Pathophysiology of shock

Oxygen delivery (DO_2) vs. oxygen consumption (VO_2)

$VO_2 = \varepsilon \cdot DO_2$

VO_2 → [mol/min]

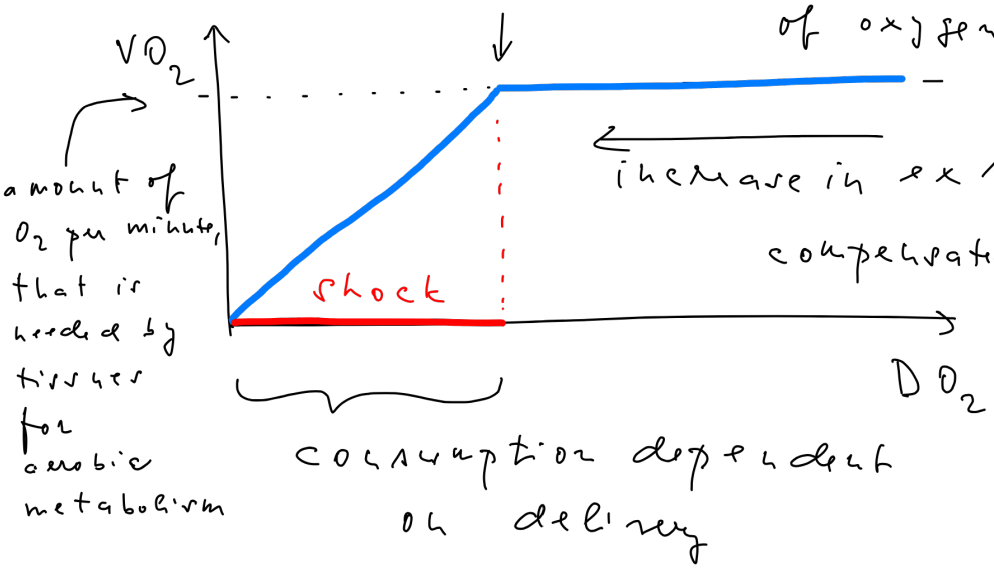
ε ↑ extraction of oxygen

$DO_2 = k \cdot CO \cdot [Hb] \cdot SaO_2$

CO ↑ cardiac output

[Hb] ↑ concentration of hemoglobin

SaO_2 ↑ arterial saturation of O_2

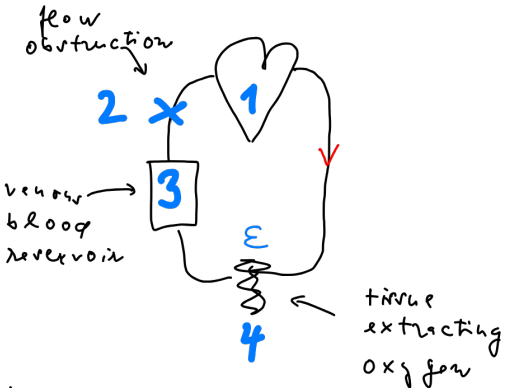


Shock = $\varepsilon \cdot DO_2 \leq VO_2$ aer from circulatory reserve

Shock is a condition of sick a low DO_2 (and extraction of O_2), that is not sufficient for aerobic metabolism of tissues

⇒ anaerobic metabolism → ↑ rate of lactate

causes of shock



1. cardiogenic shock (↓ CO)

- acute myocardial infarction
  acute valvular disease (IE, rupture of papillary muscle)
  severe tachycardia or bradycardia
  myocarditis
  acute cardiomyopathy (tako-tsubo, post-tachycard)
  ⋮

2. obstructive shock (↓ CO)

- pericardial tamponade
- pulmon. embolism
- tension pneumothorax

3. hypovolemic shock (↓ CO)

- bleeding
- dehydration (preying (zvracení, průjmy, pocení, diuretika ...)
- fluid redistribution extravascular (relative hypovolemia)
  - pancreatitis
  - ascites
  - sepsis
- low tonus of veins

