جامعة الملك سعود
King Saud University

Shock

Index:
Red: important Grey: extra information Green: doctor's notes yellow: numbers
Purple: only in female slides Blue: only in male slides

Physiology Team437

Physiology 437 teamwork

MED437
KING SAUD UNIVERSITY

OBJECTIVES

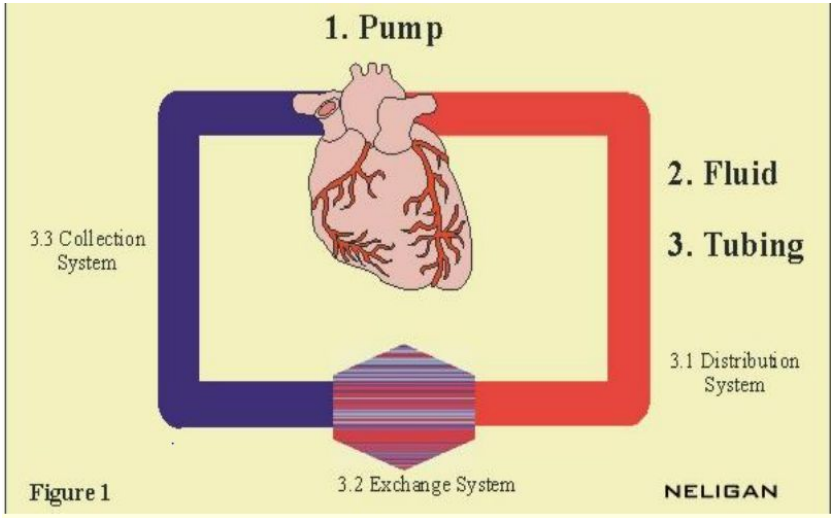
by the end of this lecture you will be able to:

- Types and causes of shock.
- Define circulatory shock.
- Mechanisms responsible for the irreversible phase of hemorrhagic shock.
- Body compensatory mechanisms during reversible phases of hemorrhagic shock.
- Define shock and state the pathophysiological classification of shock.
- Describe the pathways leading to shock and decreased tissue perfusion.
- Discuss the stages of a hypovolemic shock.
- Explain how stage III hypovolemic shock might result in major organs failure.
- Discuss the different compensatory mechanisms during a hypovolemic shock.
- Describe the positive feedback mechanisms in the irreversible stage of a hypovolemic shock.

What is Shock ?

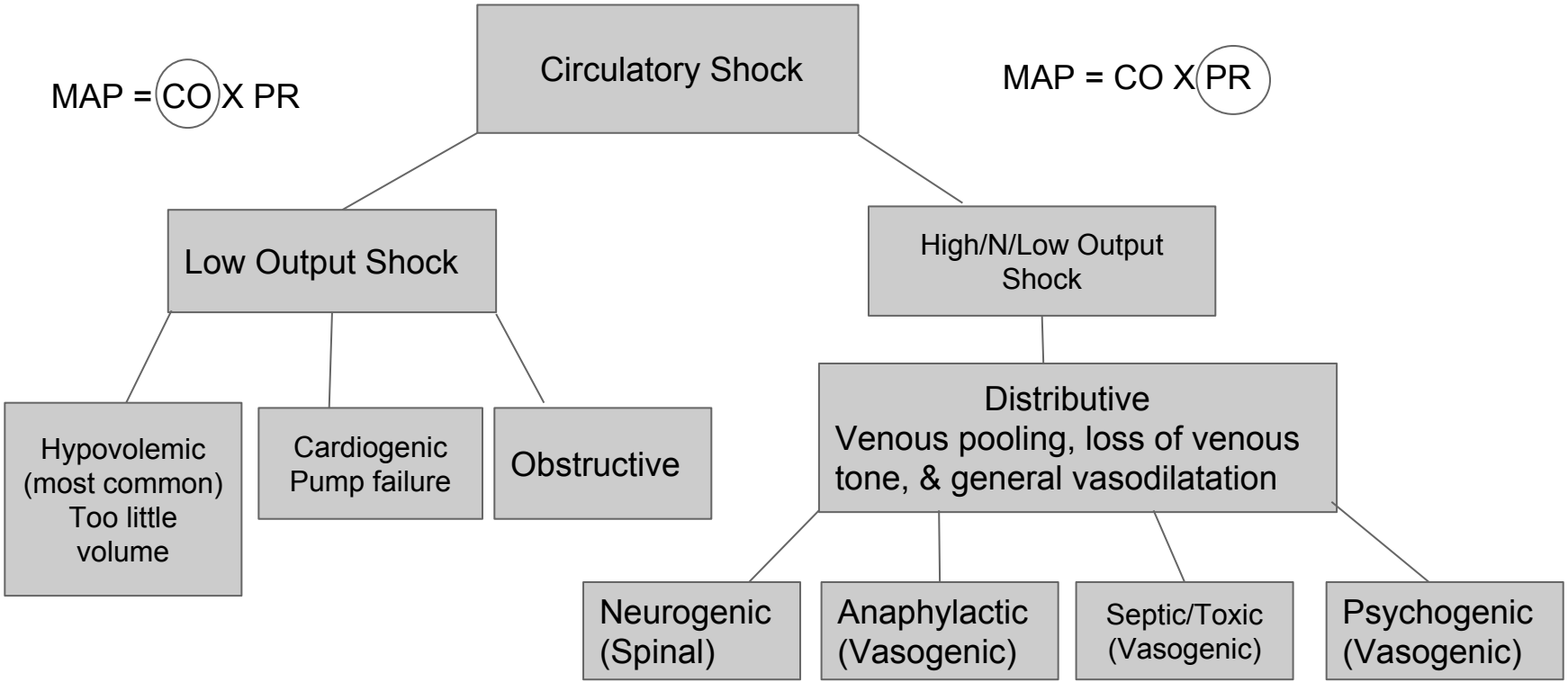
- Shock is defined as an acute circulatory failure leading to inadequate tissue perfusion and end organ injury.

