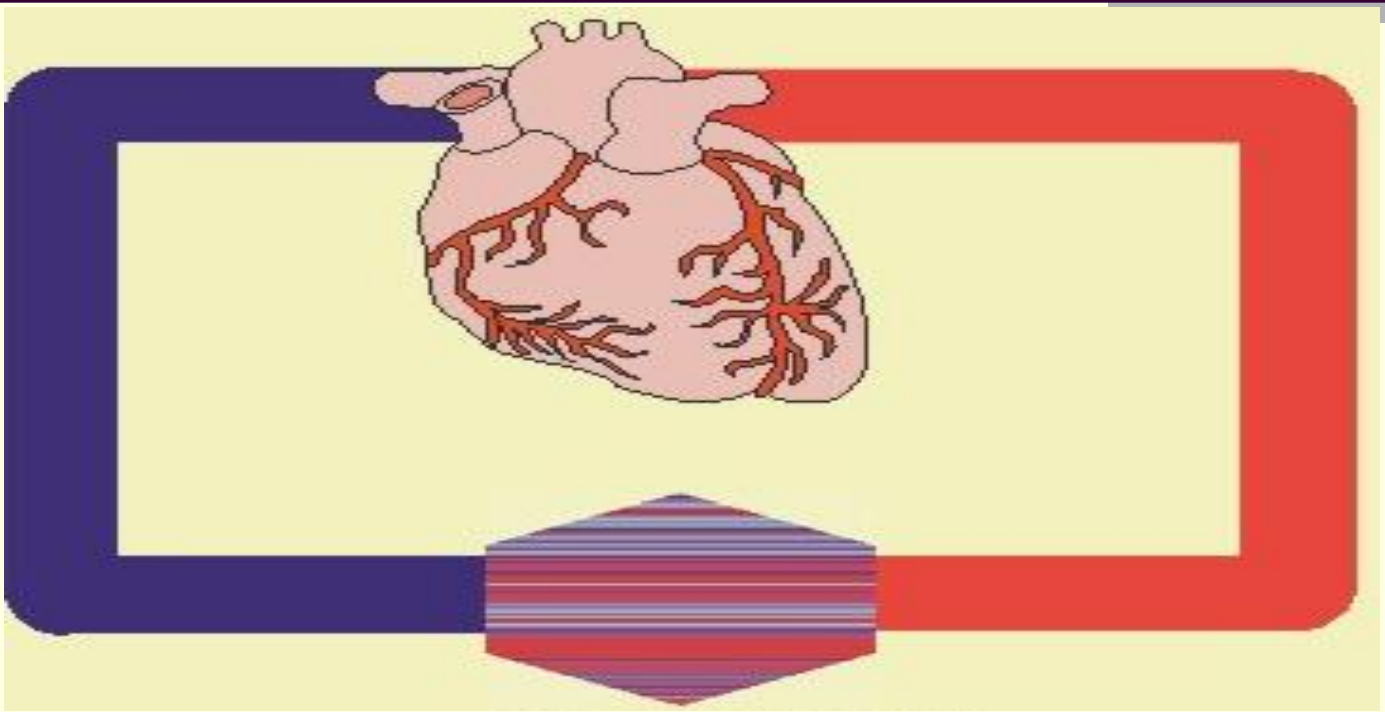
SHOCK



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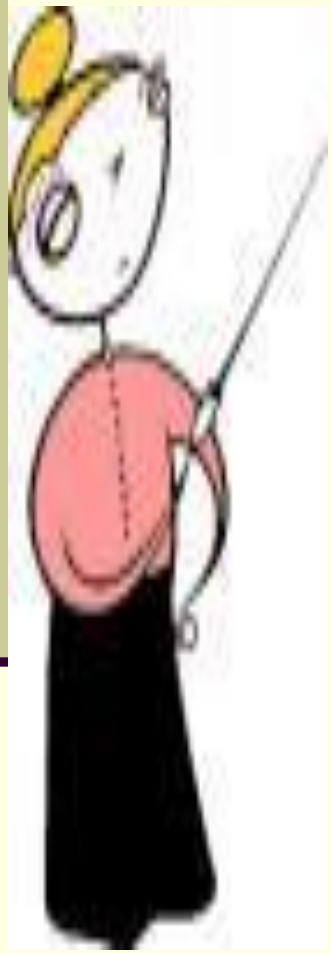
LECTURE OUTLI NES / OBJECTIVES



STUDENTS ABLE TO UNDERSTAND:

Define shock, list the types and causes of shock

Pathophysiological classification of shock

Describe the pathways leading to shock

Discuss the stages of a hypovolemic shock.

Understand the body compensatory mechanisms during the hemorrhagic shock

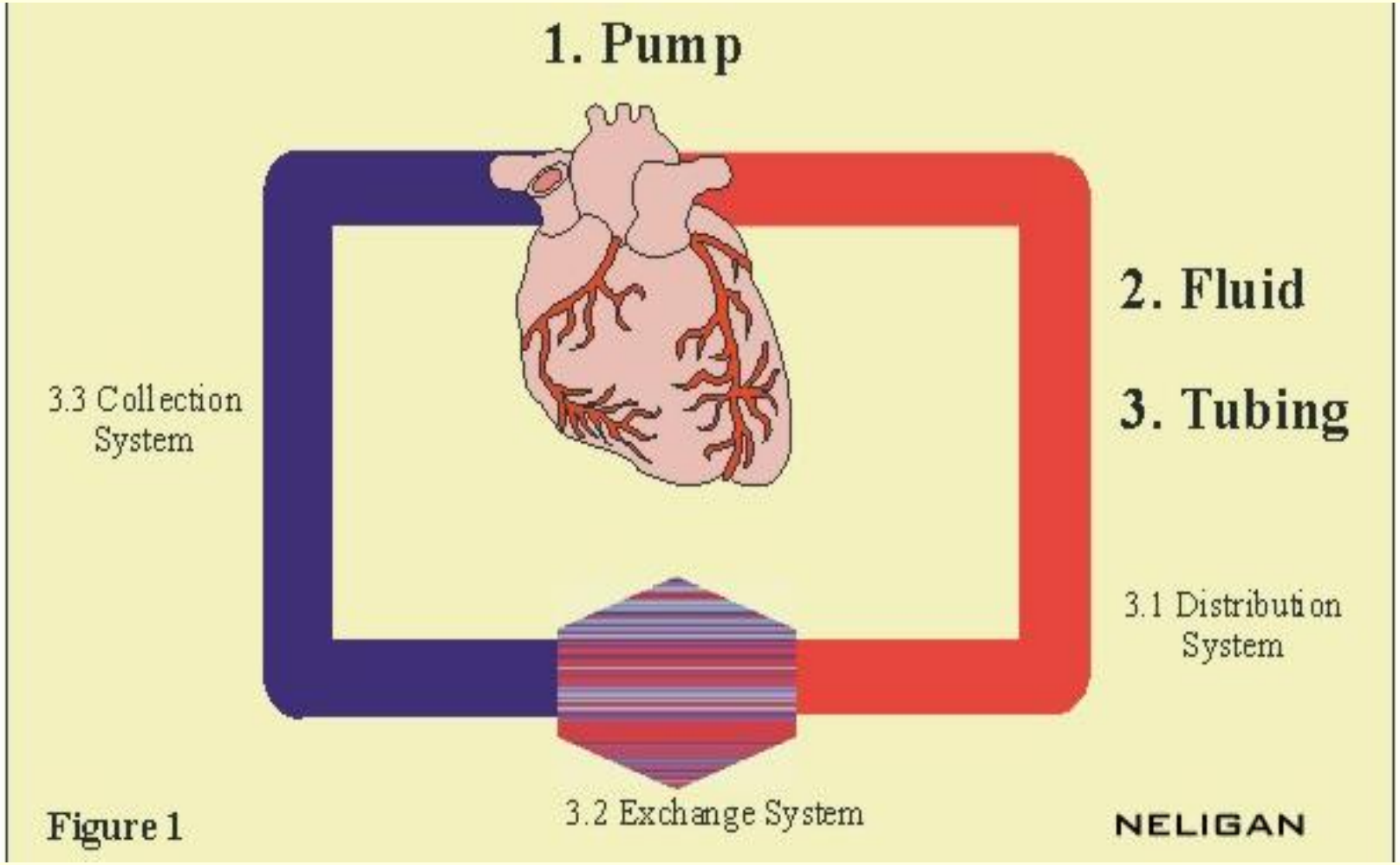
Discuss the different compensatory mechanisms during a hypovolemic shock.

