Shock

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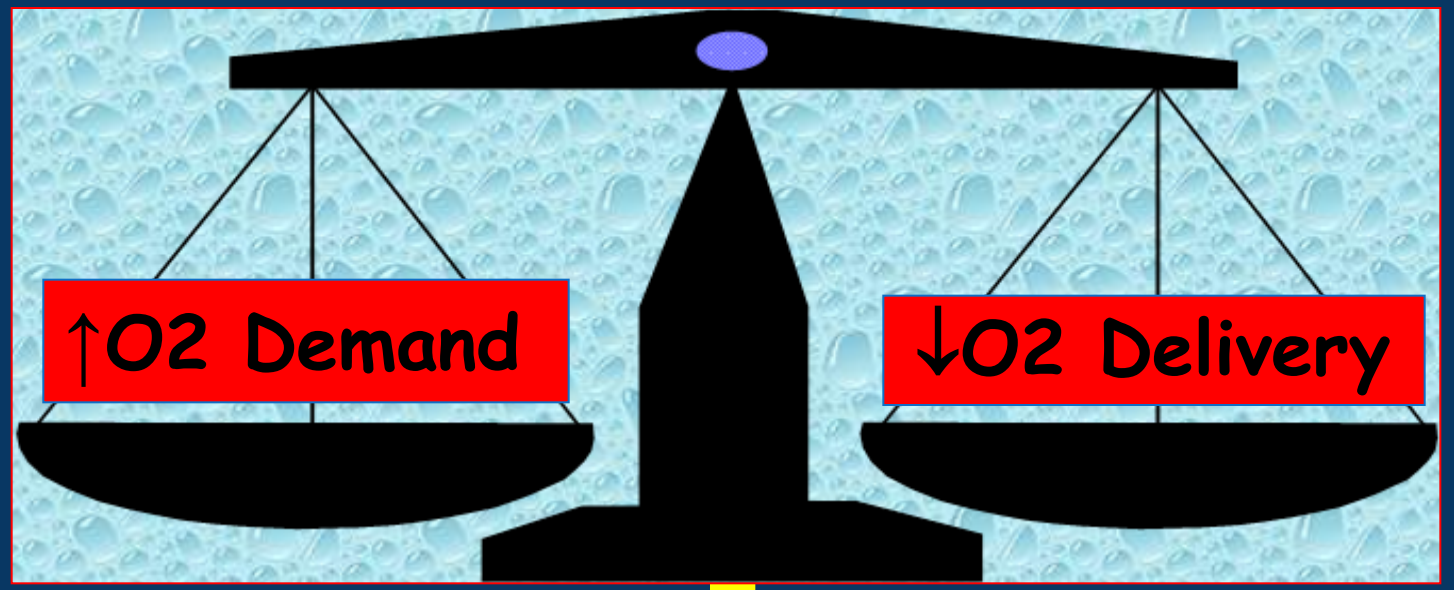
Outline for Today

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

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

Physiologic Determinants

O_2 consumption = O_2 Delivery – O_2 Return

$VO_2 = CO(C_aO_2-C_vO_2)$

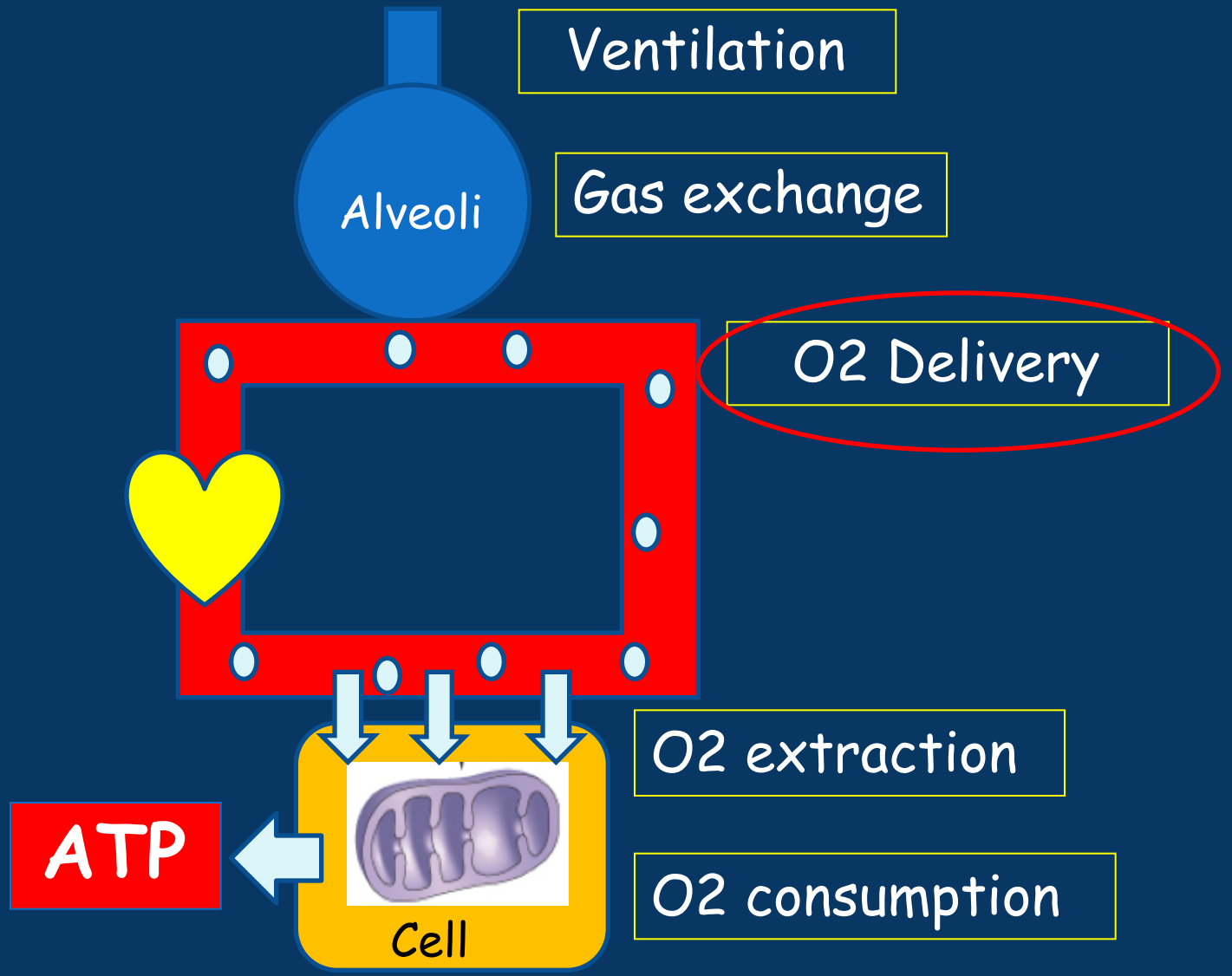
$VO_2 = CO*1.34*Hgb*(S_aO_2-S_vO_2)$

CO = cardiac output

C_aO_2 and C_vO_2 = arterial and mixed venous oxygen content

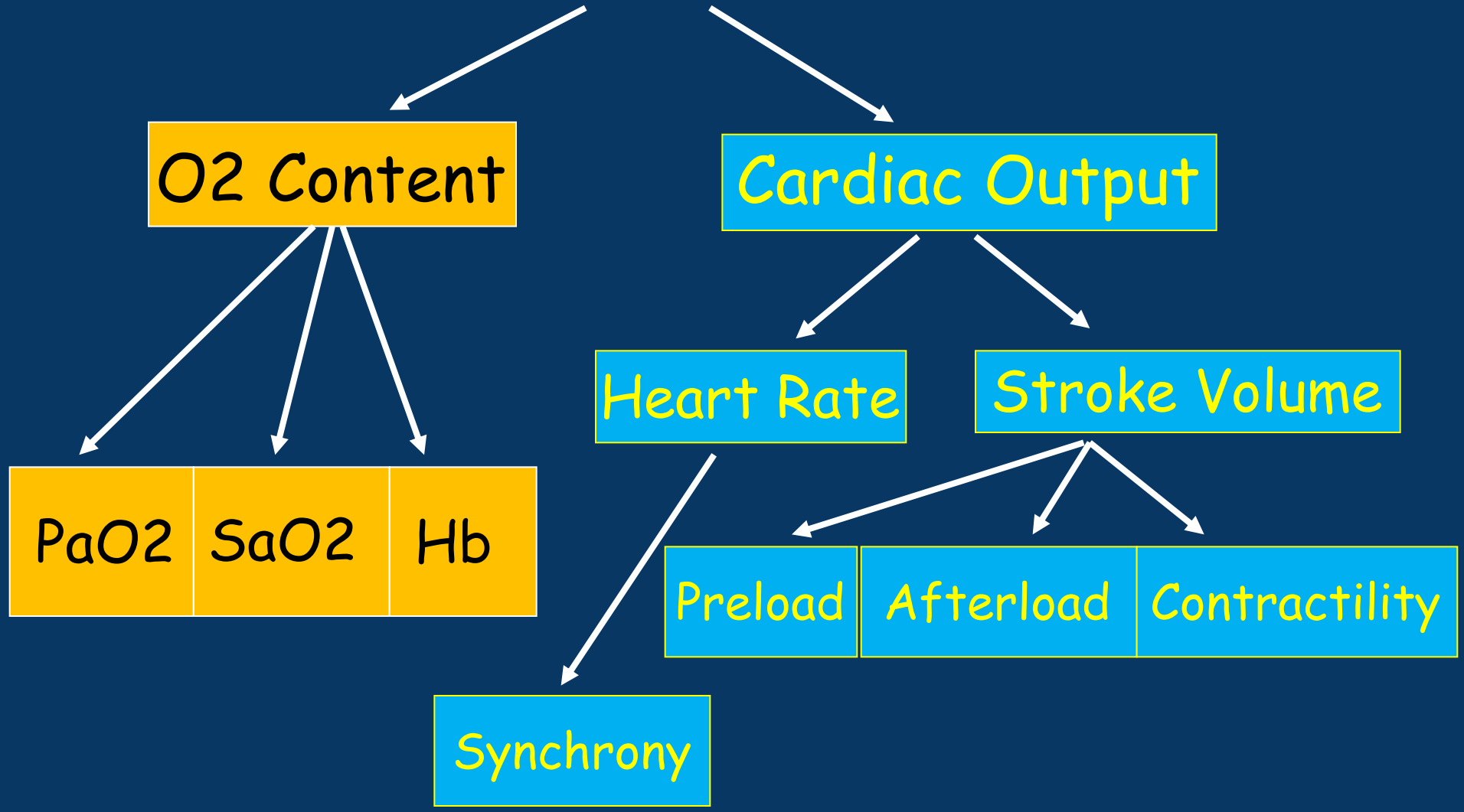
S_aO_2 and S_vO_2 = arterial and mixed venous oxygen saturation

