MUNI
MED

MUNI
MED

Shock

Definition of shock

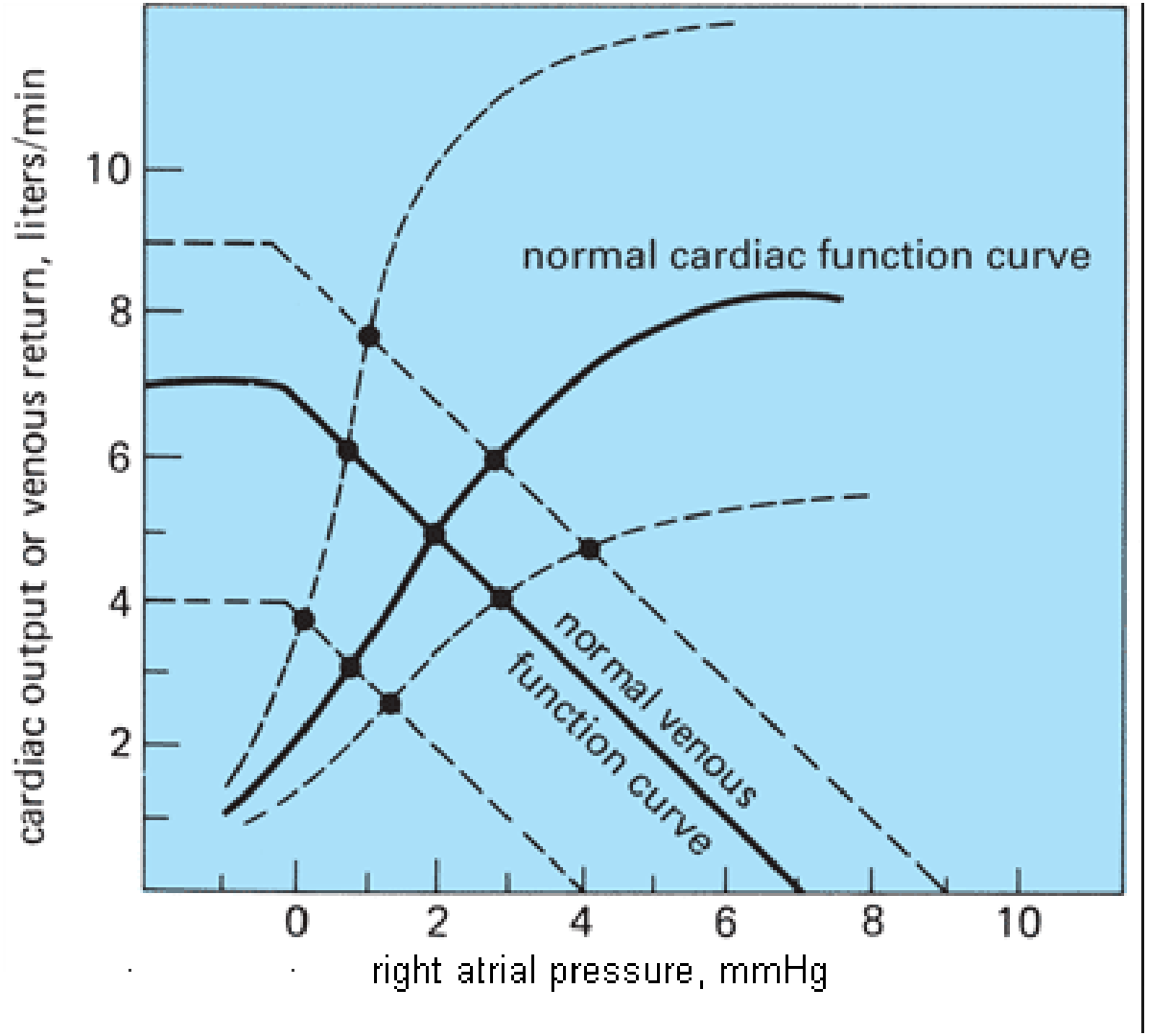
- Severe tissue hypoperfusion resulting in low supply of oxygen to the organs
- Systemic hypotension (of various causes) is present
- $P = Q \times R$
- $Q \sim CO = SV \times f$
- CO depends on

  a) cardiac function

  b) venous return (→preload)
- R – systemic resistance (mostly arterioles) - afterload