Clinical features and management

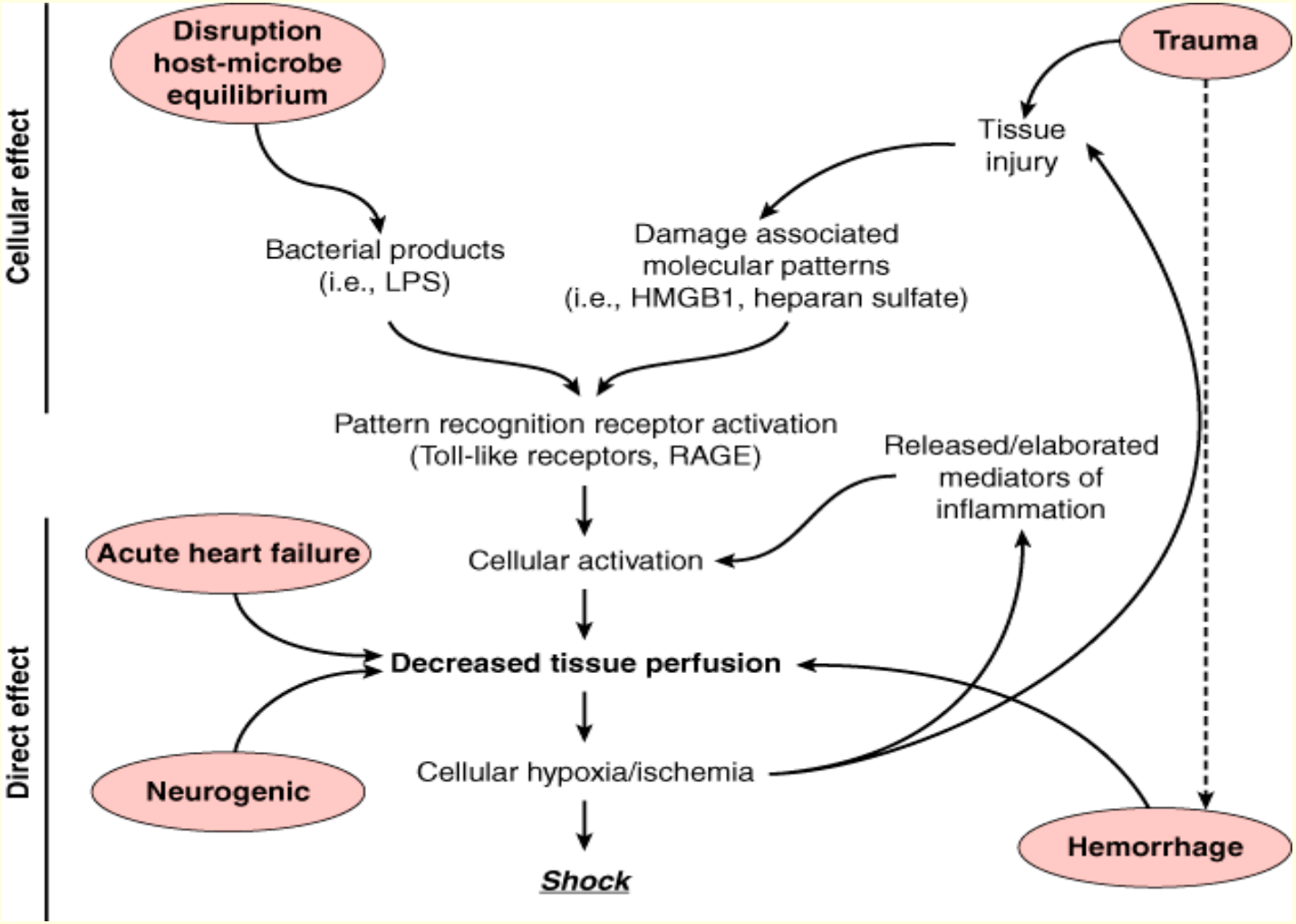
WHAT IS SHOCK?

- Shock is defined as an acute circulatory failure leading to inadequate tissue perfusion and end organ injury.
- The main feature of circulatory shock is loss of fluid from the circulating blood volume, so that adequate circulation to all parts of body cannot be maintained.

WHAT IS SHOCK?



WHAT IS SHOCK?

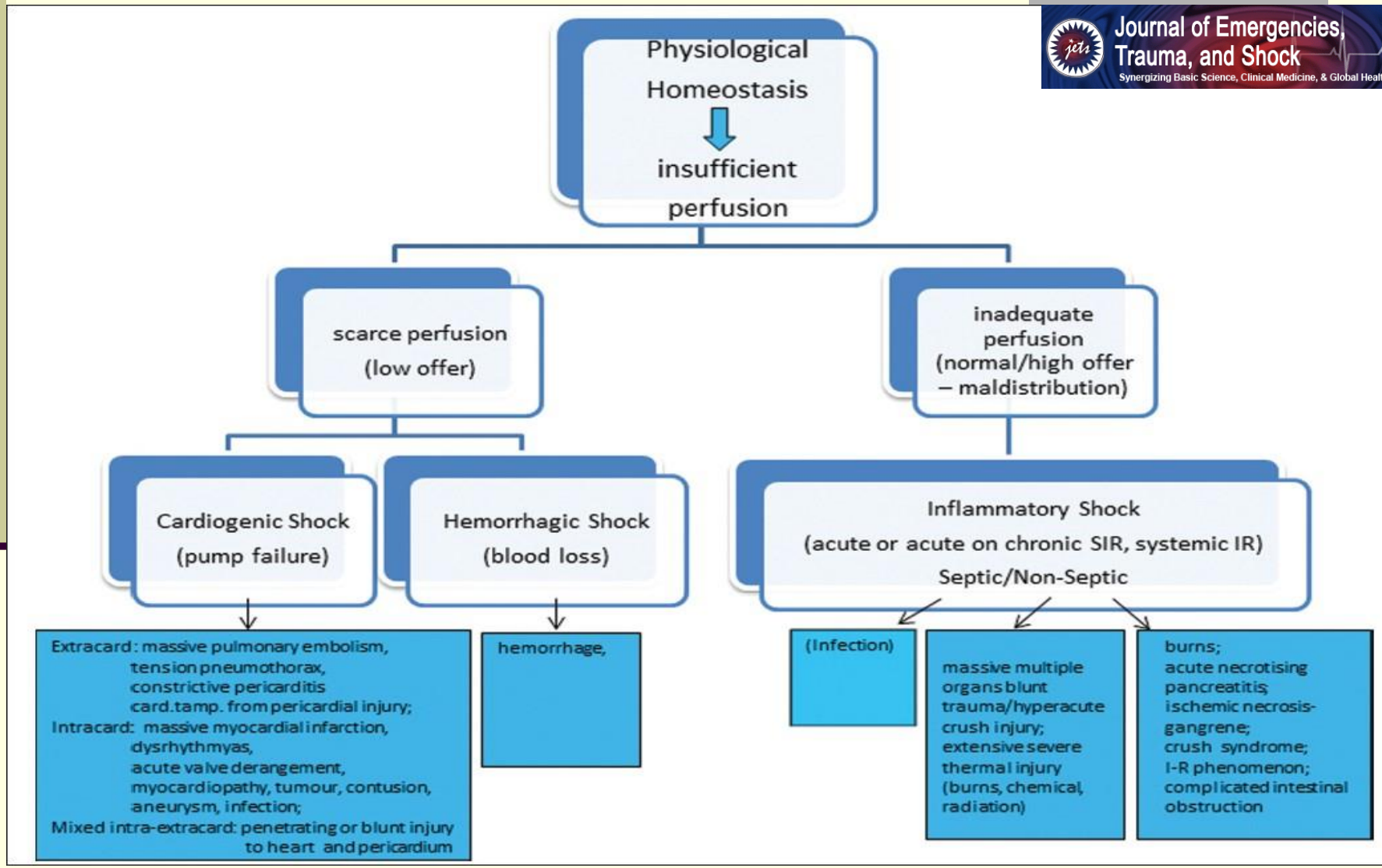


CLASSIFICATION OF SHOCK

1. Hypovolumic Shock
2. Cardiogenic Shock
3. Neurogenic Shock
4. Vasogenic Shock
    i. Anaphylactic shock
    ii. Septic shock

CLASSIFICATION OF SHOCK

Journal of Emergencies, Trauma, and Shock
Synergizing Basic Science, Clinical Medicine, & Global Health



PHYSIOLOGICAL CAUSES OF SHOCK

Circulatory shock caused by decreased cardiac output

Shock usually results from inadequate cardiac output.

Two types of factors can severely reduce cardiac output:

- 1. Cardiac abnormalities that decrease the heart to pump blood. These includes MI, toxic heart, severe heart valve dysfunction, heart arrhythmias.
- Circulatory shock results from diminished cardiac pumping ability is called cardiogenic shock. 85% people die who develop cardiogenic shock
- 2. Factors decrease venous return also decrease cardiac output because the heart cannot pump blood that does not flow into it. The common cause of decreased venous return is diminished blood volume, decreased vascular tone

GENERAL MECHANISM

Flow= Pressure

Adequate Flow = Adequate pressure

In adequate flow = In adequate pressure [Hypo perfusion]

Adequate perfusion = No Shock

Hypo perfusion = Shock

GENERAL MECHANISM

In Adequate pump:

Inadequate preload Poor contractility

Preload is the end-diastolic volume (EDV) at the beginning of systole. Directly allied to degree of stretch
It is related to ventricular filling.

Excessive after load Inadequate heart rate

Afterload is the ventricular pressure at the end of systole. Force against which heart contract to eject the blood.

