

# Physiology of Shock

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# Overview

- Introduction
- Classification
- Overview of types of shock
- Physiological basis
- Compensated vs decompensated shock

# What is shock?



• VS



# What is shock?

- First described by Le Dran a French surgeon in 1737
- Shock is a state of peripheral circulatory failure that is characterized by inadequate perfusion of the peripheral tissues.
- During shock, the *systolic* arterial pressure is usually <90 mm Hg, and the *mean* arterial pressure is <70 mmHg
- Leads to organ failure and death



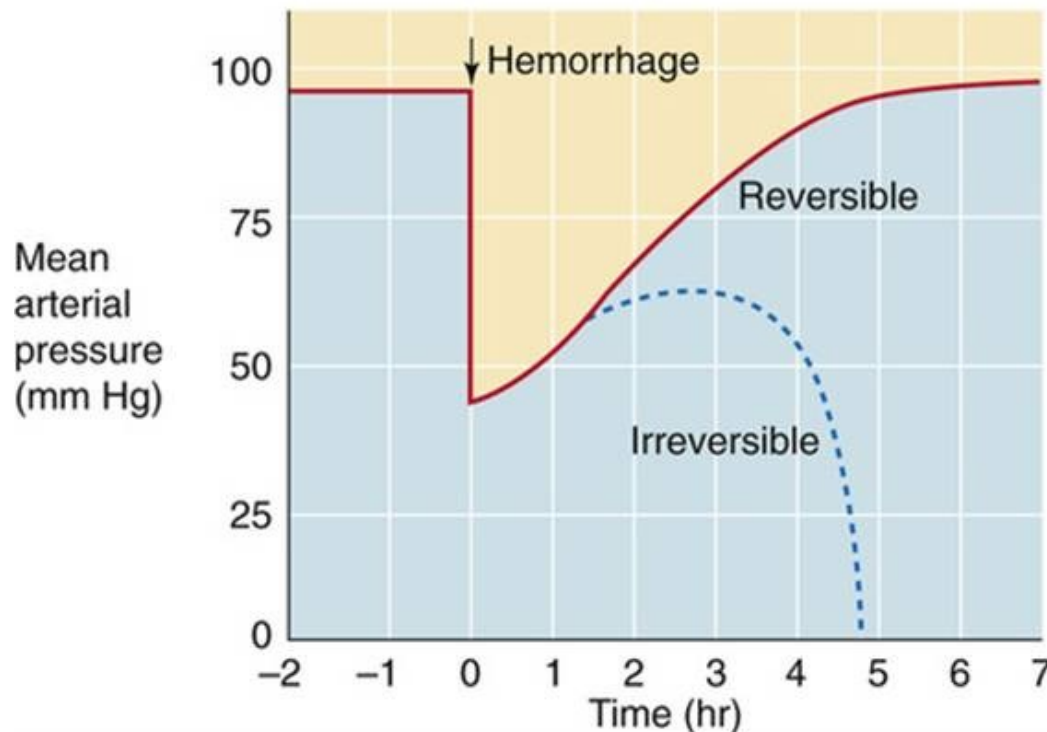
# Classification of shock

- Hypovolemic shock
- Cardiogenic shock
- Obstructive shock
- Distributive shock
  - Neurogenic shock
  - Septic Shock
  - Anaphylactic shock

# Hypovolaemic shock

- Large hemorrhages, in which one loses 30% or more of total blood volume produce **hypovolemic shock**.
- Clinical features
  - narrowing of the pulse pressure
  - sensation of faintness when sitting or standing.
  - cold and moist (i.e., “clammy”) skin
  - rapid and weak pulse
  - urine output drops to  $<25$  mL/hr, even if fluid intake had been normal