- 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

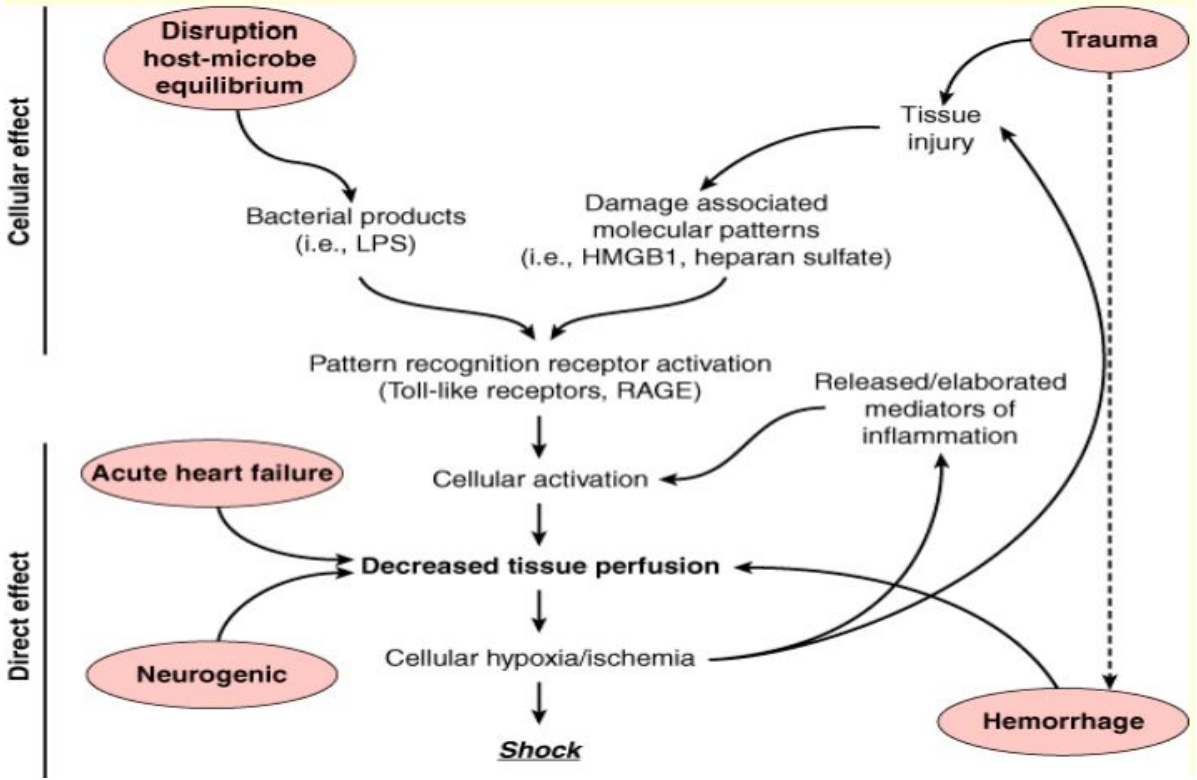


ONLY in female slides

Types of Circulatory Shock



What is Shock ?



Classification of Shock

1. Hypovolemic Shock

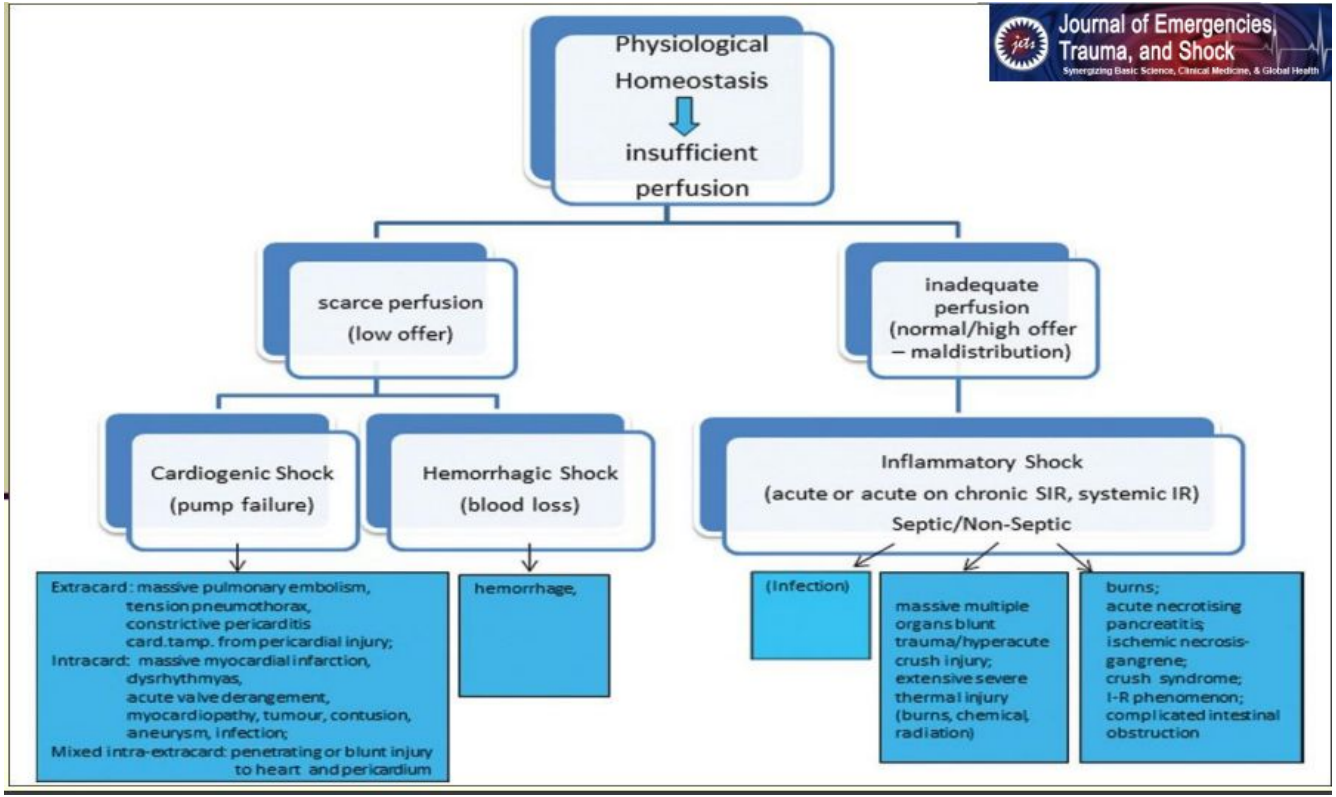
2. Cardiogenic Shock

3. Neurogenic Shock

4. Vasogenic Shock

- Anaphylactic shock
- Septic shock

Classification of Shock



Physiological Causes of Shock

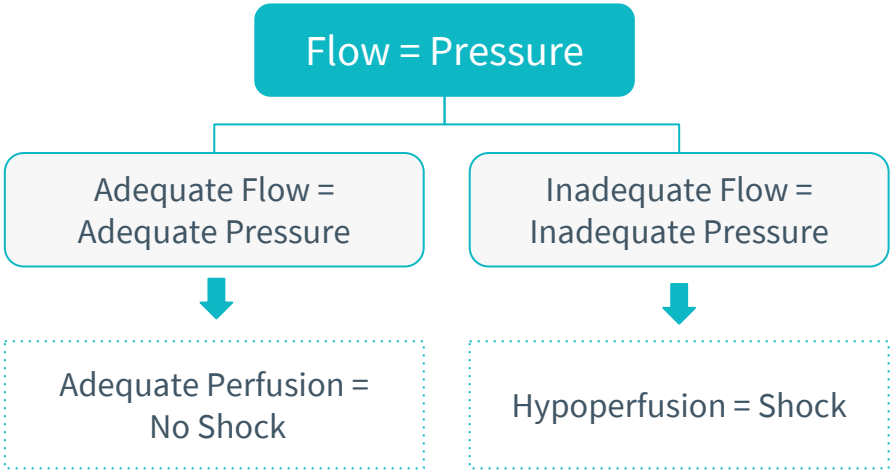
Circulatory shock caused by decreased cardiac output
Shock usually results from inadequate cardiac output.
Two types of factors can severely reduce cardiac output:

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



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

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

Inadequate Pump

- Inadequate preload
- Poor Contractility
- Excessive afterload
- Inadequate heart rate

Inadequate Fluid Volume

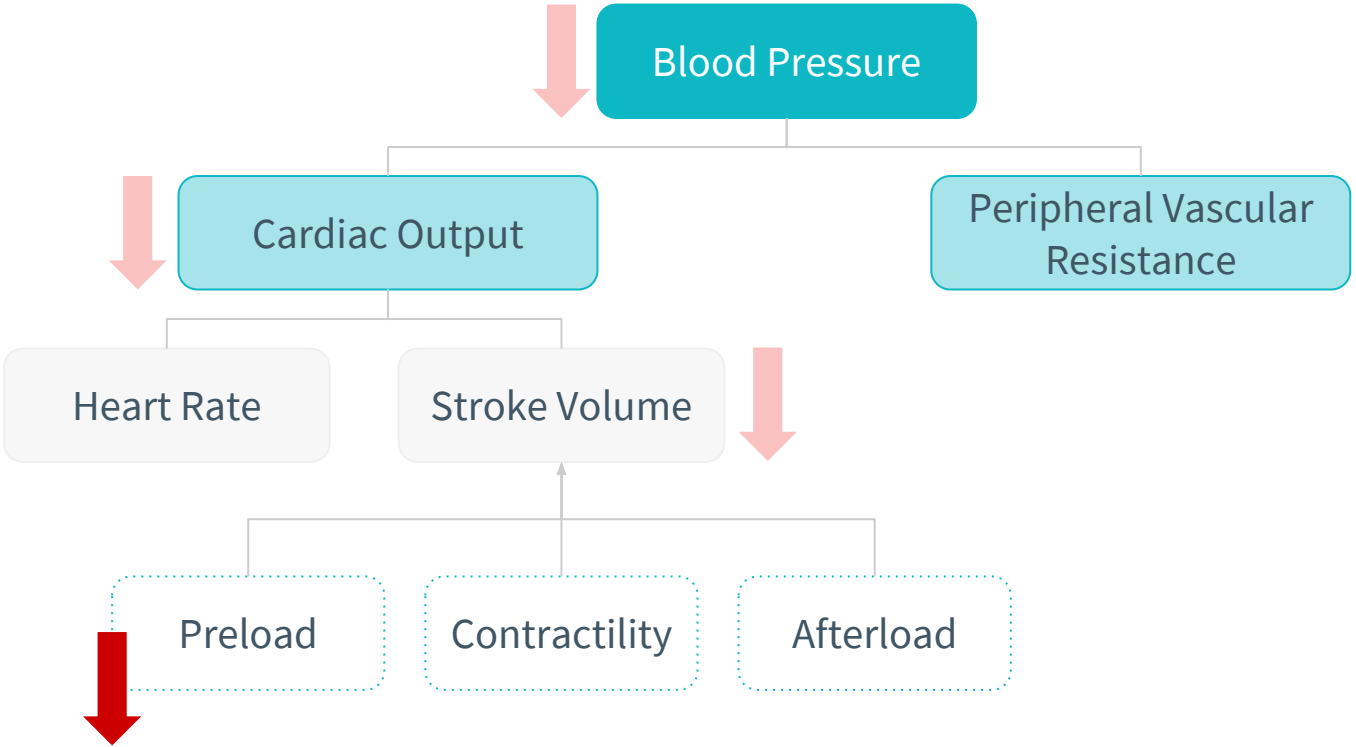
- Hypovolemia

Inadequate Container

- Excessive dilation
- Inadequate systemic vascular resistance

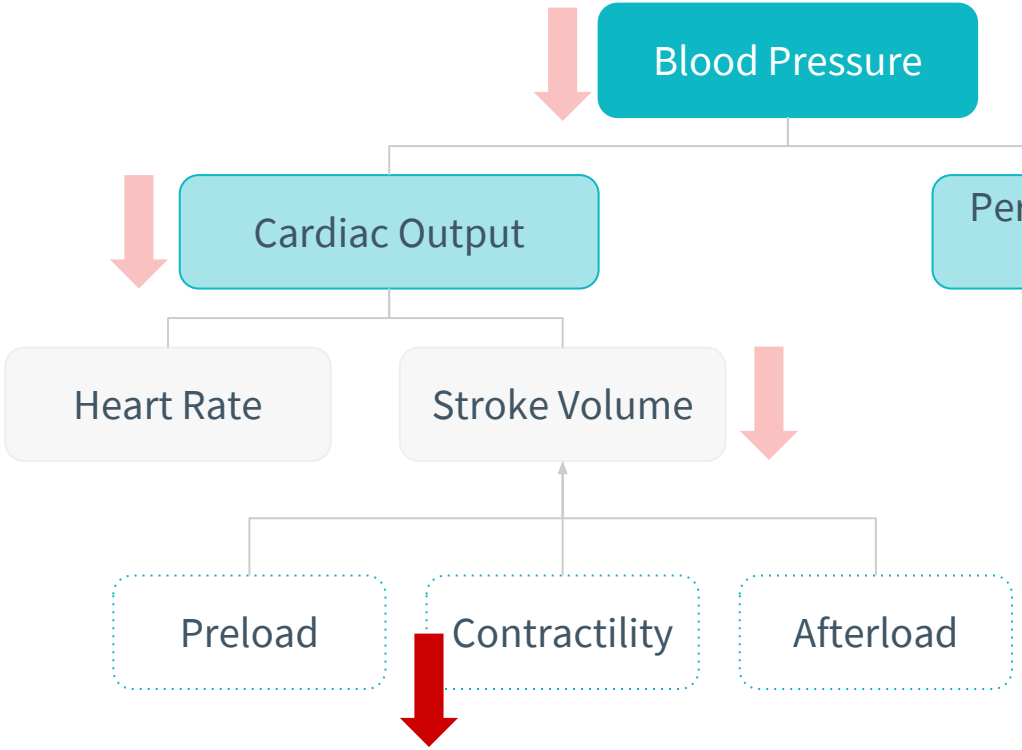
General Mechanism
Hypovolemic Shock (Hemorrhage, Dehydration)