MUNI
MED

Cardiac and venous function



MUNI MED

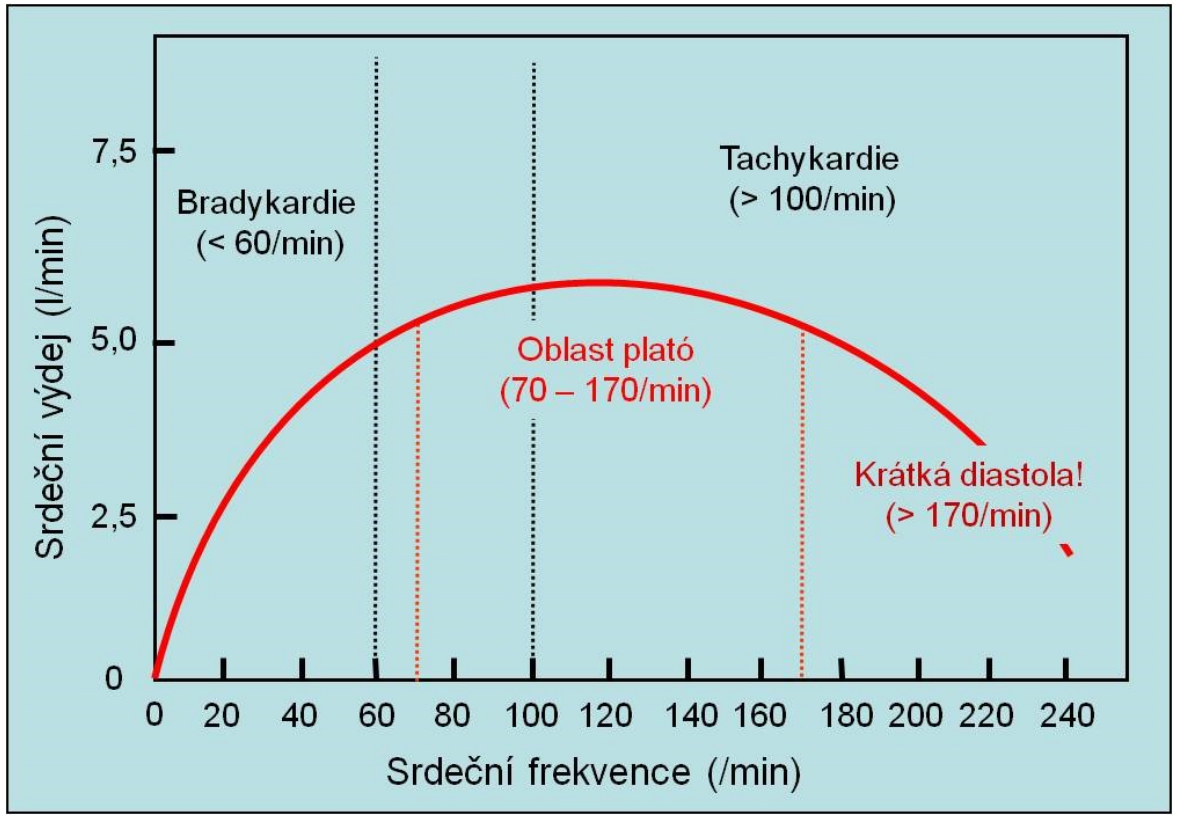
Phases of shock

- Compensation of initiating cause
- Decompensation
- Refractory shock

MUNI
MED

Compensatory mechanisms and their limits

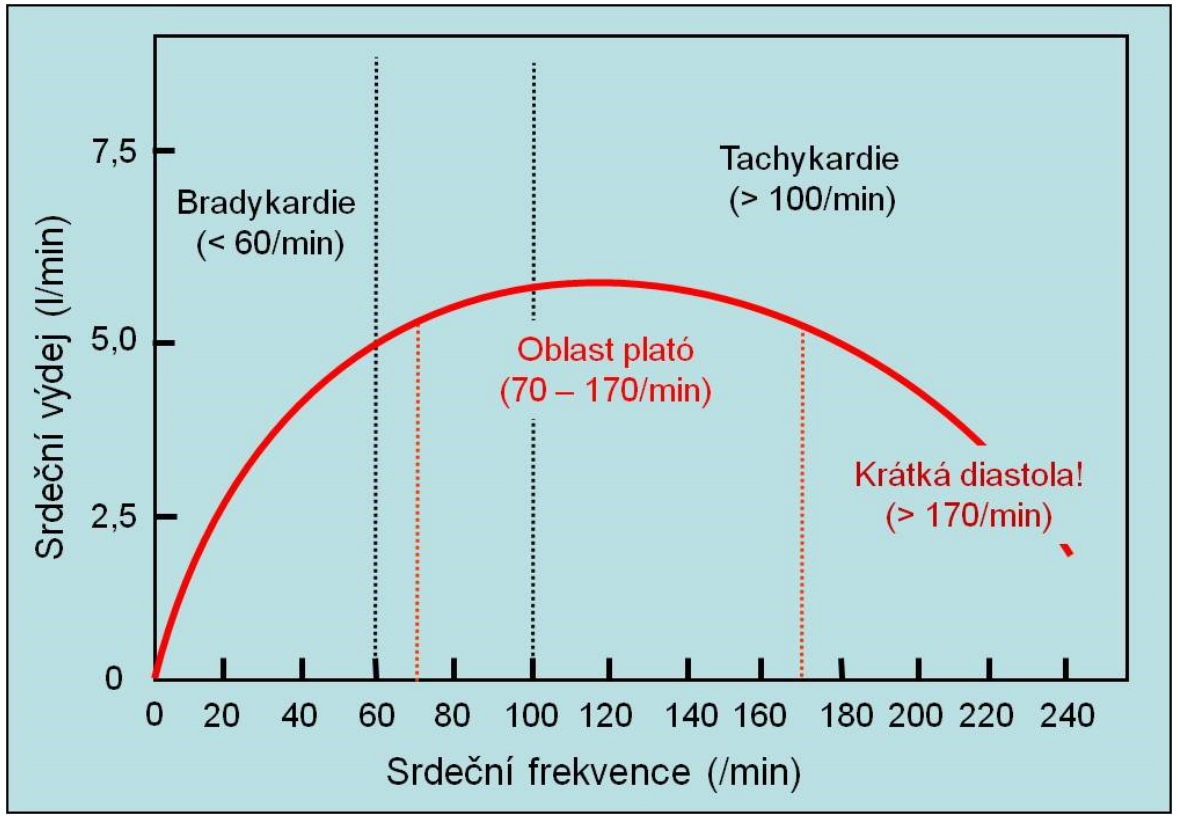
- Activation of sympathetic nervous system
  (tens of seconds)
- Activation of RAAS (cca 1 hour)
- Vasoconstriction (if possible)
- Vasodilatation in some tissues
  (esp. myocardium)
- Positively inotropic effect of SNS (if possible)
  - but at cost of higher metabolic requirements of the heart



MUNI MED

Compensatory mechanisms and their limits

- Increased heart rate
  - but CO decreases in high HR (>150 bpm)
- Keeping circulating volume by lower diuresis
  - but at cost of acute renal failure
- Shift to anaerobic metabolism
  - but at cost of ↓ ATP a ↑ lactate (acidosis)
- Shift of saturation curve of hemoglobin to right (↑2,3-DPG)
- Hyperglycemia
  - but decreased utilization of Glc in the periphery