Oxygen Delivery to Tissues



Oxygen Delivery Components

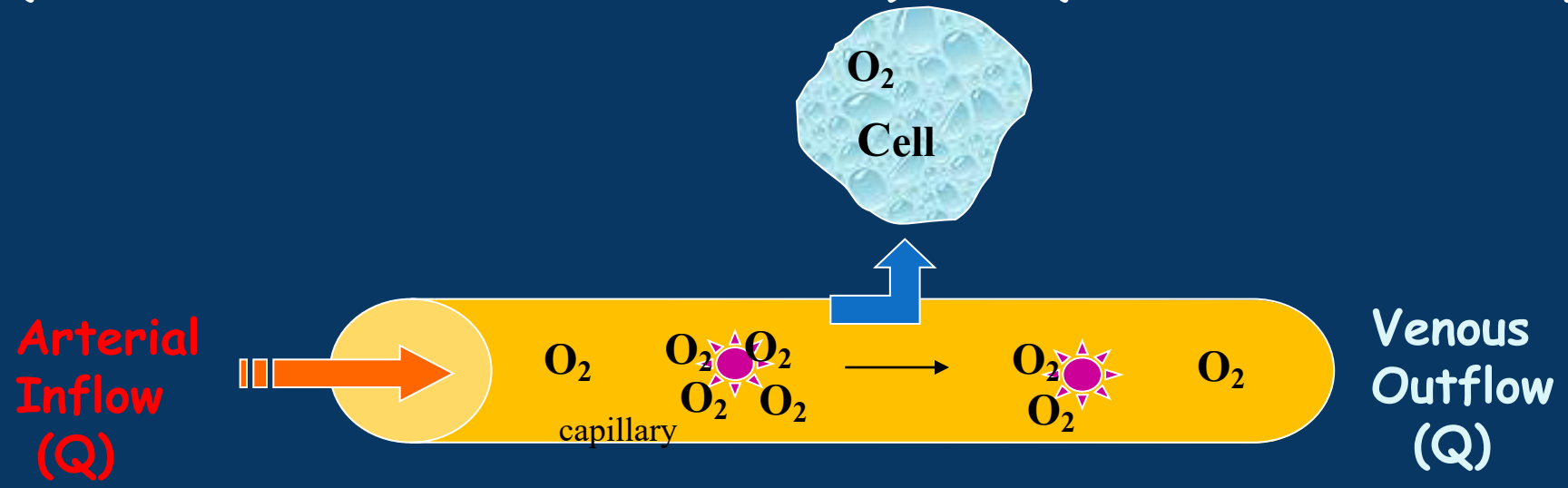
Oxygen Delivery Components



Oxygen Content of Blood

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

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



(Adapted from the ICU Book by P. Marino)