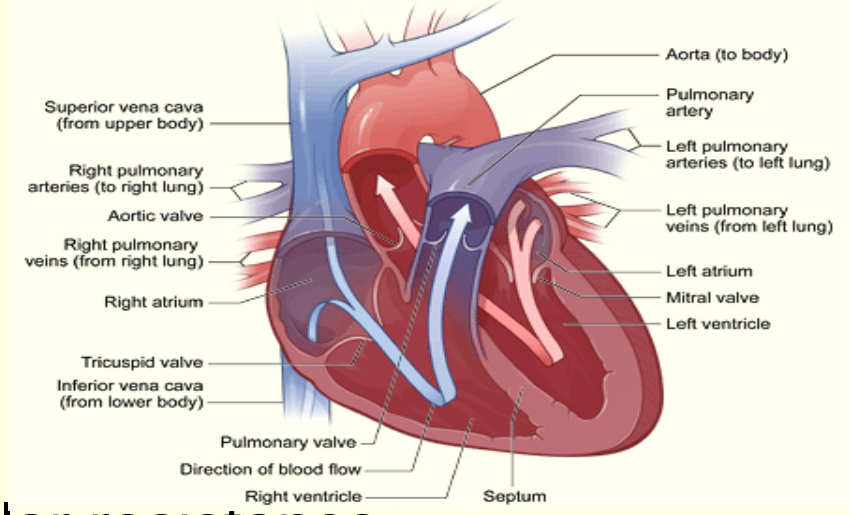
In Adequate Fluid Volume:

Hypovolumia

In adequate container:

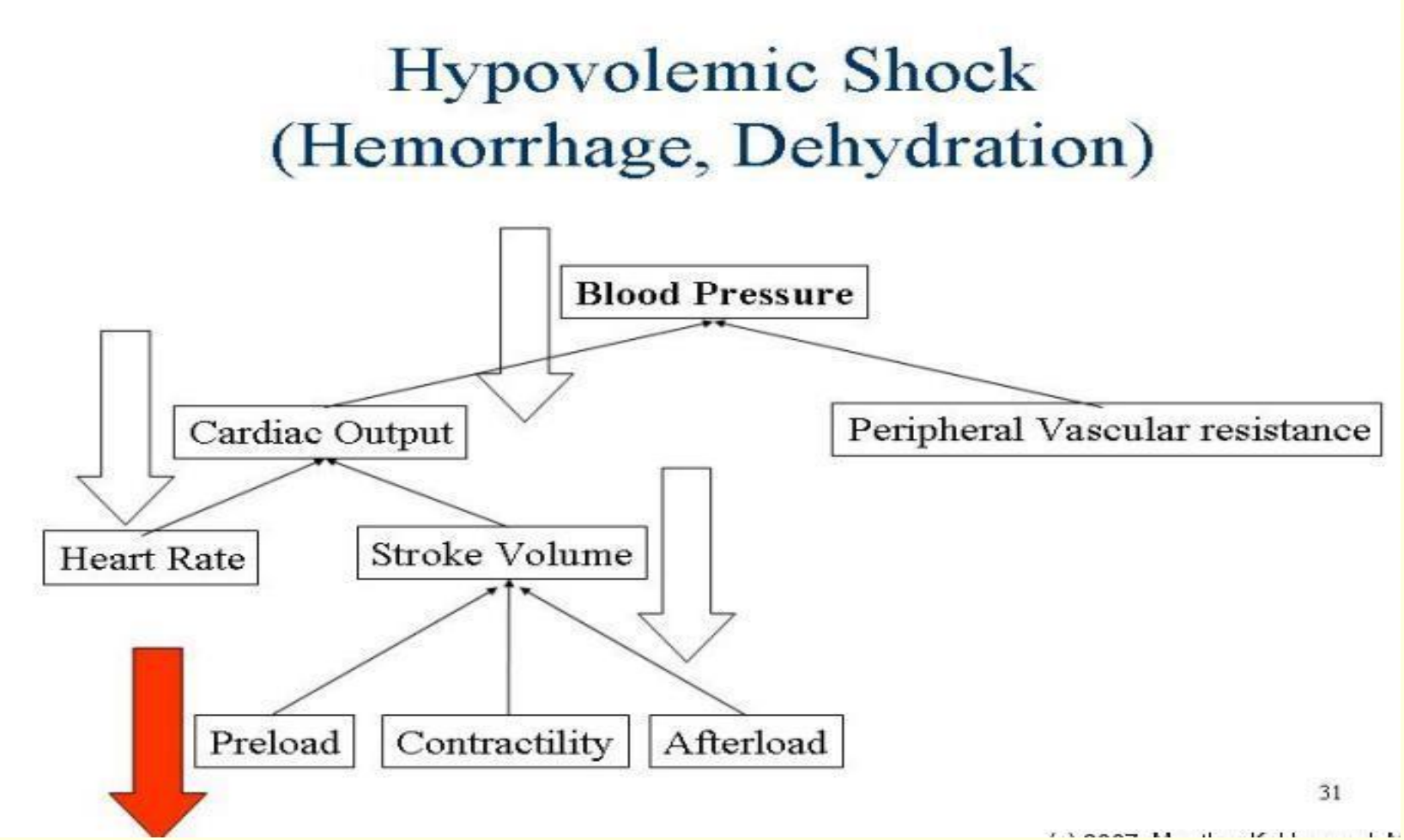
Excessive dilation

Inadequate systematic vascular resistance



Guyton, pp 118,

GENERAL MECHANISM



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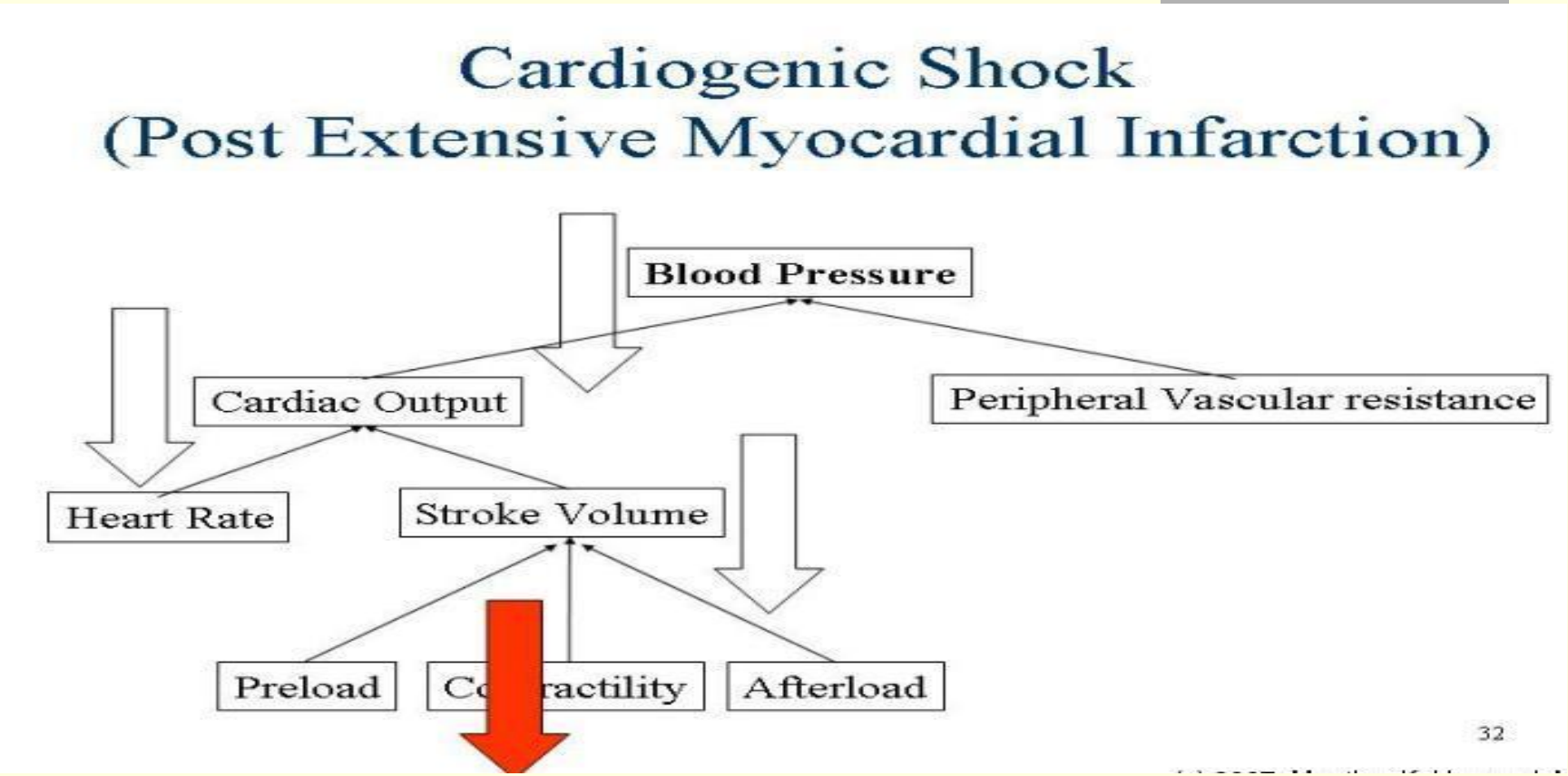
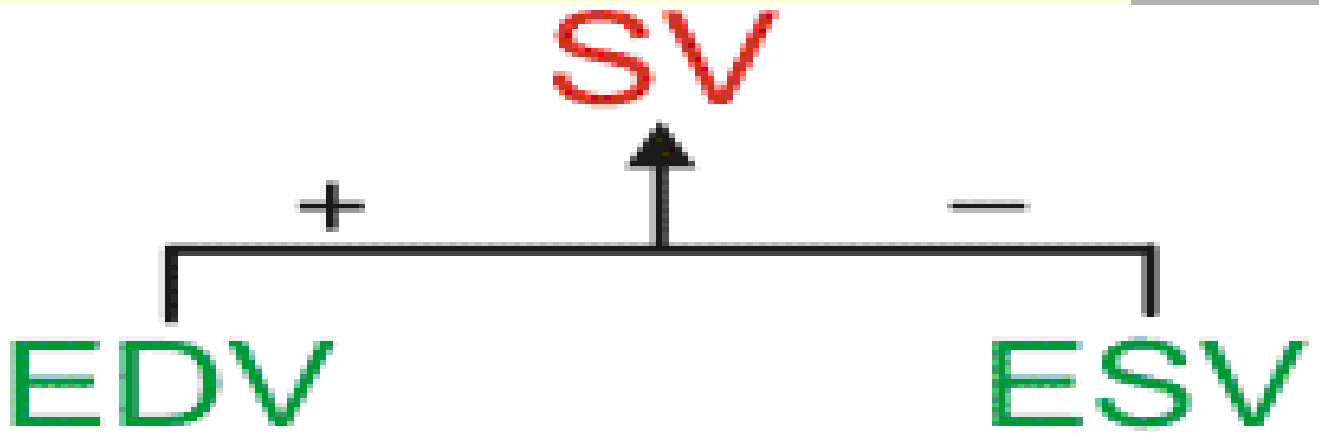