MUNI MED

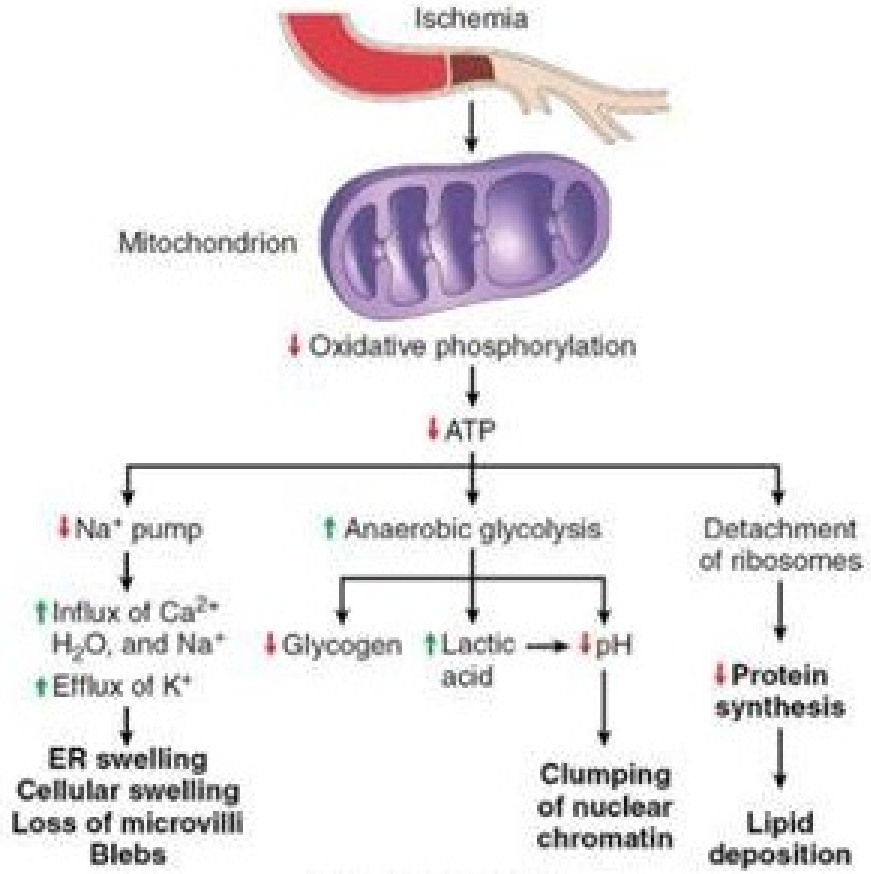
Decompensated shock

- ↓ BP
- ↓ diuresis
- Brain hypoperfusion – involvment of mental functions
- Acrocyanosis (in peripheral hypoperfusion)
- Tachypnoe
- Treatment – colloid solutions, catecholamines

MUNI
MED

Shock at the cellular level

- Mitochondrial dysfunction (result of hypoxia)
  - lower production of ATP
- ↑ ROS production by dysfunctional mitochondria
- Failure of ion pumps
  (e.g. Na/K ATP-ase →↑intracelular Ca^{2+})
- Lysosomal abnormalities
  - release of lysosomal proteases
- ↓ intracelular pH



MUNI MED

Refractory shock – Vicious circles

1) Vasodilatation ↔ hypoperfusion

- Endothelial cells contain two isoforms of nitric oxid synthase – constitutive (eNOS) and inducible (iNOS)
- In lasting hypoxia of endothelial cells there is increased iNOS activity (primarily physiological mechanism)
- ↑NO increases vasodilation and hypoperfusion

2) Myocardial hypoxia ↔ lower contractility

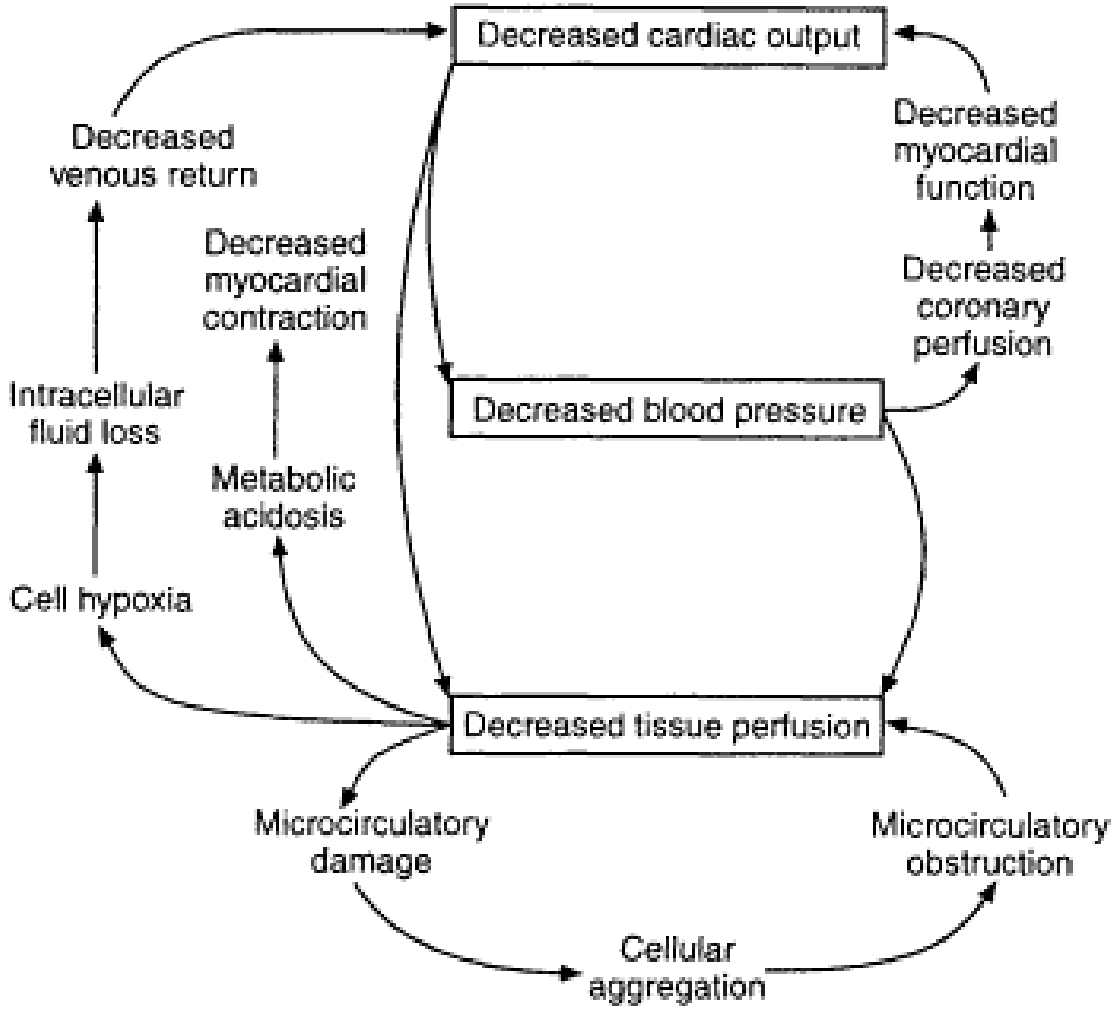
- Lower myocardial perfusion leads into ↓CO, which further reduces coronary flow
- Myocardium does not benefit from the shift of Hb saturation curve
  - efficiency of O_2 extraction is already at its maximum