Glucose

2x ATP

Pyruvic Acid

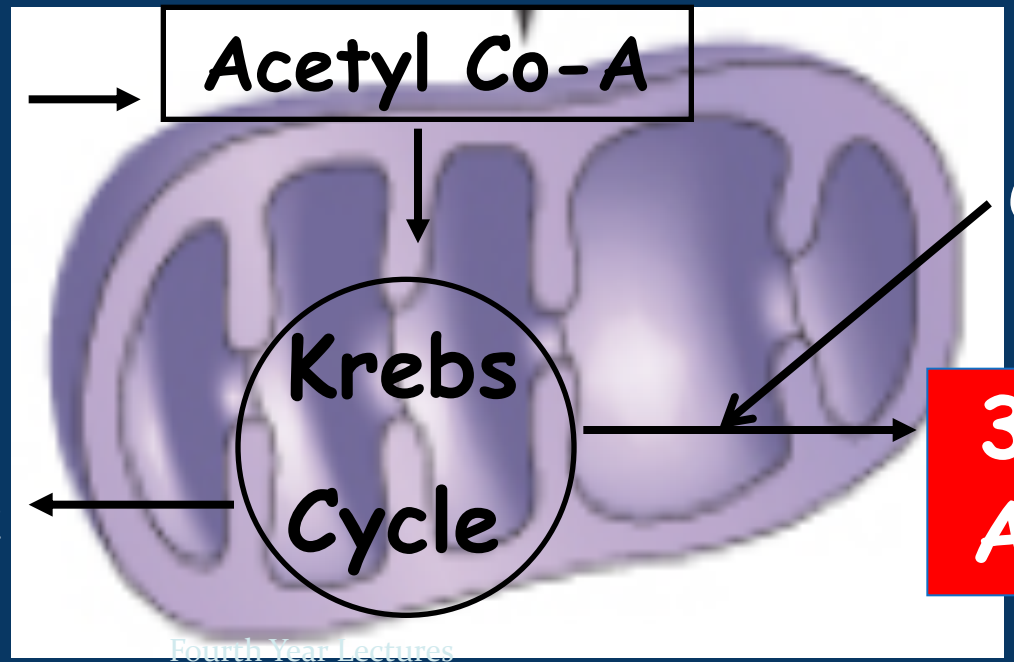
Lactic Acid

Anaerobic

Fatty Acids

Amino Acids

Acetyl Co-A



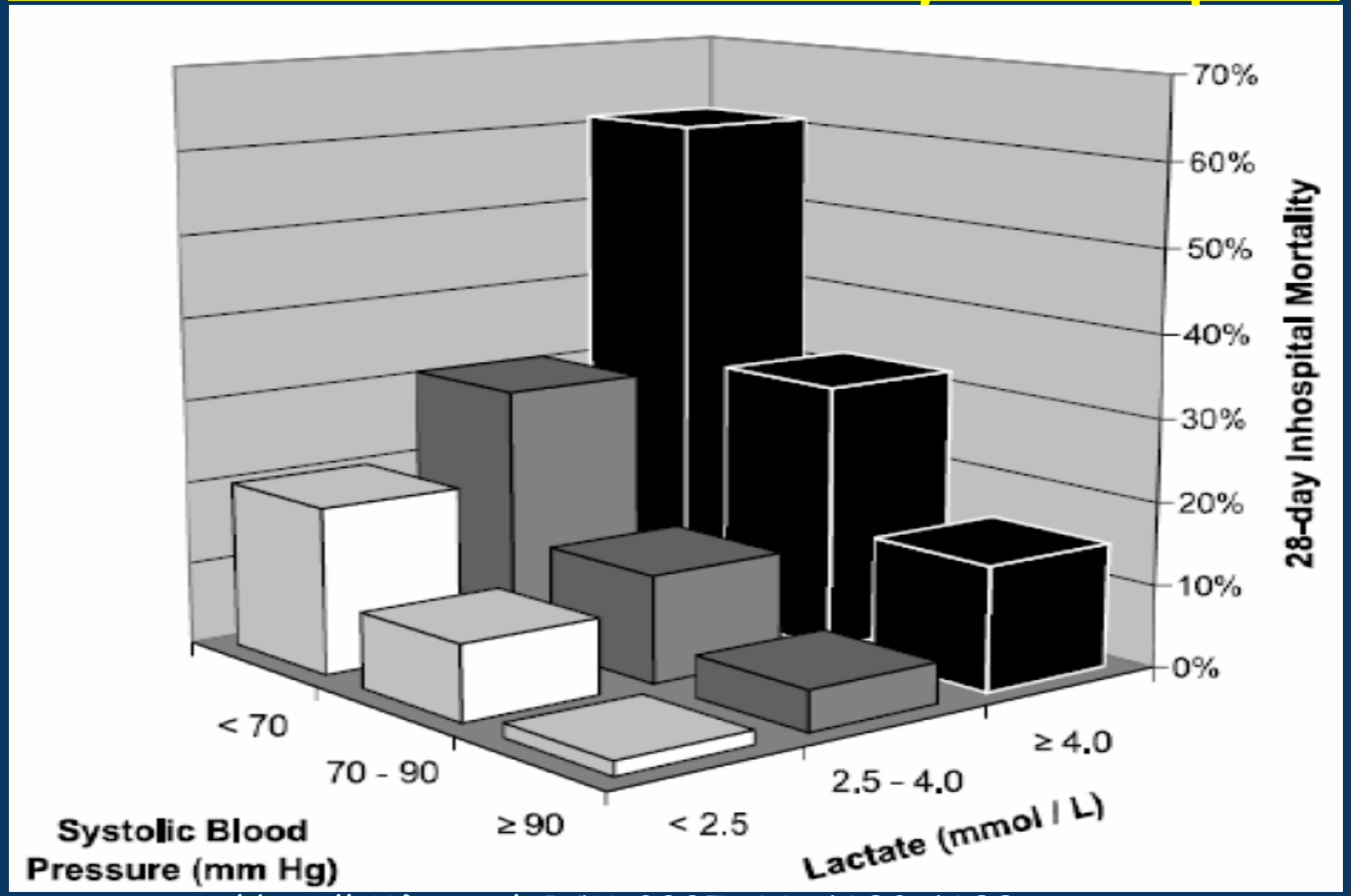
O_2

CO_2

38x ATP

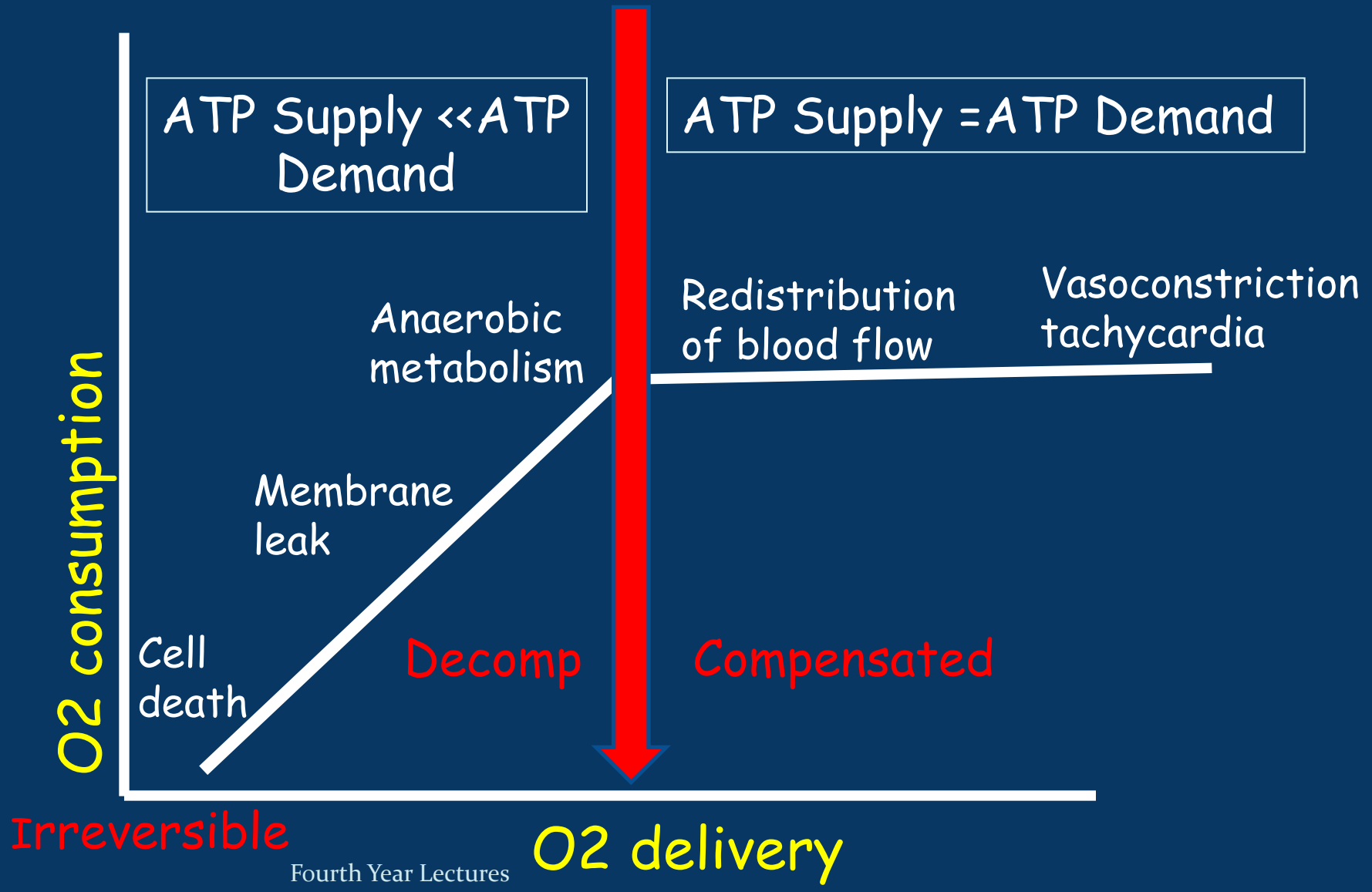
Aerobic

Lactate, BP & Mortality in Sepsis



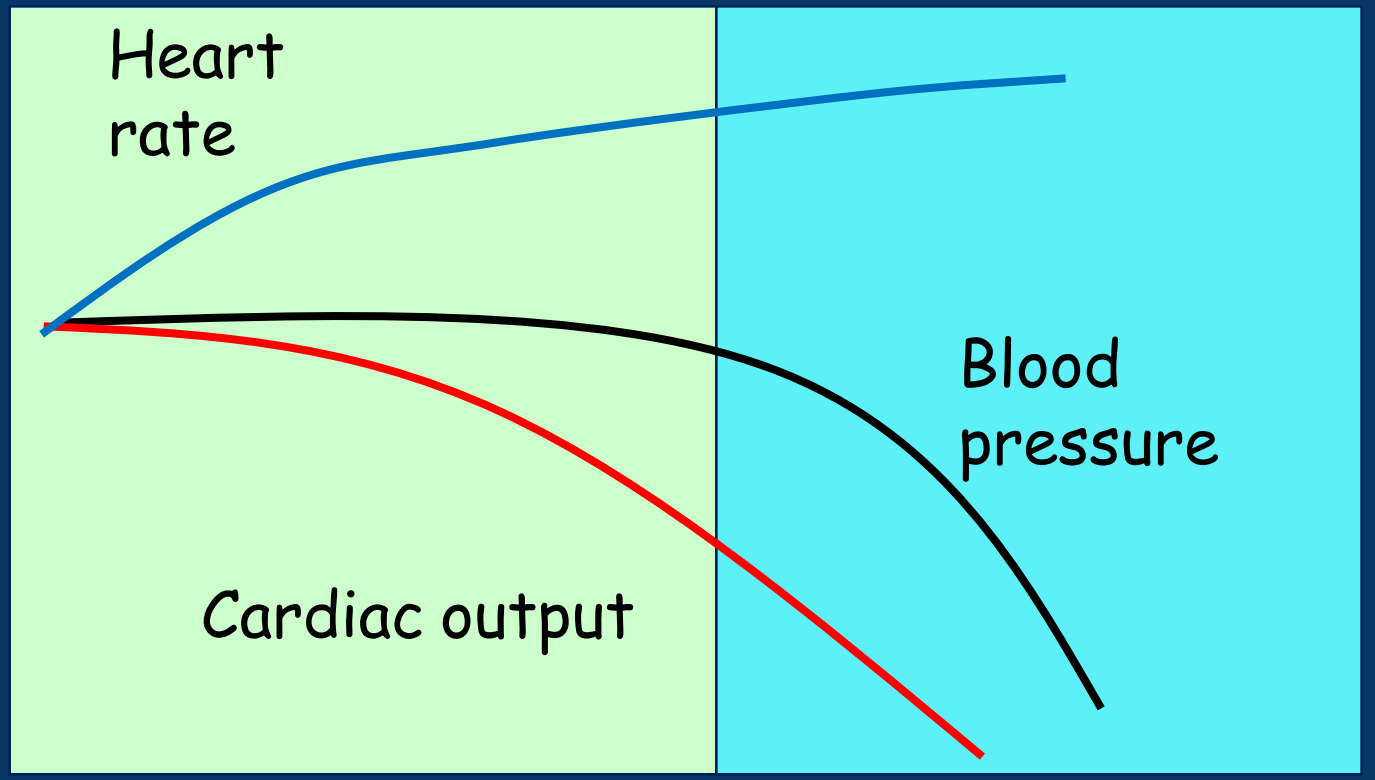
Howell MD et al. ICM 2007; 33: 1892-1899