Heart becomes incapable of contracting with sufficient force to pump enough blood into the peripheral arterial tree. Cardiac shock occurs when more than 40% of the left ventricle is infarcted and death occurs in about 85 % of patients once they develop cardiac shock.

GENERAL MECHANISM

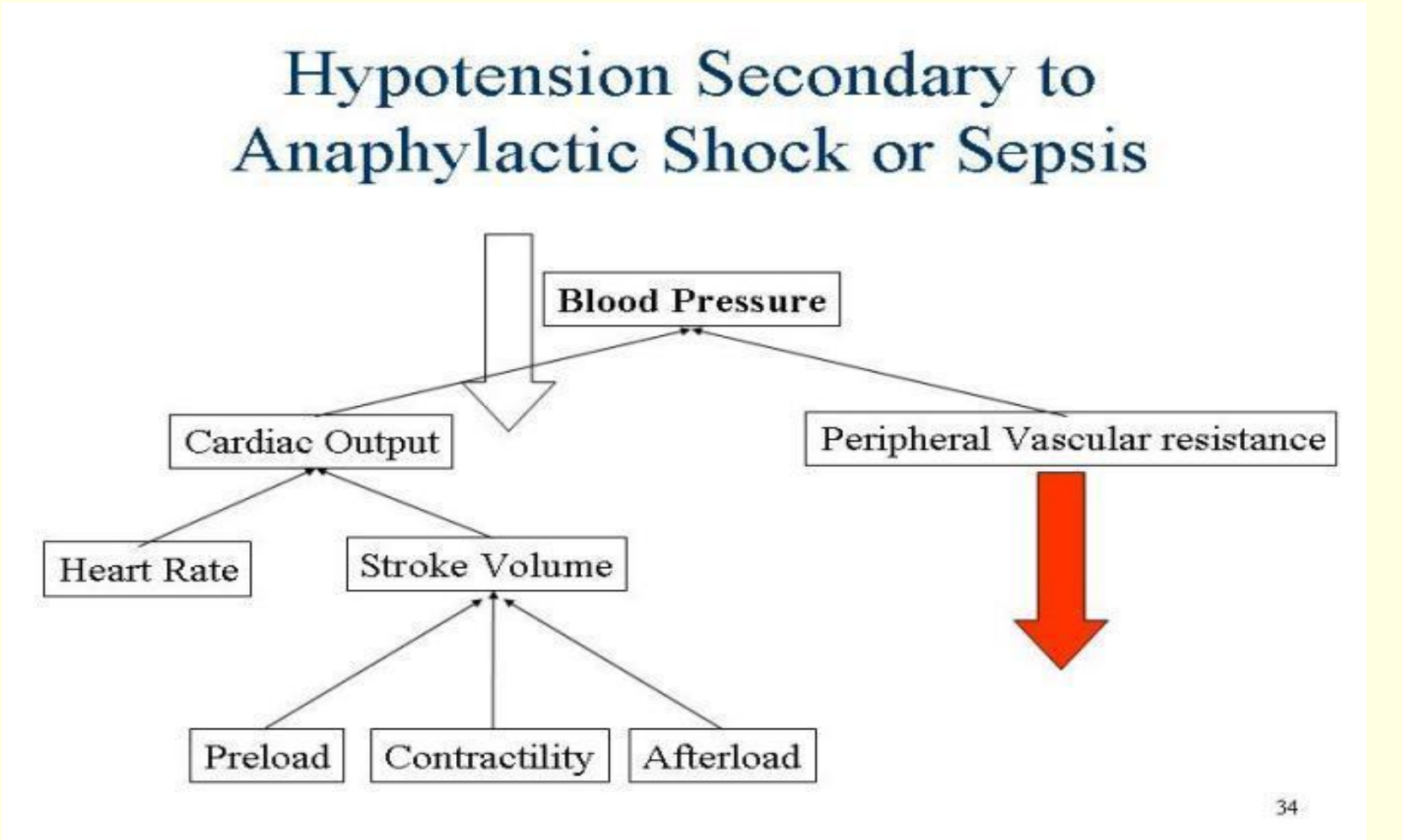


↑ Preload → ↑ SV (↑ EDV)

↑ Afterload → ↓ SV (↑ ESV)

↑ Inotropy → ↑ SV (↓ ESV)

GENERAL MECHANISM



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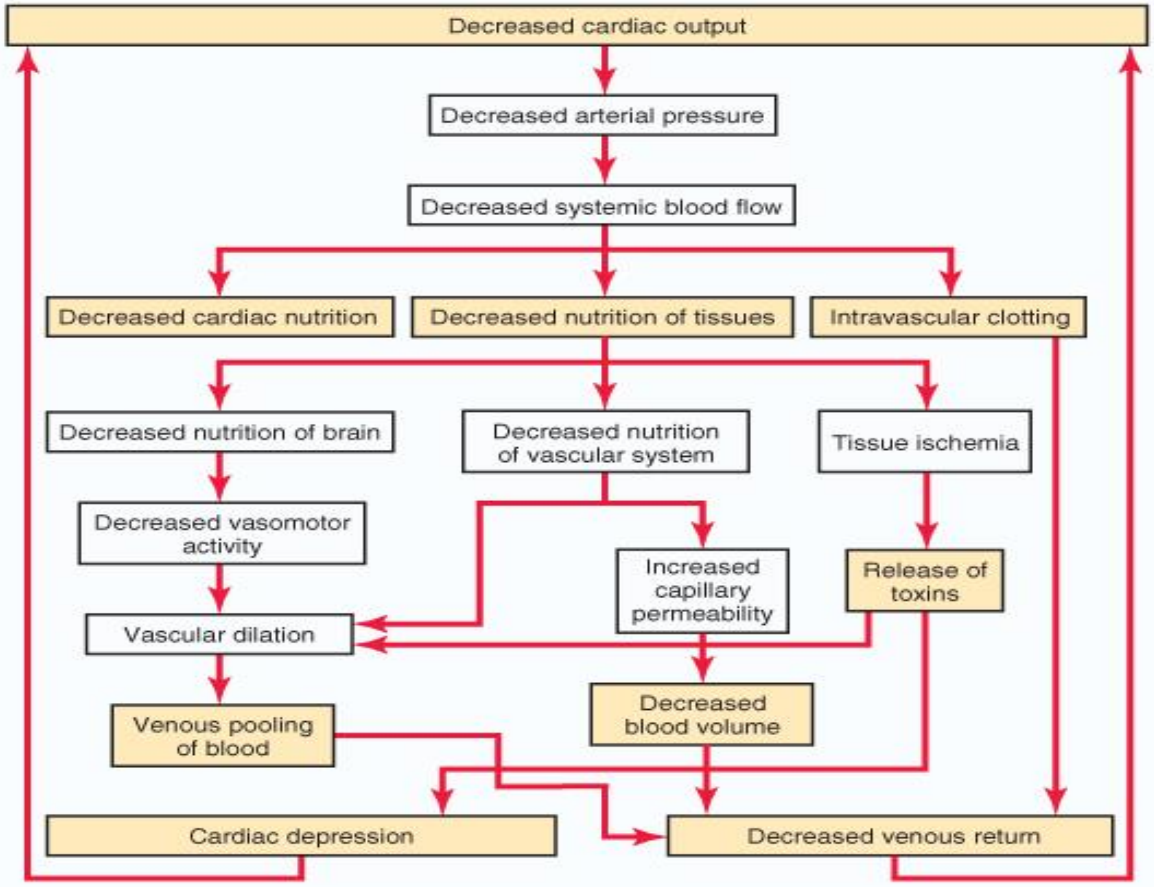