3) Brain hypoperfusion ↔ ↓SNS activity

- Lower perfusion of vasomotor centre leads first into SNS hyperactivity, which is then followed by its supression
- That leads into ↓brain perfusion

MUNI MED

Other vicious circles in refractory shock



MUNI MED

Forms of shock

a) Hypovolemic shock (i.e. absolute fluid loss)
   - low preload

b) Distributive („warm“) shock
   - low resistance, afterload, CO might be increased

c) Cardiogennic shock
   - normovolemia, normodistribution, low CO in bad cardiac function

d) Obstructive shock
   - low preload of one ventricle in normovolemia and subsequent lowering of CO
   - pathophysiology similar to cardiogennic shock

MUNI
MED

Cardiac and venous function in shock

Hypovolemic shock

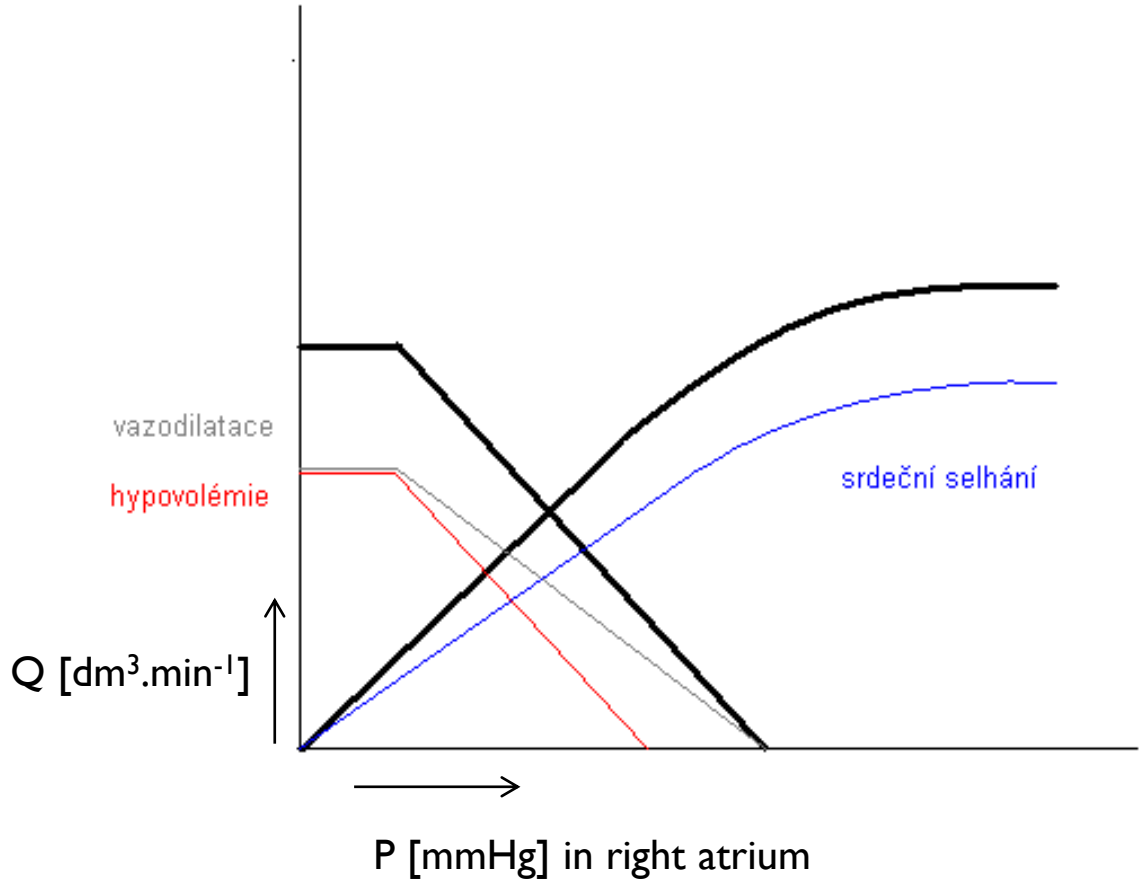
- compensation by the vasoconstriction and cardiac mechanisms

Distributive shock

- compensation by cardiac mechanisms (vasoconstriction is usually impossible)