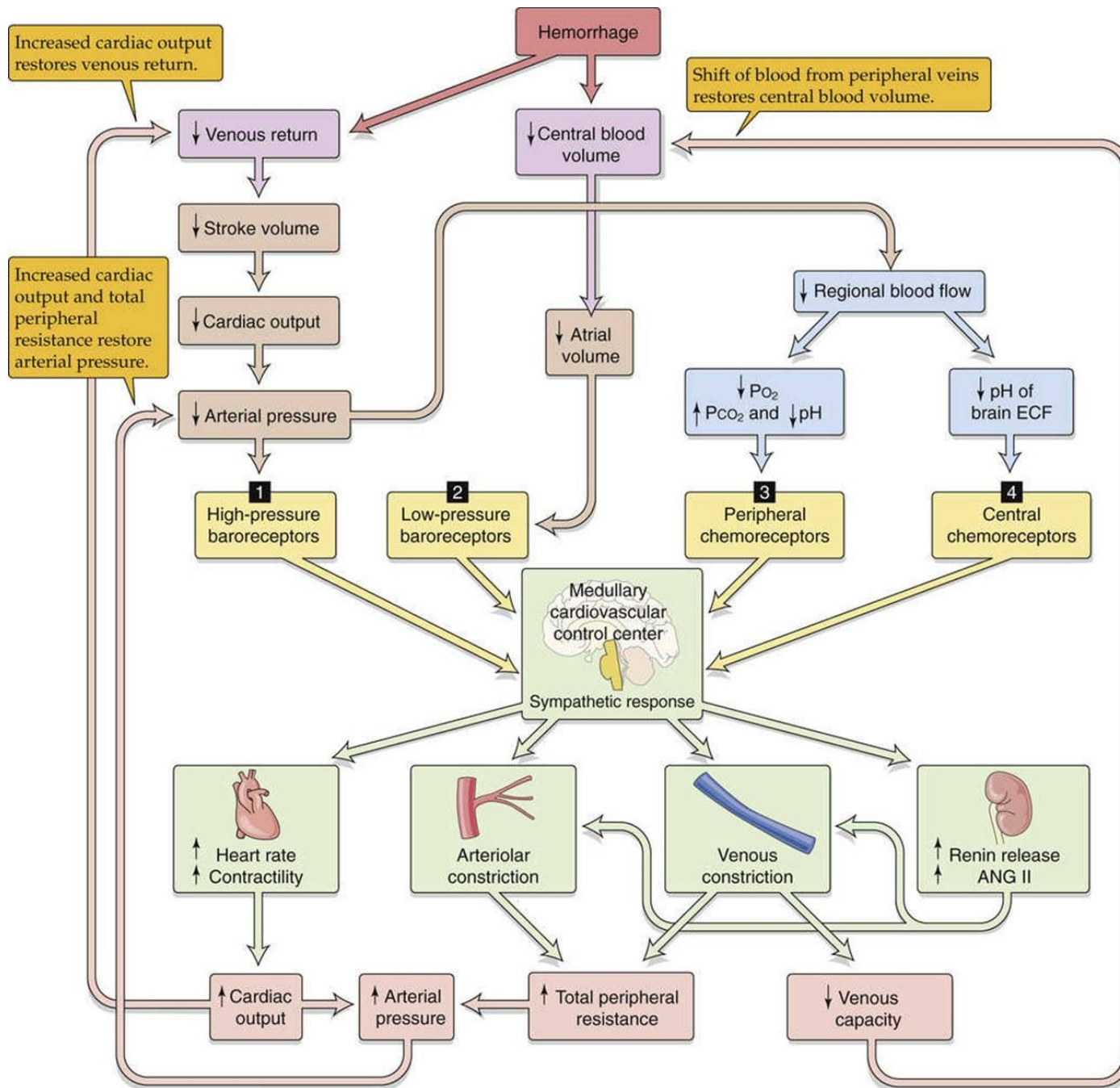
- After its abrupt initial fall, arterial pressure tends to return to normal
- Although blood pressure falls irreversibly in some cases



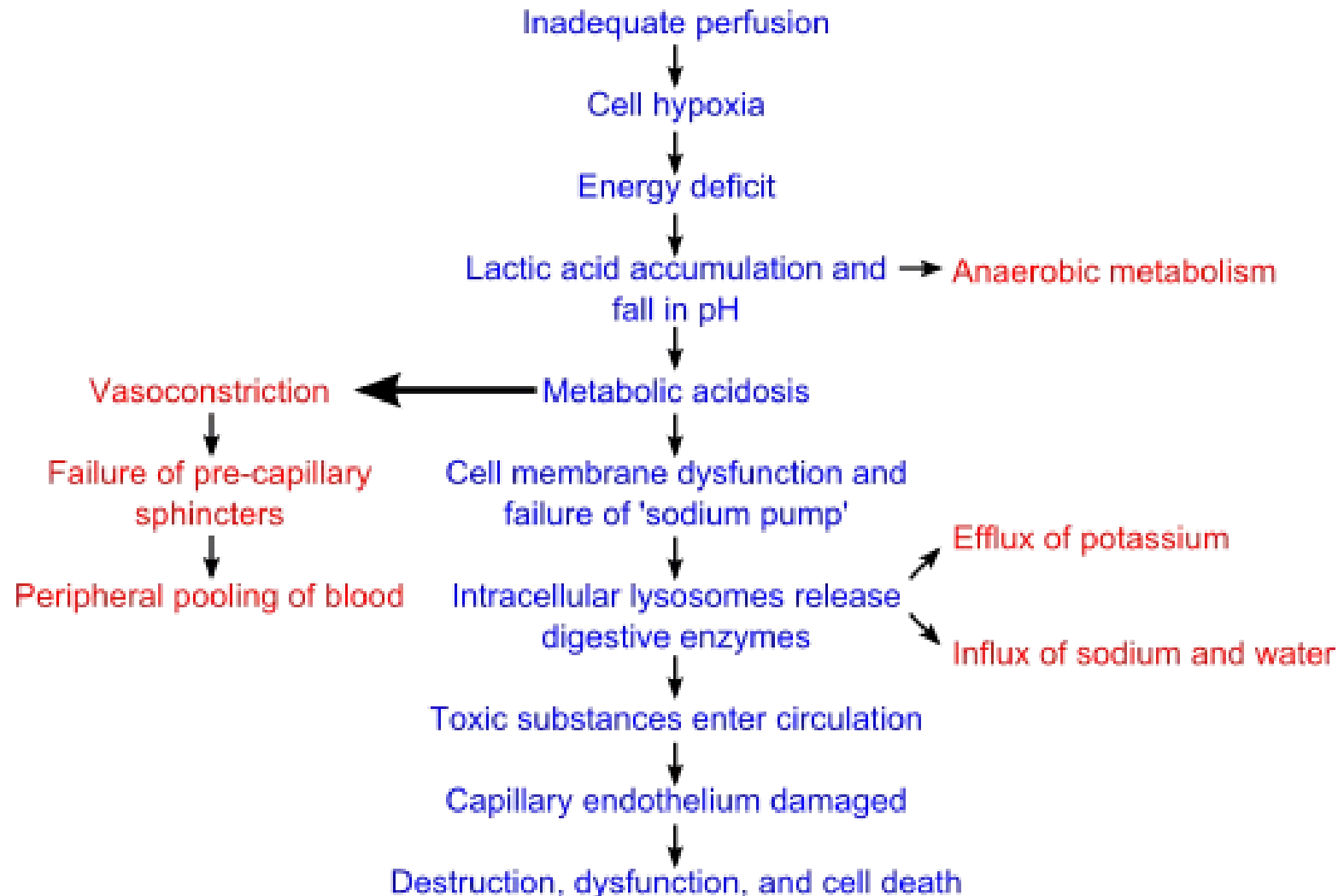
# How to restore blood pressure?