Stages of shock



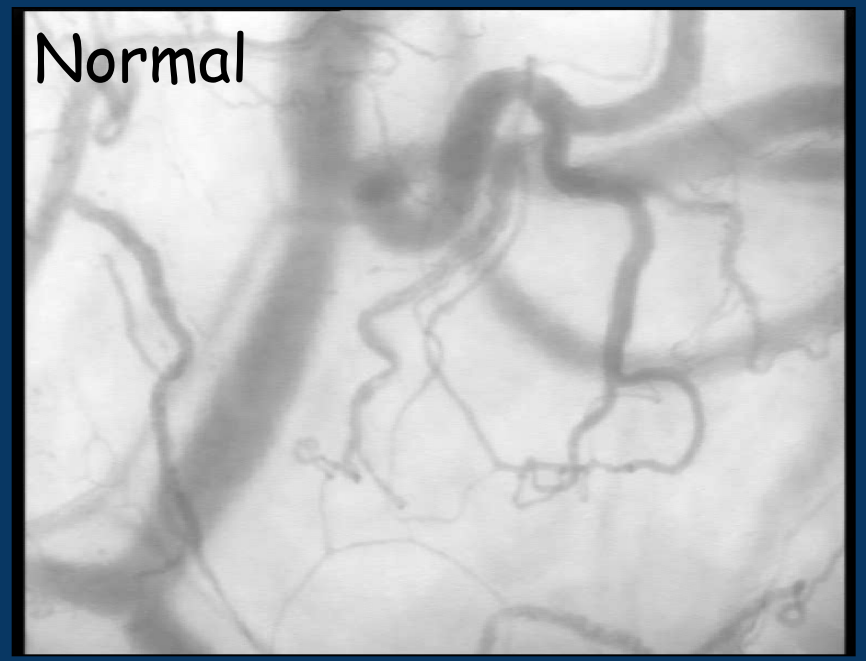
Hemodynamic Response to Shock

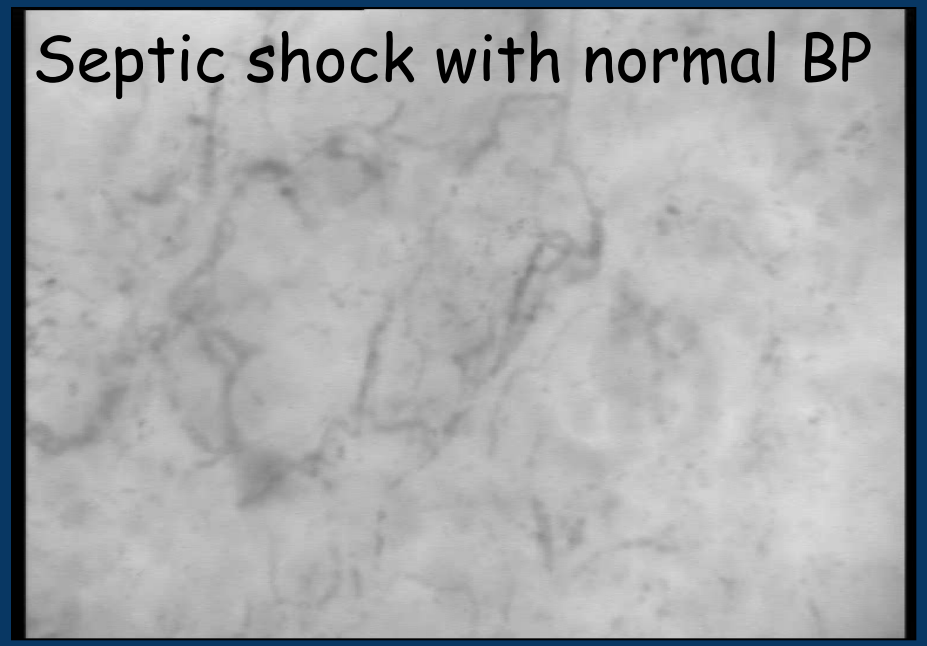
J Carcillio. Fluid Resuscitation of Hypovolemic Shock. ICM 2006;32:958



Key Issues In Shock

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

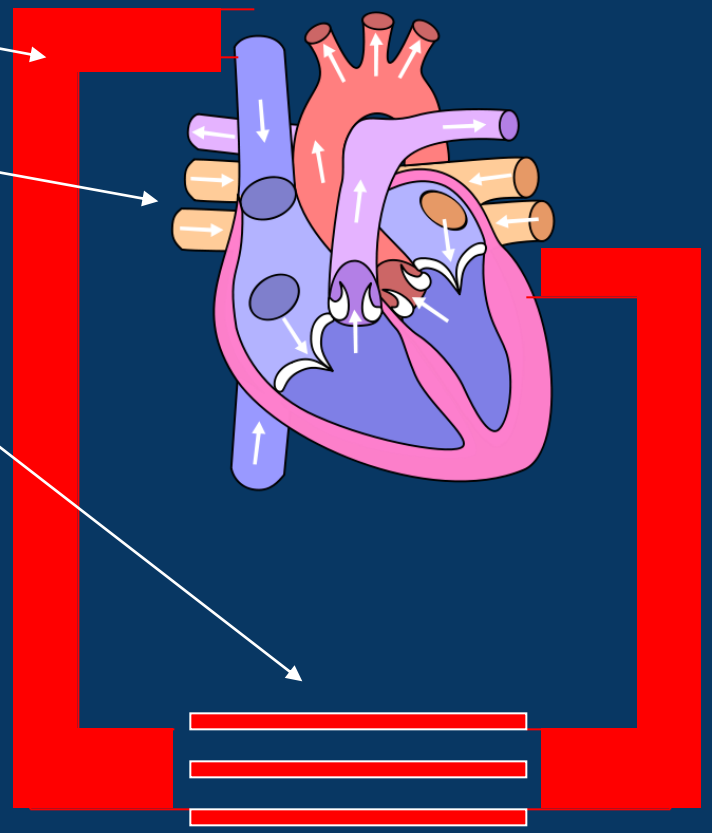




De Baker CCM 2006 34 :403-408

Key Elements of Blood Pressure

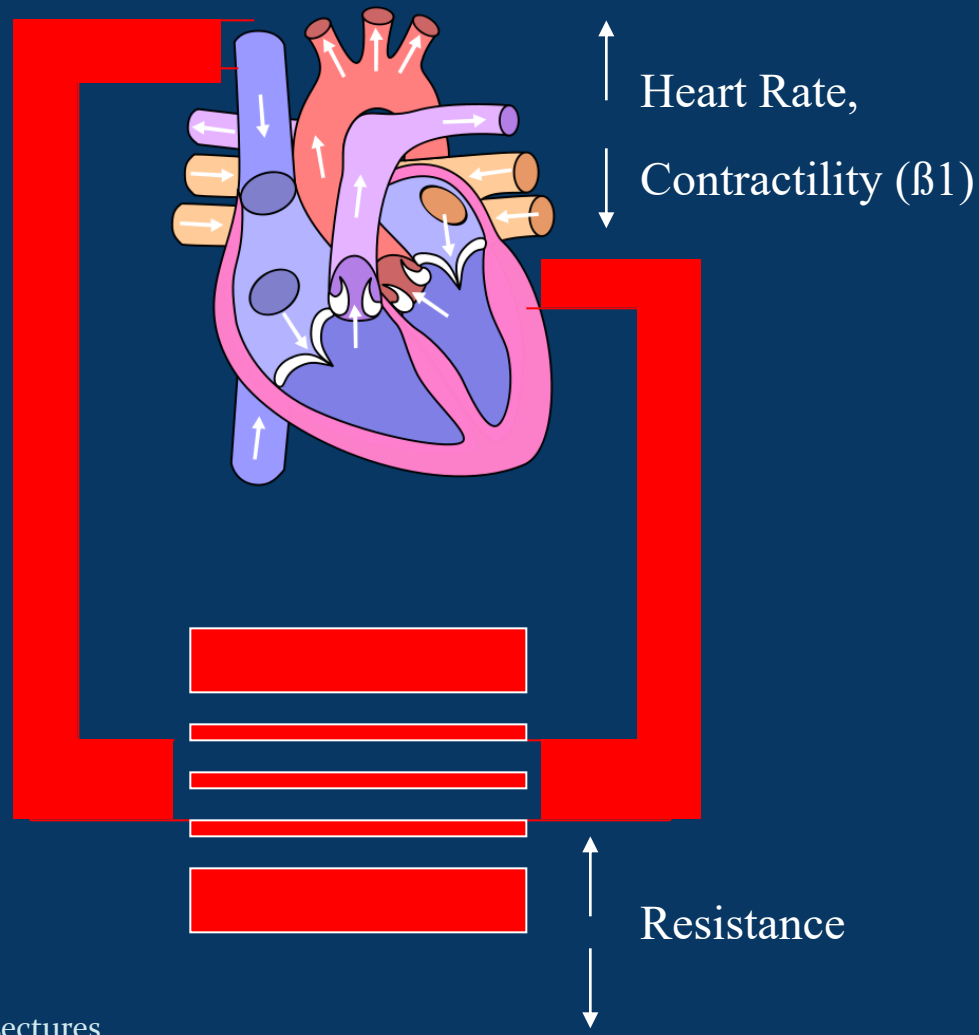
- Fluid
- Pump
- Pipes



Mean Arterial Pressure (MAP)

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

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



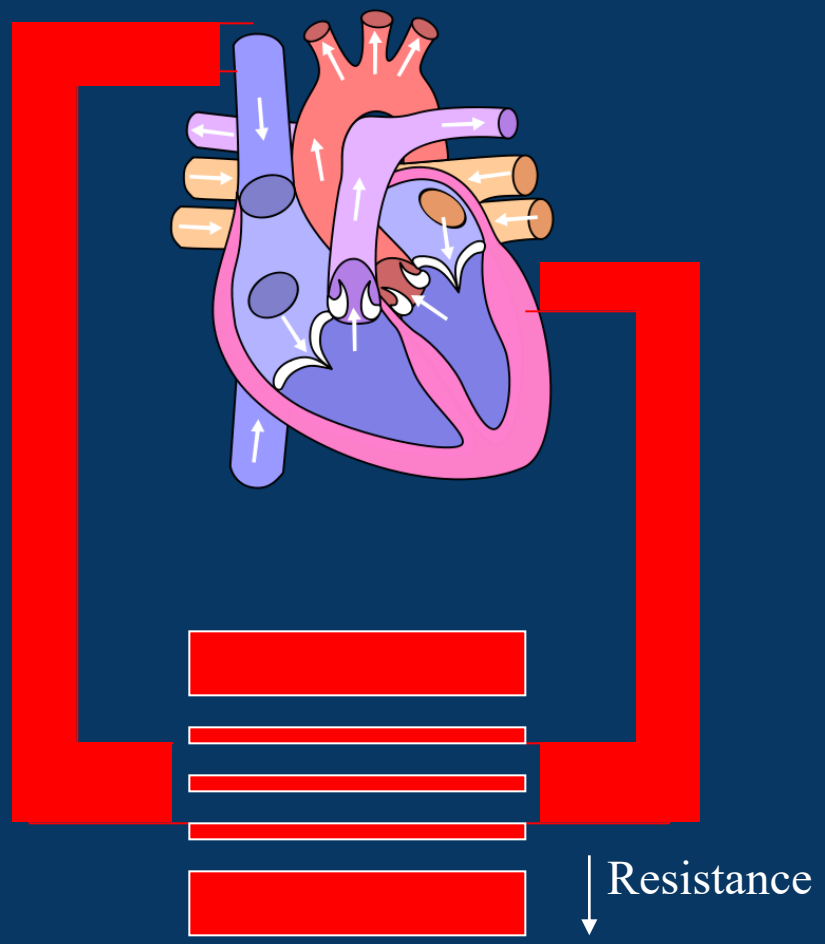
Etiologies of Shock(Distributive)