ONLY in male slides



ONLY in male slides

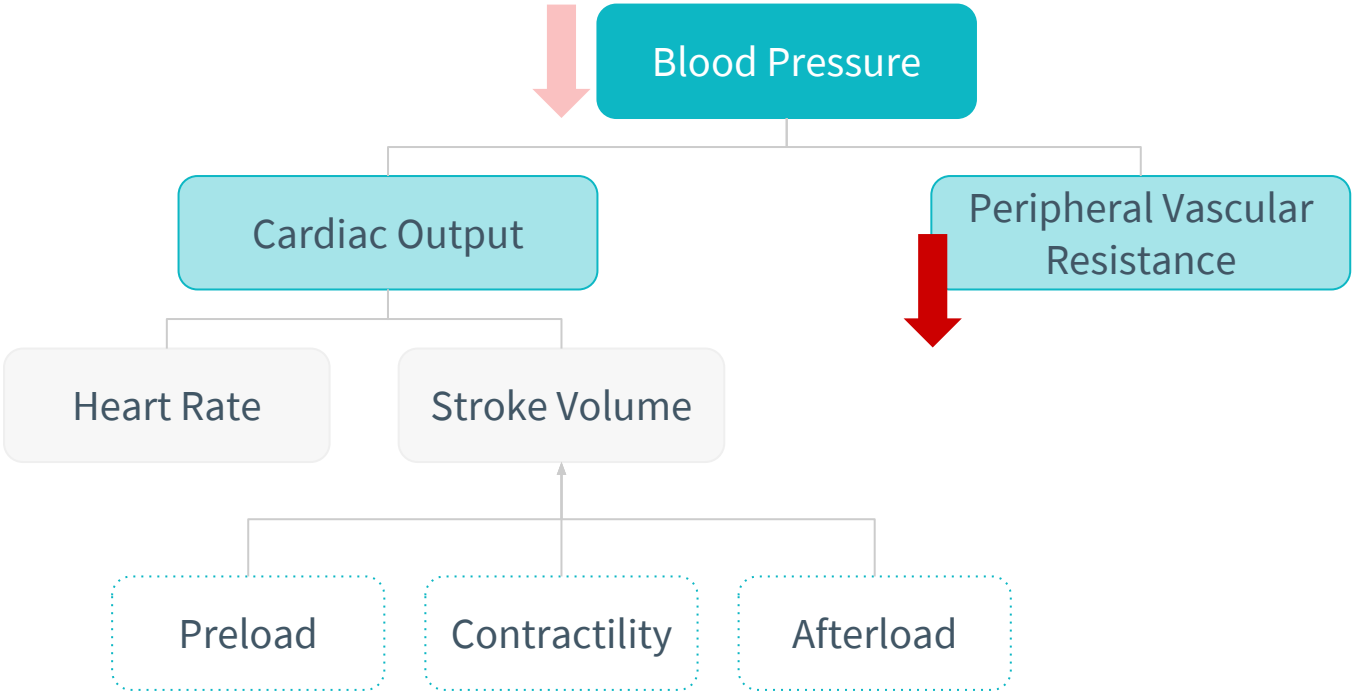
General Mechanism
Cardiogenic Shock (Post Extensive Myocardial Infarction)



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.