- First - circulatory control mechanisms act on the heart and blood vessels to restore cardiac output and to increase peripheral resistance.
- Second- mechanisms of capillary exchange and fluid conservation restore the intravascular volume.





# Cellular level changes



# Hypovolemic Shock -Causes

## ✚ Hemorrhagic

- Trauma
- Gastrointestinal
- Retroperitoneal

## ✚ Fluid depletion (nonhemorrhagic)

- External fluid loss
  - Dehydration
  - Vomiting
  - Diarrhea
  - Polyuria
- Interstitial fluid redistribution
  - Thermal injury
  - Trauma
  - Anaphylaxis

# Hypovolemic Shock Hemodynamics

	CO	SVR	PWP	EDV
Hypovolemic	↓	↑	↓	↓
Cardiogenic	↓	↑	↑	↑
Obstructive	↓	↑↑	↑	↑
afterload	↓	↑	↑	↓
preload	↓	↑	↓	↓
Distributive	↓	↑	↓	↓
pre-resusc	↑	↓	↑	↑
post-resusc				

# Case 1

- 34 year old involved in a motor vehicle accident arrived to emergency room with blood pressure of 70/30 and heart rate of 140/min



# Question

- **Which is typical of hypovolemic shock?**

(a) High SVR

(b) High cardiac output

(c) High oxygen delivery

(d) Normal wedge pressure

# Cardiogenic shock

Reduced cardiac function from direct myocardial damage or mechanical abnormality leading to reduced CO and BP

# Cardiogenic Shock



## Myopathic

- Myocardial infarction (hibernating myocardium)
- Left ventricle/ Right ventricle



