Shock

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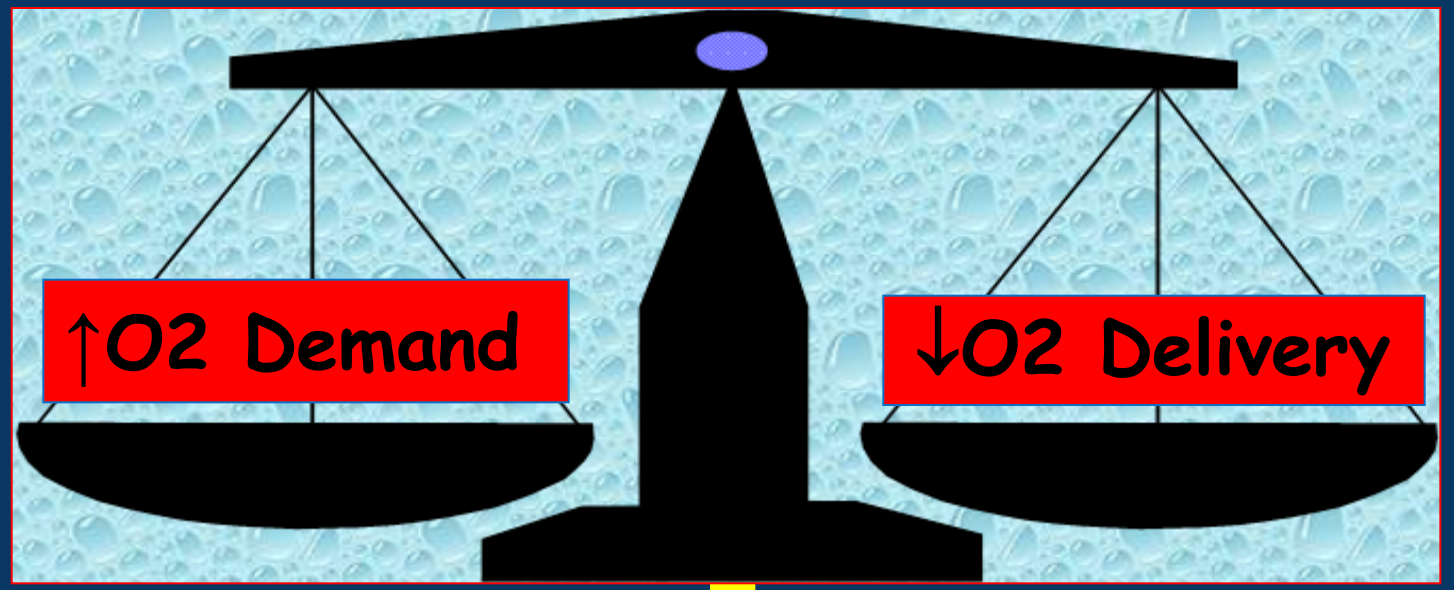
edited by : INSAF IYAD

Outline for Today

- Definition
- Ramifications
- Physiologic determinants
- Classification
- Approach to the patient with shock

Shock is:



Reduced Tissue Perfusion

Cellular Hypoxia & Energy Failure

Definition

- Shock is not:
  - an absolute blood pressure measurement
  - an independent diagnosis

(shock doesn't alway mean hypotension)

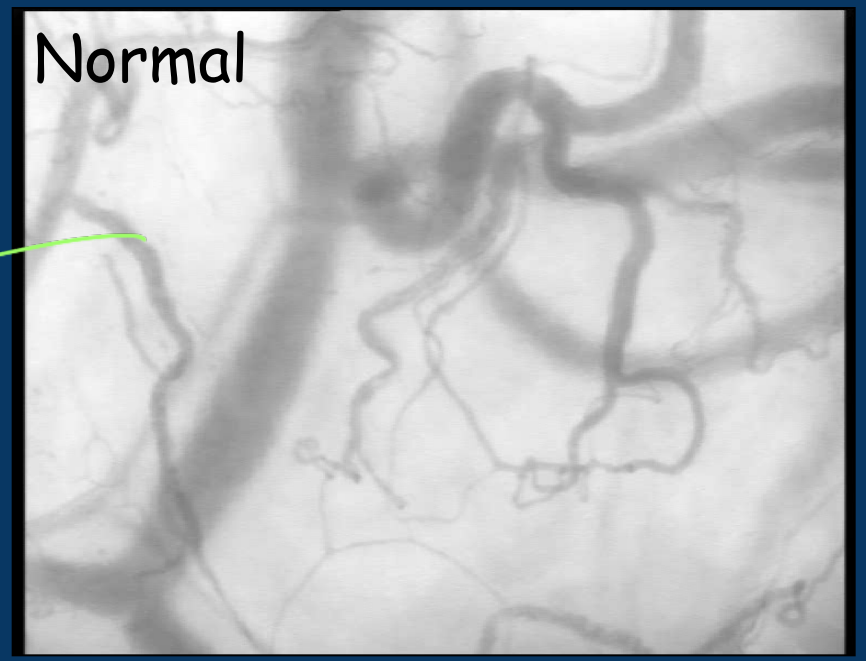
- Shock is:
  - a physiologic state in which significant, systemic reduction in tissue perfusion results in decreased tissue oxygen delivery

↓

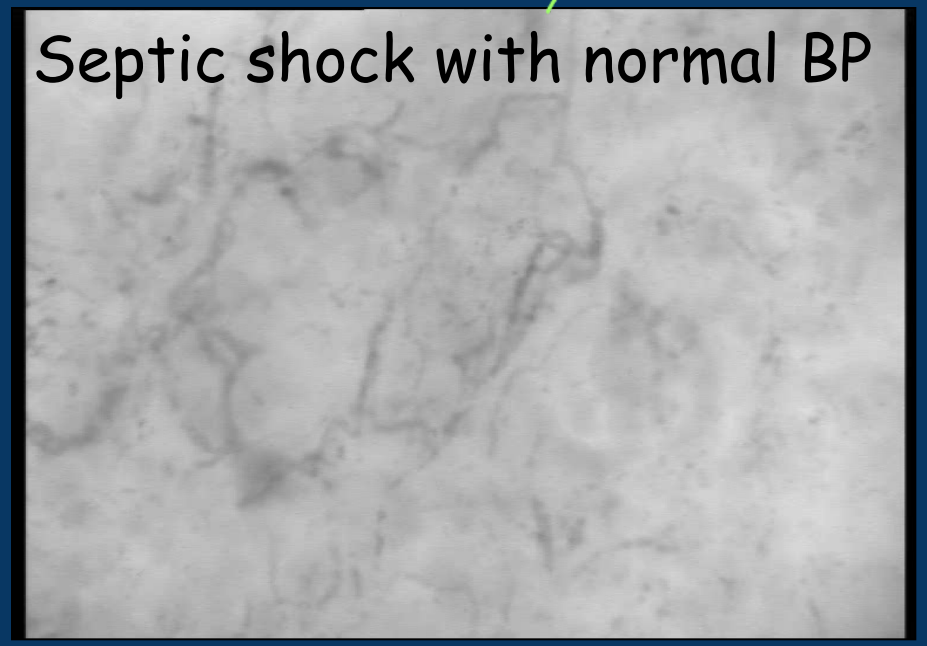
tissue injury & death

Key Issues In Shock

- Falling BP = LATE sign.
- Pallor, tachycardia, slow CFT, restlessness
  = Shock until proven otherwise.
- BP is NOT same as perfusion.



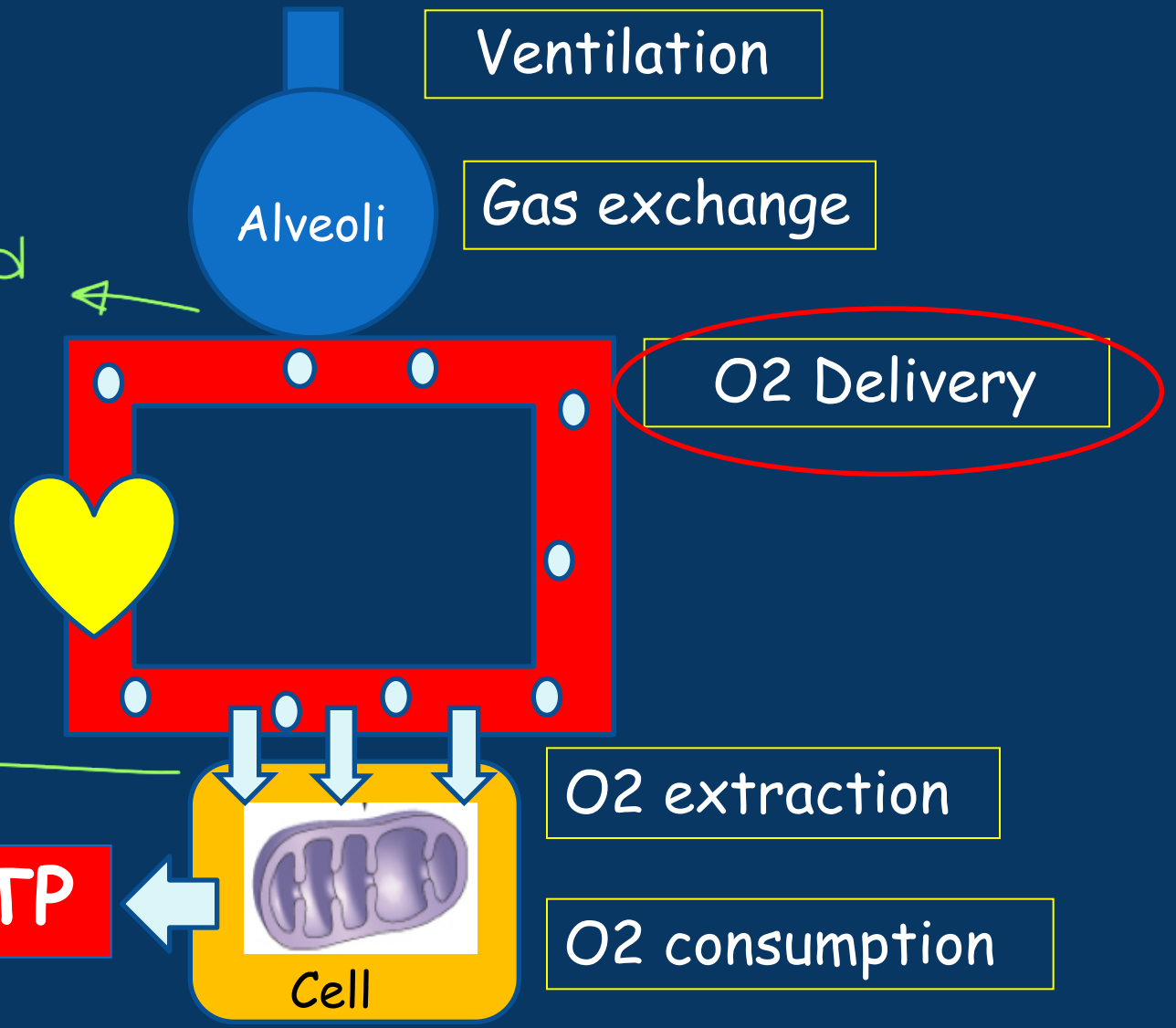
normal vessels



vasoconstriction even with normal pressure

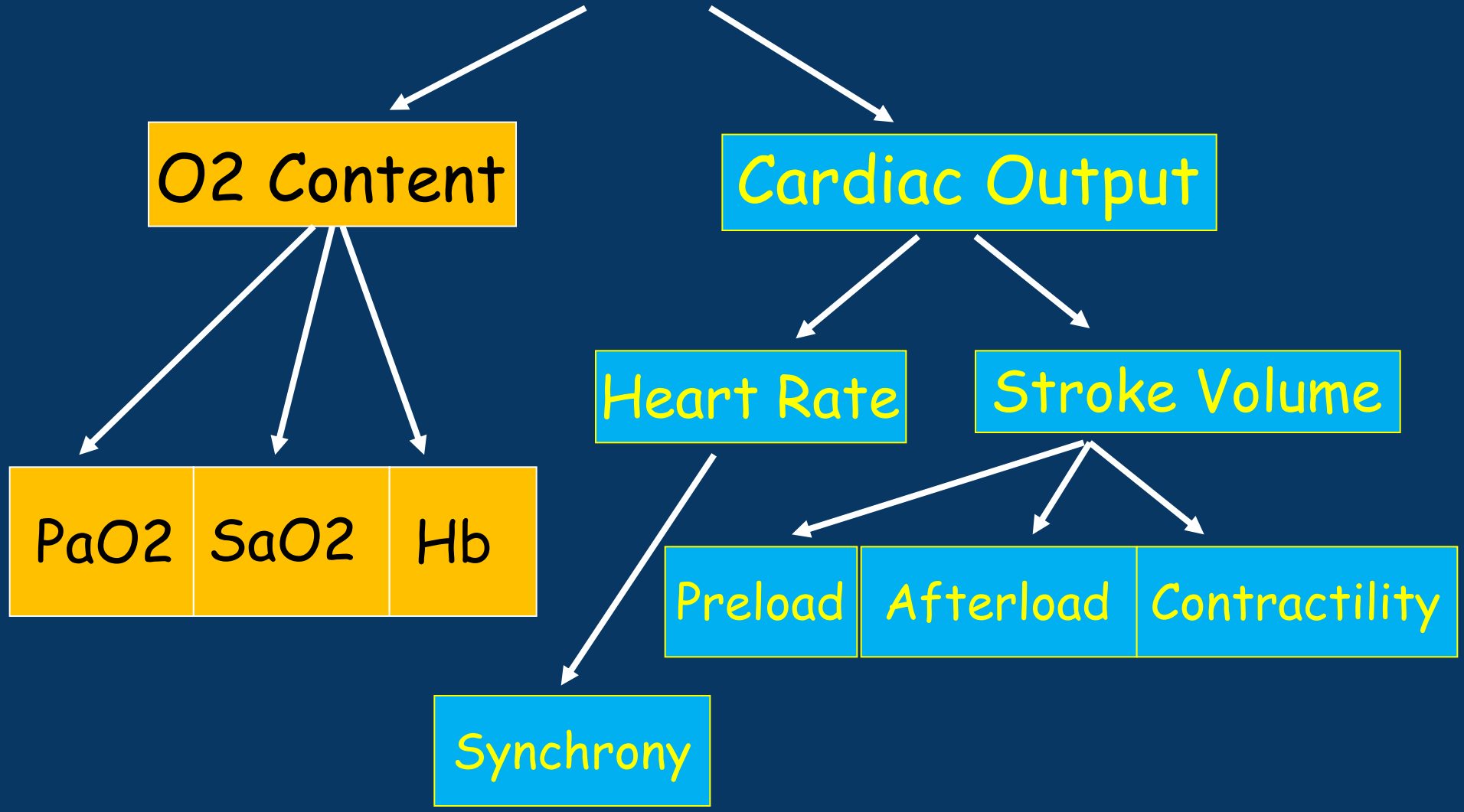
De Baker CCM 2006 34 :403-408

Oxygen Delivery to Tissues



Oxygen Delivery Components

Oxygen Delivery Components



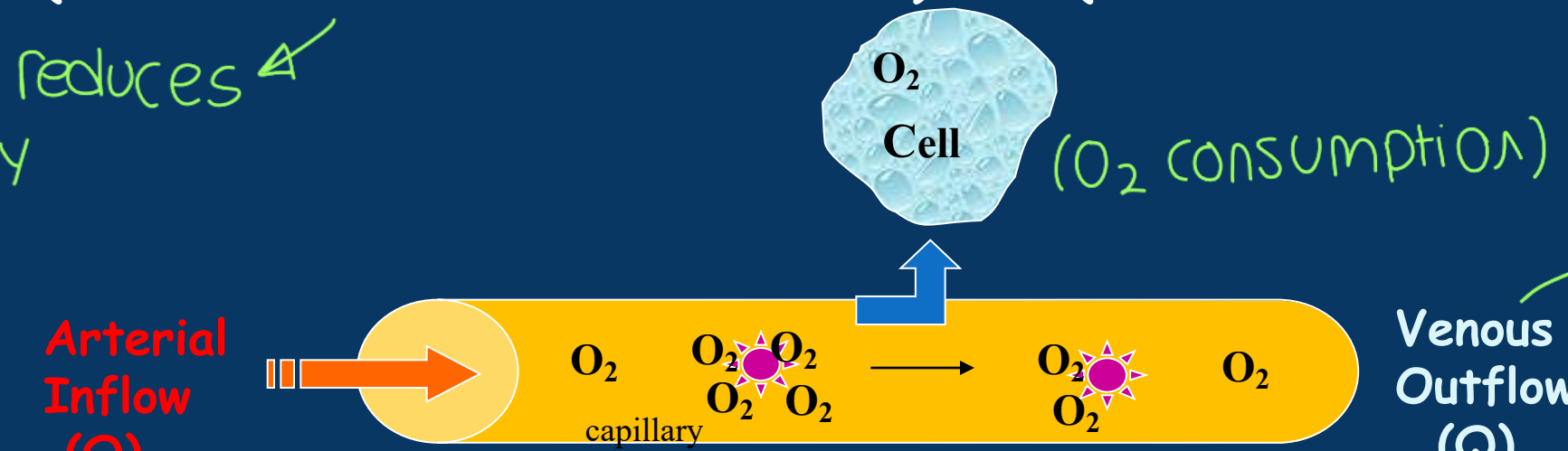
Oxygen Content of Blood

=(O2 carried by Hb) + (O2 in solution)

= (1.34 x Hb x Sats x 0.01) + (0.023 x PaO2)

dissolution coefficient

anemia reduces delivery



من أعلى rt atrium (central venous P) pulm. Aiwgl (mixed venous saturation

(Adapted from the ICU Book by P. Marino)

Glucose

2x ATP

Pyruvic Acid

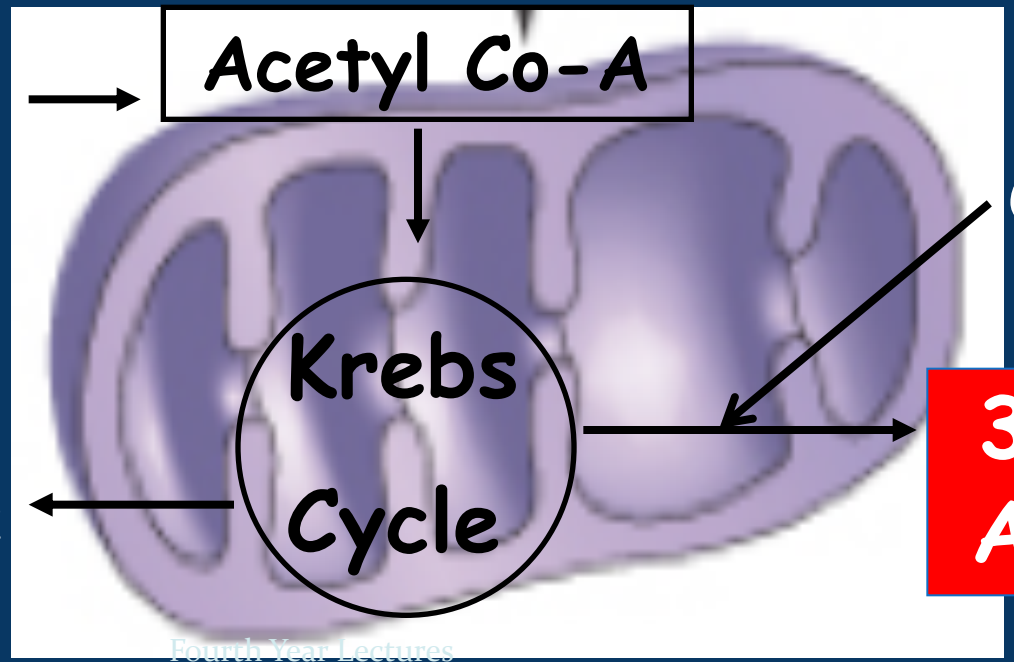
Lactic Acid

Anaerobic

Fatty Acids

Amino Acids

Acetyl Co-A



O_2

CO_2

38x ATP

Aerobic

↓ ATP leads to cellular edema & then rupture → tissue hypoperfusion ↗ injury → organ dysfxn & death.