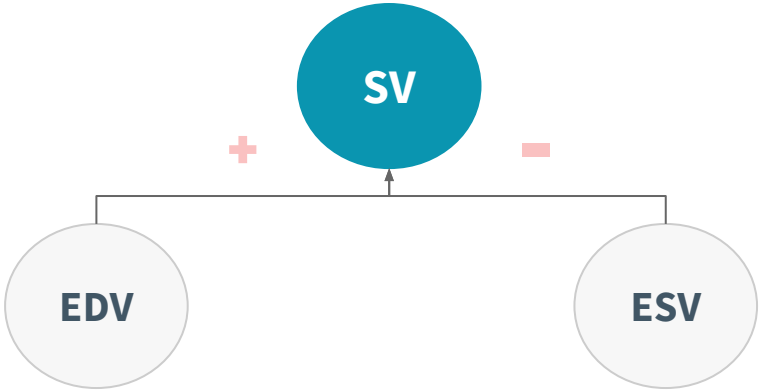
ONLY in male slides

General Mechanism
Hypotension Secondary to Anaphylactic Shock or Sepsis



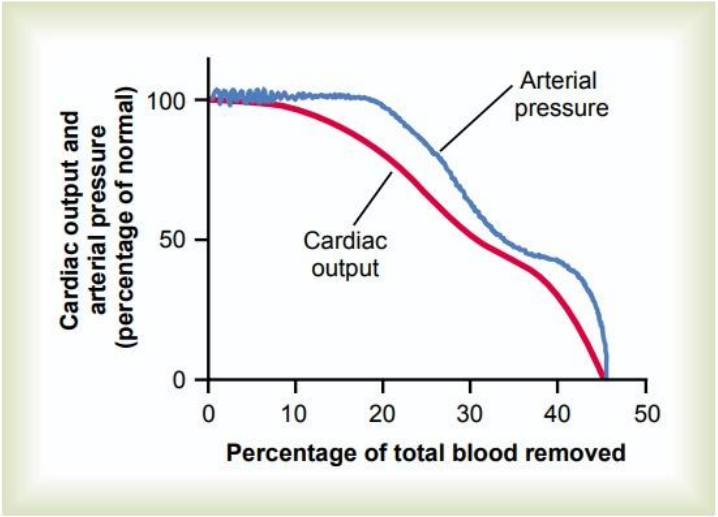
ONLY in male slides

General Mechanism



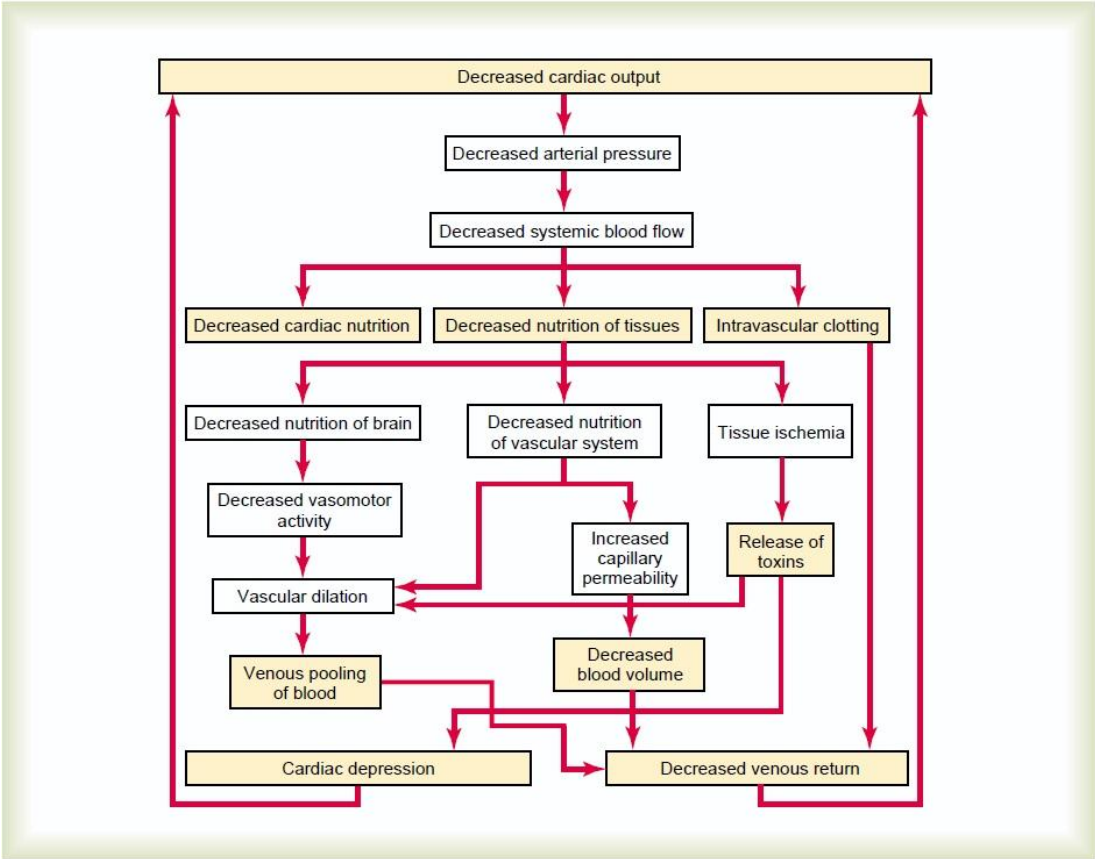
↑ Preload → ↑ SV (↑ EDV)
↑ Afterload → ↓ SV (↑ ESV)
↑ Inotropy → ↑ SV (↓ ESV)

Effect of hemorrhage on cardiac output and arterial pressure



General Mechanism

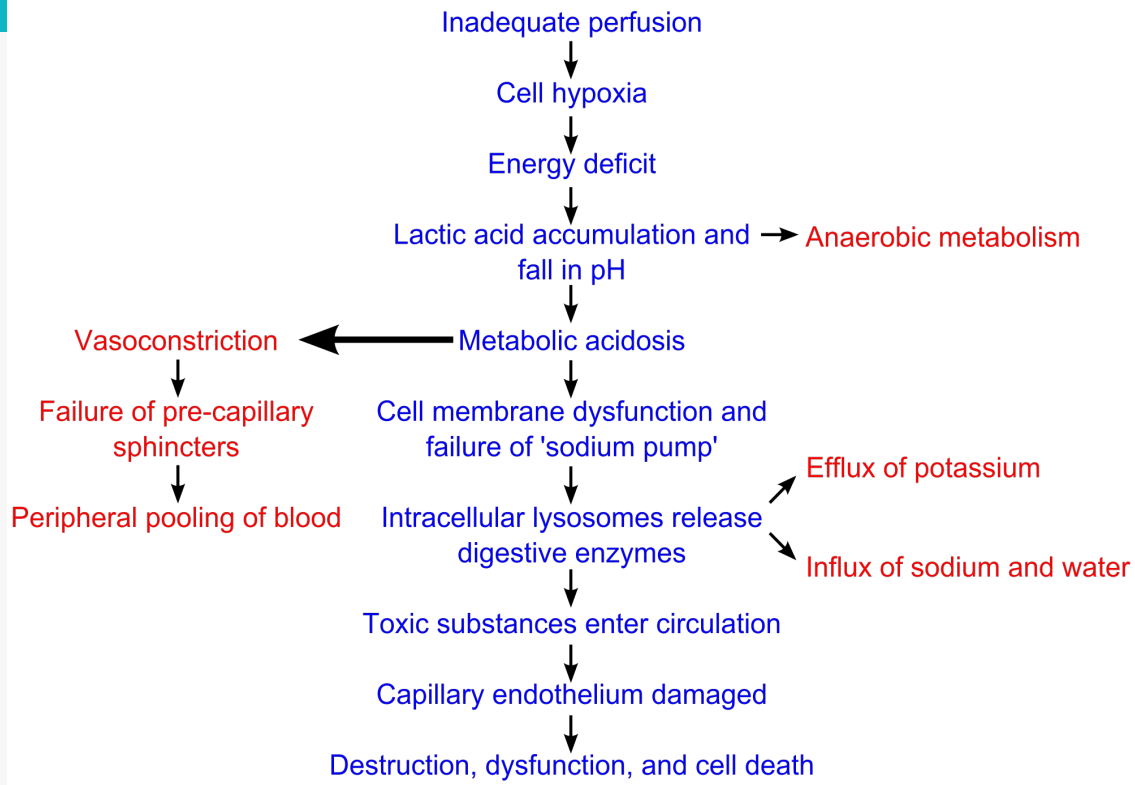
ONLY in male slides



Males' Version

Females' Version

General Mechanism



ONLY in female slides

Mechanical Changes and 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+] & [Ca2+]).
→ **Lysosomes, nuclear membranes & mitochondrial breakdown**.

2. After 3-5 hrs of shock:

→ precapillary sphincters dilate, venules are still constricted.
→ blood stagnation in capillaries.
→ hypoxia continue + fluid leaves to extravascular compartment.

→ further reduction in circulating blood volume.

3. Granulocytes accumulation at injured vessels:

→ **free radicals release.**
→ further tissue damage.

ONLY in female slides

Mechanical Changes and 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.

Stages of Shock

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

A non-progressive stage (Compensated stage)

The normal circulatory compensatory mechanisms eventually cause full recovery without help from outside therapy.

- Changes can be reversed by compensatory mechanism (neurohormonal activation) or by treatment.
- Defense mechanisms are successful in maintaining perfusion.
- Non-progressive.

A progressive stage

Without therapy, shock worse until death.