↓ 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)

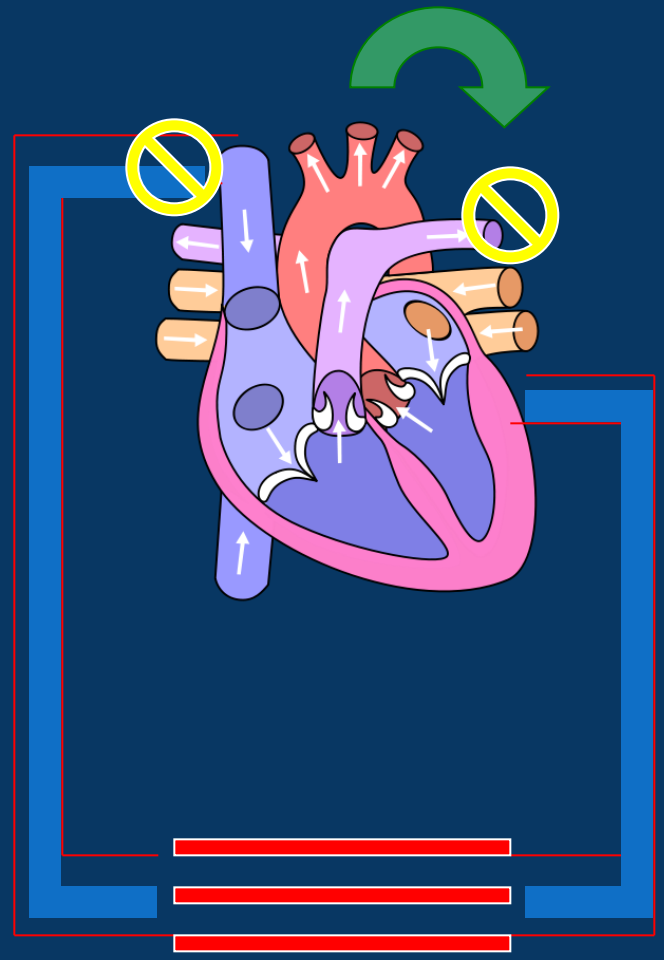
MAP – CVP = (SV x HR) x SVR

- Low Stroke Volume:

Venous return & Outflow obstruction

"Obstructive"

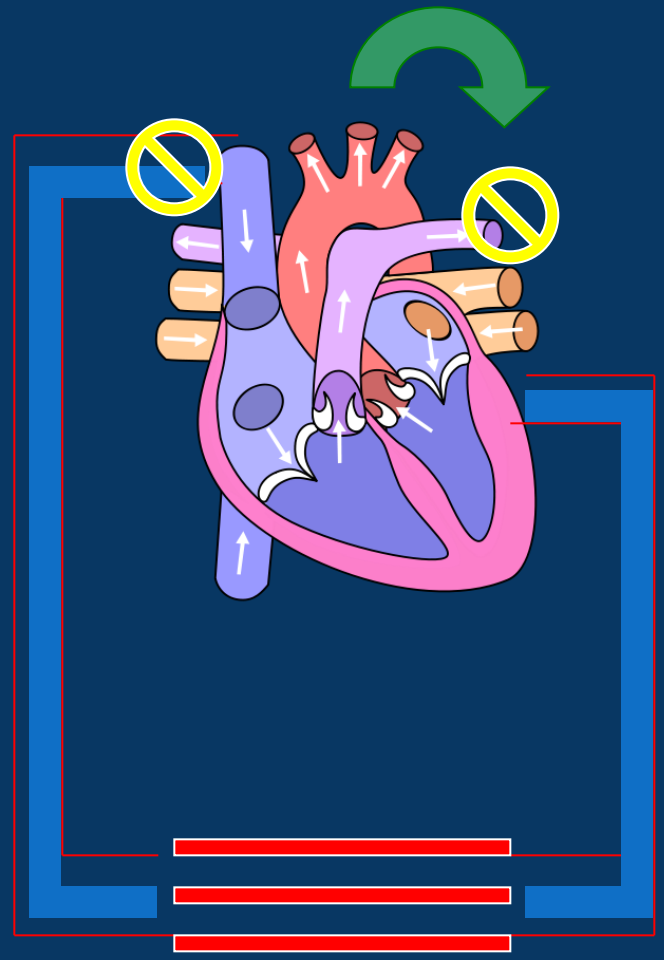
Tamponade, tension pneumothorax, PEEP, Pulmonary embolism



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**

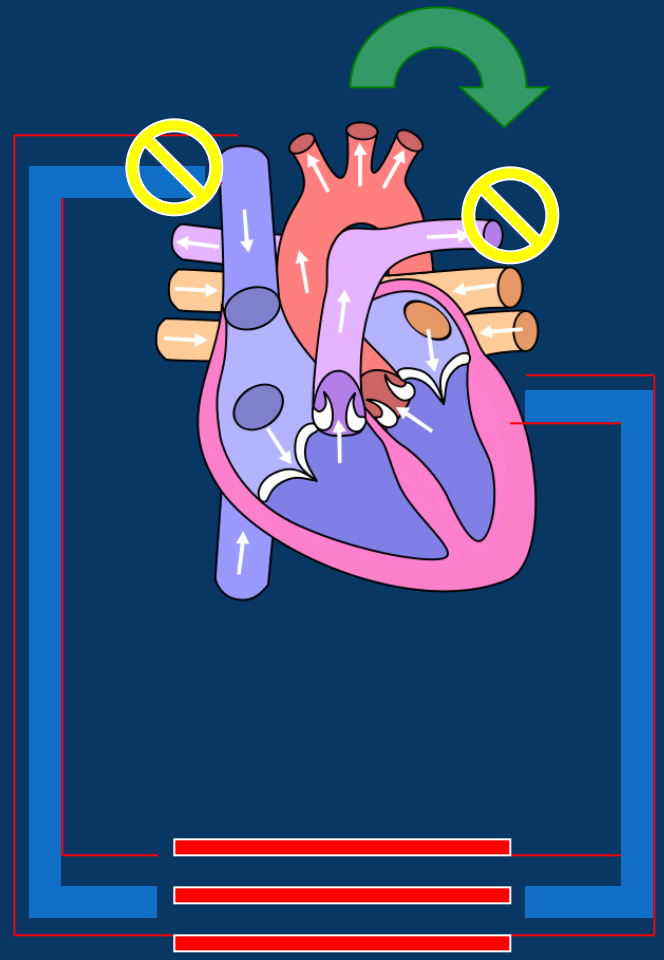
  Myocardial infarct, valvular defect

- Abnormal heart rate:

  "Cardiogenic"

  Tachycardia (short filling time)

  Bradycardia



- 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

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

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
- 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

- 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

- 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

- 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

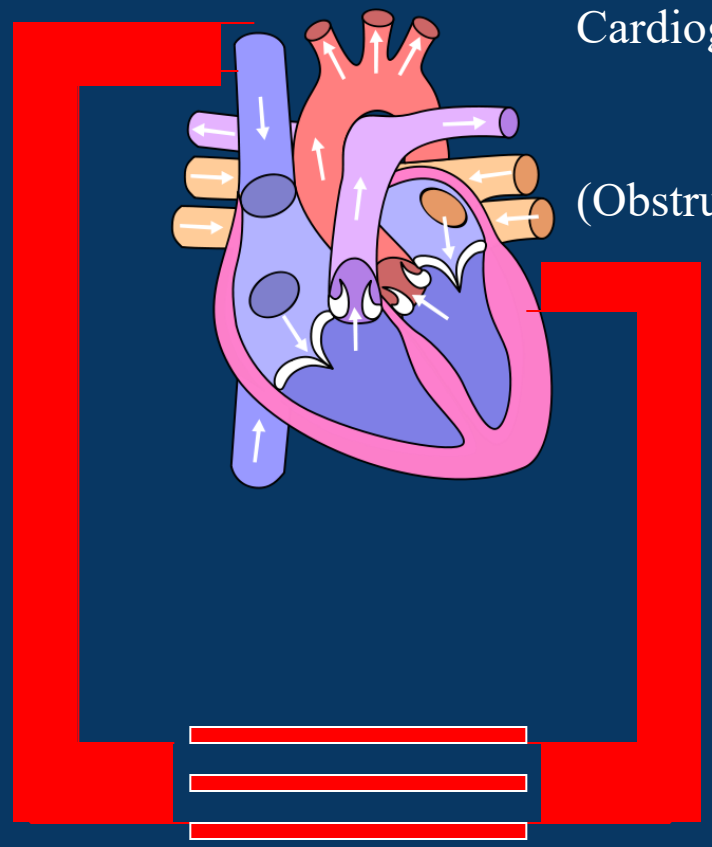
- IL-6, also produced predominantly by the macrophage, is the best predictor of prolonged recovery and development of multiple organ failure after shock