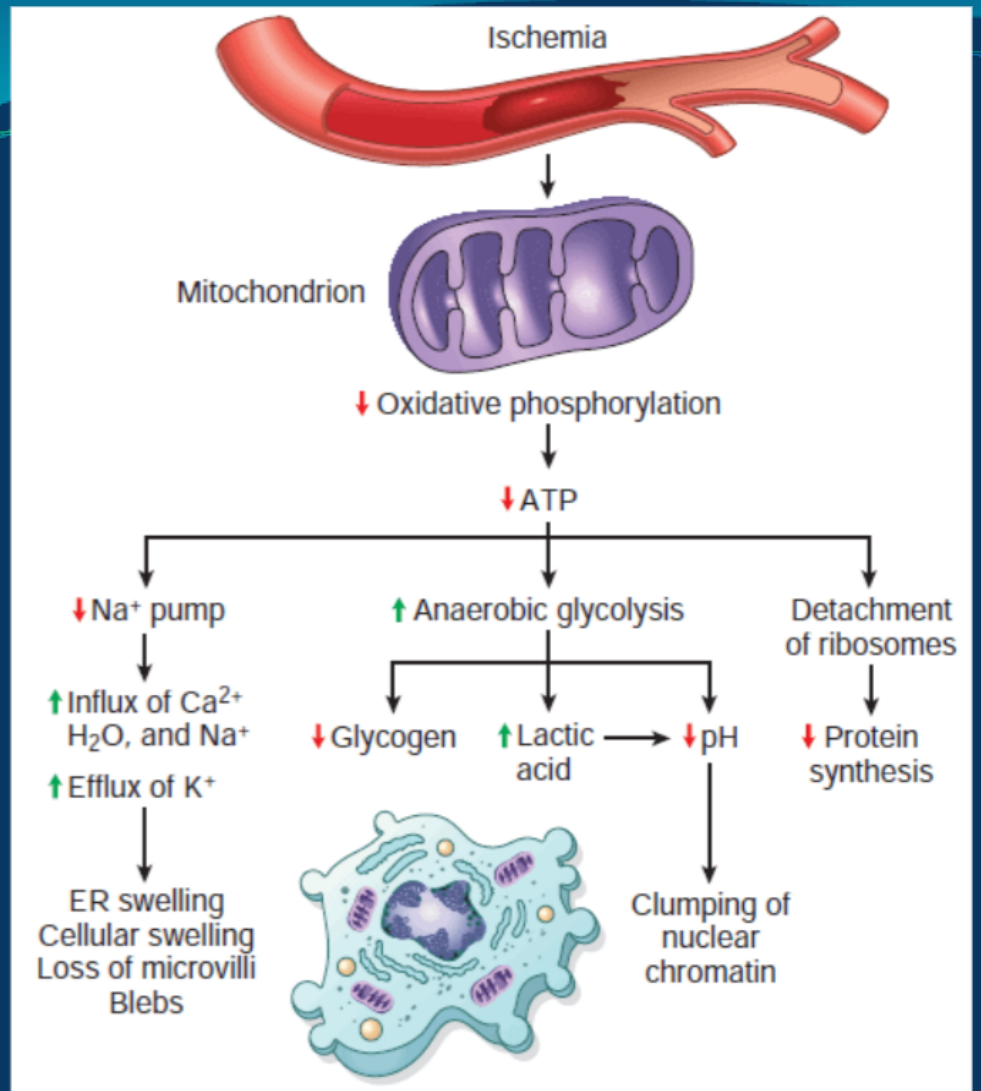


Figure 2.24 Functional and morphologic consequences of decreased intracellular adenosine triphosphate *(ATP)* in ischemic cell injury. The morphologic changes shown here are indicative of reversible cell injury. Further depletion of ATP results in cell death, typically by necrosis. *ER*, Endoplasmic reticulum.

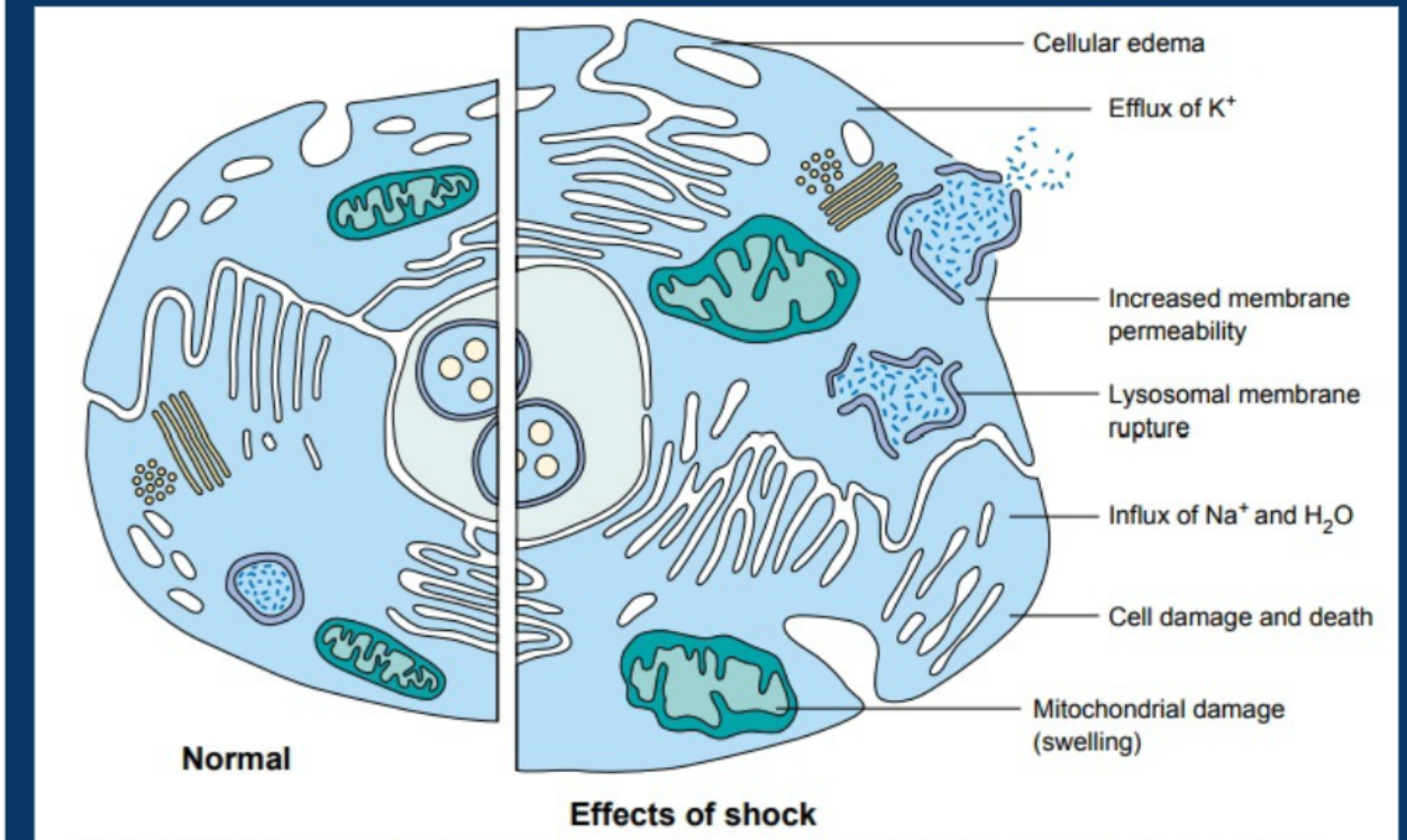
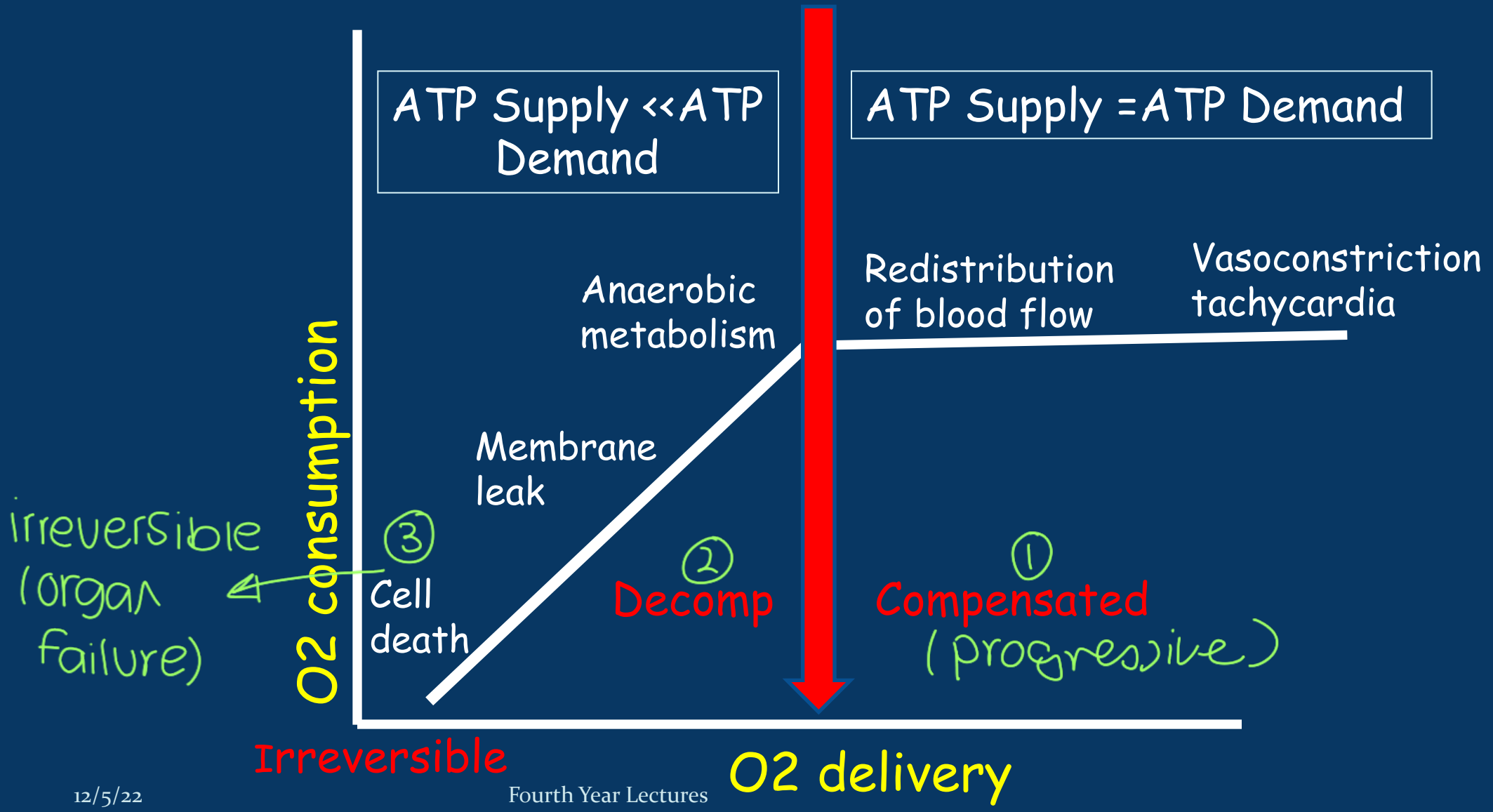


FIGURE 15-1 Cellular effects of shock. The cell swells and the cell membrane becomes more permeable, and fluids and electrolytes seep from and into the cell. Mitochondria and lysosomes are damaged, and the cell dies.

Ramifications of Shock

- Can lead to irreversible cell and tissue injury ultimately resulting in:
  - end-organ damage
  - multi-system organ failure
  - death
- Mortality from shock remains high:
  - cardiogenic shock from AMI - 60-90%
  - septic shock - 35-40%
  - hypovolemic shock - varies depending on disease state

Stages of shock (3 Stages)



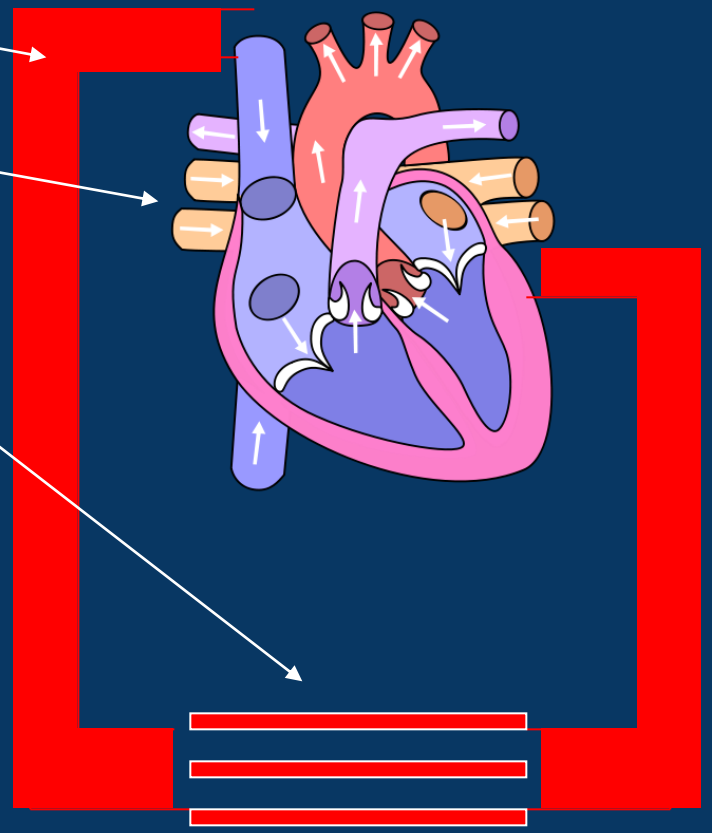
Key Elements of Blood Pressure

important for blood flow

intravascular Compartment (preload)

- Fluid
- Pump
- Pipes

Systemic vascular resistance SVR (after load)



Physiologic Determinants

O_2 consumption = O_2 Delivery – O_2 Return

$VO_2 = CO(C_aO_2-C_vO_2)$

$VO_2 = CO*1.34*Hgb*(S_aO_2-S_vO_2)$

CO = cardiac output

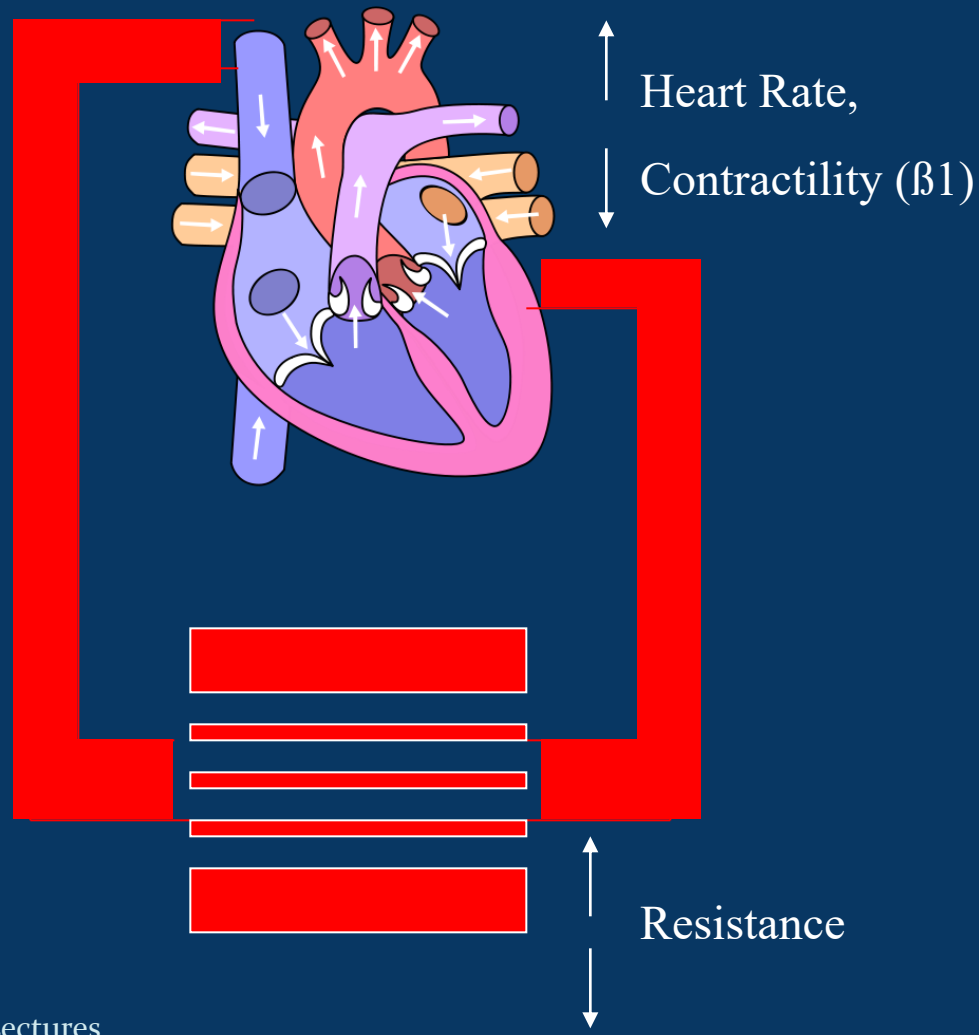
C_aO_2 and C_vO_2 = arterial and mixed venous oxygen content