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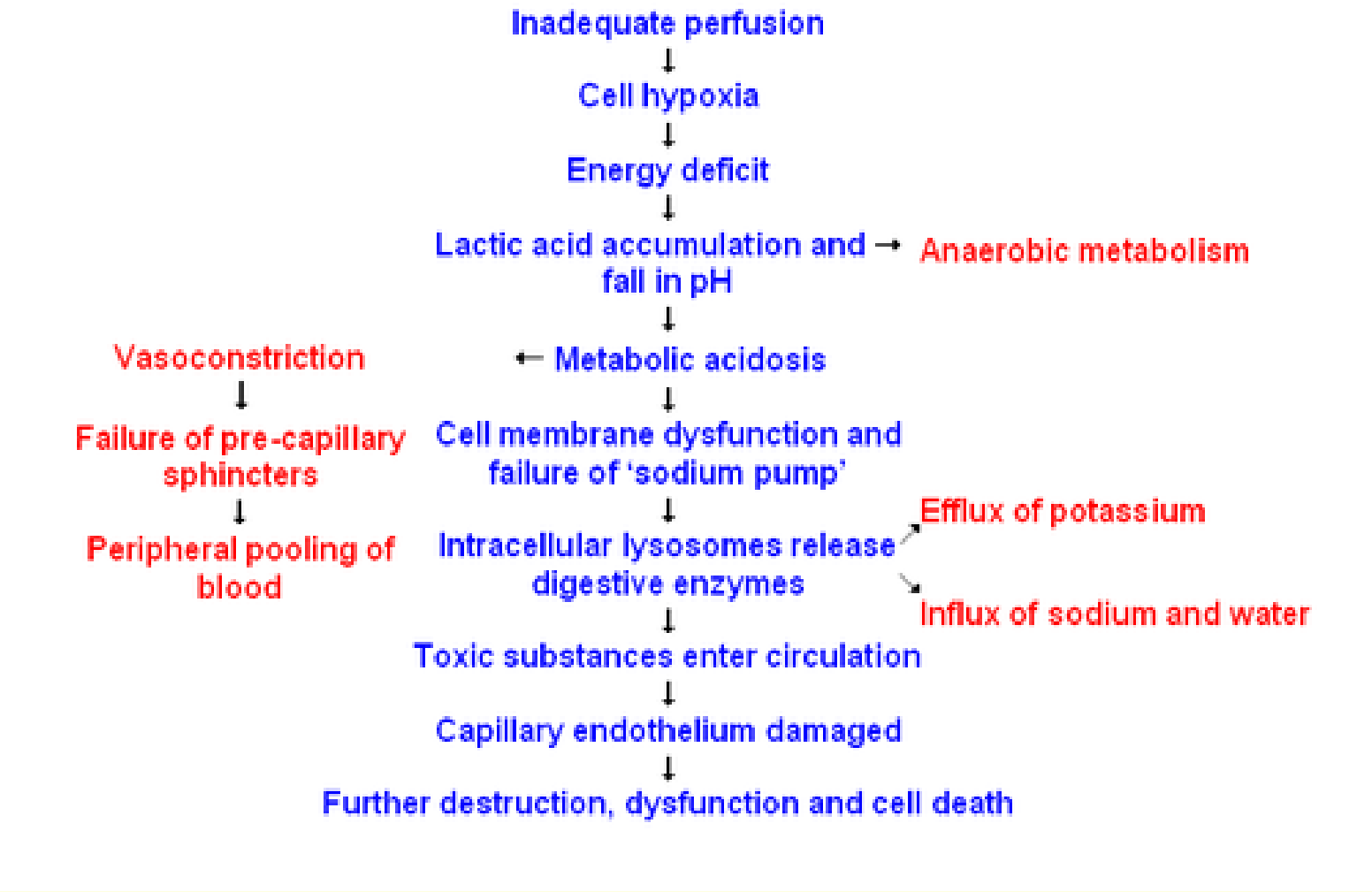

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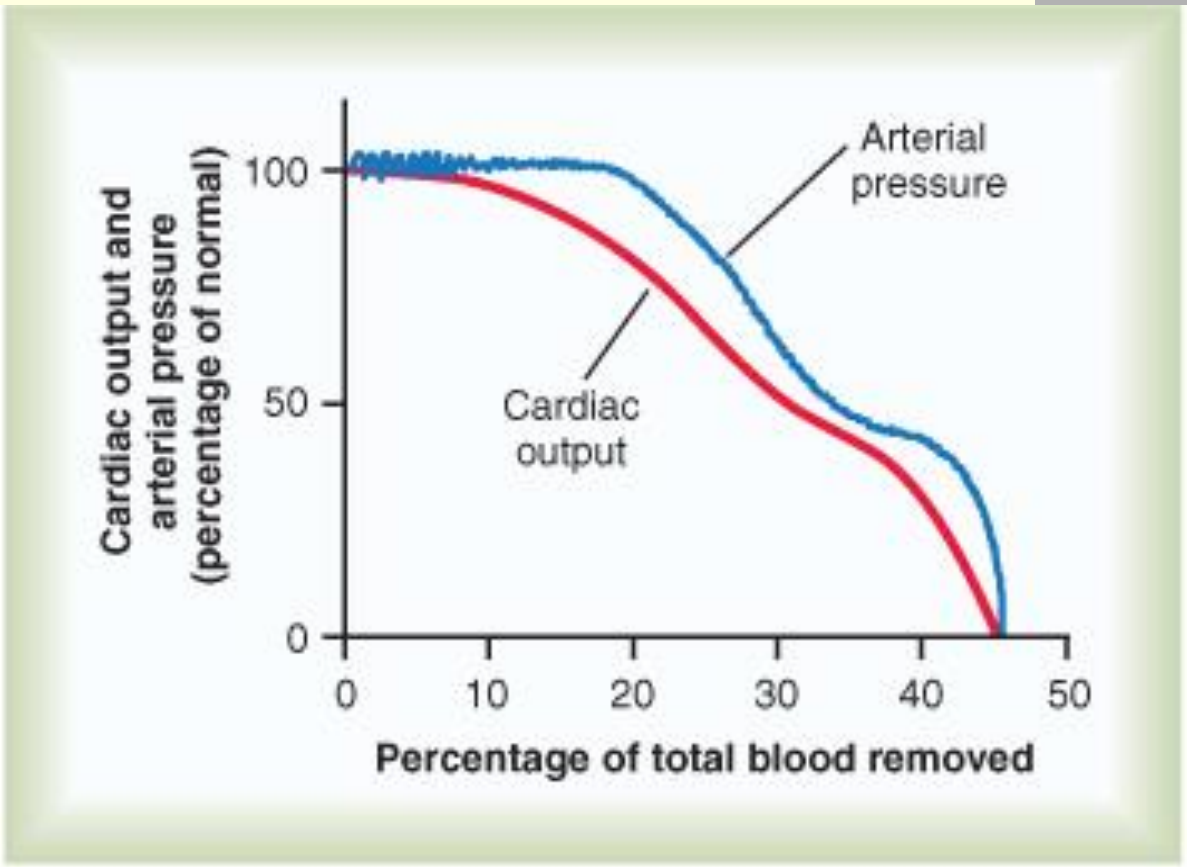


Effect of hemorrhage on cardiac output and arterial pressure

STAGES OF SHOCK

Stages of Shock: Circulatory shock change with different degrees of severity, shock is divided into following major stages:

1. A non-progressive stage (Compensated stage): The normal circulatory compensatory mechanisms eventually cause full recovery without help from outside therapy.

2. A progressive stage: Without therapy, shock worse until death.

3. An irreversible stage: Shock progressed to an extent that all forms of known therapy are inadequate to save the life, even though, for the moment, the person is still alive.

HYPOVOLUMIC SHOCK:

CAUSES OF HYPOVOLUMIC SHOCK

[Decreased Blood Volume]

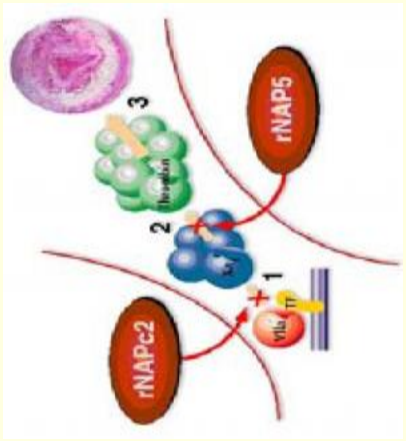
- Hemorrhage [Trauma, GI bleed, ruptured aneurysm]
- Surgery
- Burns [Loss of plasma]
- Vomiting and Diarrhea [Fluid Loss]