- 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

Classification of Shock

- Hypovolemic
- Cardiogenic
- Distributive (vasodilatory)
- Obstructive

Types of Shock



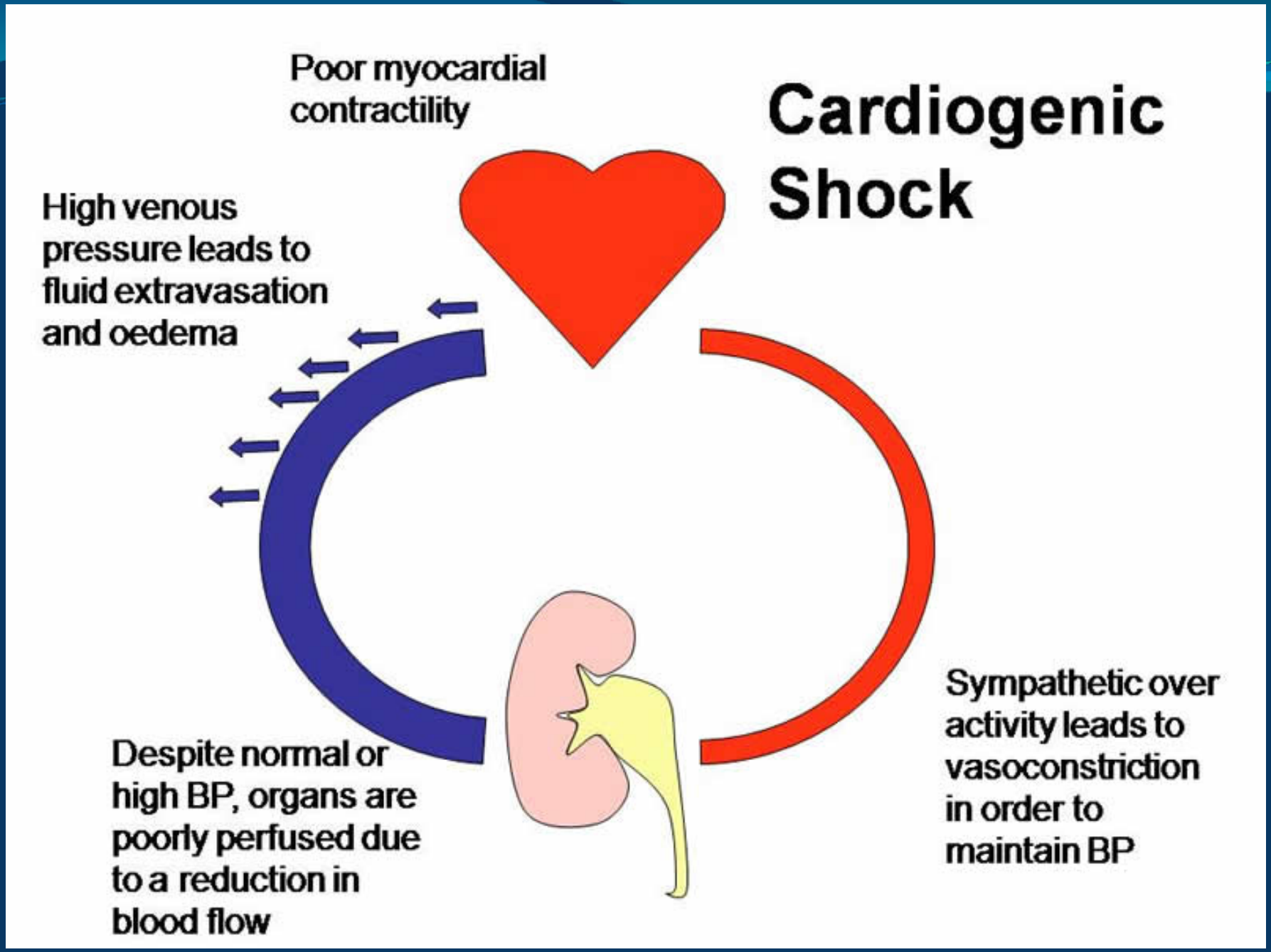
Cardiogenic

(Obstructive)

Hypovolemic

Distributive

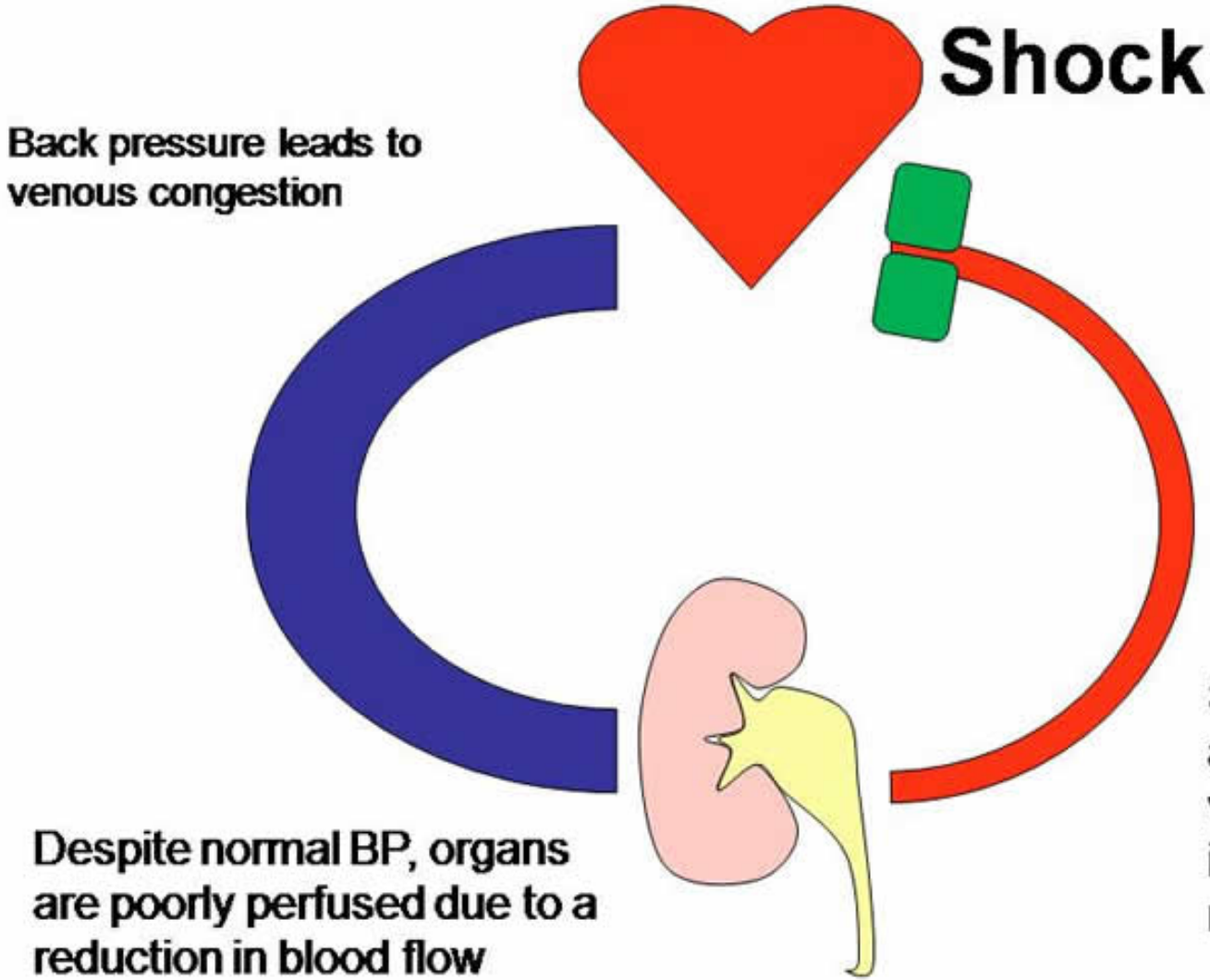
Cardiogenic Shock



Cardiogenic

- Myocardial infarction
- Myocardial contusion
- Myocarditis
- Acute valvular failure
- Arrhythmia
- Acute ventricular septal wall defect