S_aO_2 and S_vO_2 = arterial and mixed venous oxygen saturation

Mean Arterial Pressure (MAP)

- MAP - CVP = Cardiac Output x SVR
- Cardiac Output (CO)= HR x Stroke Volume

MAP $_{-\ CVP}$ = (HR x SV) x SVR



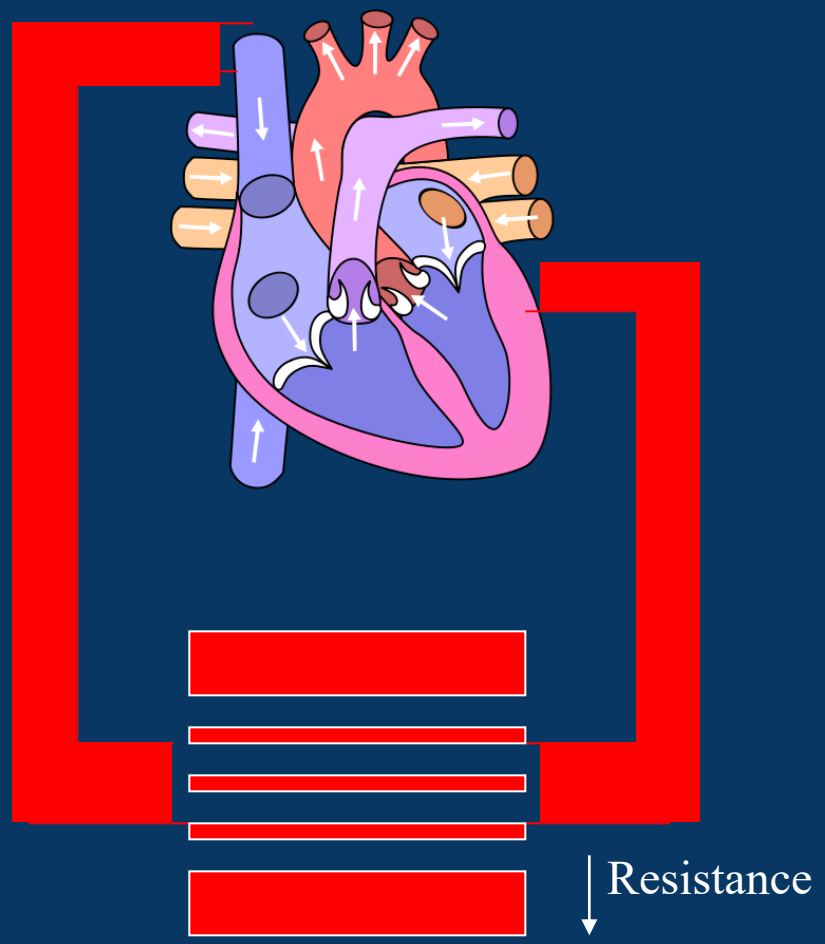
Etiologies of Shock(Distributive)

↓ MAP – CVP = (SV x HR) x SVR

- Low vascular resistance: **"Distributive"**

  Sepsis, anaphylaxis

  Other: adrenal insufficiency, myxedema coma, drug reaction, toxic shock syndrome, neurogenic



Etiologies of Shock (obstructive)

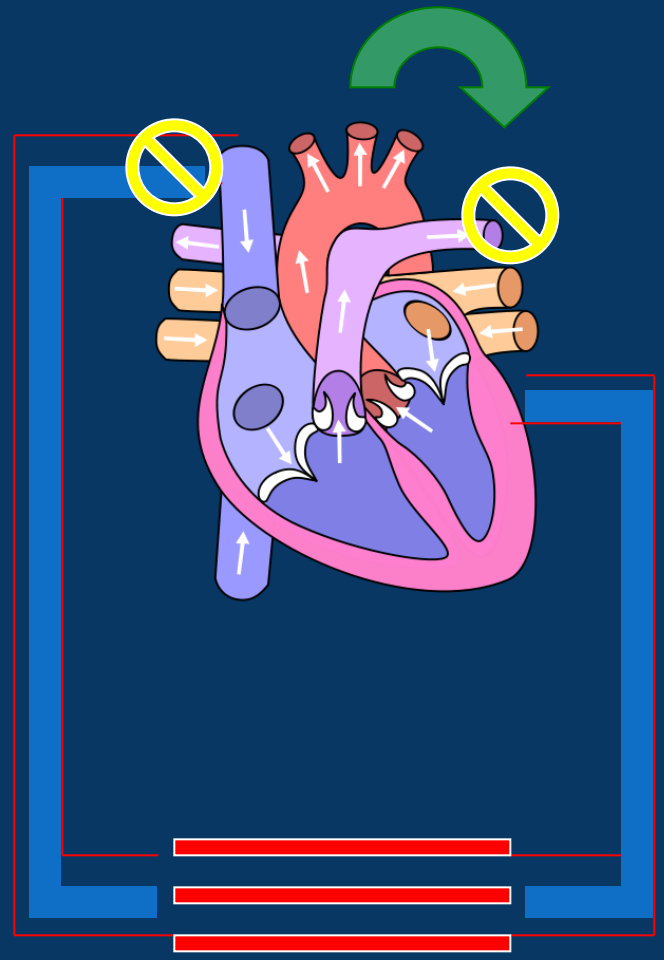
MAP – CVP = (SV x HR) x SVR

- Low Stroke Volume:

Venous return & Outflow obstruction

"Obstructive"

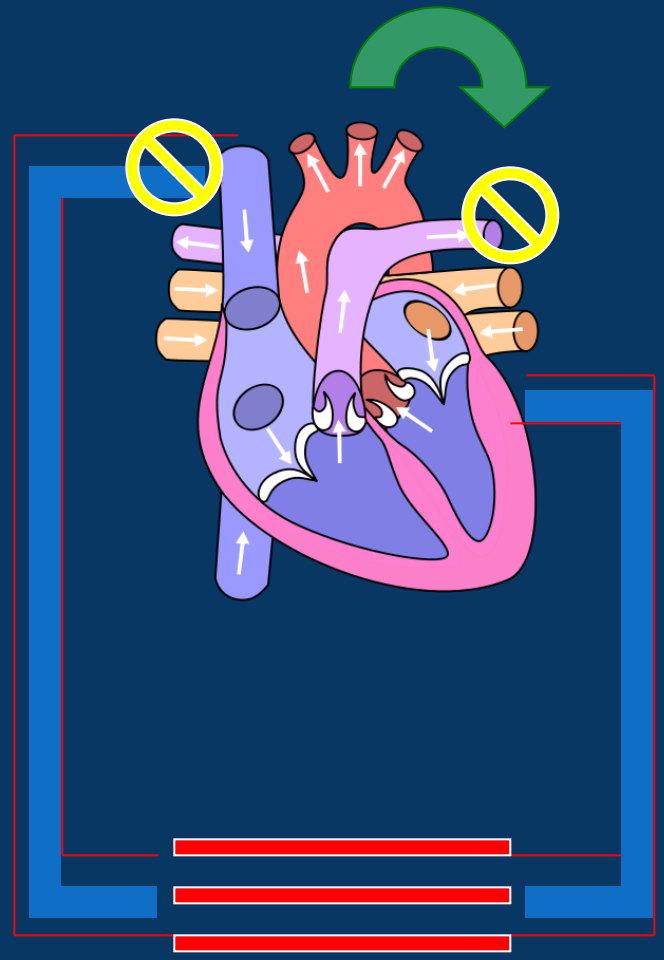
Tamponade, tension pneumothorax, PEEP, Pulmonary embolism



Etiologies of Shock (Hypovolemic)

↓ MAP – CVP = (SV x HR) x SVR

- Low Stroke Volume:
  - ↓ Intravascular volume:
    "Hypovolemic"
    Dehydration, hemorrhage, 3rd space



Etiologies of Shock (Cardiogenic)

↓ MAP – CVP = (SV x HR) x SVR

- ↓ Low Stroke Volume:

  Ejection: **"Cardiogenic**

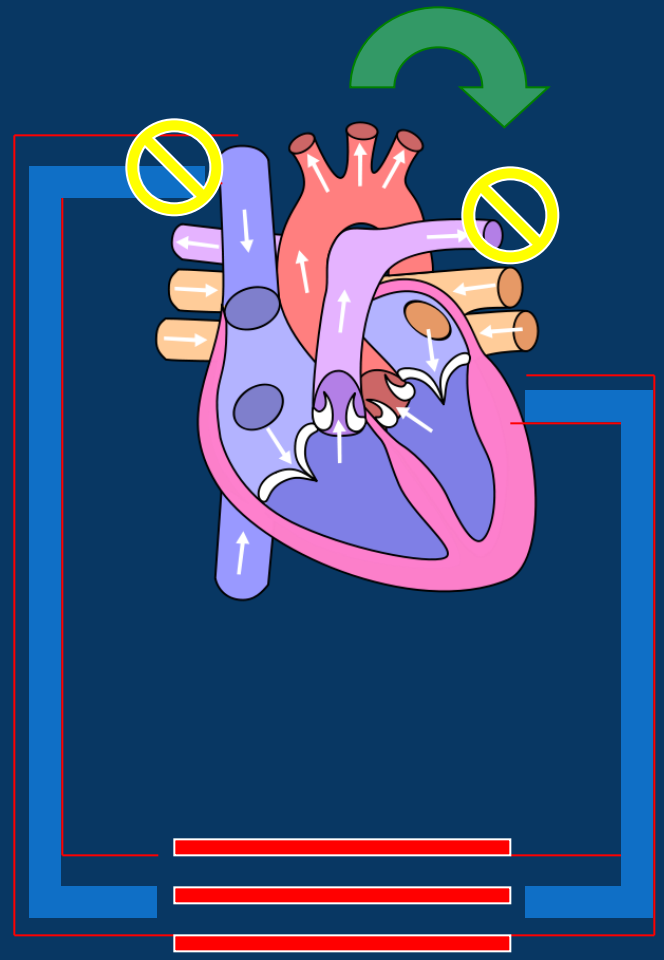
  Myocardial infarct, valvular defect

- Abnormal heart rate:

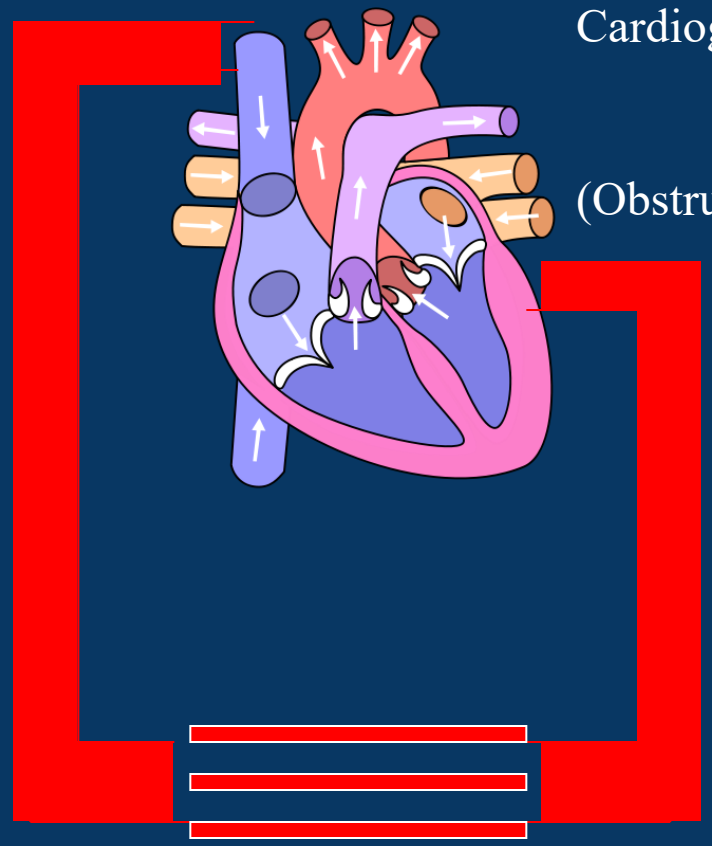
  "Cardiogenic"

  Tachycardia (short filling time)

  Bradycardia



Types of Shock



Cardiogenic → pump problem

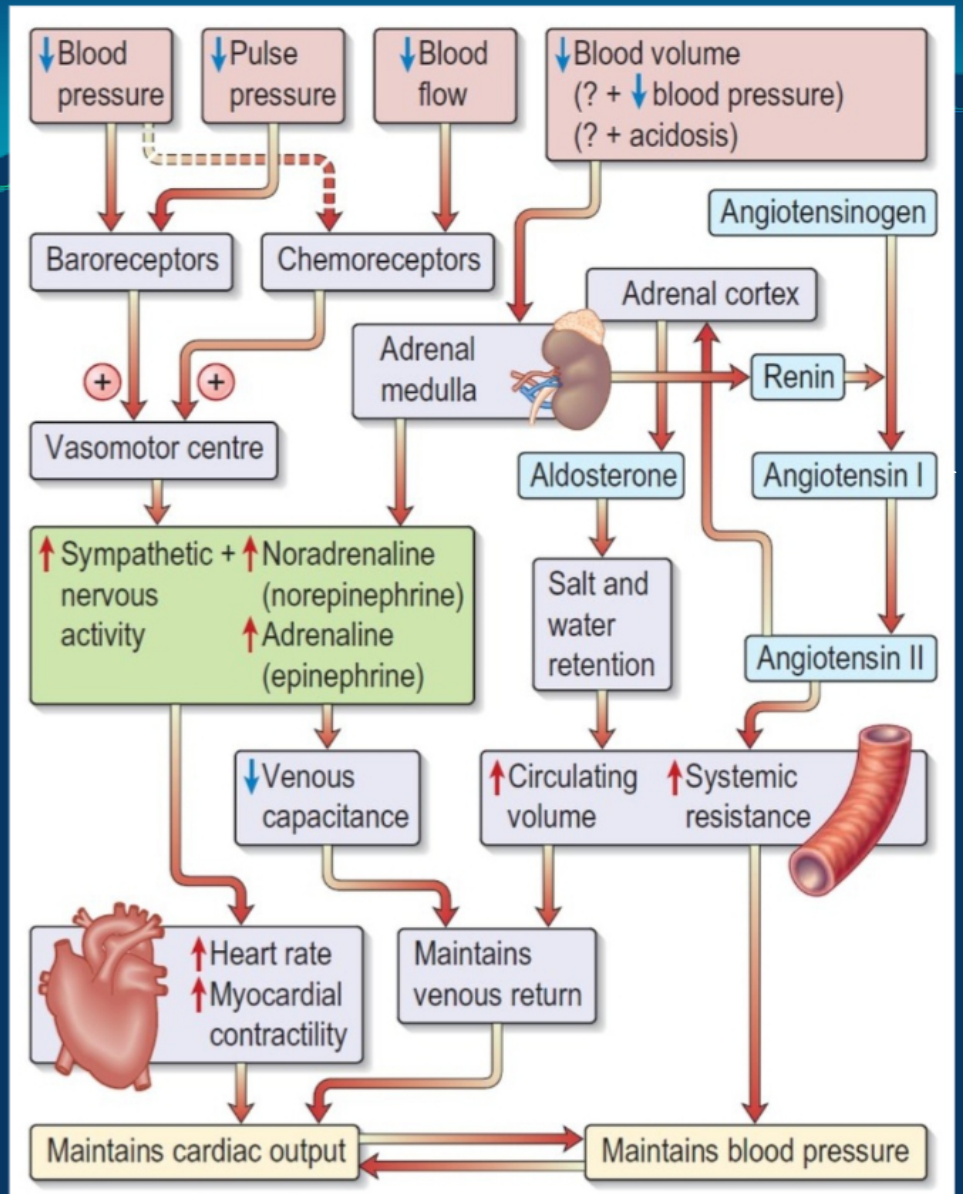
(Obstructive) → heart not able to fill or eject

Hypovolemic → intravascular compartment problem (preload)

Distributive → SVR problem (afterload)

- The clinical manifestations of shock are the result of:

1- autonomic neuroendocrine responses
2- cardiovascular response
3- pulmonary response
4- renal response
5- cellular response
6- metabolic derangement
7- inflammatory response



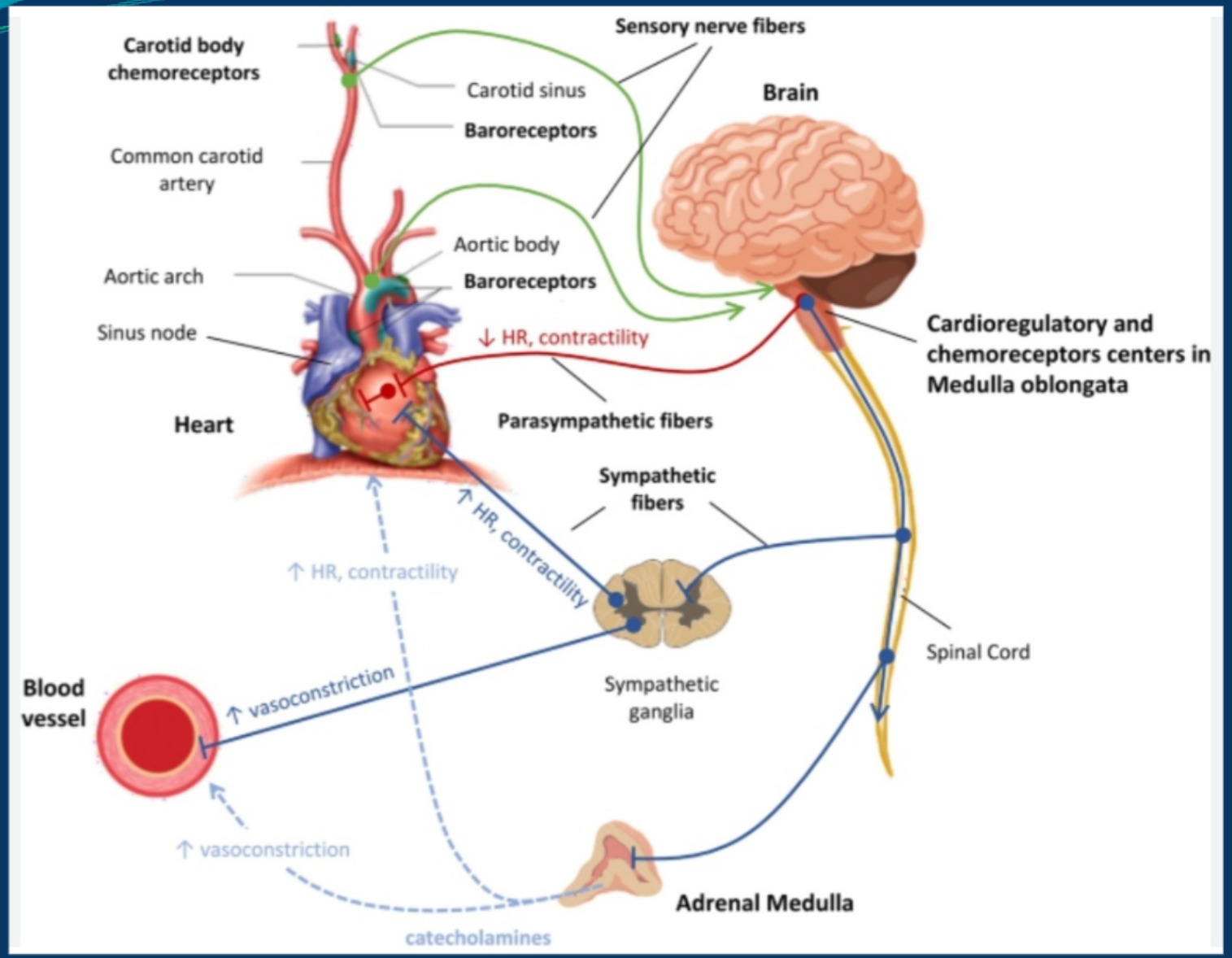
The sympatho-adrenal response to shock showing the effect of increased catecholamines on the left of the diagram and the release of angiotensin and aldosterone on the right. Both mechanisms result in maintaining the cardiac output in shock.