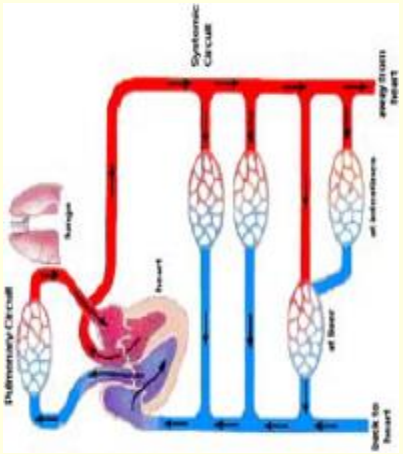
HYPOVOLUMIC SHOCK

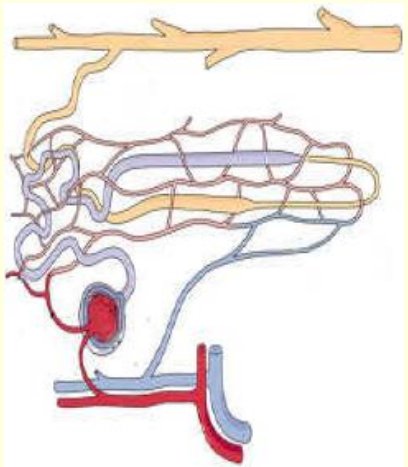
The human body responds to acute hemorrhage by activating four major physiologic systems:

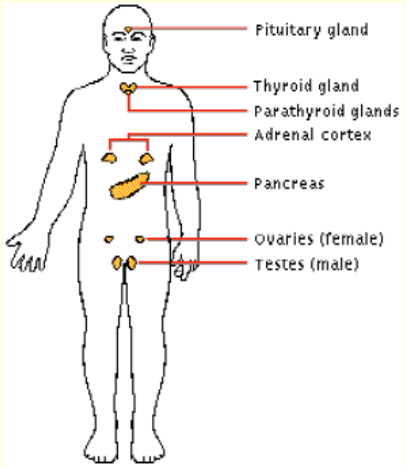
i. Hematologic, ii. Cardiovascular, iii. Renal

iv. Neuroendocrine system.

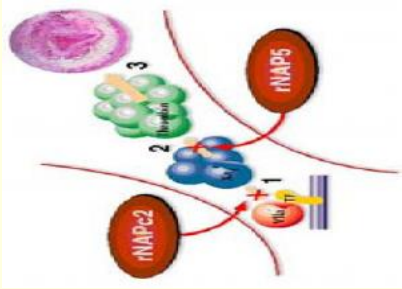








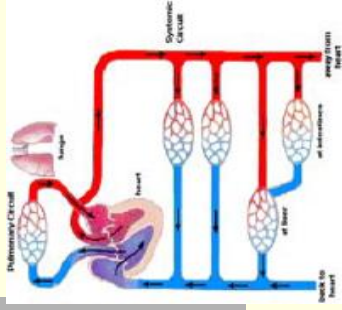
HYPOVOLUMIC SHOCK



Hematologic System

- Activating the coagulation cascade
- Contracting the bleeding vessels (via local thromboxane A2 release)
- Platelets activated which form an immature clot on the bleeding source
- The damaged vessel exposes collagen, which subsequently causes fibrin deposition and stabilization of the clot.

HYPOVOLUMIC SHOCK



Cardiovascular System

- Increase heart rate, increase myocardial contractility and constricting peripheral blood vessels.
- This response occurs secondary to an increase secretion of norepinephrine and a decrease in vagal tone (regulated by the baroreceptors in the carotid arch, aortic arch, left atrium, and pulmonary vessels).
- The CVS also responds by redistributing blood to the brain, heart, and kidneys and away from skin, muscle, and GI tract.

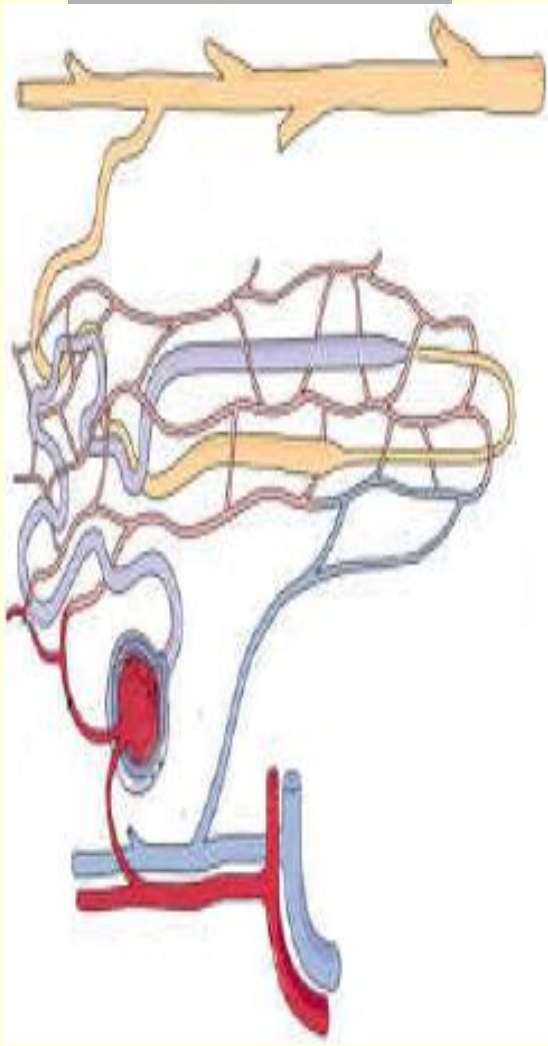
HYPOVOLUMIC SHOCK

Renal System

- The kidneys respond to hemorrhagic
- shock by stimulating an increase in renin

secretion from the juxtaglomerular apparatus

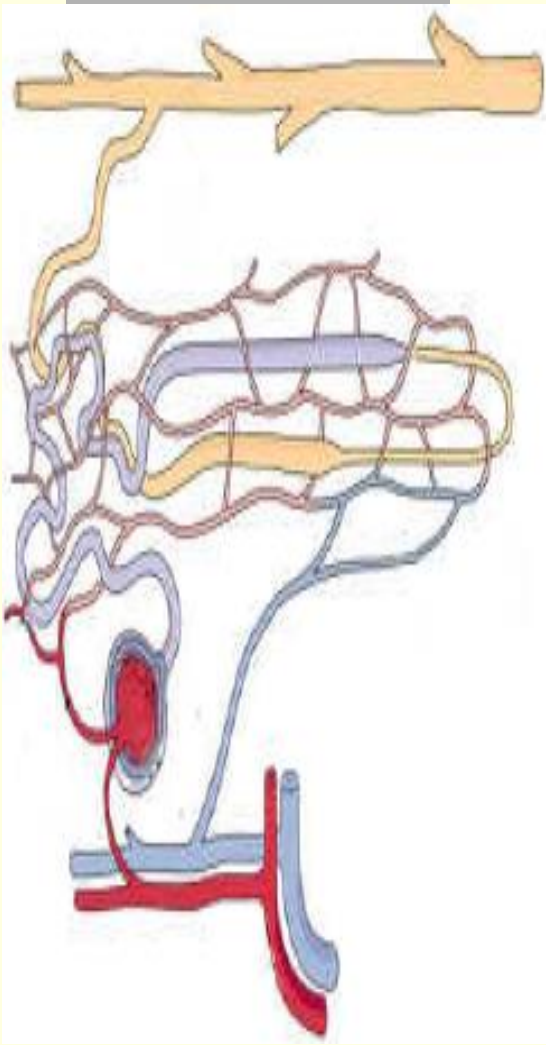
Renin → Angiotensinogen …. Lungs and Liver → Angiotensin I , Angiotensin II