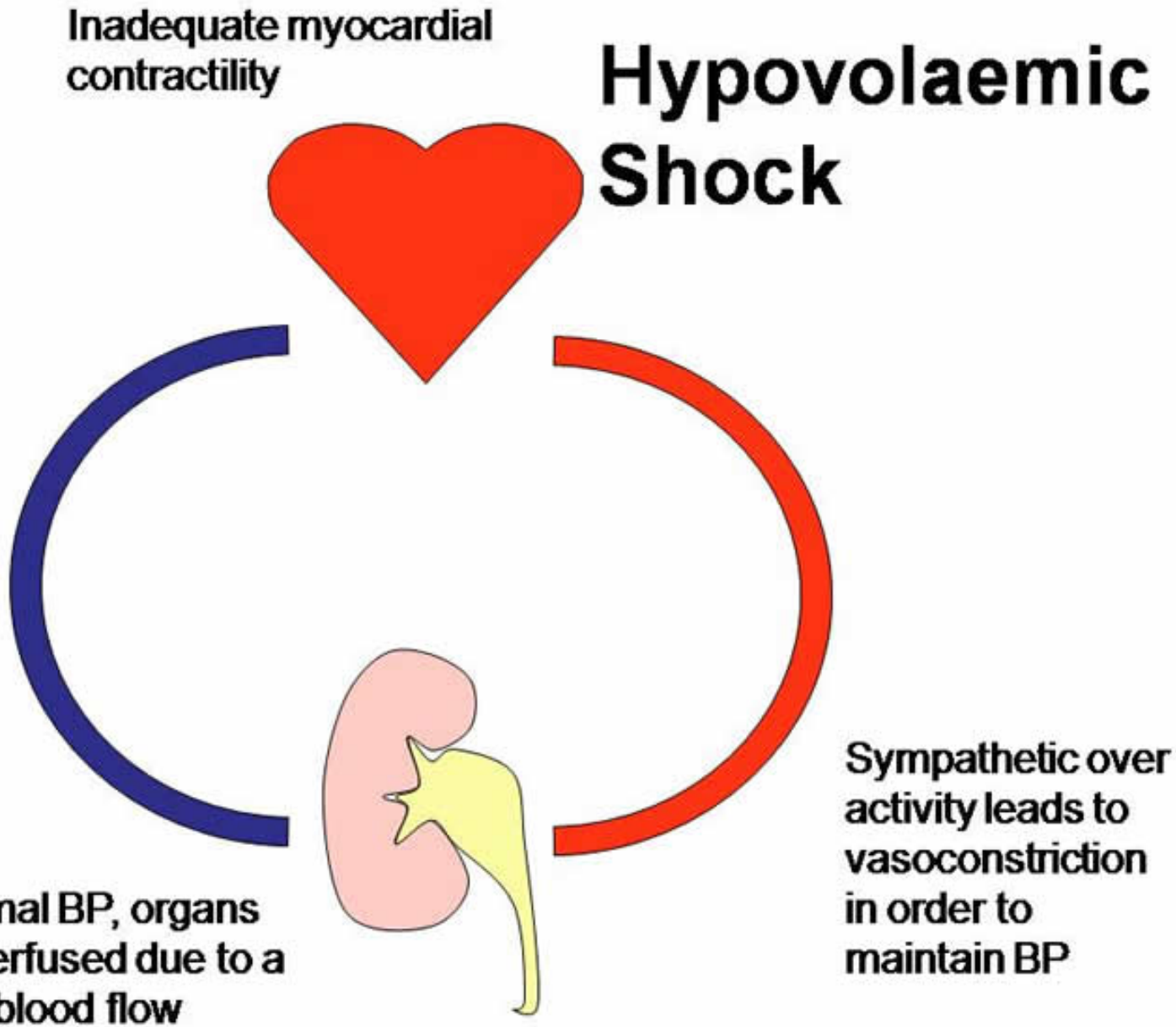
Obstructive Shock



Obstructive

- Pulmonary embolus
- Cardiac tamponade
- Tension pneumothorax

Hypovolaemic Shock

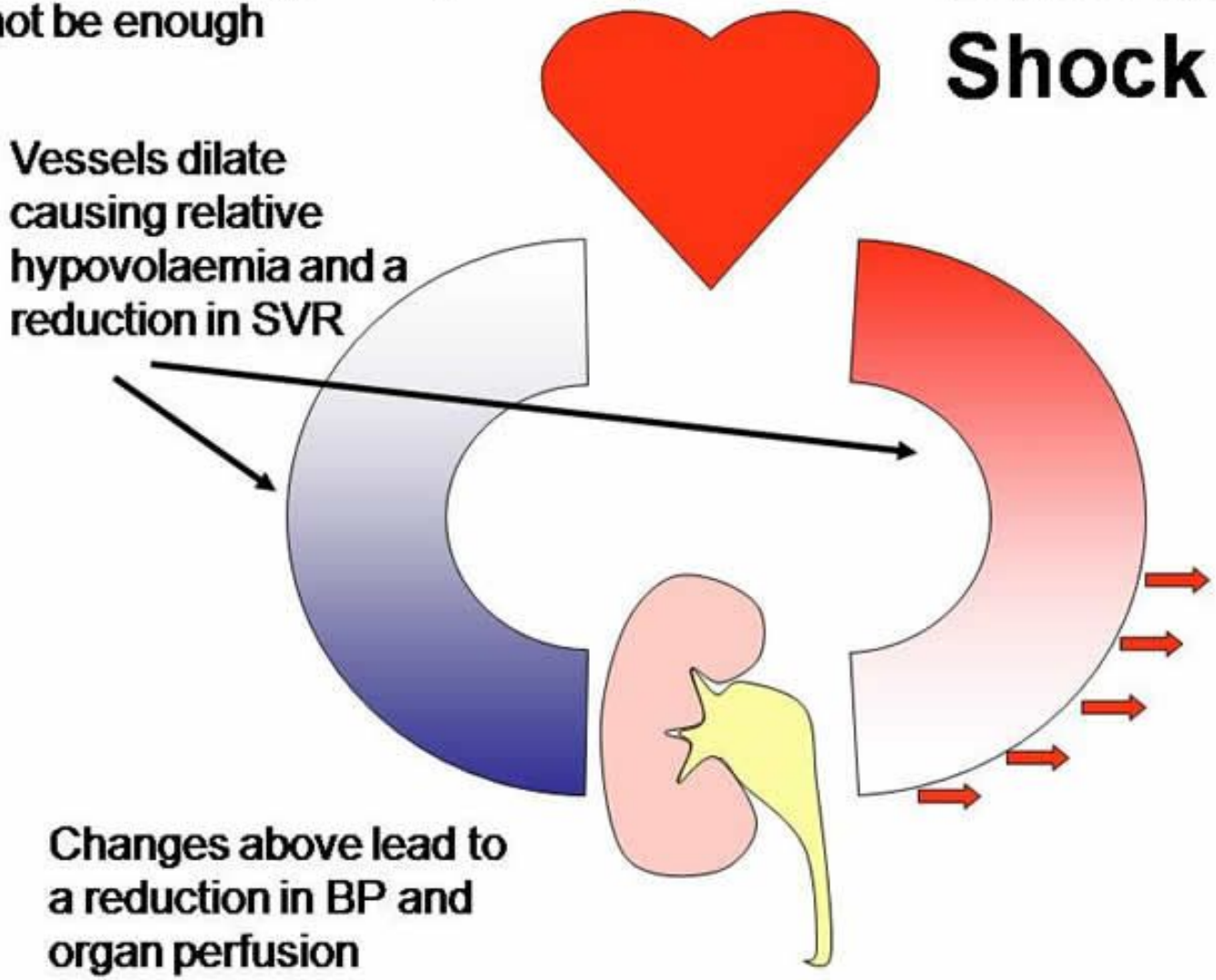


Hypovolaemic

- Fluid depletion
  - Vomiting and diarrhoea
  - Burns
  - Polyuria
- 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



Vessels dilate causing relative hypovolaemia and a reduction in SVR

Capillary leak worsens hypovolaemia and causes oedema (including pulmonary)

Changes above lead to a reduction in BP and organ perfusion

Distributive

- Sepsis
- Neurogenic
- Anaphylaxis

Key Issues

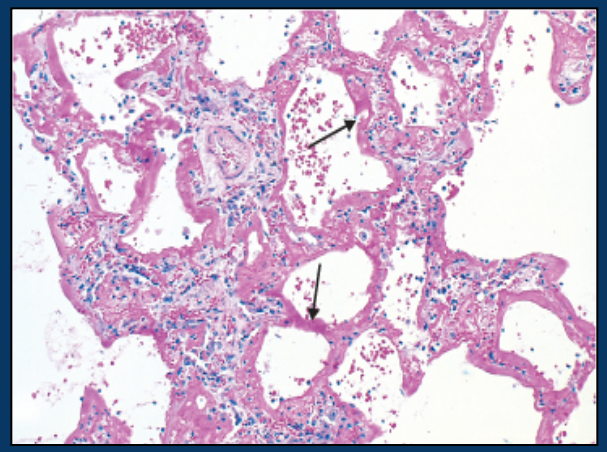
Recognize & **Treat** during compensatory shock phase

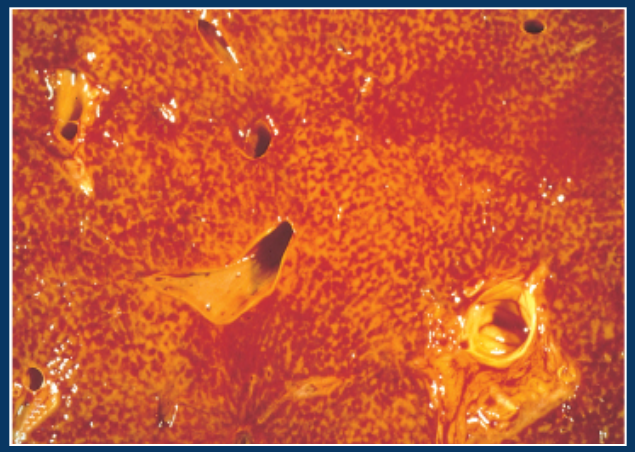
Mortality
increase 2-fold for every hour
in treatment delay.