* Sympathoadrenal response to Shock

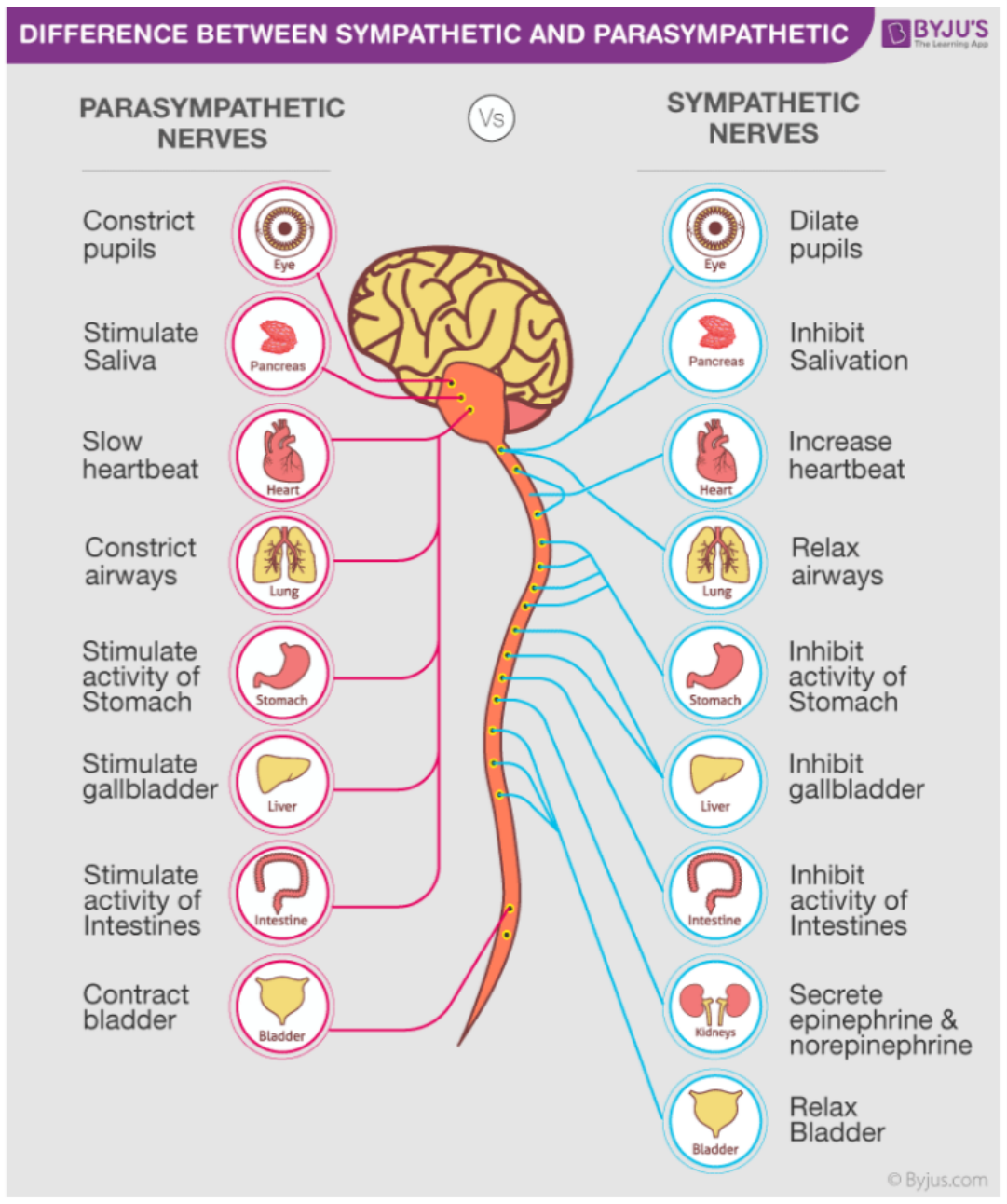
① baro & chemo receptors detect drop in pressure → Symp. Stimulation

② RAS

③ ADH System

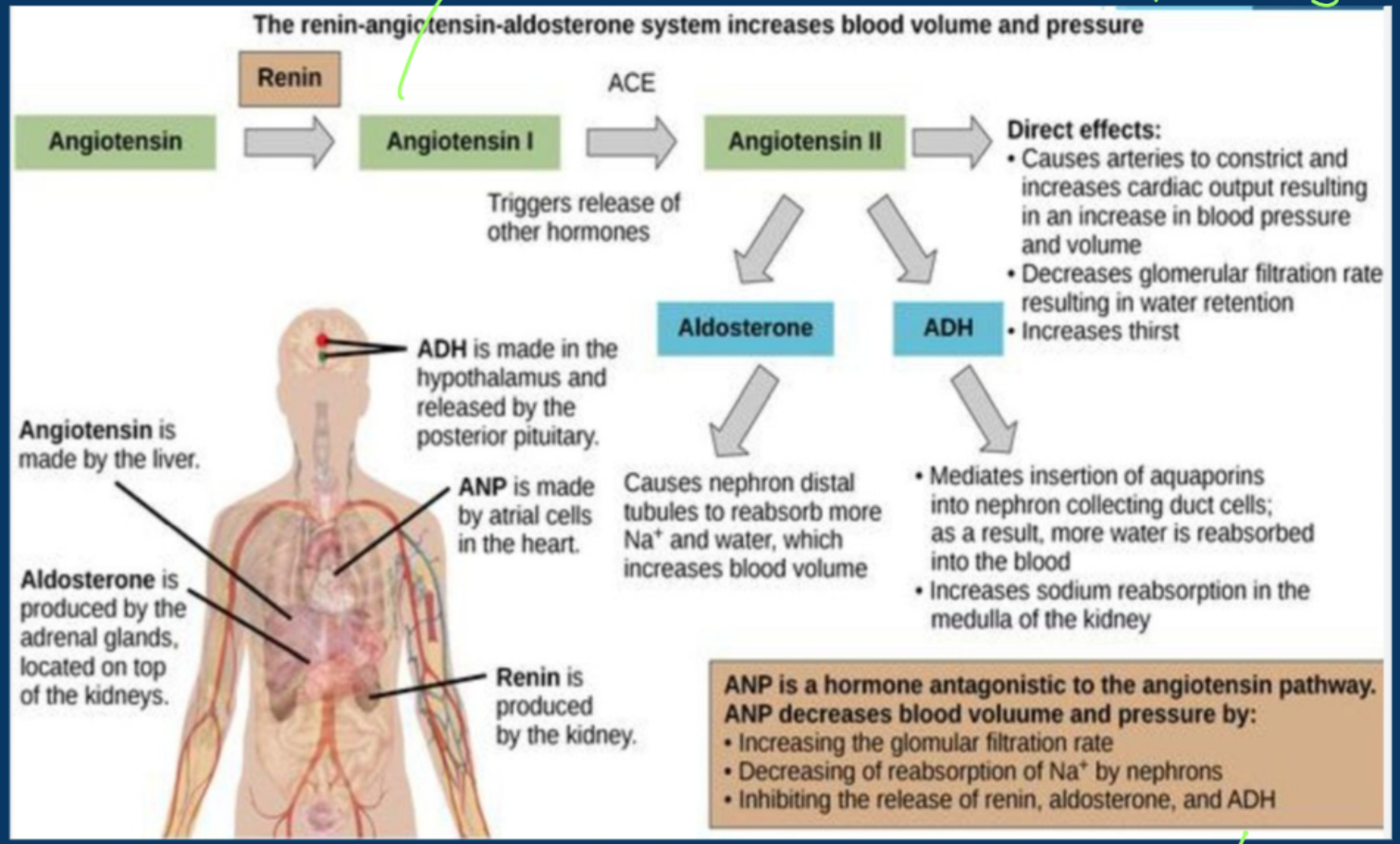


baroreceptors & chemoreceptors send hypotension & changes signal to brain which sends signals to adrenals & spinal cord → vasoconstriction, ↑HR, ↑ cardiac contractility to increase perfusion



Sympathetic responses are common for all types of shock

bi products of this step (ang 1.7, ang 1.8)
→ opposite effect of ang II



Opposite effect (responses to atrial distension)

angiotensin 2
① vasoconstriction
② Na^+ & water retention
③ Stimulates aldosterone which increases reabsorption of Na^+ & water

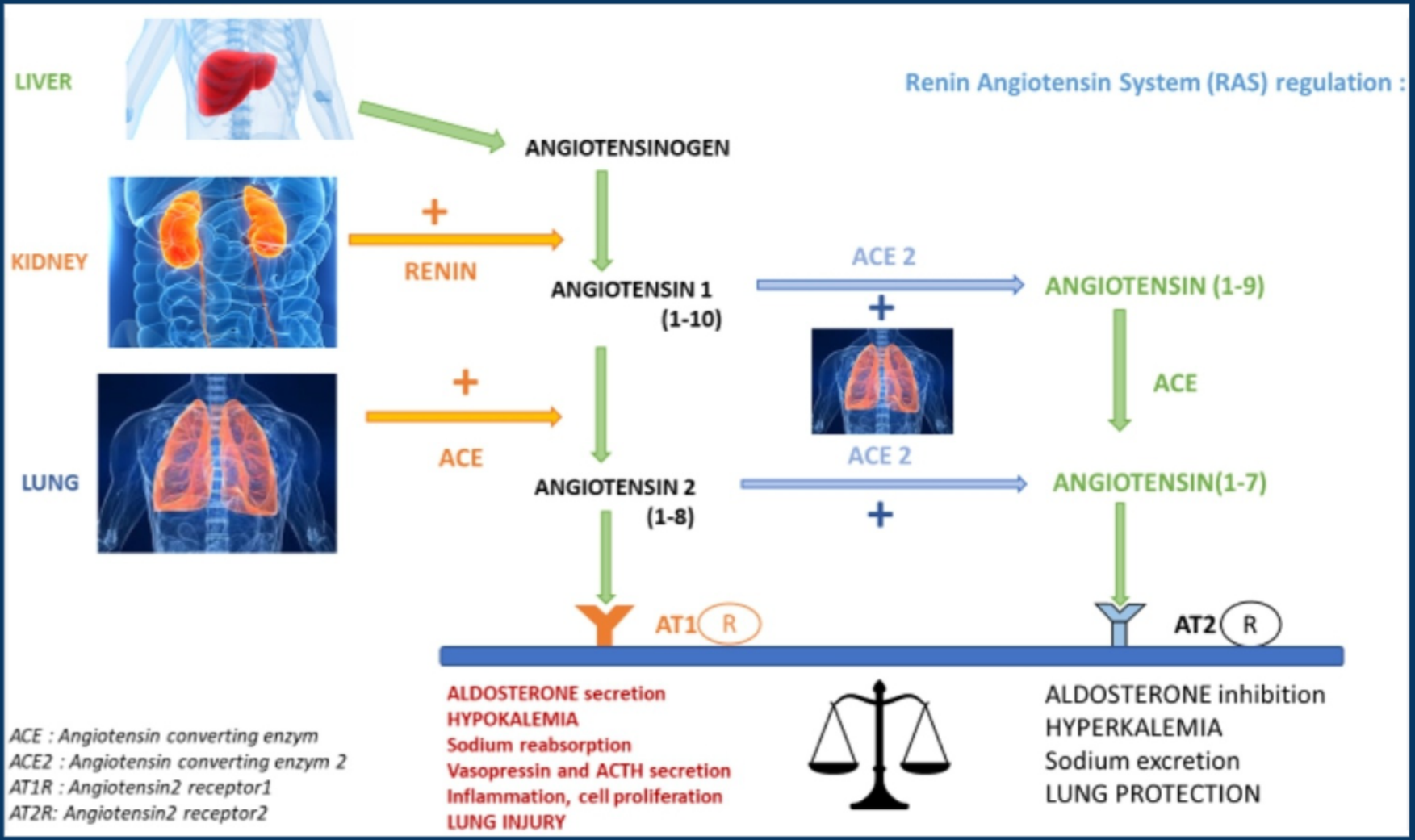
ADH
sensation of thirst
(synthesis in hypothalamus & secreted by posterior pituitary) → water & electrolyte retension

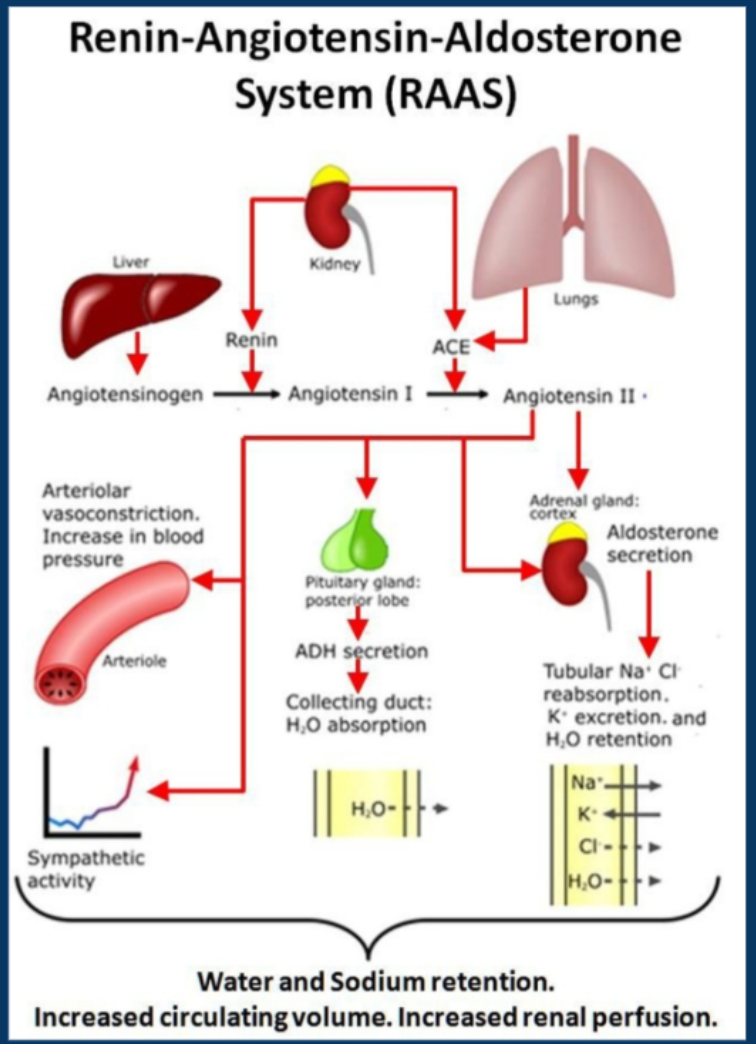
→ vagus (parasymp.) → ⊕ Symp.

Neuroendocrine response

- Hypotension, and hypoxia are sensed by baroreceptors and chemoreceptors, which contribute to an autonomic response
- Release of norepinephrine induces arterial vasoconstriction (redistribution of blood flow from the skin, skeletal muscle, kidneys, and splanchnic viscera to heart and brain)
- Reduced vagal activity increases the heart rate and cardiac output
- Constriction of venous capacitance vessels, which augments venous return

- Renin-angiotensin-aldosterone axis activated
- Vasopressin increased and causes vasoconstriction and enhance water reabsorption


LIVER
KIDNEY
LUNG
Renin Angiotensin System (RAS) regulation :
ANGIOTENSINOGEN
RENIN
ANGIOTENSIN 1
(1-10)
ACE
ANGIOTENSIN 2
(1-8)
ACE 2
ANGIOTENSIN (1-9)
ACE
ACE 2
ANGIOTENSIN(1-7)
AT1 R
AT2 R
ALDOSTERONE secretion
HYPOKALEMIA
Sodium reabsorption
Vasopressin and ACTH secretion
Inflammation, cell proliferation
LUNG INJURY
ALDOSTERONE inhibition
HYPERKALEMIA
Sodium excretion
LUNG PROTECTION
ACE : Angiotensin converting enzym
ACE2 : Angiotensin converting enzym 2
AT1R : Angiotensin2 receptor1
AT2R: Angiotensin2 receptor2


Renin-Angiotensin-Aldosterone System (RAAS)
Liver
Kidney
Lungs
Renin
ACE
Angiotensinogen
Angiotensin I
Angiotensin II
Arteriolar vasoconstriction. Increase in blood pressure
Arteriole
Pituitary gland: posterior lobe
ADH secretion
Collecting duct: H2O absorption
Adrenal gland: cortex
Aldosterone secretion
Tubular Na+ Cl- reabsorption. K+ excretion. and H2O retention
Sympathetic activity
Water and Sodium retention.
Increased circulating volume. Increased renal perfusion.