## Myocardial contusion (trauma)



## Myocarditis



## Cardiomyopathy



## Post-ischemic myocardial stunning



## Septic myocardial depression



## Pharmacologic

- Anthracycline cardiotoxicity
- Calcium channel blockers



## Mechanical

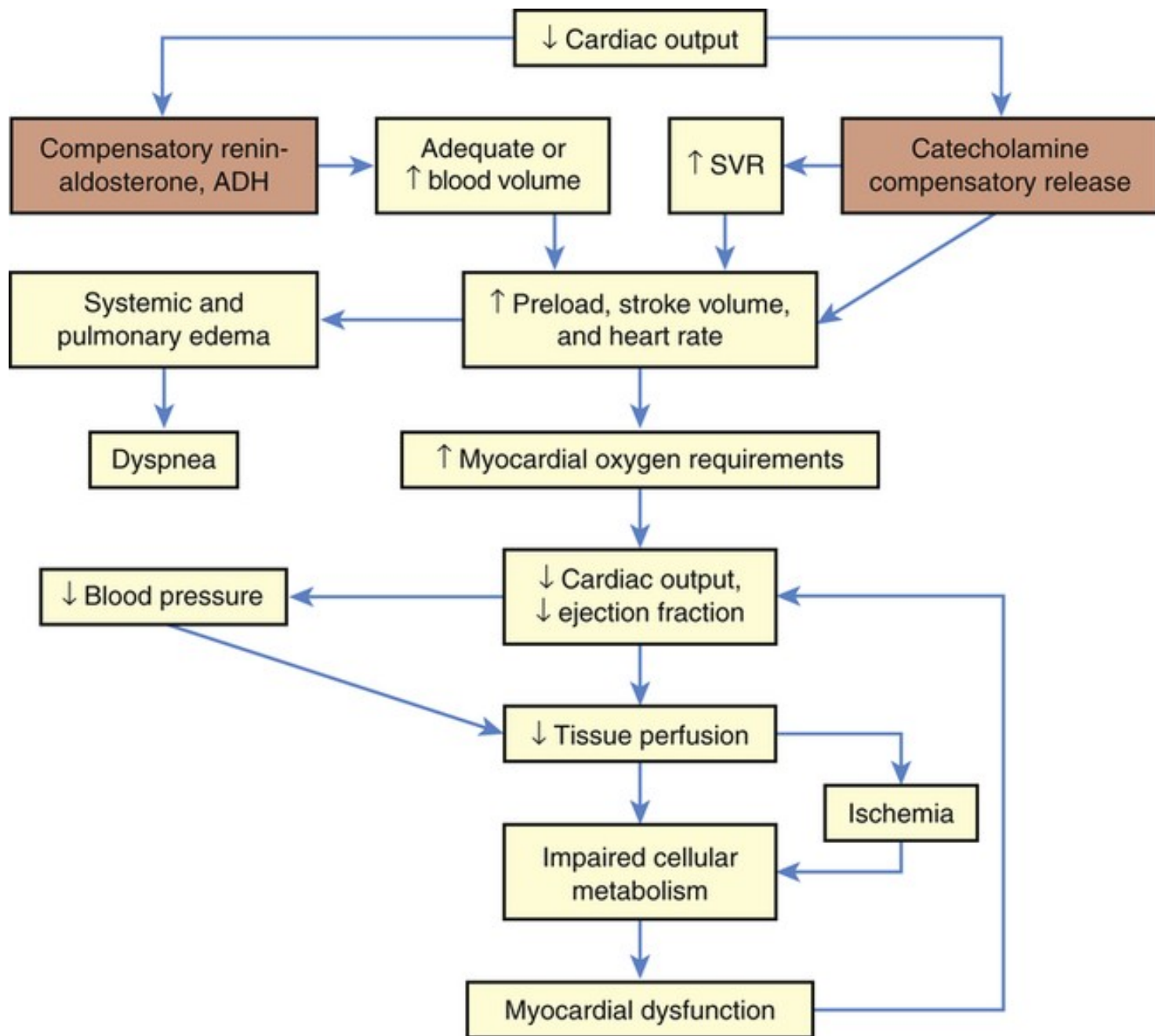
- Valvular failure (stenotic or regurgitant)
- Hypertrophic cardiomyopathy
- Ventricular septal defect



## Arrhythmic

- Bradycardia
- Tachycardia





# Cardiogenic Shock Hemodynamics

	CO	SVR	PWP	EDV
Hypovolemic	↓	↑	↓	↓
Cardiogenic	↓	↑	↑	↑
Obstructive				
afterload	↓	↑↑	↑	↑
preload	↓	↑	↑	↓
Distributive				
pre-resusc	↓	↑	↓	↓
post-resusc	↑	↓	↑	↑

# Case 2

- 54 year old with acute onset chest pain arrived to emergency room with blood pressure of 70/30 and heart rate of 140/min



# Question

**Which is typical of cardiogenic shock?**

- (a) Low SVR
- (b) High cardiac output
- (c) Low oxygen delivery
- (d) Low wedge pressure

# Obstructive shock

- Secondary to obstruction to the cardiovascular unit
- Leads to inadequate diastolic filling or decreased systolic function secondary to increase in after load
- Leads to reduce CO and BP

# Extracardiac Obstructive Shock

## Impaired diastolic filling (decreased ventricular preload)

- Direct venous obstruction (vena cava)
  - Intrathoracic obstructive tumors
- Increased intrathoracic pressure
  - Tension pneumothorax
  - Mechanical ventilation (with excessive pressure or volume depletion)
  - Asthma
- Decreased cardiac compliance
  - Constrictive pericarditis
  - Cardiac tamponade

## Impaired systolic contraction (increased ventricular afterload)

- Right ventricle
  - Pulmonary embolus (massive)
  - Acute pulmonary hypertension
- Left ventricle
  - Saddle embolus
  - Aortic dissection

# Obstructive Shock Hemodynamics

	CO	SVR	PWP	EDV
Hypovolemic	↓	↑	↓	↓
Cardiogenic	↓	↑	↑	↑
Obstructive				
afterload	↓	↑↑	↑	↑
preload	↓	↑	↑	↓
Distributive				
pre-resusc	↓	↑	↓	↓
post-resusc	↑	↓	↑	↑