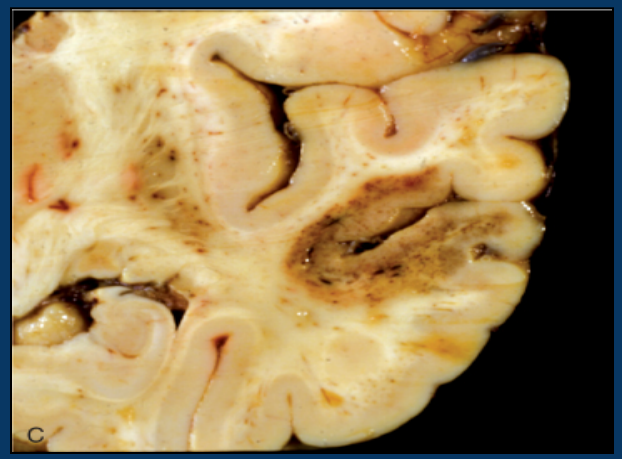
Han, Carcillo. Pediatrics 2003;112:793-799

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



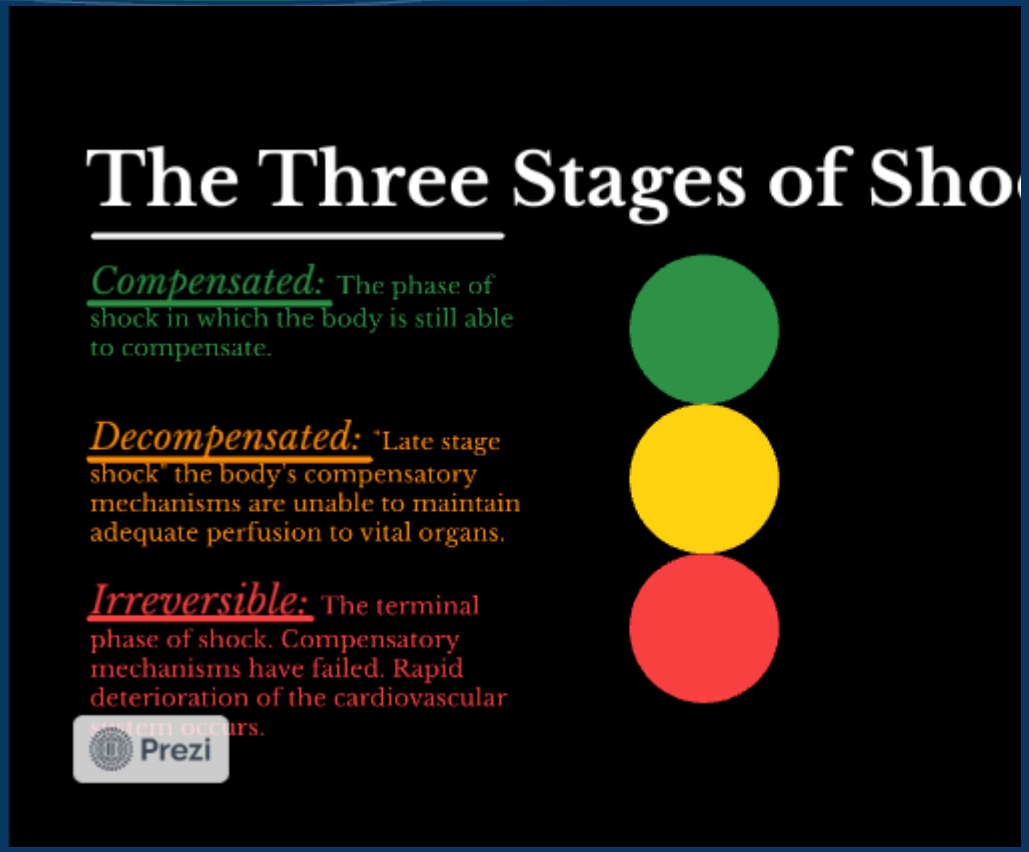




Robbins & Cotran Pathologic Basis of Disease: 2005

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



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

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

Hemodynamics in Shock

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	↑	↑	↓	↑	↓

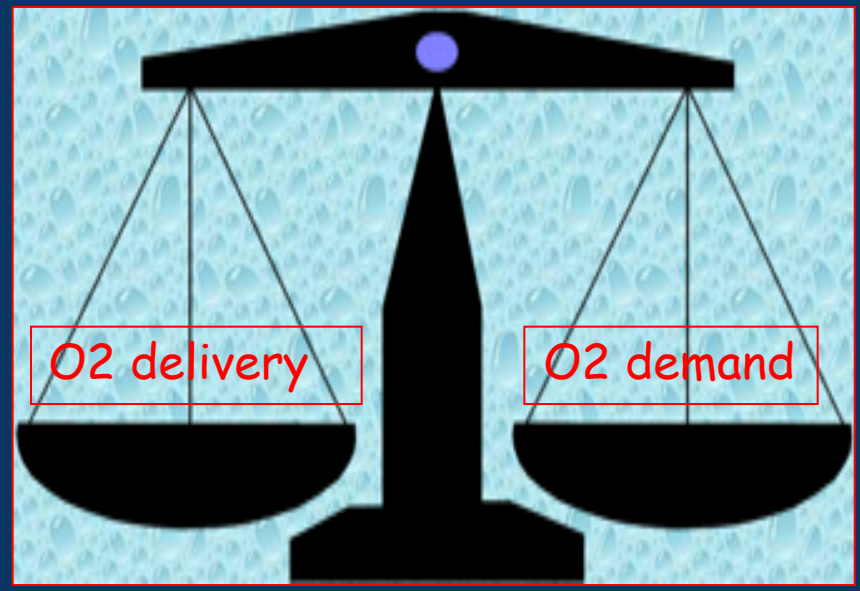
Shock states coexist

Changing hemodynamics

Individualize treatment

Treatment principles

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



ABCDE

Resuscitation Priorities

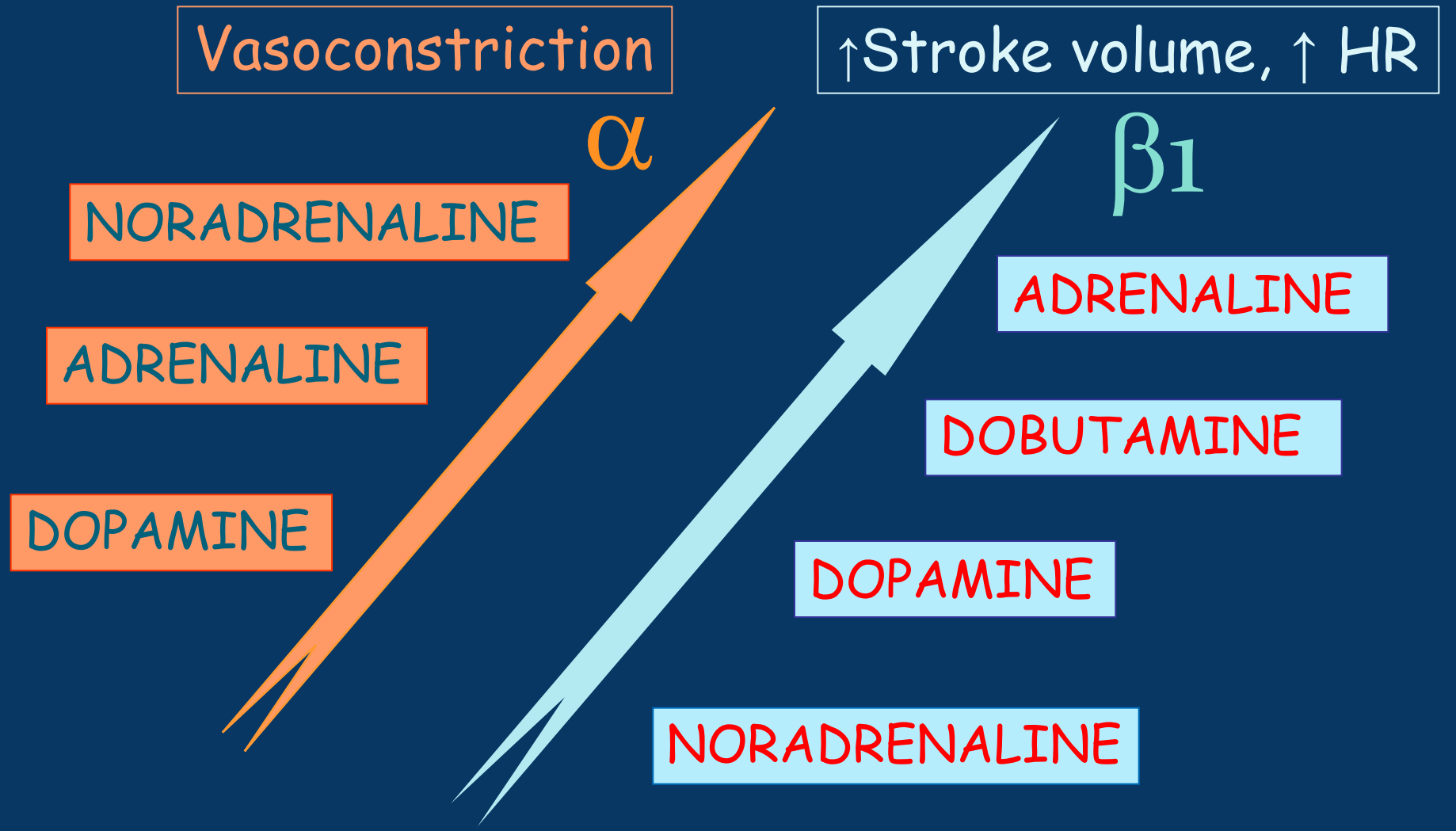
Increase O2 delivery

- 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

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
+++	++	+++

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

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