Cardiovascular response

- An increase in heart rate is a useful but limited compensatory mechanism to maintain cardiac output
- Increased filling pressures of heart (cardiogenic , obstructive) stimulates release of BNP to secrete sodium and volume to relieve the pressure on the heart
- Prolonged hypotension , acidosis , sepsis, ischemia, trauma , hypothermia all impair myocardial contractility and reduce the SV and decrease CO (shock induced cardiomyopathy)

Pulmonary response

- Relative increase in PVR , particularly in septic shock
  ↳ acidosis لحموضة ال
- Shock-induced tachypnea cause respiratory alkalosis and reduces tidal volume
- Acute lung injury and ARDS may complicate shock

Renal response

- The physiologic response of the kidney to hypoperfusion is to conserve salt and water (by decreasing GFR) which together with increased aldosterone and vasopressin is responsible for reduced urine amount

- This may leads to: acute renal failure , acute tubular necrosis , rhabdomyolysis

Cellular response

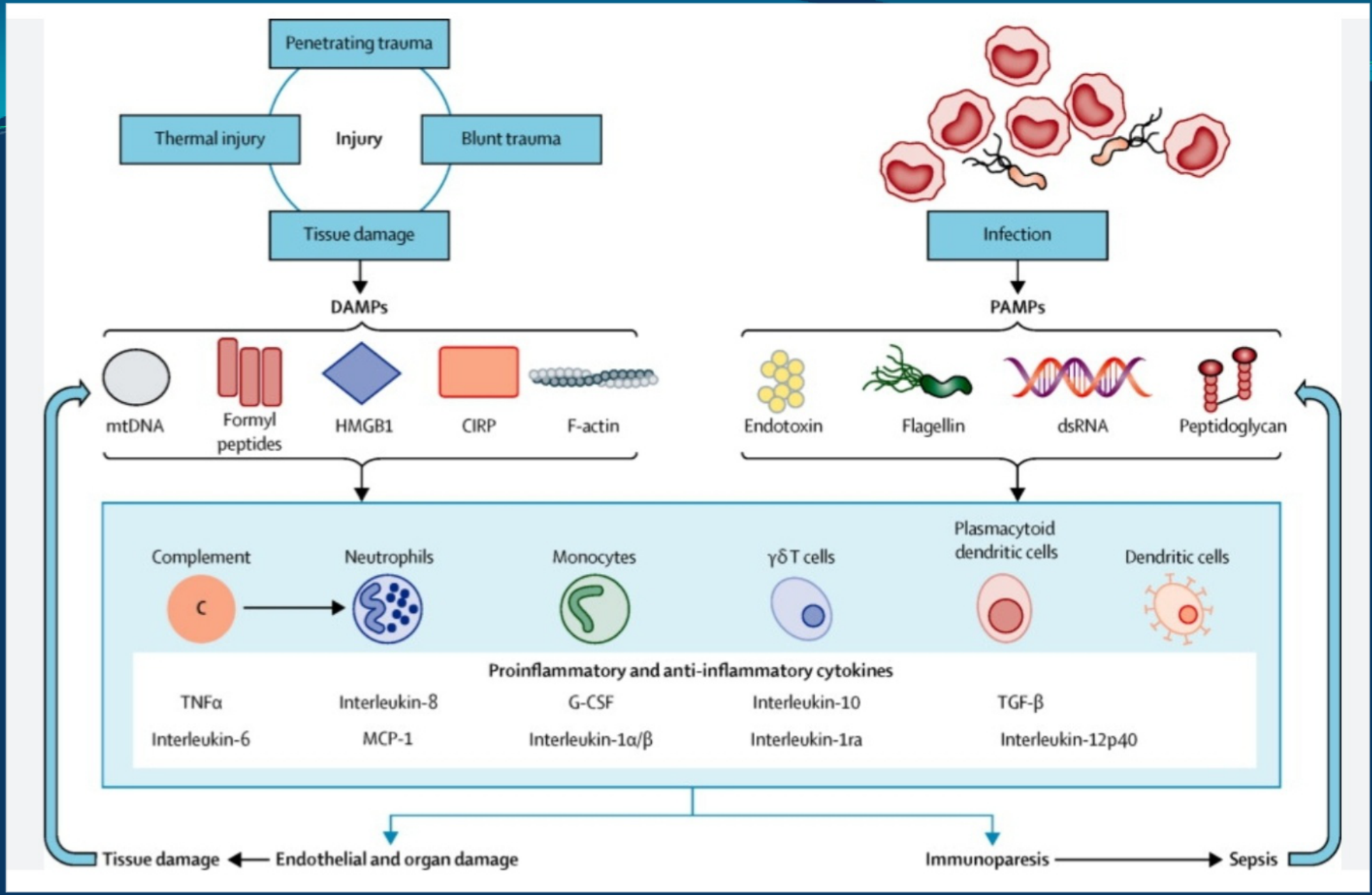
- Mitochondrial dysfunction leads to decrease in ATP and accumulation of hydrogen ions, lactate, and other products of anaerobic metabolism
- Dysfunction of cell membranes, leads to increase in intracellular sodium and water, leading to cell swelling, which interferes further with microvascular perfusion
- Cellular membrane receptors become poorly responsive to the stress hormones insulin, glucagon, cortisol, and catecholamines
- Homeostasis of calcium is lost with accumulation of calcium intracellularly and a concomitant extracellular hypocalcaemia

Metabolic derangement

↳ acidosis (↑ lactic acid, cell death, cell hypoperfusion)

- As shock progresses, lysosomal enzymes are released into the cells with subsequent hydrolysis of membranes, resulting in cellular death

- These pathologic events give rise to the metabolic features of hemoconcentration, hyperkalemia, hyponatremia, prerenal azotemia, hyper- or hypoglycemia, and lactic acidosis



inflammatory response

① ↑WBCs

② endoth. damage

③ immuno suppression (infxn)

Inflammatory response

- The **complement cascade**, activated through both the classical and alternative pathways, generates the anaphylatoxins C3a, C4a ,C5a

- Activation of the **coagulation cascade** causes microvascular thrombosis, with subsequent fibrinolysis leading to repeated episodes of ischemia and reperfusion

↑ coagulation, ↓ fibrinolysis, release of tumor necrosis factor → more hypotension & lactoacidosis

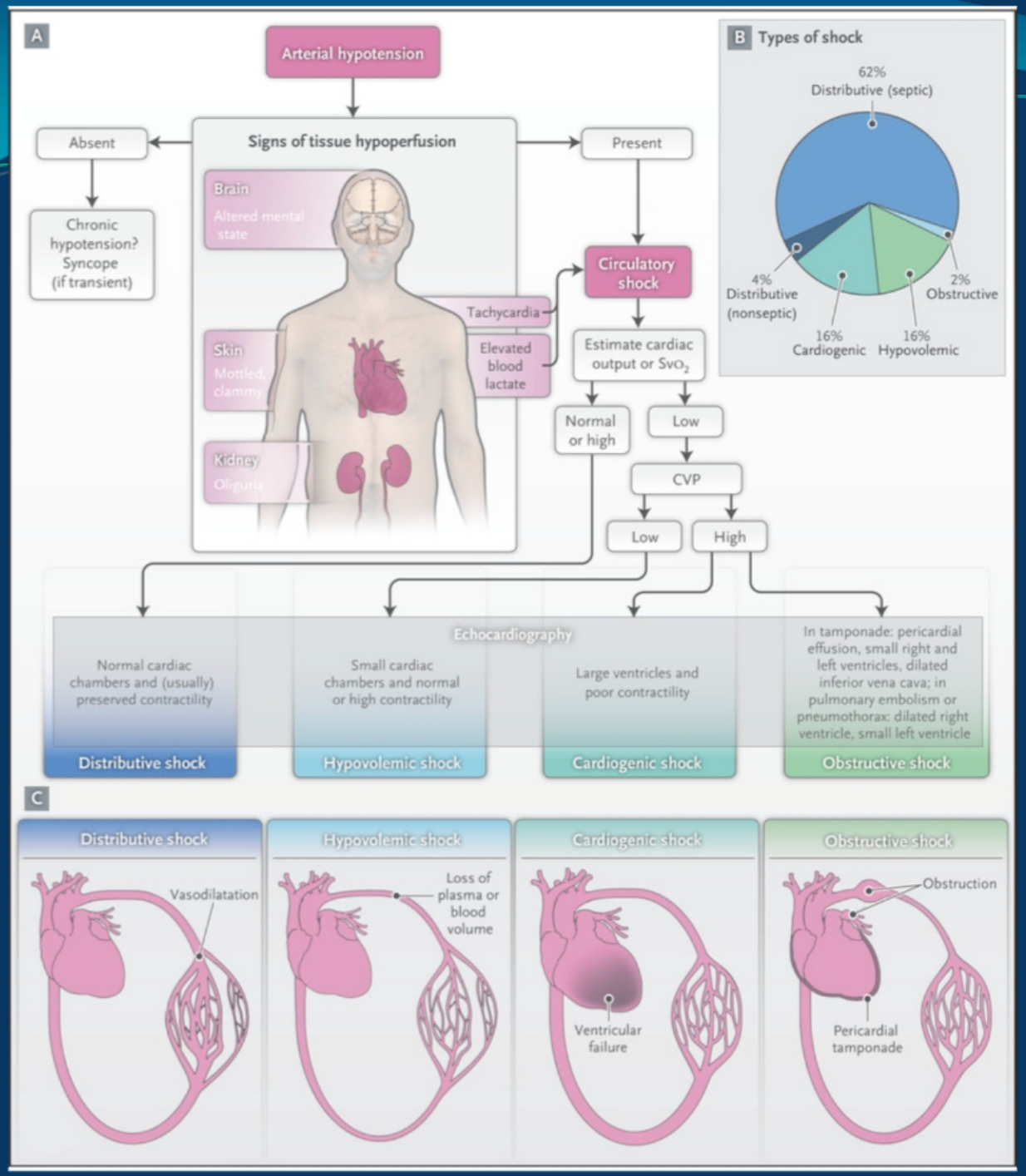
- Thrombin, potent proinflammatory can cause expression of adhesion molecules on endothelial cells and activation of neutrophils causing secondary injury because of the release of toxic oxygen radicals

- Platelet-activating factor causes pulmonary vasoconstriction, bronchoconstriction, systemic vasodilation, increased capillary permeability, and activates macrophages and neutrophils

- TNF α produced by activated macrophages causes hypotension, lactic acidosis, and respiratory failure

Classification of Shock

- Hypovolemic
- Cardiogenic
- Distributive (vasodilatory)
- Obstructive



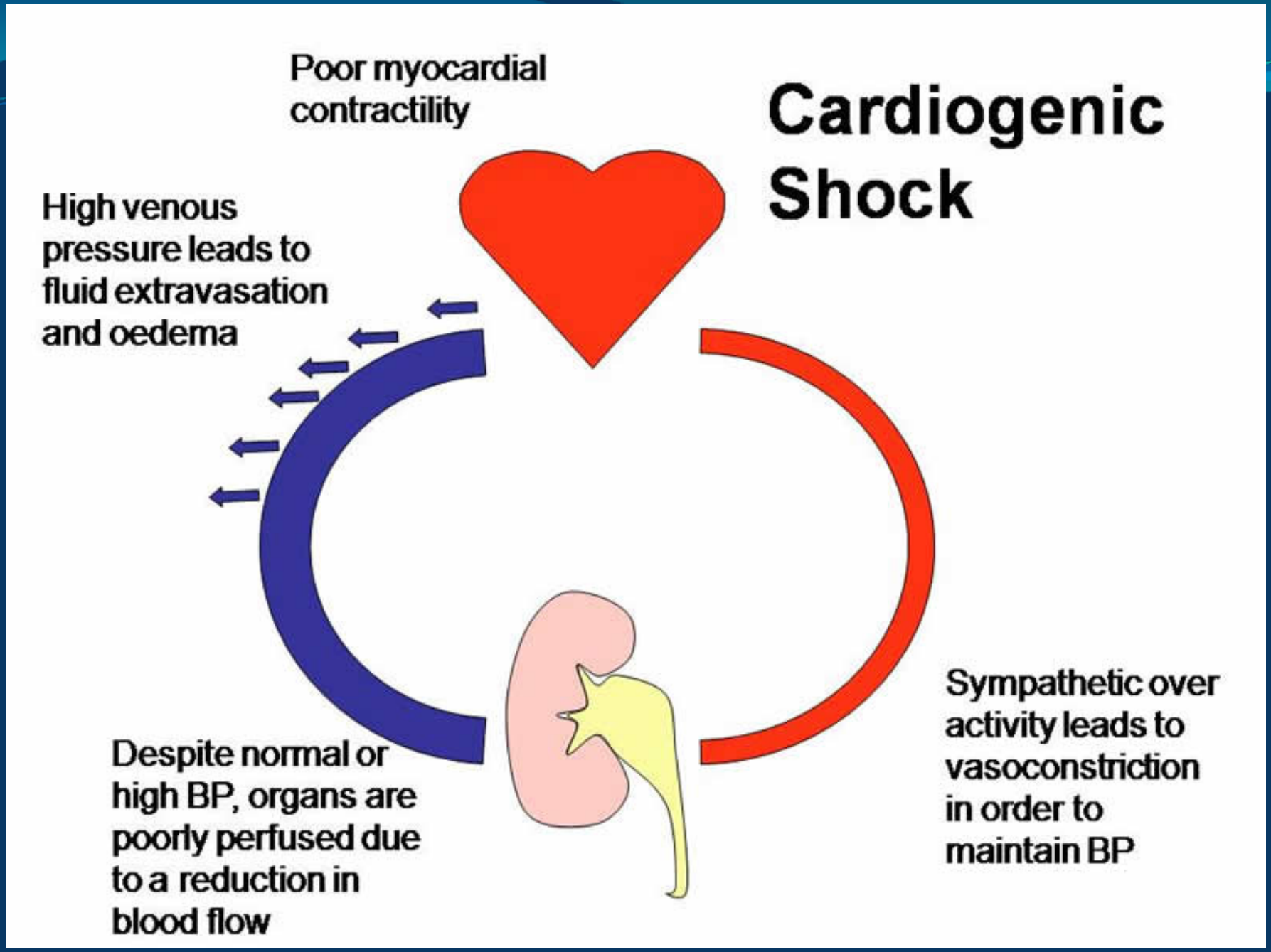
① distributive → ↓ after load

② hypovolemic → ↓ pre load

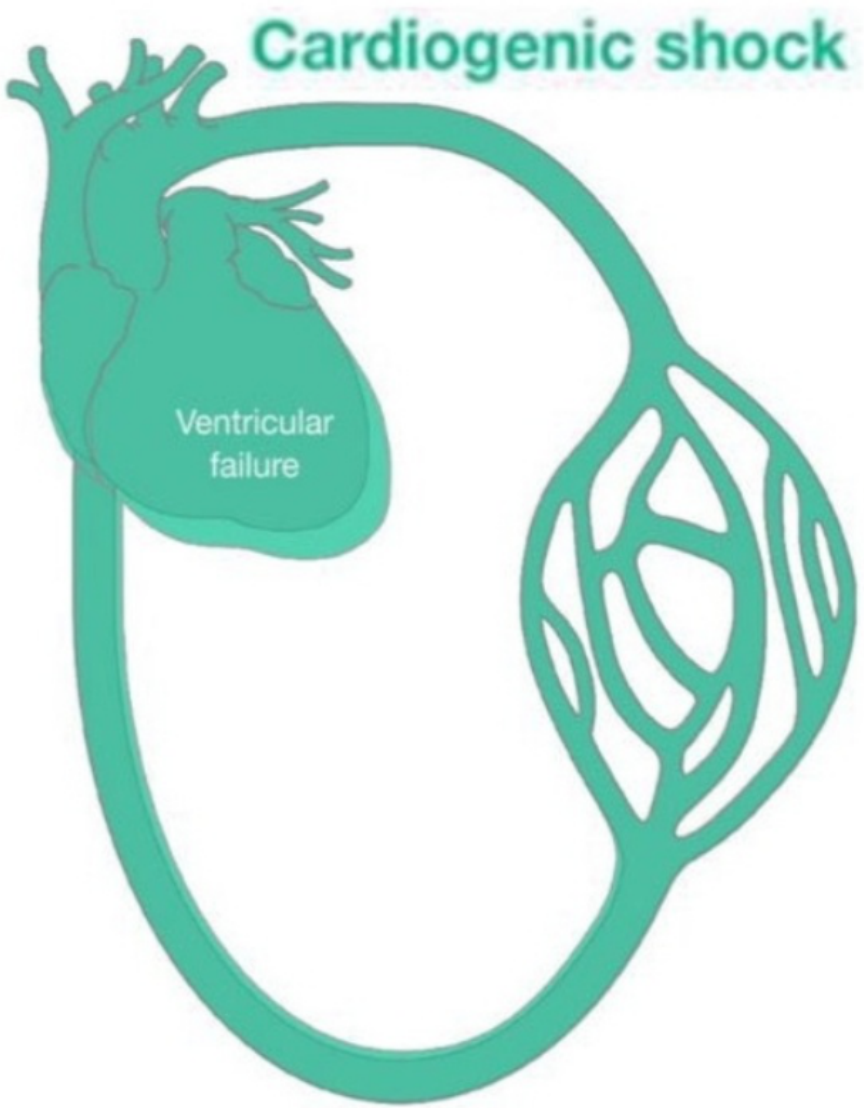
③ cardiogenic → pump failing
(↑ afterload / venous congestion cuz heart can't pump)

④ obstructive
↳ lt side: coarctation
↳ rt side: pulm. embolism
↳ pericardial
(venous congestion, severe vasoconst., ↑ afterload)

Cardiogenic Shock



DIAGRAM



SIGNS OF CIRCULATORY SHOCK PATHWAY

Arterial hypotension and usually tachycardia

↓

Signs of tissue hypoperfusion present

- Altered mental state
- Mottled, clammy skin
- Oliguria
- Elevated blood lactate

↓

Circulatory shock

↓

Low estimated cardiac output or SvO_2

↓

High central venous pressure

↓

Cardiogenic circulatory shock

SYMPTOMS

Cardiogenic shock is characterized by low cardiac output and inadequate oxygen transport. It can be the result of a myocardial infarction, end-stage cardiomyopathy, advanced valvular disease, severe myocarditis, or severe cardiac arrhythmias.