# Distributive shock

- Characterized by vasodilation leading to decrease in preload leading to hypotension with a normal or increased cardiac output
- Myocardial depression occurs



# Distributive Shock

- Septic (bacterial, fungal, viral, rickettsial)
- Toxic shock syndrome
- Anaphylactic, anaphylactoid
- Neurogenic (spinal shock)
- Endocrinologic
  - Adrenal crisis
  - Thyroid storm
- Toxic (e.g., nitroprusside, bretylium)

# Distributive Hemodynamics

	CO	SVR	PWP	EDV
Hypovolemic	↓	↑	↓	↓
Cardiogenic	↓	↑	↑	↑
Obstructive	↓	↑↑	↑	↑
afterload	↓	↑	↑	↓
preload	↓	↑	↑	↓
Distributive	↓	↑	↓	↓
pre-resusc	↓	↑	↓	↓
post-resusc	↑	↓	↑	↑

# Septic shock

- Presence of infection associated with a systemic inflammatory response
- Results in physiologic alterations at the capillary endothelial level.

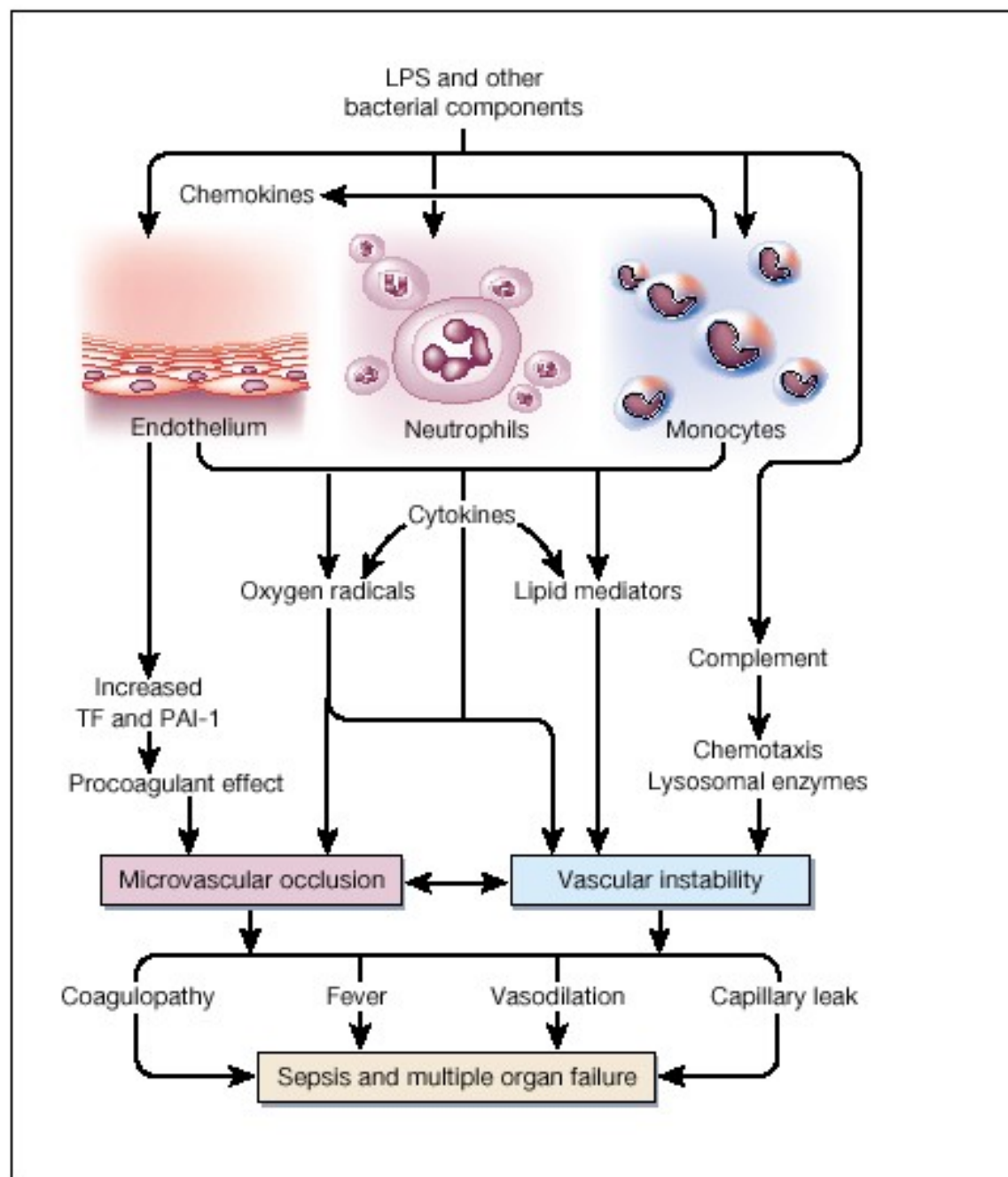
# Septic shock-Features

- High cardiac output
- Hypotension
- A large pulse pressure
- A low diastolic pressure
- Warm extremities with good capillary refill.