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

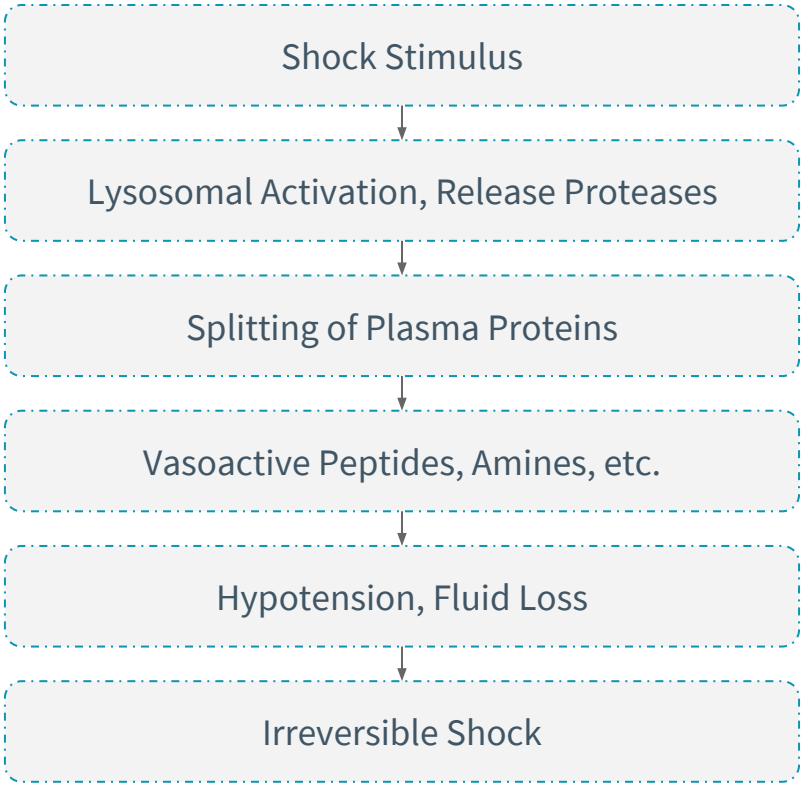
An irreversible stage

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.

- Complete failure of compensatory mechanisms.
- Can lead to death.

Possible Mechanisms that Lead to Developing Irreversible Shock

ONLY in female slides



HYPOVOLEMIC SHOCK

- Low CO due to:

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

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

i. Hematologic

ii. Cardiovascular

iii. Renal

iv. Neuroendocrine system.

CAUSES OF HYPOVOLEMIC SHOCK:

[Decreased Blood Volume]

1. Hemorrhage or blood loss : external or internal (commonest) [Trauma, GI bleed, ruptured aneurysm]
2. Surgery
3. Burns [Loss of plasma]
4. [Fluid loss] Vomiting, diarrhea, excess sweating, dehydration & trauma.

HYPOVOLEMIC 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.

- **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.

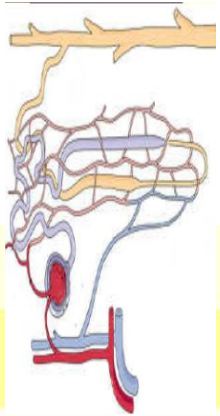
HYPOVOLEMIC SHOCK

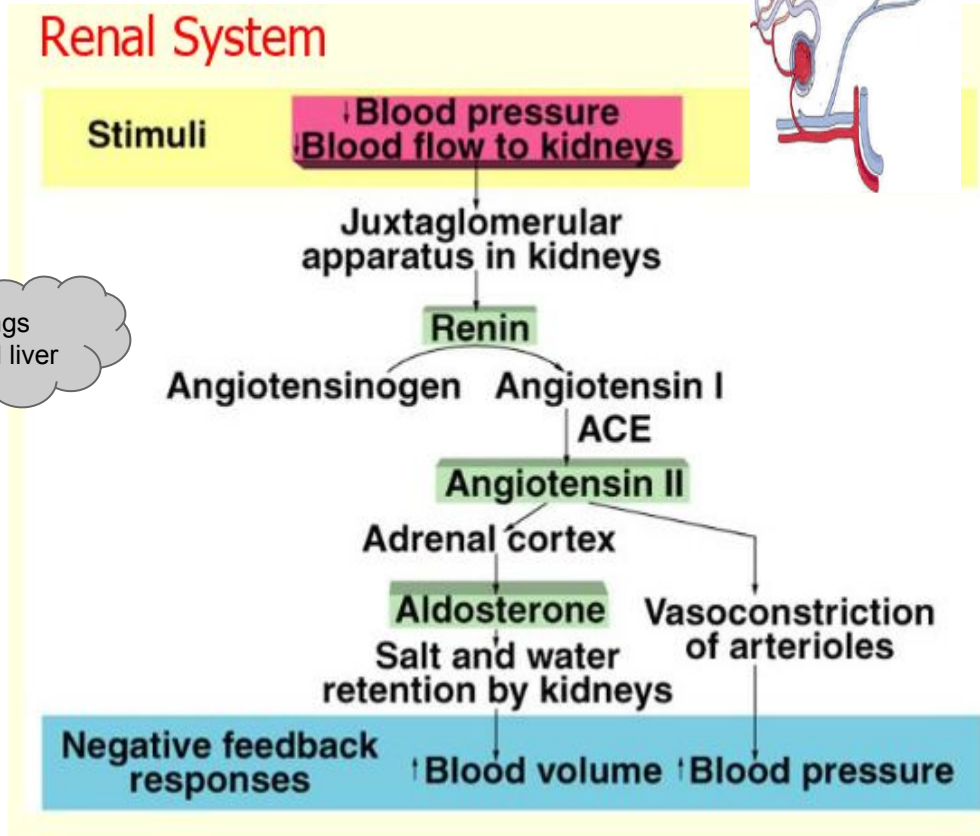
- **Renal system**
  - The kidneys respond to hemorrhagic shock by stimulating an increase in renin secretion from the juxtaglomerular apparatus.