ECHOCARDIOGRAPHIC SIGNS

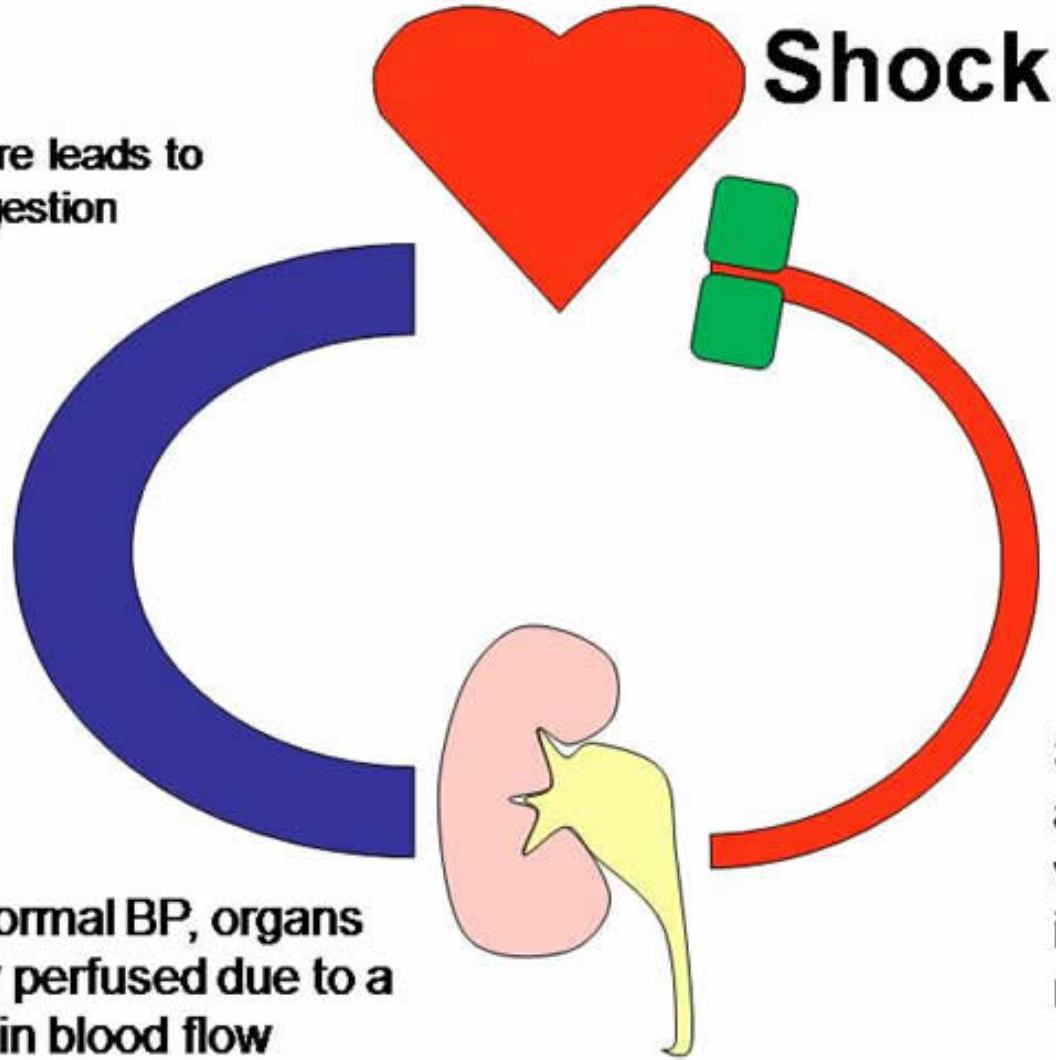
Large ventricles and poor contractility.

Cardiogenic

- Myocardial infarction
- Myocardial contusion (trauma)
- Myocarditis
- Acute valvular failure (dilatative cardiomyopathy)
- Arrhythmia
- Acute ventricular septal wall defect (mechanical complication of MI)

Obstructive Shock

myocardium contracts against high afterload



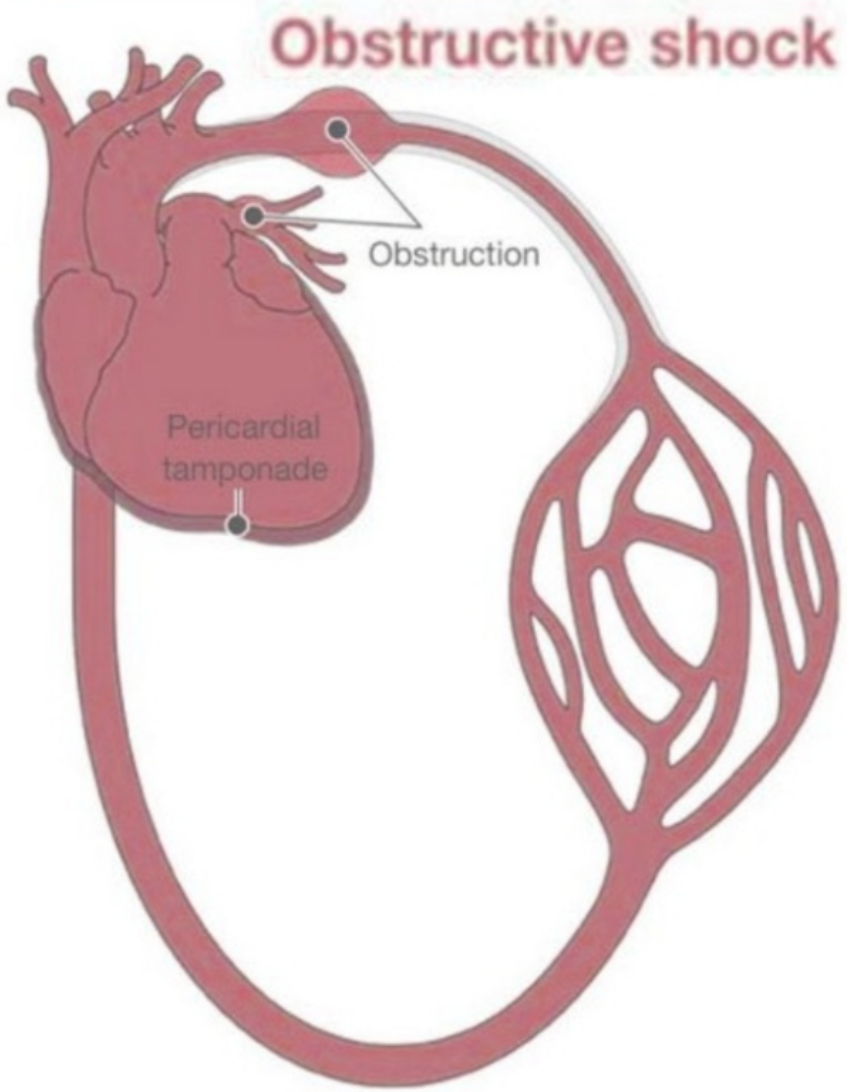
Back pressure leads to venous congestion

Sympathetic over activity leads to vasoconstriction in order to maintain BP

Despite normal BP, organs are poorly perfused due to a reduction in blood flow

Very high Preload

DIAGRAM



SIGNS OF CIRCULATORY SHOCK PATHWAY

Arterial hypotension and usually tachycardia

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Signs of tissue hypoperfusion present

- Altered mental state
- Mottled, clammy skin
- Oliguria
- Elevated blood lactate

↓

Circulatory shock

↓

Low estimated cardiac output or SvO_2

↓

High central venous pressure

↓

Obstructive circulatory shock

SYMPTOMS

Obstructive shock is characterized by a blockage in blood flow caused by a massive pericardial effusion, cardiac tamponade, or tension pneumothorax.

ECHOCARDIOGRAPHIC SIGNS

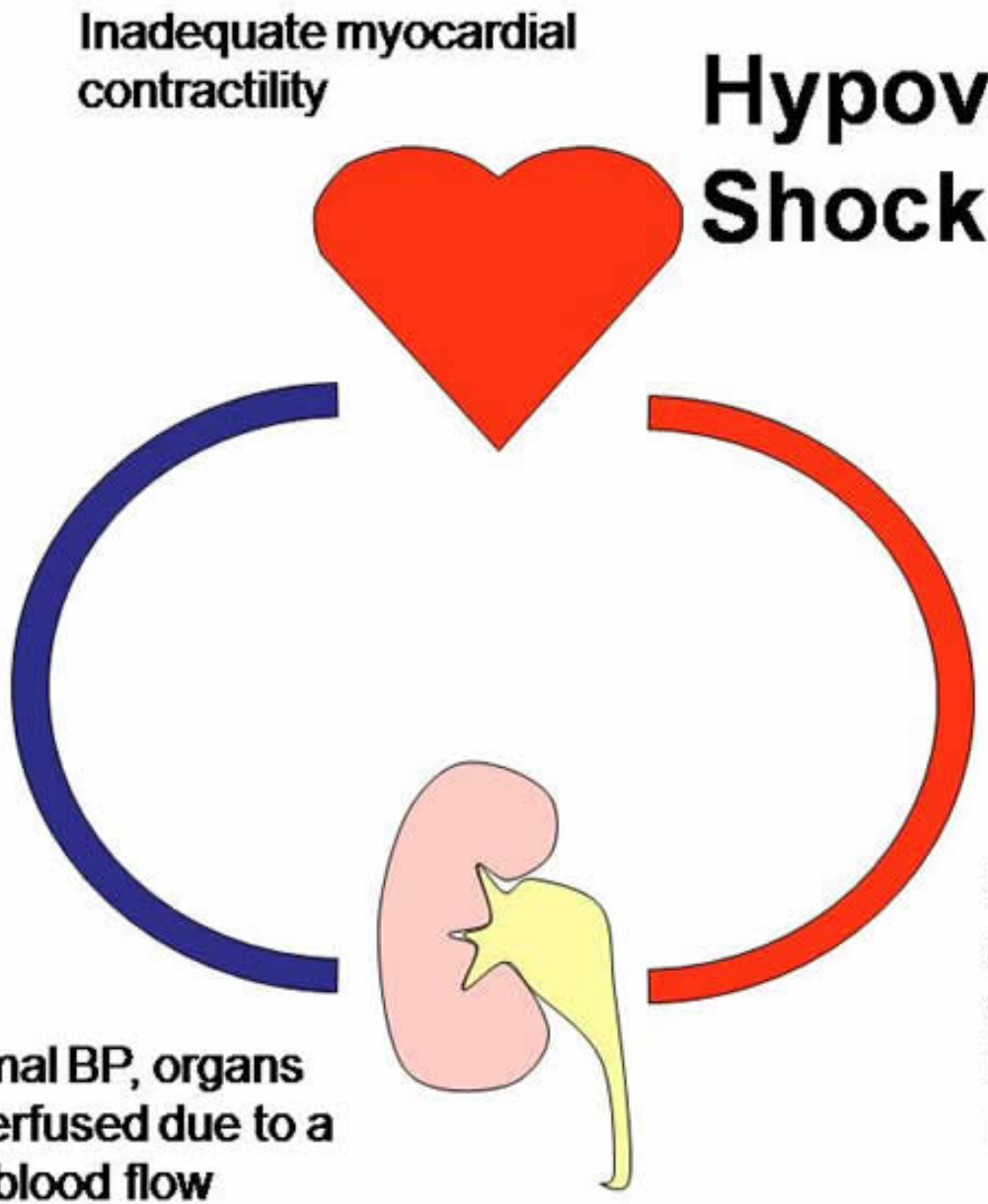
Depends on the cause. Pulmonary embolism or pneumothorax: dilated right ventricle, small left ventricle. Tamponade: pericardial effusion, small left and right ventricles, dilated inferior vena cava.

Obstructive

- Pulmonary embolus
- Cardiac tamponade
- Tension pneumothorax
- Coarctation of aorta

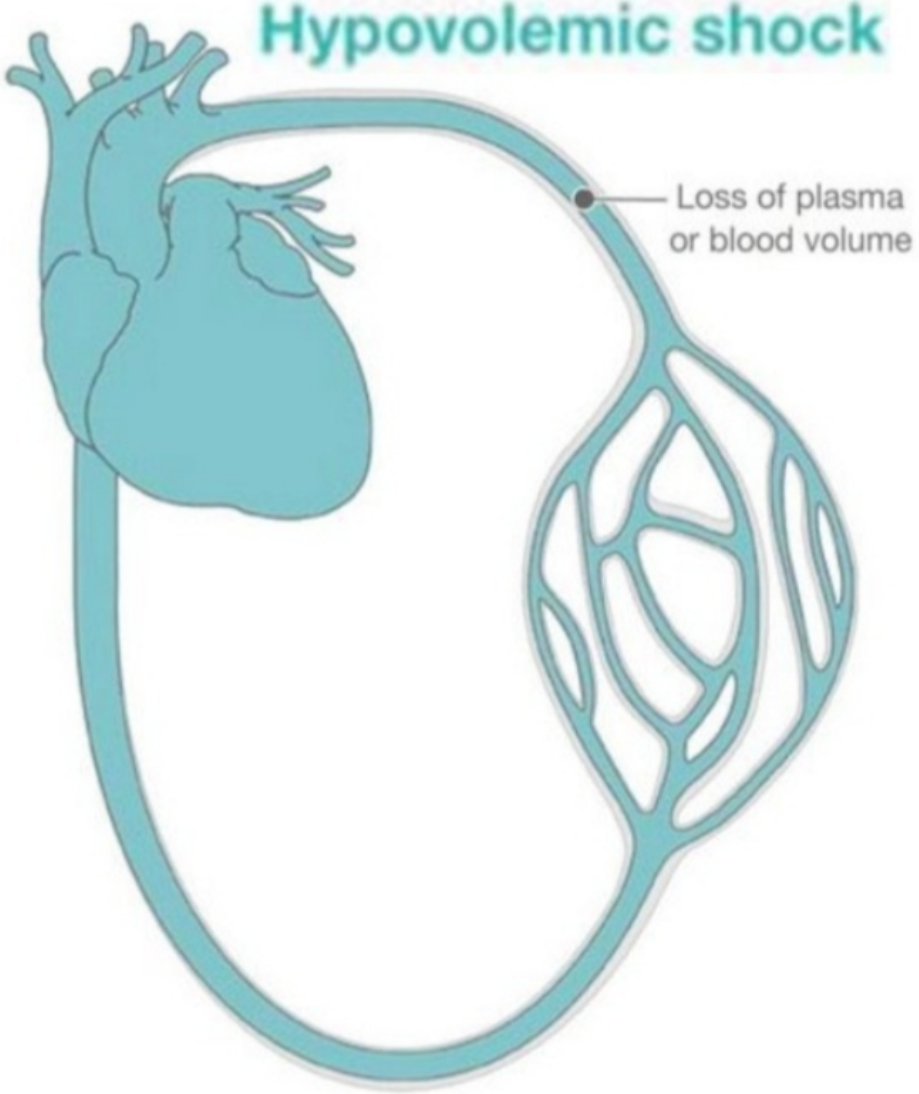
- Intravascular Compartment
- Preload

Hypovolaemic Shock



DIAGRAM

Hypovolemic shock



SIGNS OF CIRCULATORY SHOCK PATHWAY

Arterial hypotension and usually tachycardia

↓

Signs of tissue hypoperfusion present

- Altered mental state
- Mottled, clammy skin
- Oliguria
- Elevated blood lactate

↓

Circulatory shock

↓

Low estimated cardiac output or SvO_2

↓

Low central venous pressure

↓

Hypovolemic circulatory shock

SYMPTOMS

Hypovolemic shock is characterized by internal or external fluid loss leading to organ failure.

ECHOCARDIOGRAPHIC SIGNS

Small cardiac chambers and normal or high contractility.

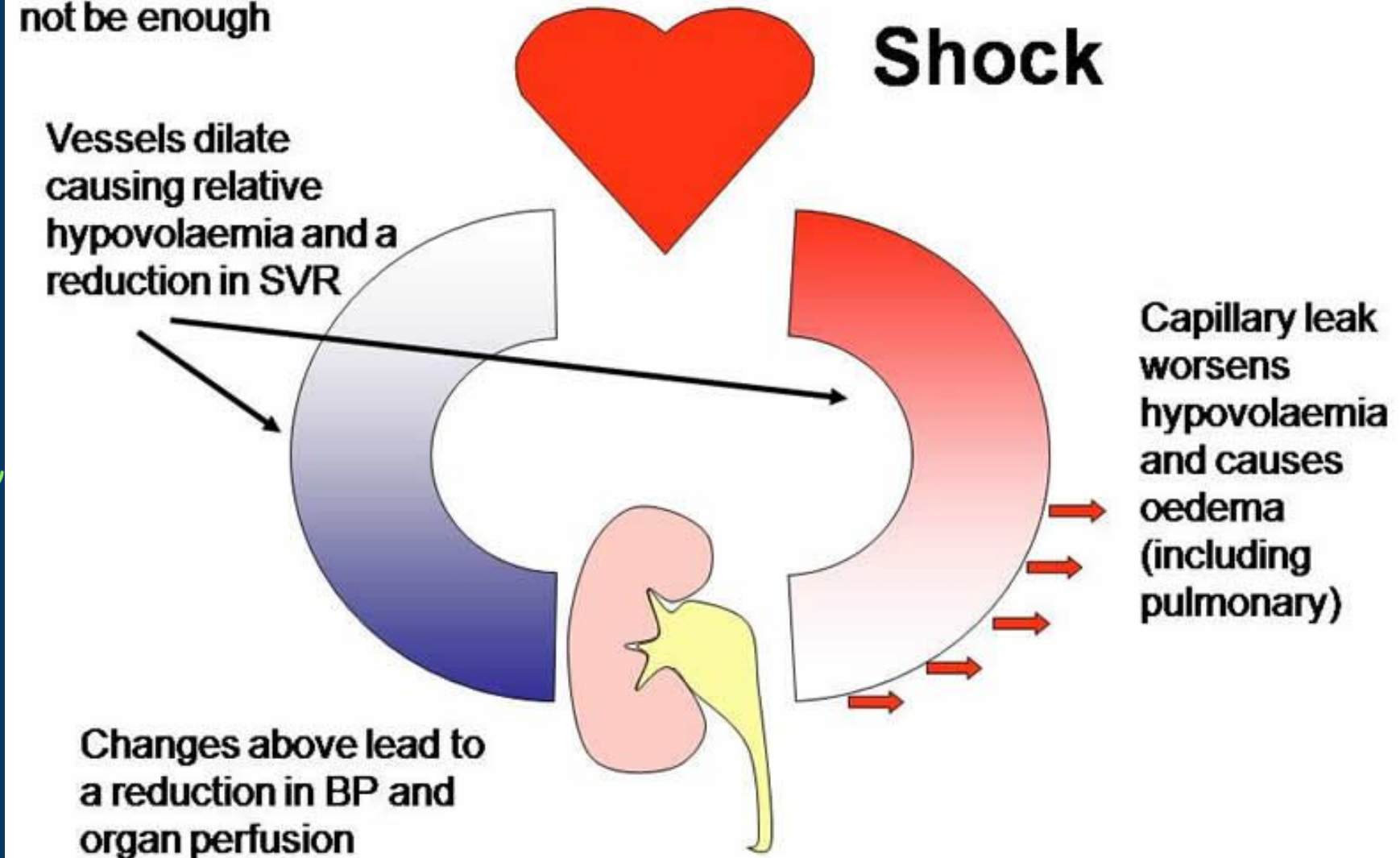
Vincent JL, De Backer D. Circulatory shock. N Engl J Med. 2013;369(18):1726-34.