HYPOVOLUMIC SHOCK

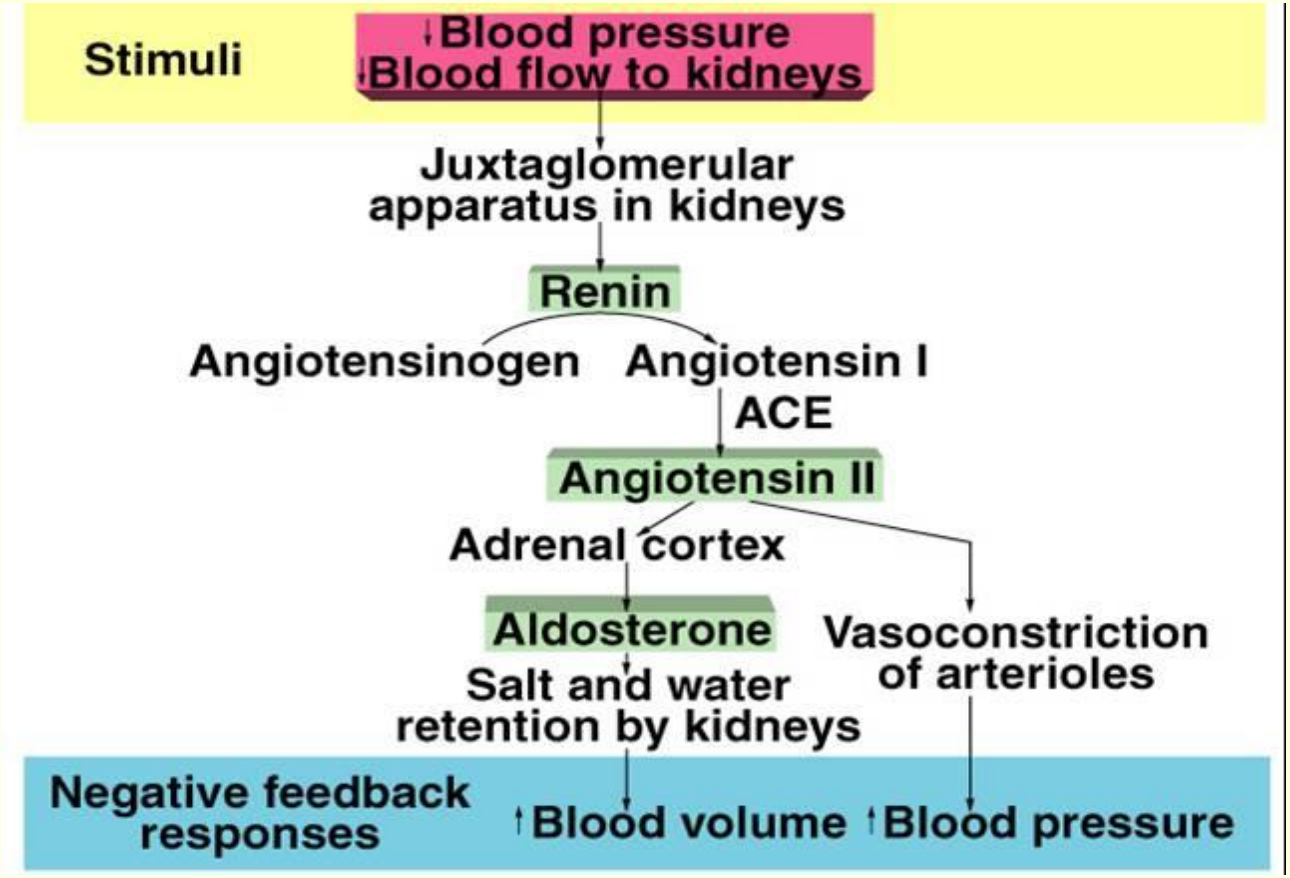
Renal System

Angiotensin II has two main effects, both of which help to reverse hypovolemic shock, vasoconstriction of arteriolar smooth muscle and stimulation of aldosterone secretion by the adrenal cortex.

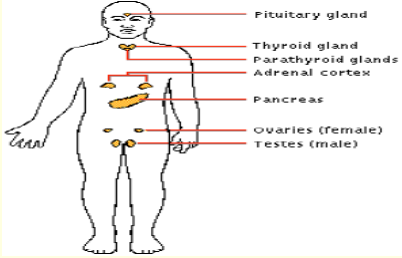


HYPOVOLUMIC SHOCK

Renal System



HYPOVOLUMIC SHOCK



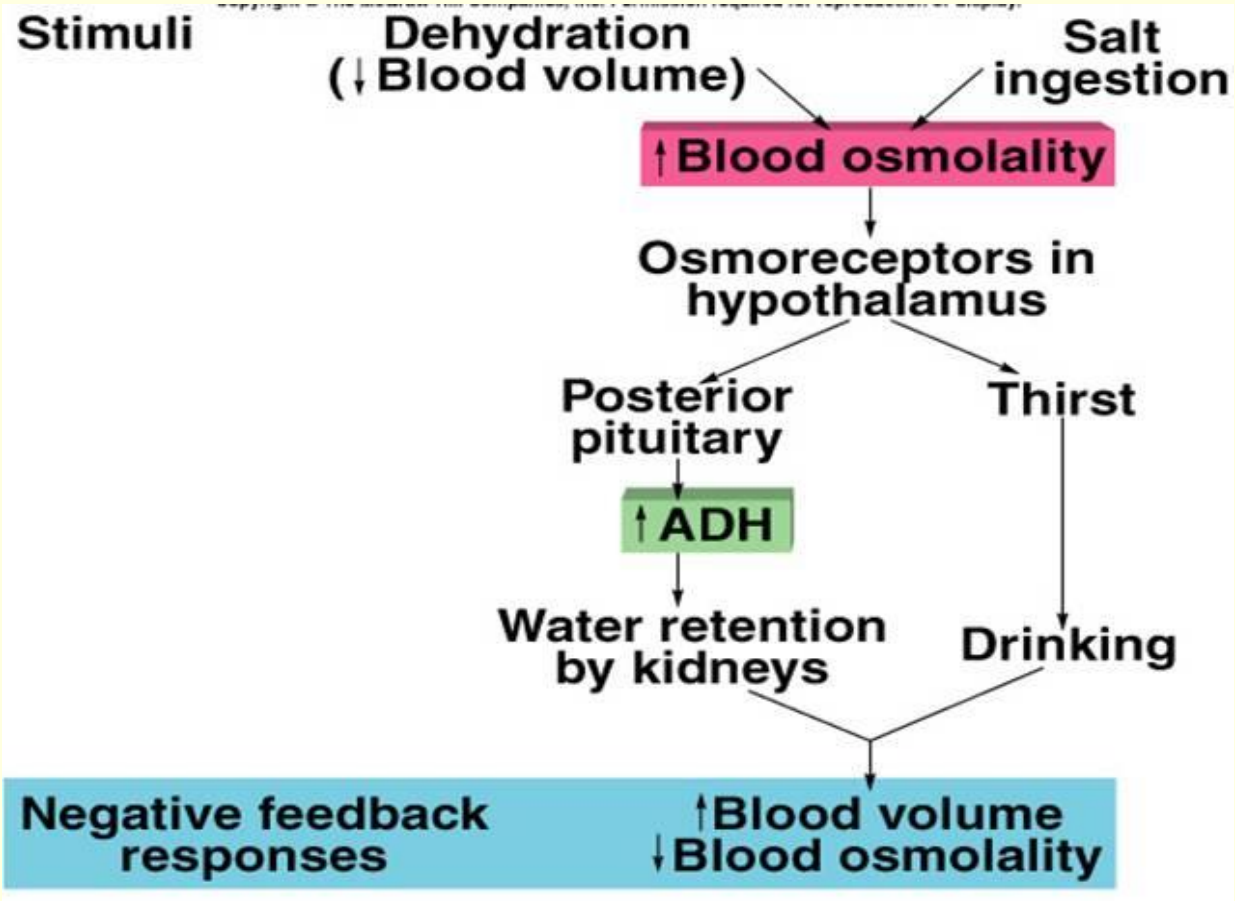
Neuroendocrine system

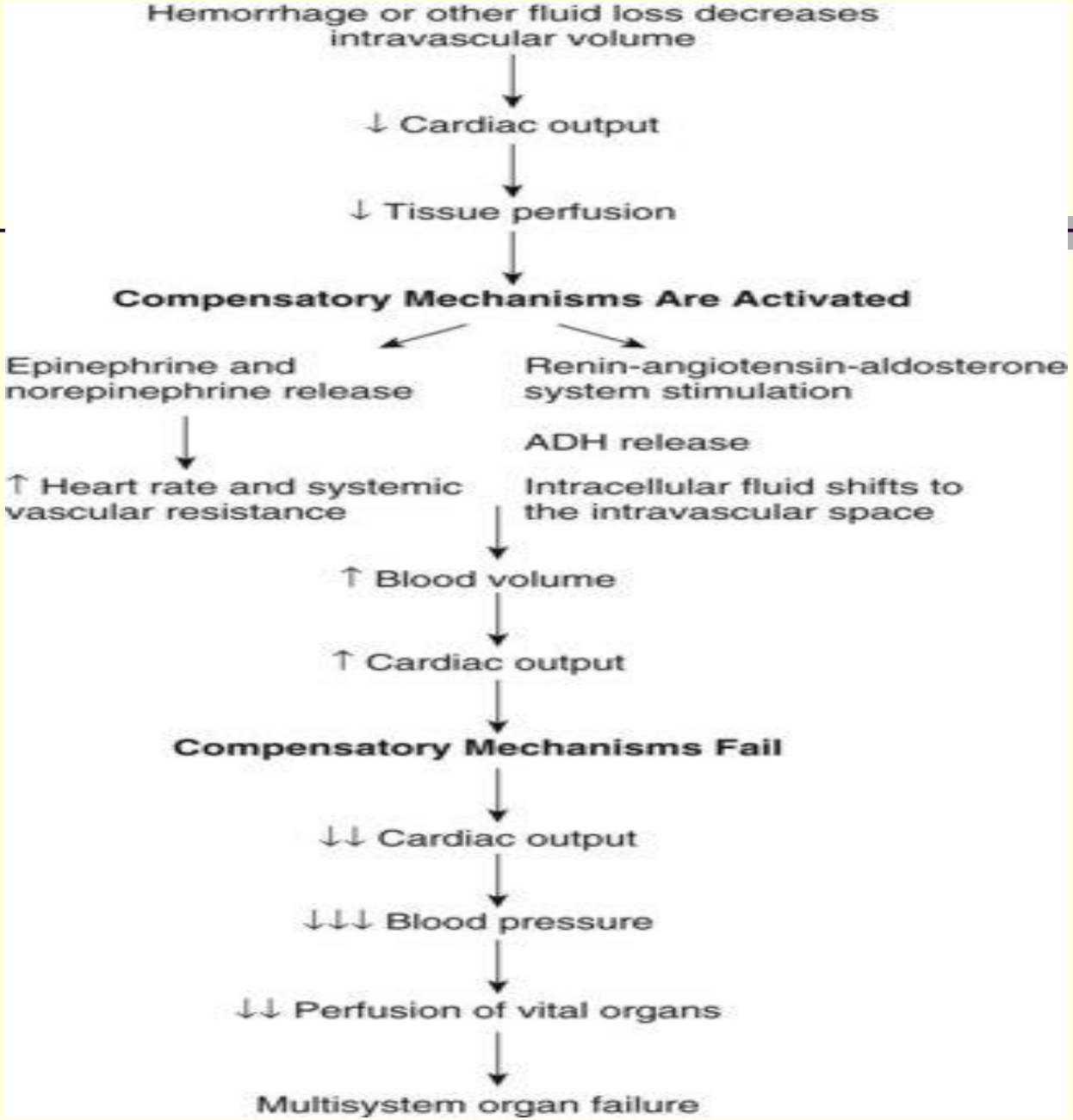
Causes an increase in circulating antidiuretic hormone (ADH)

- ADH released in response to a decrease in blood pressure (as detected by baroreceptors) and a decrease in sodium concentration
- ADH increase in reabsorption of water and salt (NaCl) by the distal tubule and the collecting ducts.