Cardiogennic (and obstructive) shock

- compensation by vasoconstriction



MUNI MED

Hypovolemic shock - causes

- Acute bleeding
- Burns, trauma
- Rapid development of ascites
- Acute pancreatitis
- Severe dehydratation
  - Vomiting, diarrhoea
  - Excessive diuresis (e.g. in diabetes insipidus)

MUNI
MED

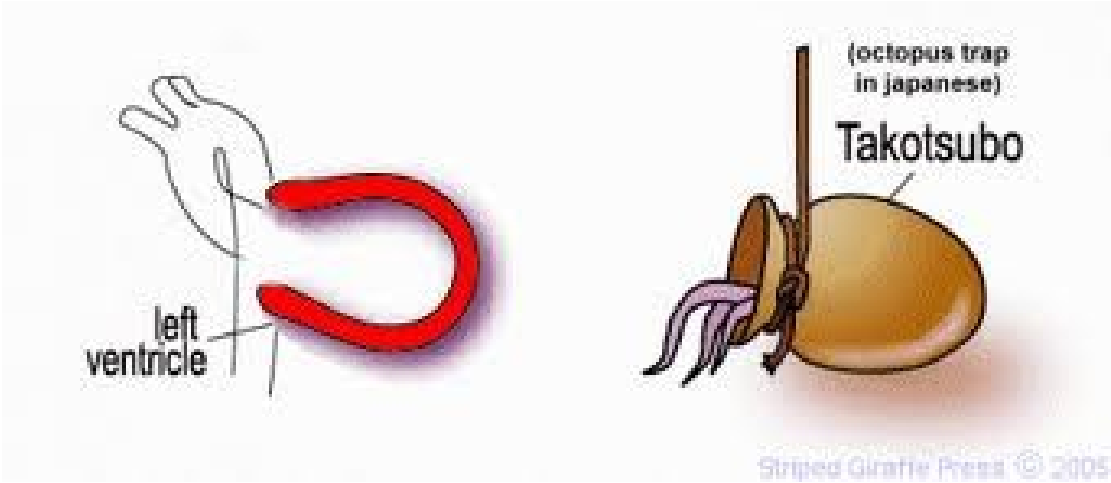
Distributive shock - causes

- Anafylactic shock
- Anafylactoid shock
  - Mediators of mast cells, but without IgE
  - E.g. snake venoms, radiocontrasts
- Septic shock
  - Role of bacterial lipopolysaccharides
  - Bacterial toxins
  - IL-1, TNF-α – stimulate synthesis of PGE_2 and NO
- Neurogennic shock
  - Vasodilatation as a result of vasomotoric centre (or its efferent pahways) impairment

MUNI MED

Cardiogennic shock - causes

- Myocardial infarction
- Arrhythmias
- Valvular disease (e.g. rupture of papillary muscles)
- Decompensation of heart failure in dilated/restrictive cardiomyopathy, amyloidosis
- Overload by catecholamines (“tako-tsubo cardiomyopathy“ – apical akinesia + basal hyperkinesia)
- Rupture of ventricular septum
- Obstructive shock
  - e.g. cardiac tamponade, massive pulmonary embolism, aortic dissection



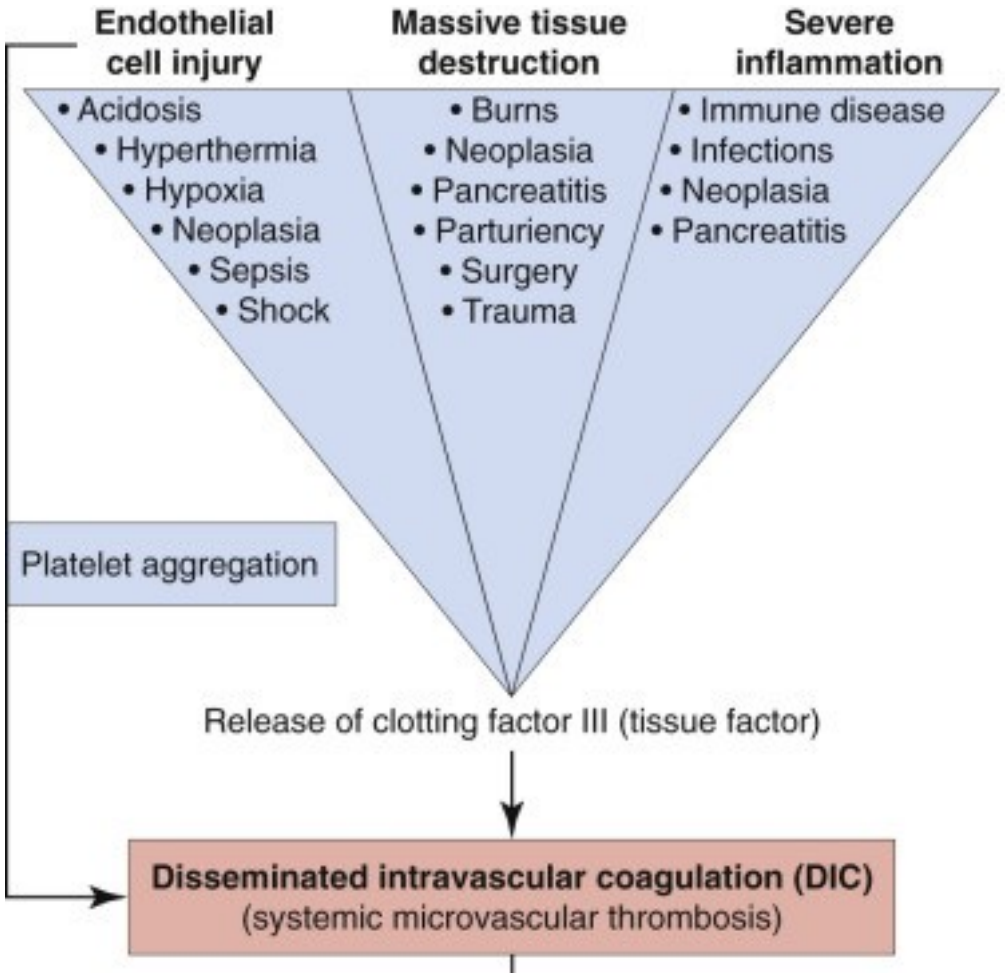
Organ complications in shock

- Lungs
  - ARDS
- Liver
  - necrosis of hepatocytes
- GIT
  - stress ulcer
  - Damage of intestinal mucosa by ischemic necrosis → sepsis
- Kidneys
  - Acute renal failure in vasoconstriction of a. afferens
  - Acute tubular necrosis during ischemia