Hypovolaemic → most responsive to management

- Fluid depletion
  - Vomiting and diarrhoea
  - Burns
  - Polyuria
    - pancreatitis
    - peritonitis
- Haemorrhagic
- Trauma
- Gastrointestinal
- Retroperitoneal

Distributive Shock

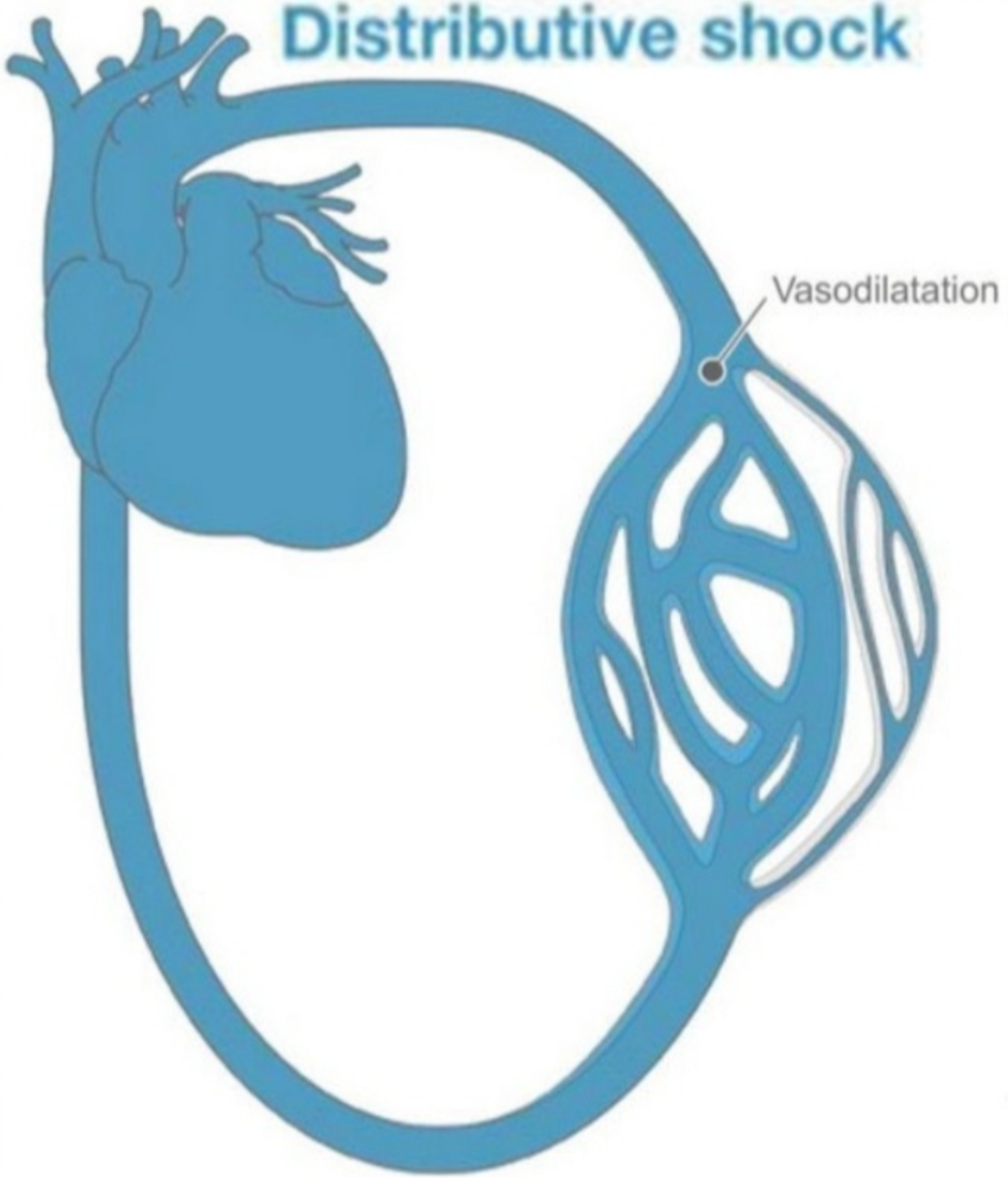
With adequate fluid therapy, the heart usually compensates by increase rate and contractility, although this might not be enough



↓afterload with leak

↓SVR → hyperdynamic heart (warm red body)

Distributive shock



Arterial hypotension and usually tachycardia

↓

Signs of tissue hypoperfusion present

- Altered mental state
- Mottled, clammy skin
- Oliguria
- Elevated blood lactate

↓

Circulatory shock

↓

Normal or high cardiac output or SvO_2

↓

Distributive circulatory shock

SYMPTOMS

Distributive shock is characterized by hypovolemia and hypotension. It is the result of vasodilatation and release of inflammatory mediators.

ECHOCARDIOGRAPHIC SIGNS

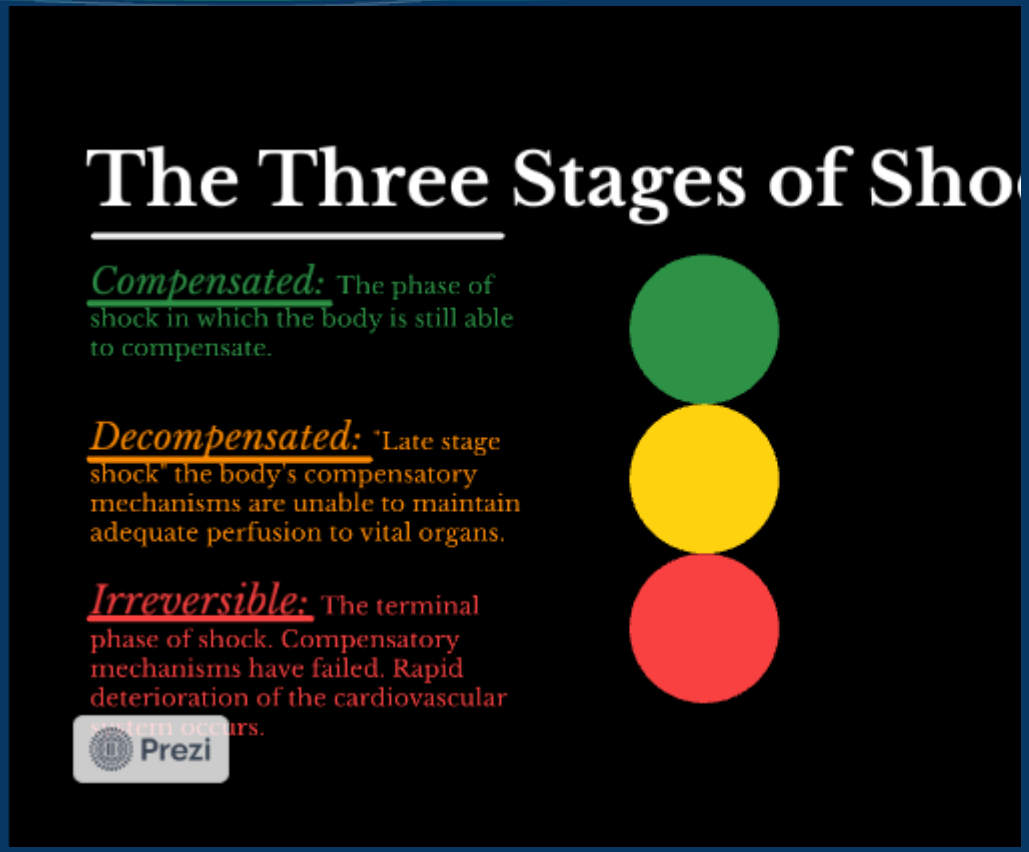
Normal cardiac chambers and (usually) preserved contractility.

Distributive

- Sepsis
- Neurogenic
- Anaphylaxis

Stages of Shock

- **Stage I Compensated**
  - Maintains end organ perfusion
  - BP is maintained usually by ↑ HR
- **Stage II Uncompensated**
  - Decreases micro-vascular perfusion
  - Sign/symptoms of end organ dysfunction
  - Hypotensive
- **Stage III Irreversible**
  - Progressive end-organ dysfunction
  - Cellular acidosis results in cell death



Key Issues

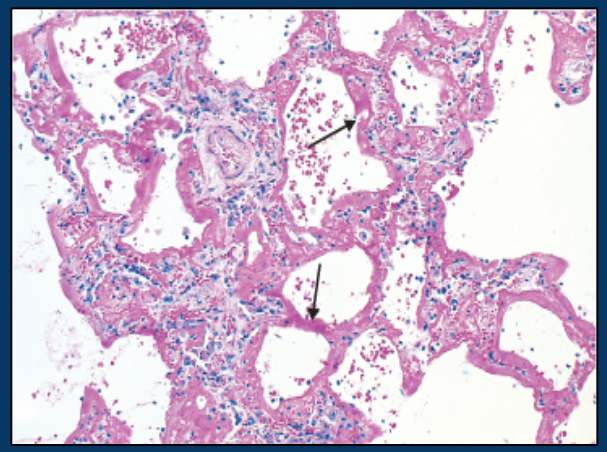
Recognize & **Treat** during compensatory shock phase

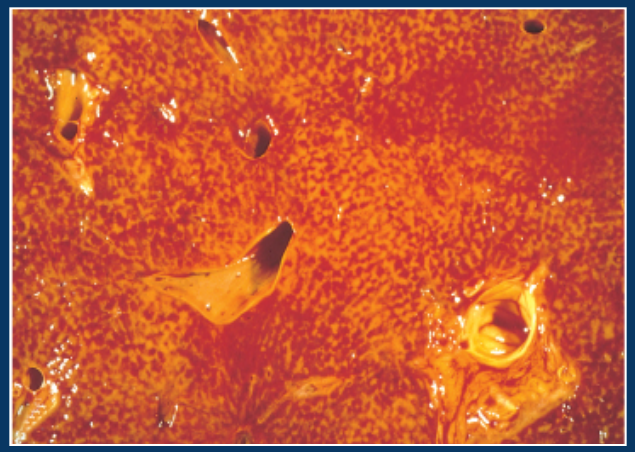
Mortality
increase 2-fold for every hour
in treatment delay.

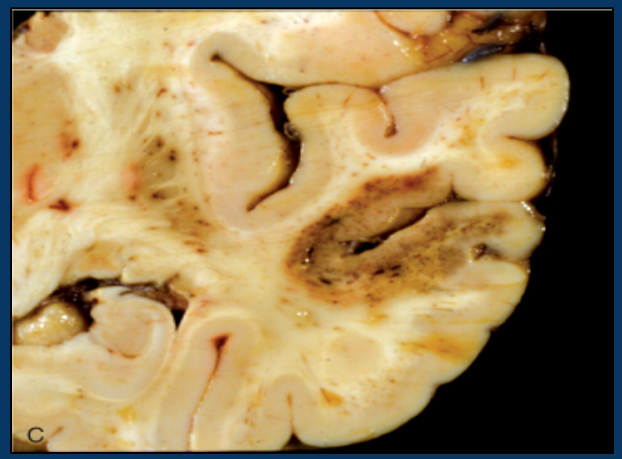
Han, Carcillo. Pediatrics 2003;112:793-799

Multisystem effect of shock

- Resp: Resp failure, ARDS
- Renal: ATN, acute renal failure
- CNS: infarcts & bleeding
- Liver: centrilobular necrosis
- GIT: bleeds, necrosis, ileus, bacterial translocation
- Haemat: DIC, vasculopathy, capillary leak



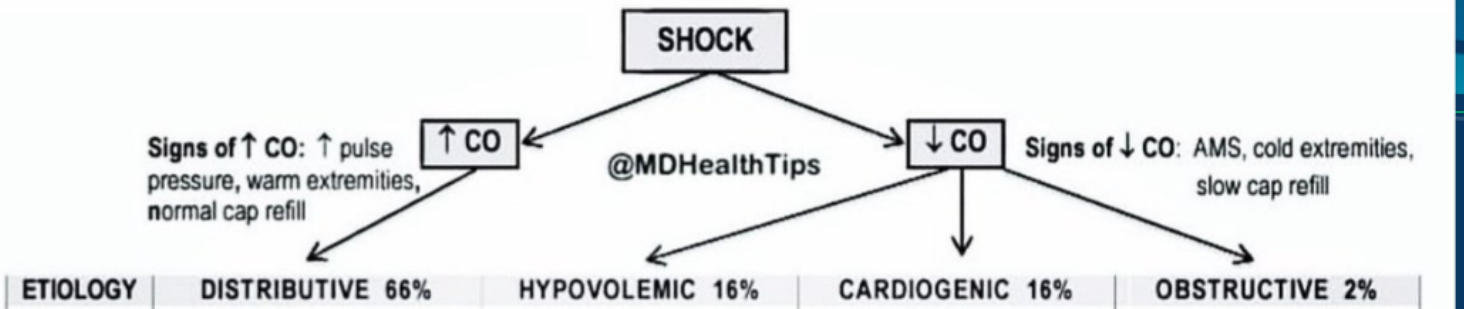


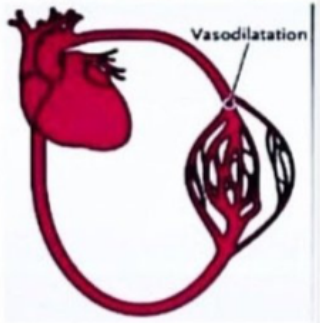
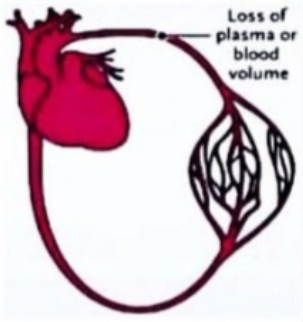
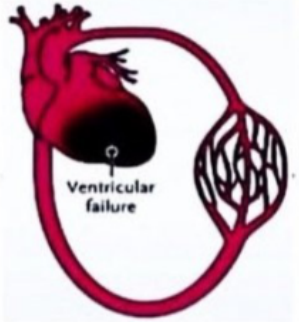
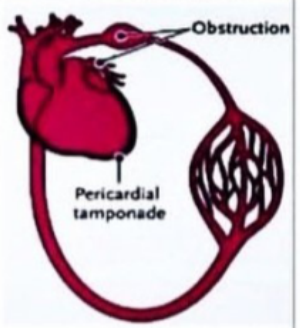


Robbins & Cotran Pathologic Basis of Disease: 2005

Common Features of Shock

- Cool, clammy skin
- Pale or ashen skin
- Bluish tinge to lips or fingernails (or gray in the case of dark complexions)
- Rapid pulse
- Rapid breathing
- Nausea or vomiting
- Enlarged pupils
- Weakness or fatigue
- Dizziness or fainting
- Changes in mental status or behavior, such as anxiousness or agitation



ETIOLOGY	DISTRIBUTIVE 66%	HYPOVOLEMIC 16%	CARDIOGENIC 16%	OBSTRUCTIVE 2%
				
PHYSIO	Decreased systemic vascular resistance and altered oxygen extraction	Low cardiac output and therefore inadequate oxygen transport		
EXAMPLE	**Sepsis/SIRS, anaphylaxis, adrenal insufficiency**, liver failure, toxins, spinal/neurogenic	**Bleed (GIB, RP bleed), third spacing (pancreatitis)**, GI losses, overdiuresis	**MI, CHF**	**PE, tension PTX, tamponade**
EXT	Warm and dry	Cold and dry	Cold and wet	Cold and dry
CVP (JVP)	↓	↓	↑	↑
CO (SvO_2)	↑ or normal	↓↓	↓↓	↓↓
SVR	↓↓	↑	↑	↑
BASIC TX	All causes: IVF, pressors Sepsis: source control, abx Adrenal: steroids Anaphylaxis: epi 0.3mg IM	Ensure adequate access! Most cases: Crystalloid HRS/SBP: Albumin Hemorrhage: pRBCs	Based on etiology consider diuresis, pressors, inotrope / inodilators, +/- PA line	PE: Heparin/lysis PTX: needle decompression Tamponade: pericardiocentesis

Each type of shock needs different management

ex:

hypovolemic → blood units

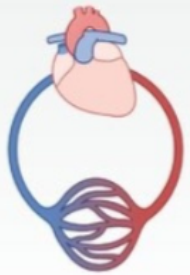
Cardiogenic → Cath

Classification of Shock

Volume

Shift — Distributive shock

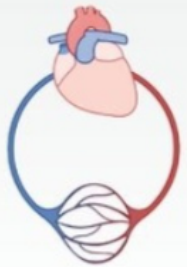
Septic	Capillary leakage
Anaphylactic Anaphylactoid Neurogenic	Vascular tone dysregulation



Vasodilation

Loss — Hypovolemic shock

Hemorrhagic (traumatic or nontraumatic)	Blood (whole)
Nonhemorrhagic (nontraumatic)	Body fluids (e.g., GI loss)
Nonhemorrhagic (traumatic)	Plasma (e.g., from burns)

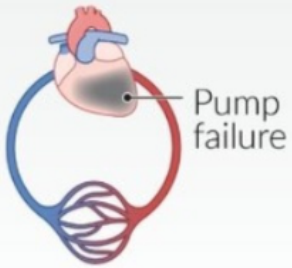


Hypovolemia

Output

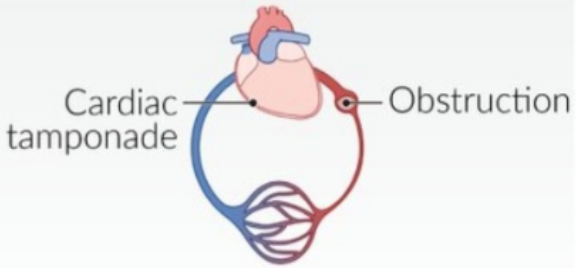
Cardiac — Cardiogenic shock

Myocardial causes	Myocardium
Arrhythmias	Conduction system
Valvular heart disease	



Extracardiac — Obstructive shock

Impaired diastolic filling	E.g., cardiac tamponade
↑ Ventricular afterload	E.g., massive PE
Obstruction of venous return	E.g., tension pneumothorax