These findings on physical examination strongly suggest a working diagnosis of septic shock.

# Physiological basis

- The normal physiologic response to localized infection includes
  - activation of host defence mechanisms that result in the influx of activated neutrophils and monocytes
  - release of inflammatory mediators
  - local vasodilation
  - increased endothelial permeability
  - activation of coagulation pathways



# Case 3

- 67 year old with fever, chills, SOB, and ugly looking abdominal wound arrived to emergency room with blood pressure of 70/30 and heart rate of 140/min



# Question

- Which is not typical of sepsis?
- (a) Low SVR
- (b) High cardiac output
- (c) Low oxygen delivery
- (d) Low wedge pressure



# Anaphylactic shock

- Anaphylaxis is an acute, potentially fatal, multiorgan system reaction caused by the release of chemical mediators from mast cells and basophils.

# Agents which can cause anaphylaxis

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Antibiotics and other drugs

Foreign proteins (e.g., horse serum. Streptokinase)

Foods (nuts, eggs, shrimp)

Allergen extracts (allergy shots)

*Hymenoptera* stings (bees, wasps)

Hormones (Insulin)

Blood Products

Aspirin

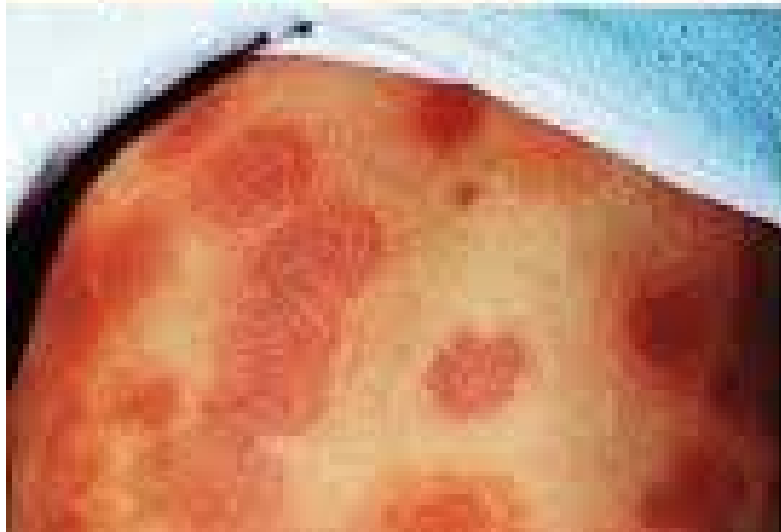
Non-steroidal anti-inflammatory drugs (NSAIDs)

Preservatives (sulfiting agents)

X-ray contrast media

Dextran

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# Physiological basis

- The physiologic responses to the release of anaphylaxis mediators include
  - smooth muscle spasm in the respiratory and gastrointestinal (GI) tracts,
  - vasodilation
  - increased vascular permeability
  - and stimulation of sensory nerve endings.
  - Increased mucous secretion
  - Increased bronchial smooth muscle tone
  - Airway edema

# Physiological basis ctd...

- Cardiovascular effects result from decreased vascular tone and capillary leakage.
- Hypotension, cardiac arrhythmias, **syncope**, and **shock** can result from intravascular volume loss, vasodilation, and myocardial dysfunction.
- Increased vascular permeability can produce a shift of 35% of vascular volume to the extravascular space within 10 minutes.

# Neurogenic shock

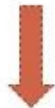
- Rarest form of shock
- Caused by spinal cord trauma
- Sudden loss of autonomic and motor reflexes below the injury level
- Sudden loss of peripheral vascular resistance
- Leads to vasodilatation and hypotension

# Neurogenic Shock

Damage to the brain and spinal cord



Loss of Sympathetic Tone:



Parasympathetic Nervous  
System is Unopposed



Uncontrolled Vasodilation



Low Blood Pressure



Hypoperfusion: Shock

# Physiological basis

- **Hypotension**
- Loss of sympathetic tone to the arterial system leads to vasodilation
- Loss of sympathetic tone to the venous system leads to pooling of blood in venous capacitance vessels causing reduced cardiac filling and CO
- **Bradycardia**
- Loss of sympathetic input from spinal cord
- Tonic parasympathetic tone is unopposed to heart