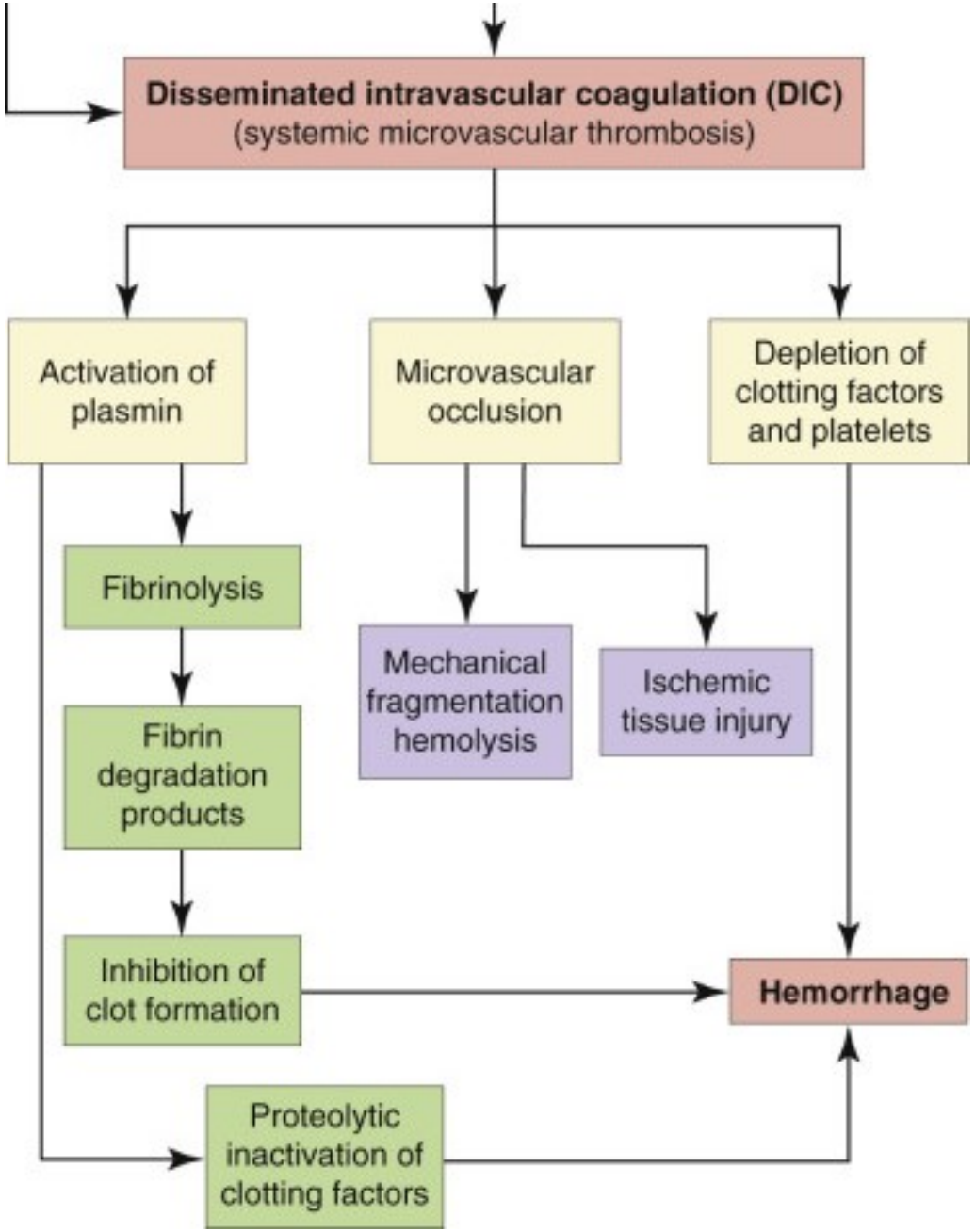
MUNI
MED

Disseminated intravascular coagulopathy (DIC)

- Systemic exposure to tissue factor
- Consequence of the vessel wall damage
- Moreover, slower blood flow contributes to the extent of coagulation reactions
- Two phases:
  1) Formation of microtrombi (with local ischemia)
  2) Bleeding as a result of consummation of coagulation factors
- DIC is especially frequent in septic shock



https://www.sciencedirect.com/topics/veterinary-science-and-veterinary-medicine/disseminated-intravascular-coagulation



https://www.sciencedirect.com/topics/veterinary-science-and-veterinary-medicine/disseminated-intravascular-coagulation