Renin → angiotensinogen → angiotensin I → Angiotensin II

Lungs and liver

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

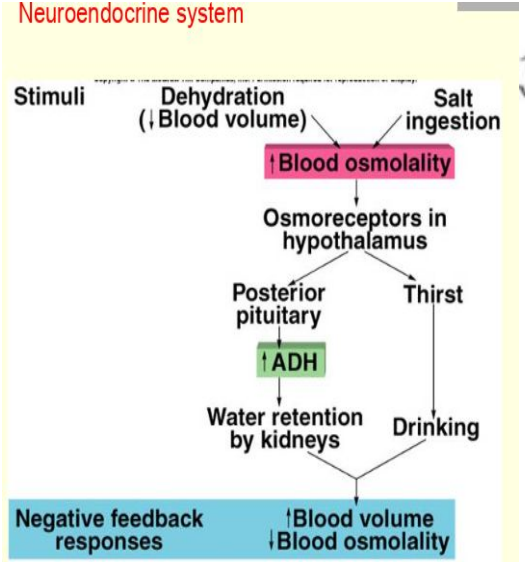


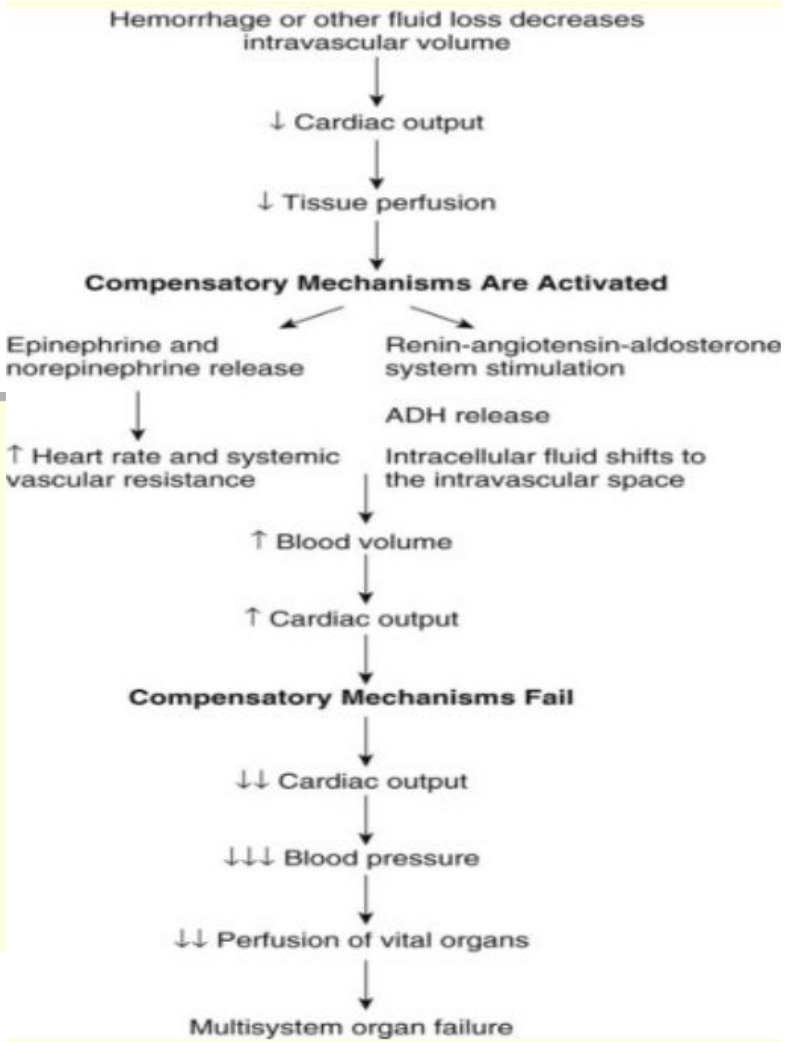


HYPOVOLEMIC SHOCK

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.





Hypovolemic Shock

- Hemorrhagic shock

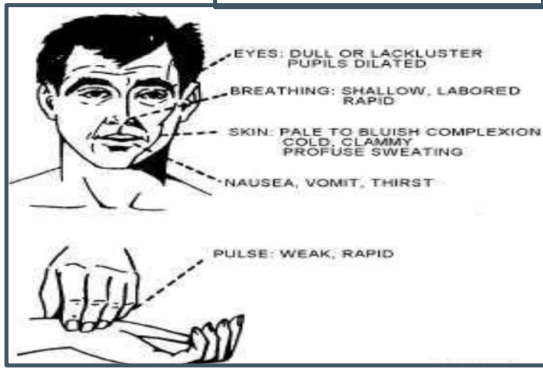
parameter	I	II	III	IV
Blood loss (mL)	<750	750-1500	1500-2000	>2000
Blood loss (%)	<15%	15-30%	30-40%	>40%
Pulse rate (beats/min)	<100	>100	>120	>140
BP	Normal	Decreased	Decreased	Decreased
Respiratory rate (bpm)	14-20	20-30	30-40	>35
Urine output (mL/hr)	>30	20-30	5-15	Negligible
CNS symptoms	Normal	Anxious	Confused	Lethargic
Fluid	Crystalloid	Crystalloid	Blood	Blood

Hypovolemic Shock: Clinical Features of Hypovolemic Shock

- Pale (due to hypoperfusion).
- Cold clammy skin (due to hypoperfusion).
- Sustained Hypotension (? ≤ 85/40 mmHg for 30 mins)
- Weak, rapid pulse (? 140/min)
- Tachycardia (sensed by baroreceptors in compensation to the ↓ MAP).
- Increased respiratory rate (sensed by chemoreceptors in compensation for hypoxia).
- Sweating
- Increased thirst
- Oliguria (low urine output) / Anuria (no urine output).
- Metabolic acidosis
- Restlessness (due to hypoperfusion).
- Blood test: lactic acidosis.

Vasoconstriction due to increased sympathetic stimulation

Clinical presentation



Type	Causes	Symptoms and signs
Hypovolemic shock	Bleeding (internal/external), dehydration (severe vomiting, severe 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 hypovolemic shock + distended jugular veins & may be absent pulse. Congestion of lungs & viscera: (CXR) 1) Interstitial pulmonary oedema. 2)Alveolar edema. 3) Cardiomegaly.
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 hypovolemic shock + distended jugular veins & pulsus paradoxus (in cardiac tamponade).
Distributive shock	Septic shock: infection ➜ release of bacterial toxins ➜ activation of NOS in macrophages ➜ production of NO ➜ vasodilation ➜ decreased vascular resistance ➜ hypotension.	Septic shock: hypotension; fever; warm due to his hyperdynamic state , sweaty skin.
Vasogenic	Anaphylactic shock: allergy (release of histamine ➜ vasodilation ➜ decreased vascular resistance ➜ hypotension.	Anaphylactic shock: skin eruptions; breathlessness, coughing; localized edema; weak, rapid pulse.
Low-resistance shock	Neurogenic shock: spinal injury ➜ loss of autonomic and motor reflexes ➜ reduction of peripheral vasomotor tone ➜ vasodilation ➜ decrease in peripheral vascular resistance ➜ hypotension.	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