# Phases of shock

- **Compensated**
  - Intrinsic regulatory mechanisms
  - Vital organ function is maintained
- **Decompensated**
  - Compromise of microvascular circulation
  - Deterioration of organ function
  - Hypotension develops
- **Refractory**
  - Damage to key organs

	compensated	Mild	Moderate	severe
Level of consciousness	Normal	Mild anxiety	Drowsy	comatose
Blood pressure	Normal	Normal	Mild hypotension	Severe hypotension
Pulse rate	Mild increase	Increased	Increased	Increased
Respiratory rate	Normal	Increased	Increased	laboured
Urine output	Normal	Normal	Reduced	Anuric
Lactic acidosis	+	++	++	+++

$$BP = SVR \times \underline{CO}$$

Anaphylactic Shock  
Neurogenic Shock  
Septic Shock  
Vasodilator Drug  
Induced Shock

$$\underline{HR} \times \underline{SV}$$

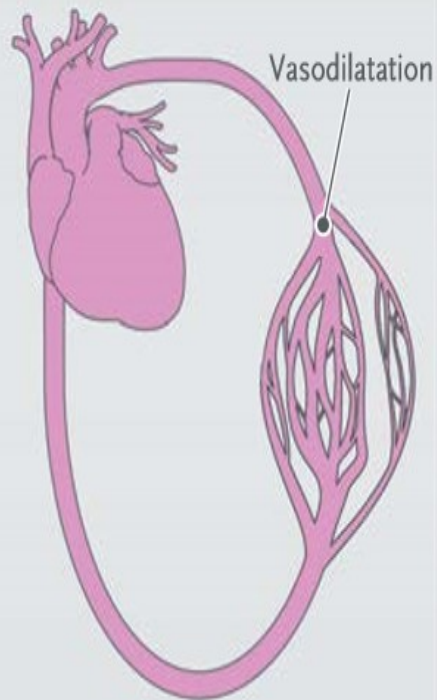
Tachycardia Extremes  
Bradycardia Extremes

$$\underline{EDV} - \underline{ESV}$$

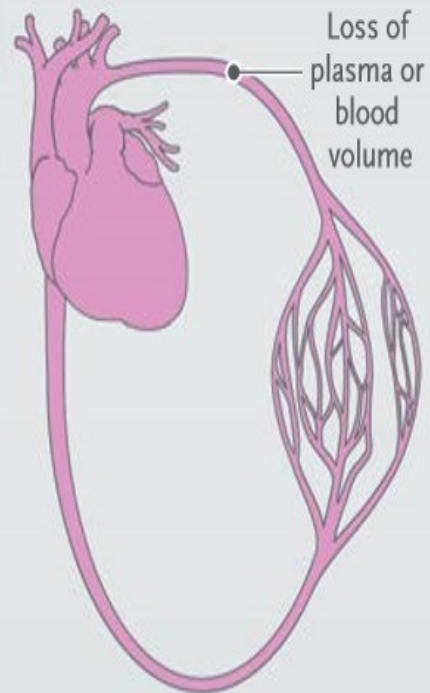
Hypovolemic Shock

Cardiogenic Shock

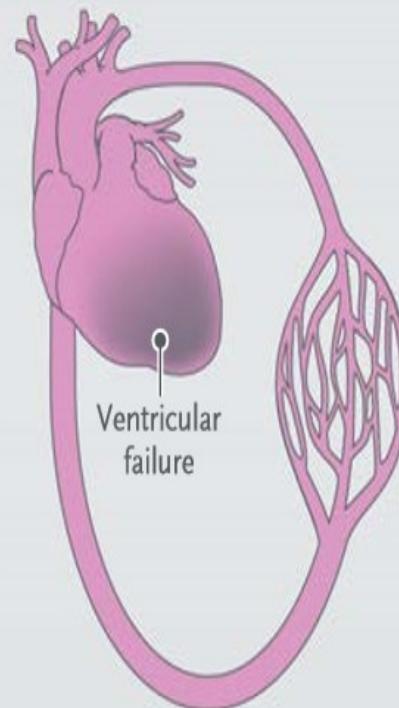
### Distributive shock



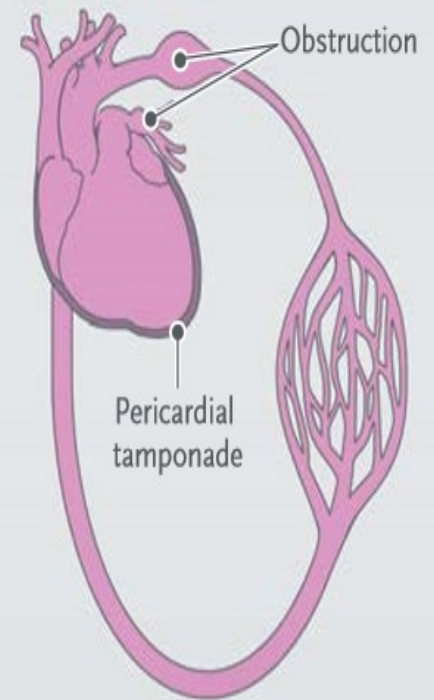
### Hypovolemic shock



### Cardiogenic shock



### Obstructive shock



## EXTRACARDIAC OBSTRUCTIVE

## CARDIOGENIC (e.g., myocardial infarction)

## HYPOVOLEMIC (e.g., hemorrhage)

## DISTRIBUTIVE (e.g., septic)

↓ diastolic filling  
(e.g., tension pneumothorax or pericardial tamponade)