MUNI MED

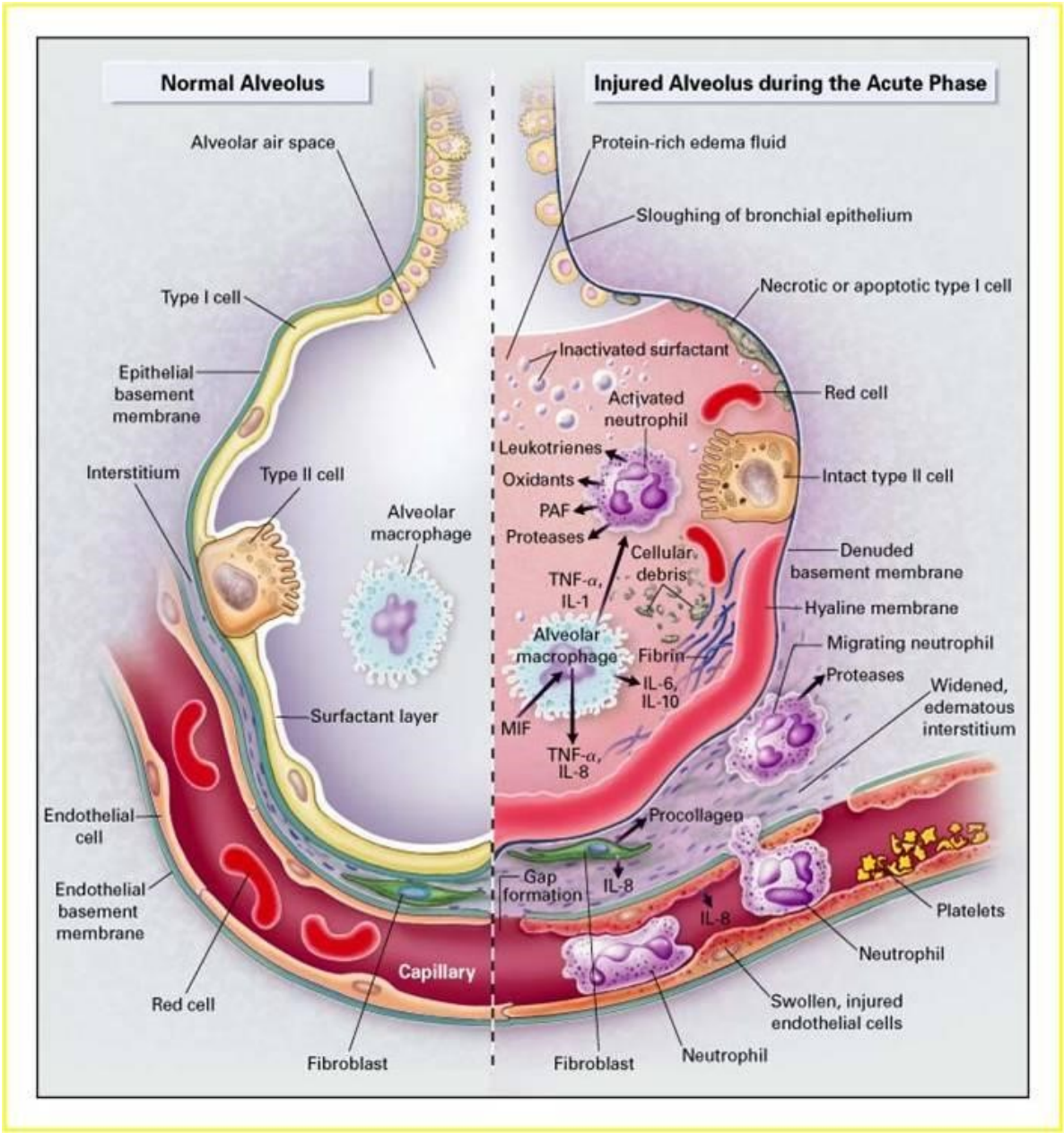
Systemic Inflammatory Response Syndrome(SIRS)

- Systemic activation of immune mechanisms
- Causes:
  - infections (sepsis)
  - Shock caused by non-infectious causes (diffuse tissue damage in hypoxia)
  - Non-compatible blood transfusions
  - Radiation syndrome (esp. GIT form)

MUNI
MED

Acute Respiratory Distress Syndrome (ARDS – „shock lung“)

- Result of lung inflammation in SIRS, pulmonary infections, aspiration of gastric juice, drowning
- Exsudative phase (hours): cytokine release, leukocyte infiltration, pulmonary edema, destruction of type I pneumocytes
- Proliferative phase: fibrosis, ↑ dead space, proliferation of type II pneumocytes
- Reparative phase: ↓ inflammation, ↓ edema, continuing fibrosis, in most cases permanent restrictive diseases