Hemodynamics in Shock

measured by Swan Ganz (pulm. A cath)

measured by echo

Physiologic variable	Preload (R)	Preload (L)	Pump function	Afterload	Tissue perfusion
Clinical measurement	**RAP/CVP**	**PCWP/LVEDP**	**Cardiac output/ index**	**SVR/TPR**	**MvO_2**
Hypovolemic · Hemorrhagic · Burns · Pancreatitis (3rd spacing)	↓	↓↓	↓	↑	↓
Distributive · Sepsis · Anaphylaxis · Addisonian crisis	↓	↓	↑	↓	↑
Cardiogenic					
LV Dysfunction · MI (LAD) · Acute myocarditis	↑	↑	↓	↑	↓
RVMI · RCA occlusion · Inferior and RV MI · Isolated RV dysfunction	↑	↓	↓	↑	↓
Obstructive					
Pulmonary Vascular · PE · Severe PH	↑	↓	↓	↑	↓
Mechanical · Pericardial tamponade · Tension pneumothorax · Constrictive pericarditis · Restrictive cardiomyopathy	↑	↑	↓	↑	↓

- RAP/CVP: rt atrial pressure/ central venous pressure
- PCWP/LVEDP: pulmonary capillary wedge pressure/ lt ventricular end diastolic pressure
- SVR/TPR: systemic vascular resistance / total peripheral resistance
- MvO_2: mixed venous O_2 content
- LAD: lt ant. descending A
- RVMI: rt ventricular MI
- RCA: rt coronary A
- SV: stroke volume
- PE: pulm. embolism
- PH: pulm. Htn

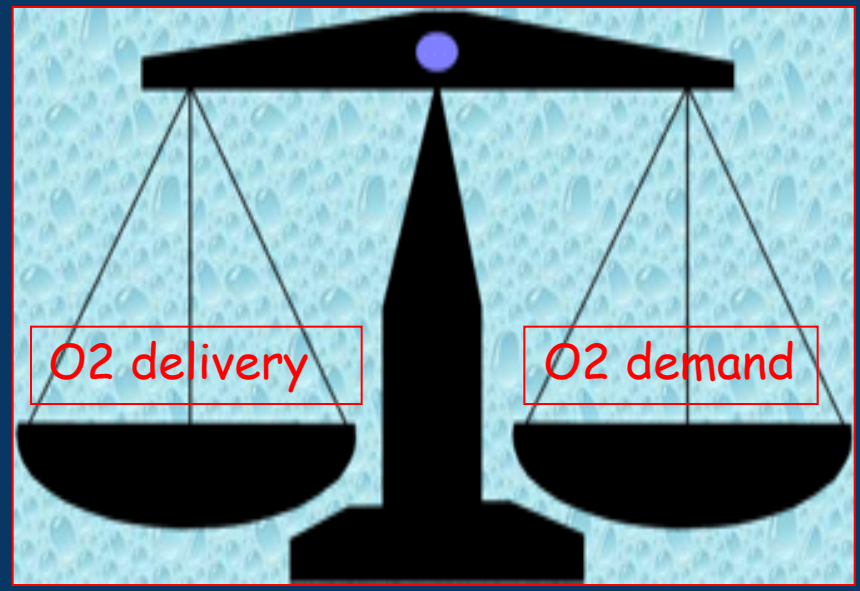
Shock states coexist

Changing hemodynamics

Individualize treatment

Treatment principles

1. Increase O2 delivery
2. Reduce O2 demand
   - Fever
   - Tachycardia
   - Tachypnea
   - Anxiety & restlessness
   - Pain
   - Seizures & shivering



ABCDE

Airway

breathing

Circulation

disabilities

exposure

Resuscitation Priorities

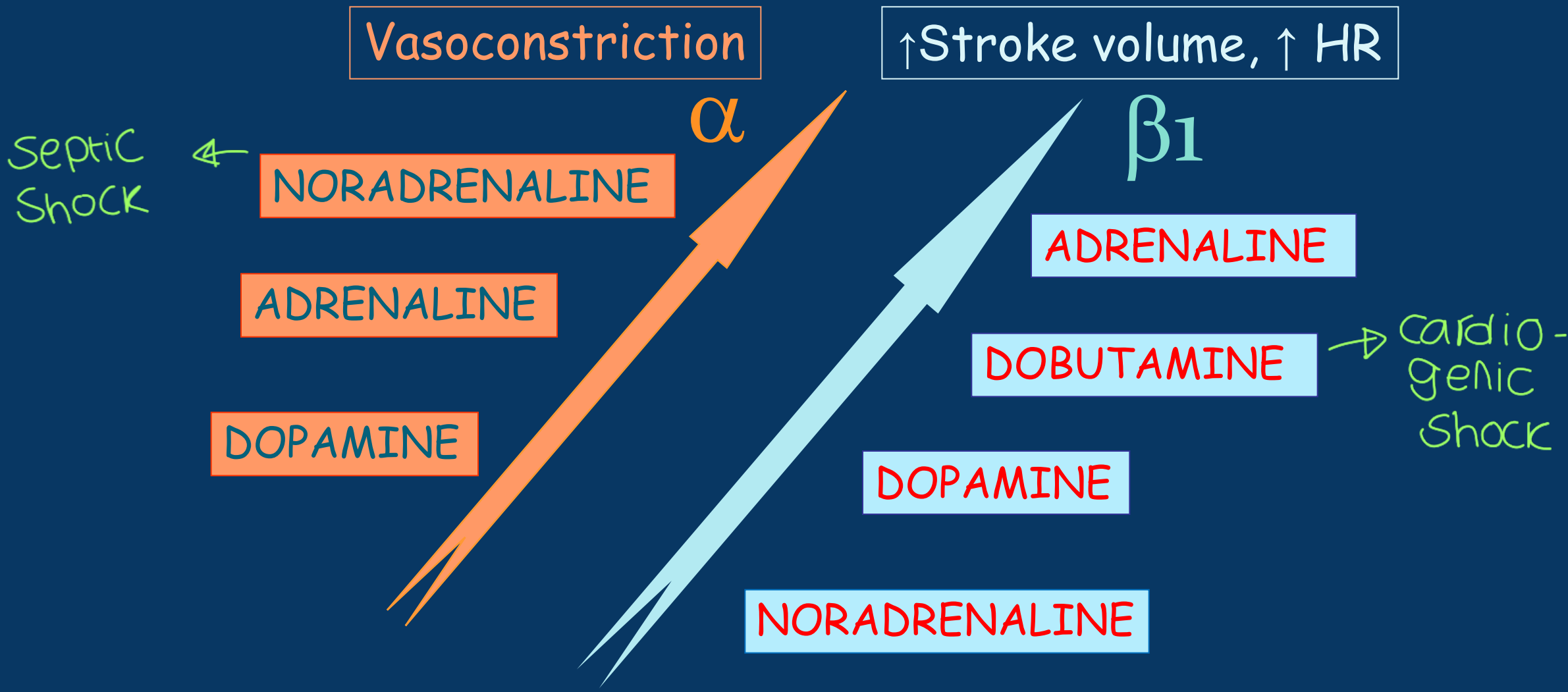
Increase O2 delivery

- V: Ventilate & Oxygenate.
- I: Infuse:
  - Fluids, fluids, fluids
  - Electrolytes
  - Blood- Hb >10
- P: ↑Pump Function:
  - Inotropes
  - Rhythm control
  - Electrolytes & glucose
- E: Etiology: - Treat the cause.

FLUID, FLUID, FLUID

- Regardless of etiology - fluid bolus x3
  - 5ml/kg cardiac
  - 10ml/kg trauma
  - 20ml/kg sepsis
- Delayed fluid resuscitation ↑ mortality.
  - *Rivers NEJM 2001, Han Pediatrics 2003*
- Reassess liver & lungs.
- Septic shock may need up to 200ml/kg.
- No evidence one is fluid superior.
  - *Finfer NEJM 2004*

Inotropes in fluid resistance



Pediatric Cardiac Intensive Care . Chang & Wernovsky

Resuscitation endpoints

- No difference between peripheral & central pulses
- Warm skin, CFT < 2sec
- Normal BP for age
- Decreasing lactate & BE
- Improving mental state
- UO >1ml/kg/h

Trend of improvement

Peters ICM 2008;34

من هذه السلايد للآخر ماكانوا موجودين بالمحاضرة 020

Noradrenaline

- Drug of choice for
  - Warm shock
  - Myocardial contractility not severely impaired
- Central line

B_1	B_2	α
+	0	++++

Dobutamine

- More expensive than dopamine
- Use to ↑contractility when BP stable
- Drug of choice for cardiacs & PHT
- Age –specific sensitivity
- Peripheral IV

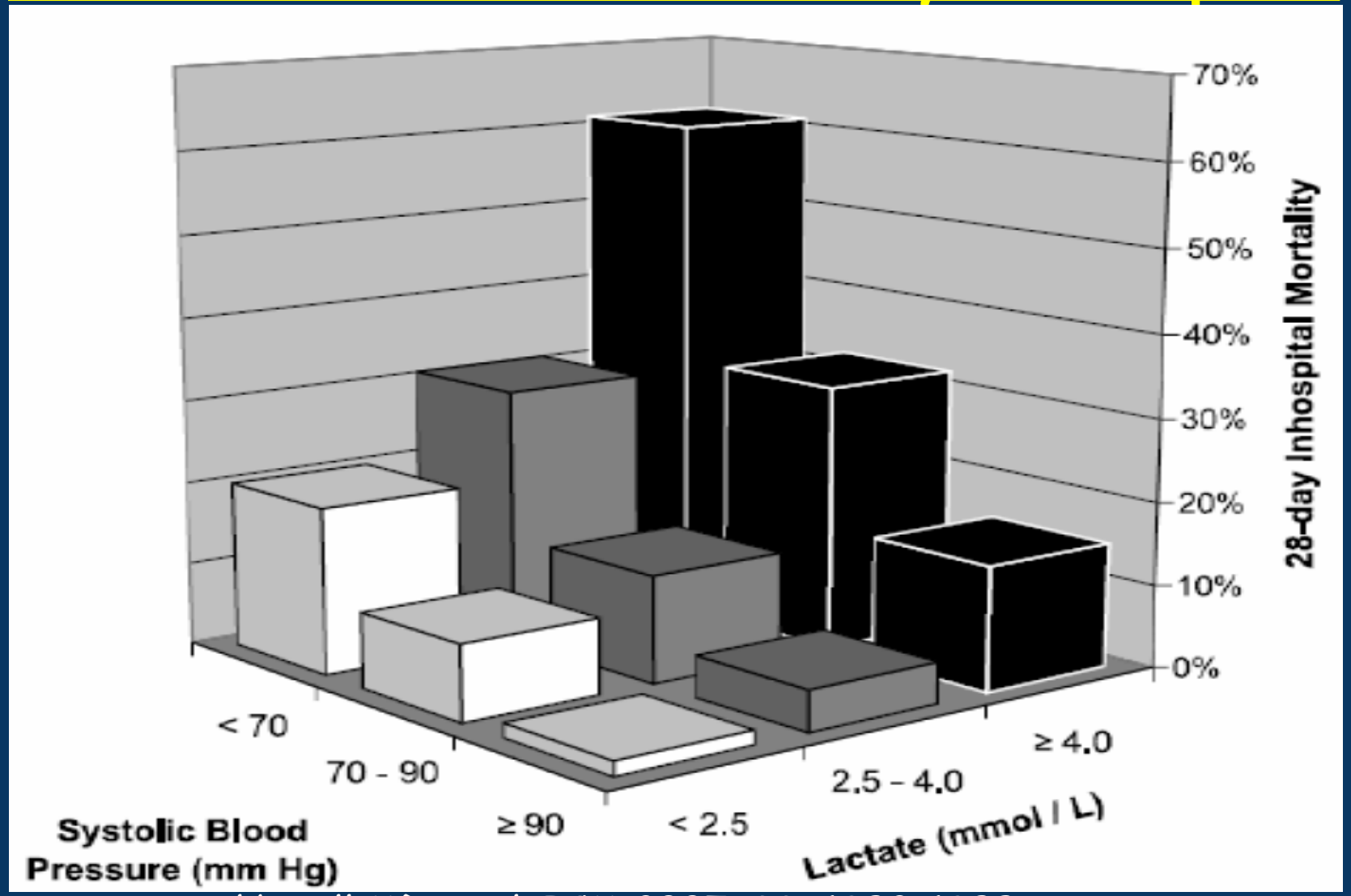
B_1	B_2	α
+++	+	+

Adrenaline

- Low dose (< 0.3mcg/kg/min) β effect - ↑Contractility
- High dose α effect - ↑ BP
- Ideally via central line
- **Side effects**
  - Renal dysfunction, gut ischaemia
  - ↑ Glucose
  - ↑ Lactate & metabolic acidosis
  - Myocardial necrosis

B_1	B_2	α
+++	++	+++

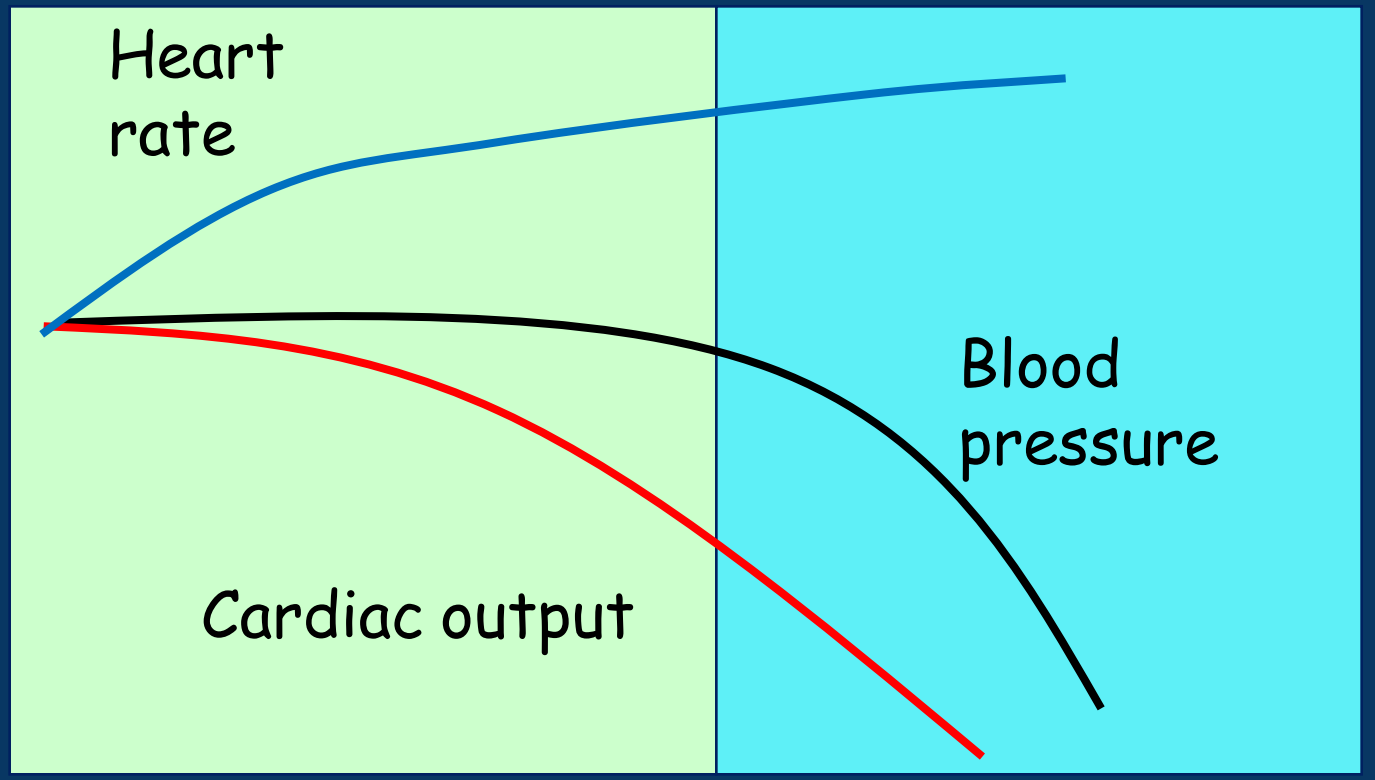
Lactate, BP & Mortality in Sepsis



Howell MD et al. ICM 2007; 33: 1892-1899

Hemodynamic Response to Shock

J Carcillio. Fluid Resuscitation of Hypovolemic Shock. ICM 2006;32:958



- IL-6, also produced predominantly by the macrophage, is the best predictor of prolonged recovery and development of multiple organ failure after shock

- Although the endothelium normally produces NO, the inflammatory response stimulates the inducible isoform of NO synthase (iNOS), which is overexpressed and produces toxic free radicals that contribute to the hyperdynamic cardiovascular response in sepsis

Measure	Hypovolaemic	Cardiogenic	Obstructive	Distributive
Preload (central venous pressure/Pulmonary artery occlusion pressure)	Decreased	Increased	increased	Decreased
Afterload (systemic vascular resistance)	Increased	Increased	Increased	Decreased
Contractility (cardiac index/stroke volume index)	Decreased	Decreased	Decreased	Increased
Oxygen delivery	Decreased	Decreased	Decreased	Increased
Systemic oxygen consumption (venous oxygen saturation	Increased	Decreased	Decreased	Decreased
Oxygen balance (venous oxygen saturation/capillary oxygen saturation)	Decreased	Decreased	Decreased	Increased

Examples	Hypovolaemic	Cardiogenic	Obstructive	Distributive
	Haemorrhage Burns Pancreatitis	Post-MI Malignant dysrhythmia Acute myocarditis	Tension pneumothorax Cardiac tamponade Pulmonary embolism	Septic shock Anaphylaxis Neurogenic shock

Common errors:	
Failure to recognize severity.	Early recognition & Rx Regular reassess
Ventilation delayed till arrest	Prioritise A & B
Crash intubation	Plan & prepare intubation
Myocardial depressant drugs for intubation.	Slow administration. •Ketamine •Fentanyl •Etomidate

Common errors:	
•No secure IV access •Wasting time on IV access	IO needle after 90 sec.
Inadequate fluid	•Fluidx3 •Pushed in •Reassess liver & lungs
Rx increase O2 demand	•Cooling •Sedation & pain control •Seizure control
Delayed antibiotics	Antibiotics within 1 hour

Not improving

- Coexisting cause of shock
- Changing hemodynamics
- Cardiogenic shock ? Echo
- Adrenal insufficiency ? Steroids
- Tension pneumothorax
- Electrolytes & glucose

Reassess ABC's & secondary survey

- Thank you for your Attention