HYPOVOLUMIC SHOCK

Neuroendocrine system




Hemorrhage or other fluid loss decreases intravascular volume
↓ Cardiac output
↓ Tissue perfusion
Compensatory Mechanisms Are Activated
Epinephrine and norepinephrine release
Renin-angiotensin-aldosterone system stimulation
ADH release
↑ Heart rate and systemic vascular resistance
Intracellular fluid shifts to the intravascular space
↑ Blood volume
↑ Cardiac output
Compensatory Mechanisms Fail
↓↓ Cardiac output
↓↓↓ Blood pressure
↓↓ Perfusion of vital organs
Multisystem organ failure

HYPOVOLUMIC SHOCK

Hemorrhagic Shock

Parameter	I	II	III	IV
Blood loss (ml)	<750	750–1500	1500–2000	>2000
Blood loss (%)	<15%	15–30%	30–40%	>40%
Pulse rate (beats/min)	<100	>100	>120	>140
Blood pressure	Normal	Decreased	Decreased	Decreased
Respiratory rate (bpm)	14–20	20–30	30–40	>35
Urine output (ml/hour)	>30	20–30	5–15	Negligible
CNS symptoms	Normal	Anxious	Confused	Lethargic

Crit Care. 2004; 8(5): 373–381.

HYPOVOLUMIC SHOCK:

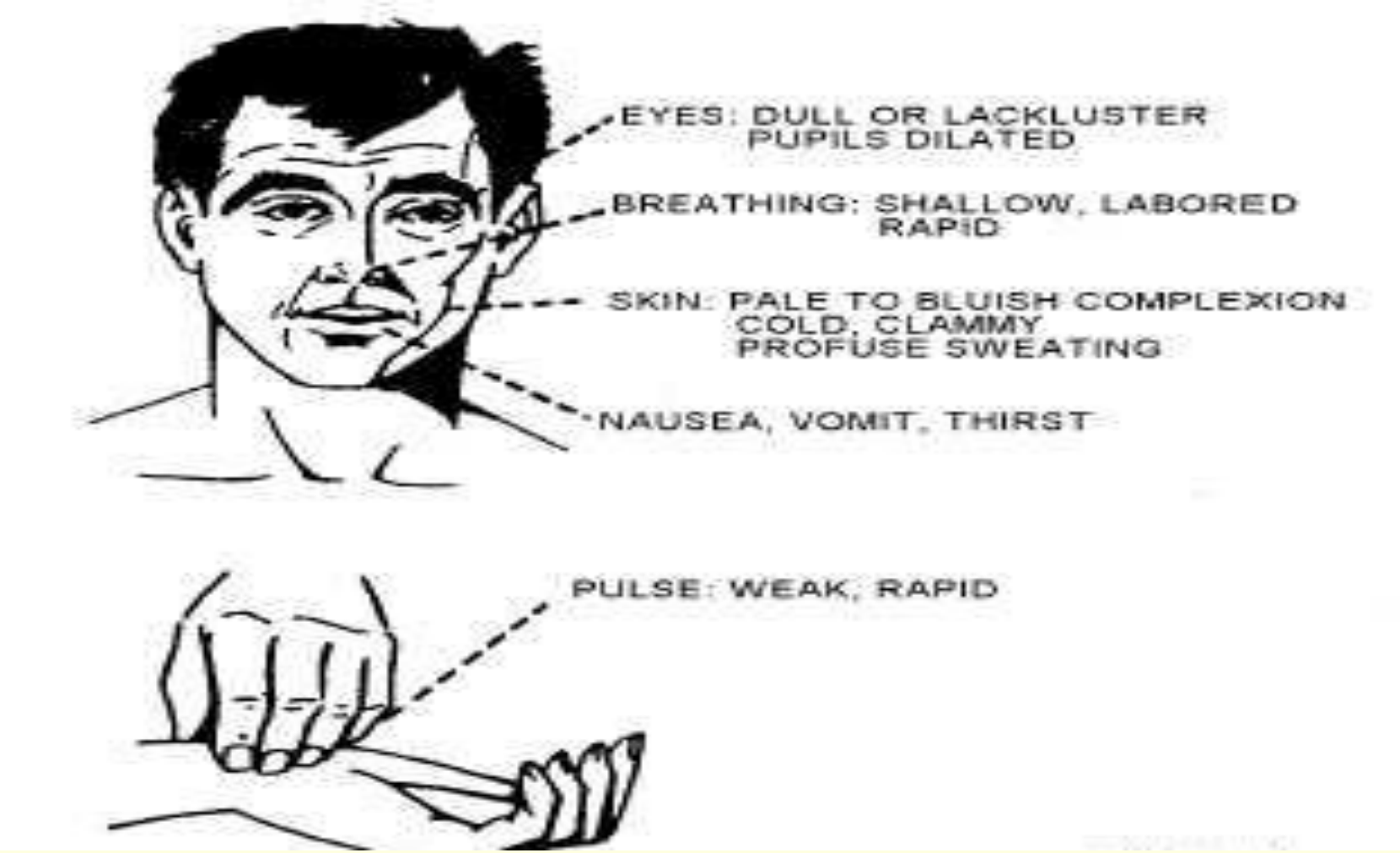
Clinical Features of Hypovolemic Shock

- Patient become
  - Pale
  - Cold clamy skin

Vasoconstriction due to increased sympathetic stimulation)

- Hypotension
- Weak, rapid pulse
- Increased respiratory rate
- Sweating
- Increased thirst
- Decreased urinary output
- Metabolic Acidosis
- Restlessness

CLINICAL PRESENTATION



TYPE	CAUSES	SYMPTOMS AND SIGNS
Hypovolemic shock	Bleeding (internal/external), dehydration (sever voming, sever diarrhea), plasma loss (as in burns) → low blood volume → decreased cardiac output → hypotension	hypotension; weak but rapid pulse; cool, clammy skin; rapid, shallow breathing; anxiety, altered mental state
Cardiogenic shock	Heart problems (e.g., myocardial infarction, heart failure; cardiac dysrhythmias) → decreased contractility → decrease in stroke volume → decreased cardiac output → hypotension	as for hypovolaemic shock + distended jugular veins & may be absent pulse
Obstructive shock	Circulatory obstruction (e.g., constrictive pericarditis, cardiac tamponade, tension pneumothorax, pulmonary embolism → reduced blood flow to lungs → decreased cardiac output → hypotension	as for hypovolaemic shock + distended jugular veins & pulsus paradoxus (in cardiac tamponade).
Distributive shock Vasogenic, Low-resistance shock	❑ Septic shock: infection → release of bacterial toxins → activation of NOS in macrophages → production of NO → vasodilation → decreased vascular resistance→ hypotension ❑ Anaphylatic shock: allergy (release of histamine → vasodilation → decreased vascular resistance→ hypotension ❑ Neurogenic shock: spinal injury → loss of autonomic and motor reflexes → reduction of peripheral vasomotor tone → vasodilation → decrease in peripheral vascular resistance → hypotension	❑ Septic shock: hypotension; fever; warm, sweaty skin ❑ Anaphylatic shock: skin eruptions; breathlessness, coughing; localized edema; weak, rapid pulse ❑ Neurogenic shock: as for hypovolemic except warm, dry skin

Manage the Emergency

- Control airway and breathing
- Maximize oxygen delivery
- Place lines, tubes, and monitors
- Get and run IVF on a pressure bag
- Get and run blood (if appropriate)
- Get and hang pressors
- Call your senior/fellow/attending

Definitive Management

- Hypovolemic – Fluid resuscitate (blood or crystalloid) and control ongoing loss
- Cardiogenic - Restore blood pressure (chemical and mechanical) and prevent ongoing cardiac death
- Distributive – Fluid resuscitate, immediate surgical control for infection

TREATMENT OF SHOCK

Treatment of Shock:

Goal: Restore normal tissue perfusion

- Blood pressure, Pulse, Respirations
- Skin Appearance
- Urine output (30-50 cc per hour)
- Hemoglobin 8-10 gm or Hematocrit 24-30

TREATMENT OF SHOCK

- While inserting IVs, draw blood for laboratories and for blood typing
- Relieve pain with IV narcotics
- Reassess
- Blood transfusion: think twice
- Vasopressors
- Antibiotics?
- Maintain IV fluids

THANK YOU