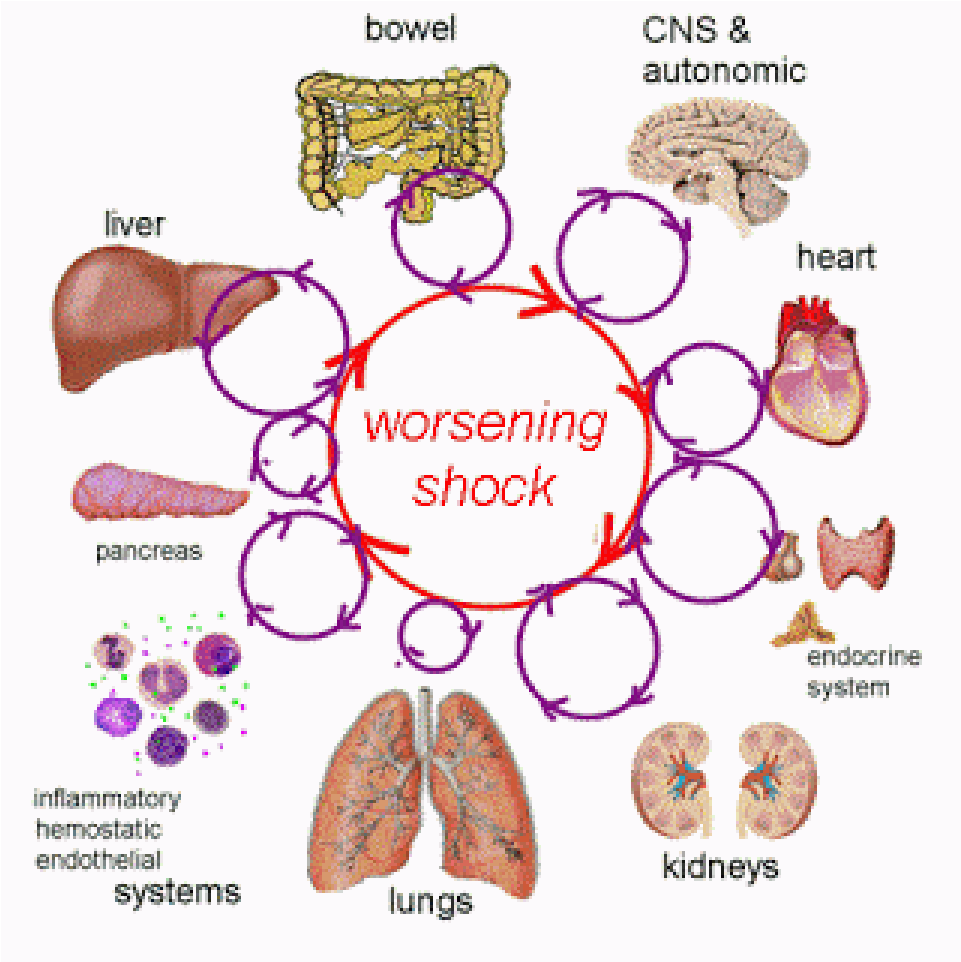


Multiorgan dysfunction syndrome (MODS)

- Failure of more organs at once

  (lungs, liver, GIT, kidneys, brain, heart)
- It can develop after initial insult

  (days or weeks)
- Hypermetabolism, catabolic stress
- Can both preceed or result from SIRS



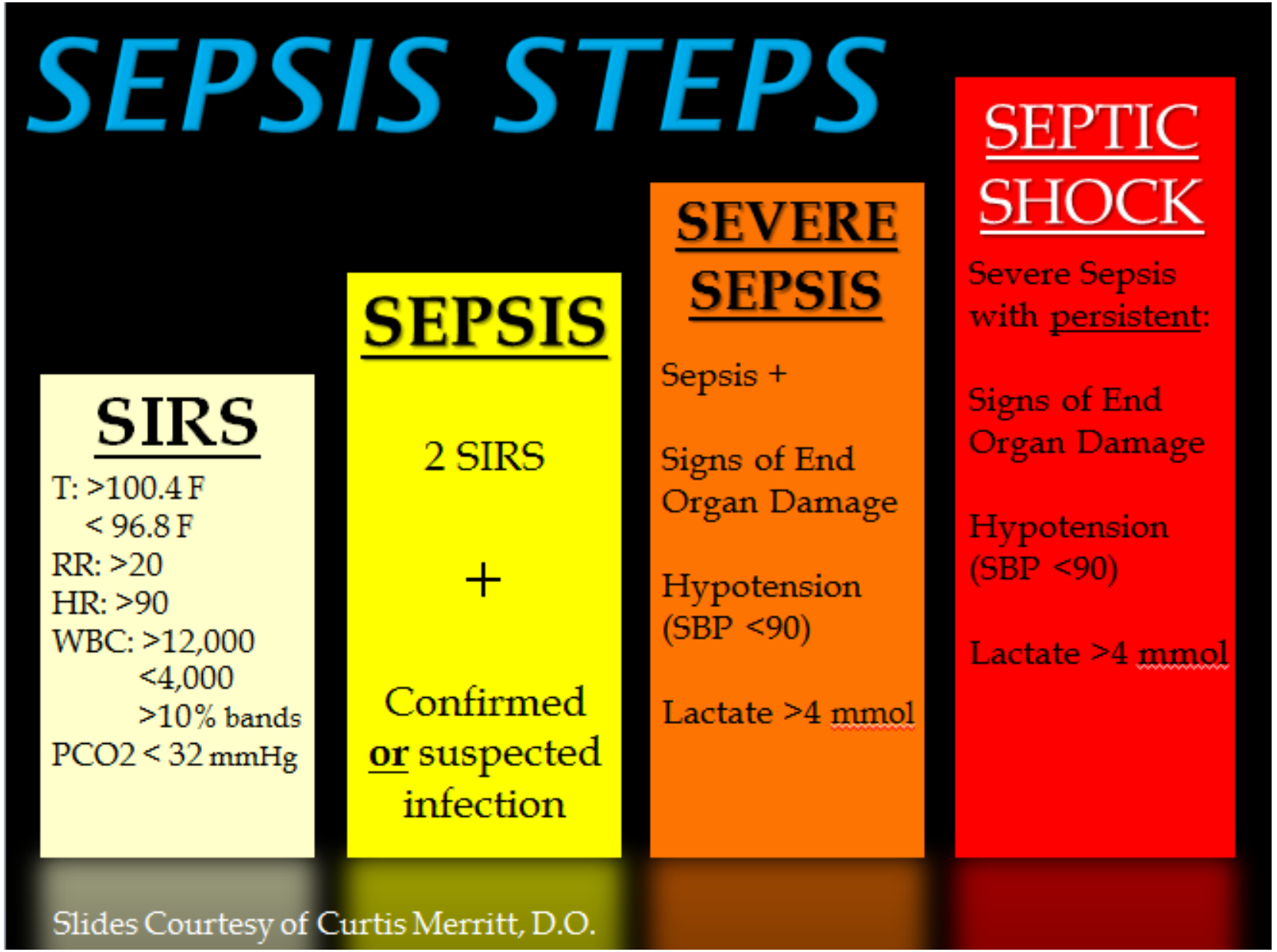
MUNI
MED

General principles of treatment

- Treatment of underlying cause
- Positively inotropic drugs, vasopressors

  (e.g. catecholamines – but: they can worsen the situation in obstructive shock)
- Colloid solutions, crystaloid solutions (but: there is a risk of edema in cardiogennic shock)
- O_2
- i.v. corticoids (anafylaxis, SIRS?)
- ATB (septic shock)
- Mechanic circulation support (cardiogennic shock)
- Anti-shock position

MUNI
MED



https://upload.wikimedia.org/wikipedia/commons/a/ad/Sepsis_Steps.png

MUNI MED

MUNI
MED