↑ ventricular afterload  
(e.g., massive pulmonary embolus)

myocardial damage

↓ preload

myocardial depression  
(↓ systolic and diastolic function)

↓ diastolic function

↓ systolic function

↓ systolic and diastolic function

↓ diastolic filling

↓ CO  
(↑ SVR)

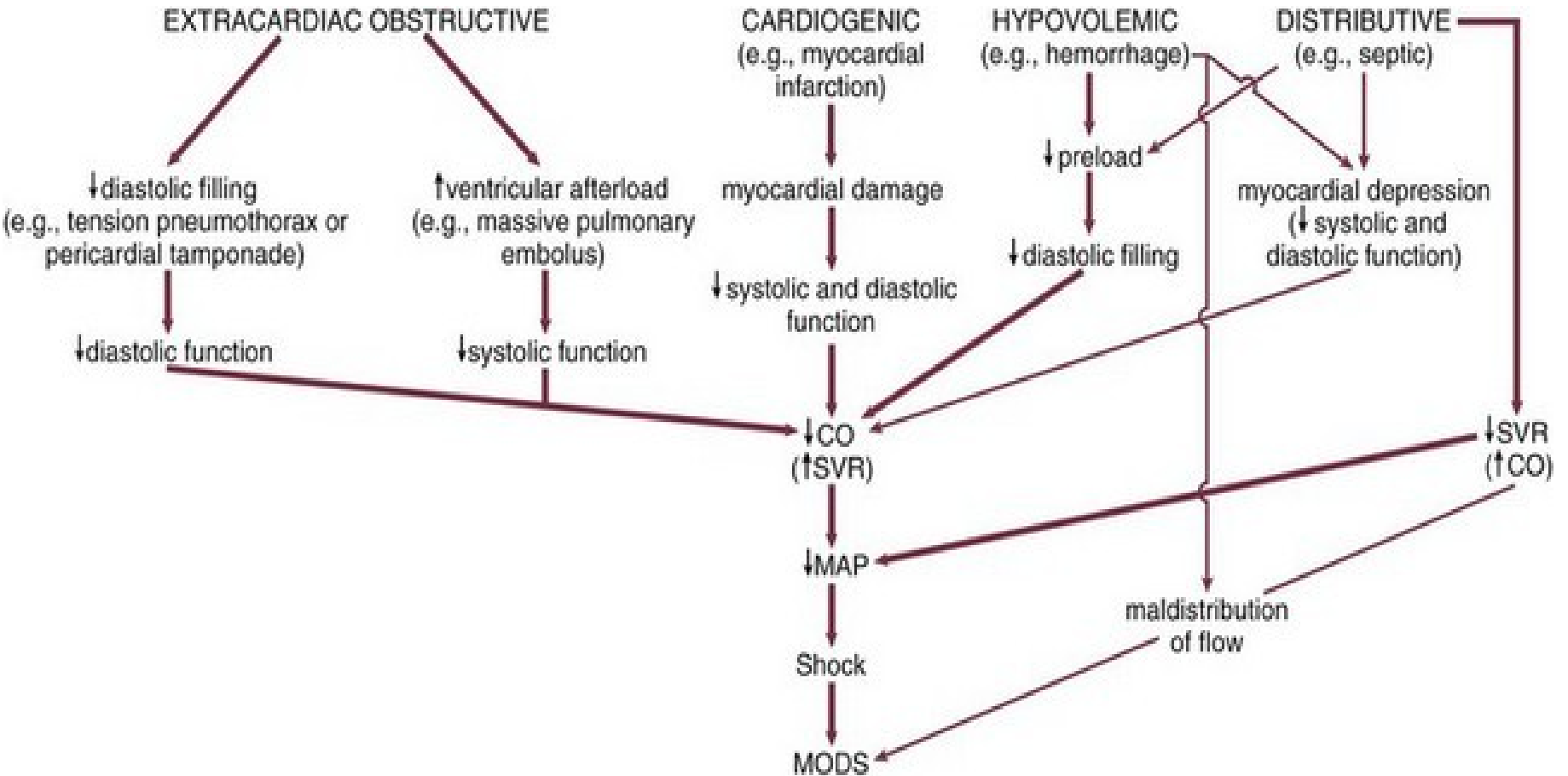
↓ SVR  
(↑ CO)

↓ MAP

Shock

MODS

maldistribution of flow



- <http://physiologyplus.com/phs-213-practice-mcq-circulatory-shock/>