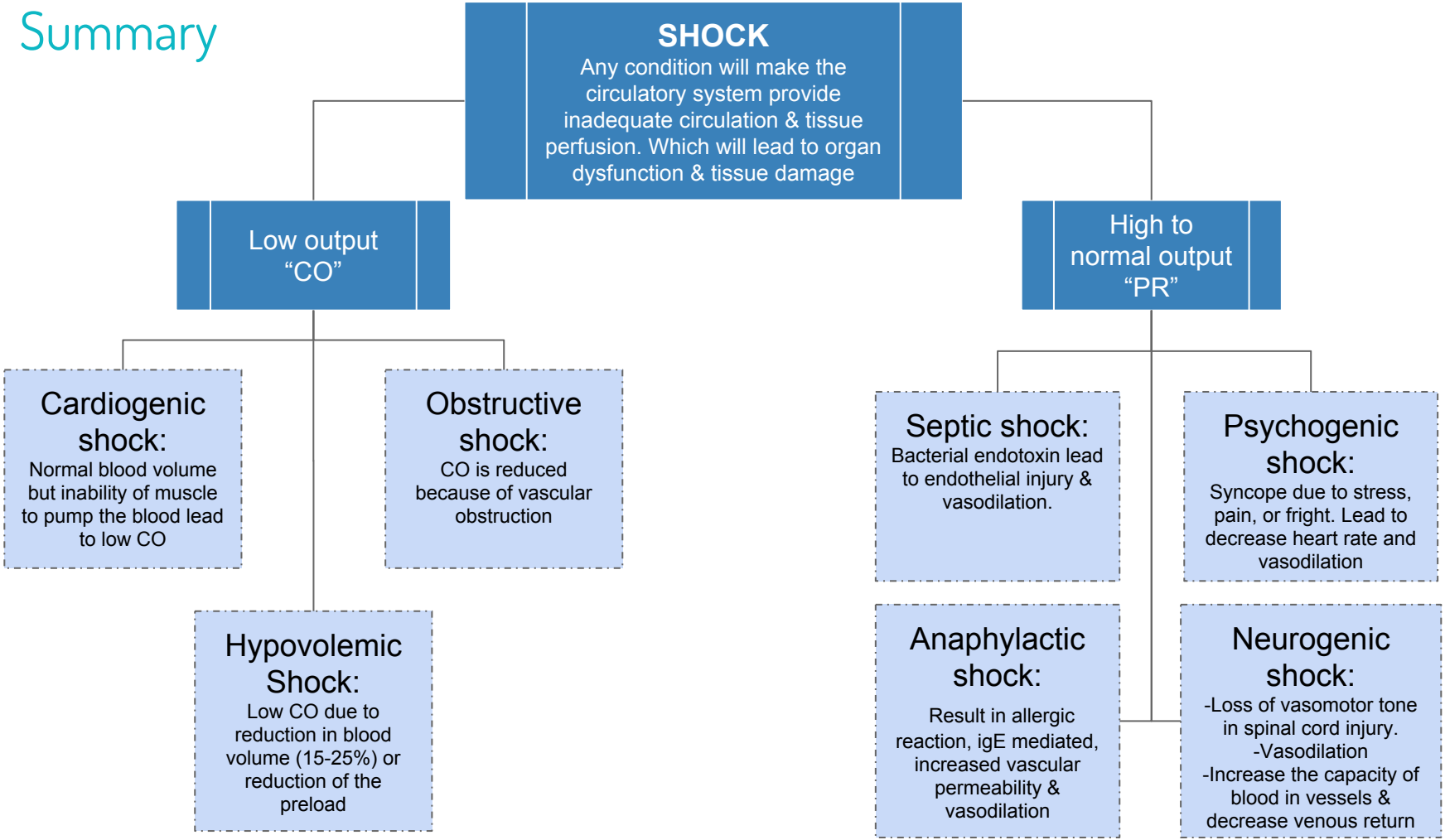
- Hypovolemic - fluid resuscitate (blood or crystalloid) and control ongoing loss.
- Cardiogenic - restore blood pressure (chemical and mechanical) and prevent ongoing cardiac death.
- Distributive - fluid resuscitate, immediate surgical control for infection.

Treatment of Shock

Goal: restore normal tissue perfusion.

- Blood pressure, pulse and respirations.
- Skin appearance.
- Urine output (30-50 cc per hour).
- Hemoglobin 8-10 gm or hematocrit 24-30.
- 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

Summary


SHOCK
Any condition will make the circulatory system provide inadequate circulation & tissue perfusion. Which will lead to organ dysfunction & tissue damage
Low output "CO"
High to normal output "PR"
Cardiogenic shock:
Normal blood volume but inability of muscle to pump the blood lead to low CO
Obstructive shock:
CO is reduced because of vascular obstruction
Hypovolemic Shock:
Low CO due to reduction in blood volume (15-25%) or reduction of the preload
Septic shock:
Bacterial endotoxin lead to endothelial injury & vasodilation.
Psychogenic shock:
Syncope due to stress, pain, or fright. Lead to decrease heart rate and vasodilation
Anaphylactic shock:
Result in allergic reaction, igE mediated, increased vascular permeability & vasodilation
Neurogenic shock:
-Loss of vasomotor tone in spinal cord injury.
-Vasodilation
-Increase the capacity of blood in vessels & decrease venous return

Quiz

- Which of the following is the cause of cardiogenic shock:

A-vena cava syndrome
B-acute valvular dysfunction
C-bacterial endotoxin

Answer : B

- Anaphylactic shock is mediated by which type of immunoglobulin?

A- igA
B- igG
C- igE

Answer : C

- Which of the following occurs after 3-5 hours of shock:

A- precapillary sphincters dilate
B- precapillary sphincters spasm
C- syncope

Answer : A

- Which of the following is compensatory mechanism effect on sympathetic system:

A- Thirst stimulation
B- Na+ retention
C- Chemoreceptors

Answer : C

- The highest point of hydrostatic pressure located in:

A- arterial end
B- venous end
C- at the center

Answer : A

- Which of the following the septic shock have the others don't in signs & symptoms:

A- cardiomegaly
B- cold & pale skin
C- warm & flushed skin

Answer : C

Thank you for checking our work

Team Leader:

العنود سلمان

Male Team:

أنس السويداء
أنس السيف
خالد شويل
ريان الموسى
سعد الهداب
سعود العطوي
سيف المشاري
عبدالجبار اليماني
عبدالرحمن آل دحيم
عمر الفوزان
فهد الحسين
نايف المطيري
نواف اللويمي
محمد الحسن
هشام الشايع
خالد العقيلي
سعد الفوزان
عبدالله الزيد
نواف اللويمي
عبدالمجيد الوردي
يزيد الدوسري

Female Team:

الآء الصويّغ
رناد المقرن
رهف الشنيبر
روان التميمي
روان مشعل
ريم القرني
ليلى الصباغ
فلوة السعوي
نورة بن حسن
نورة الحربي
نورة العثيم
مجد البراك
لينا العوهلي
عهد القرين
مها النهدي
مها بركة
سارة الفليج
هند العريعر
ريناد الغريبي
عائشة الصباغ
ميعاد النفيعي
سمية العقيفي
سارة البليهد

Any questions?

Contact us at

- twitter:@physio437
- physiologyteam437@gmail.com