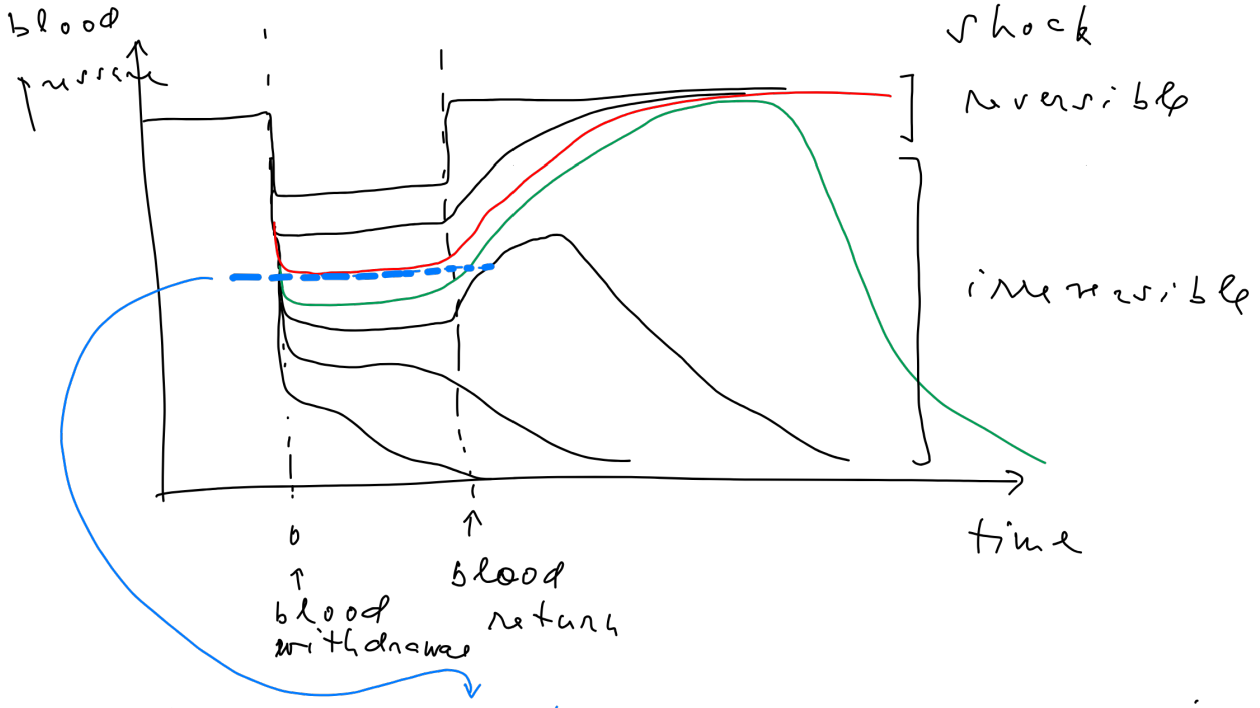
4. distributive shock (↑ CO, ↓ ε) !!

warm shock

- sepsis
- major trauma
- burns
- large surgery
- status after CPR
- severe pancreatitis
- anaphylaxis

Reversible vs. irreversible shock

- Guyton ~ 1960
  - simulation of shock through removal various amount of blood in dogs



- there is a sharp boundary between reversibility and irreversibility of shock
- cause of irreversibility? - e.g. critical loss of phosphates (ATP, ADP..) from cell

ACS (American college of surgeons) classification of bleeding according to blood loss

I. blood loss < 15% (~ < 750 ml)
- no symptoms, physiological compensation
- blood location

II. 15 - 30% (750 - 1500 ml)

- still normal BP, compensatory tachycardia (~ BP, ↑ HR) } = compensated shock
- but ↓ CO → ↑ lactate

III. 30 - 40% (1500 - 2000 ml)

- ↓ BP, ↑ HR, ↓ CO, ↑ lactate

= decompensated shock

IV. > 40% (> 2 - 2.5 l)

- irreversible shock

Symptoms of shock

- ↓ BP
- ↑ HR
- ↓ stroke volume → ↓ pressure amplitude
  ↓
  weak (thready) pulse
- vasoconstriction
  → pale cold fingers and lips
  - peripheral cyanosis of fingers and lips
  - slow capillary return (> 2 - 3 sec)
- anuria
- somnolence or anxiety
- dyspnea, tachypnea
- syncope

compensatory mechanisms of shock

$$CO = TO \cdot TF$$

preload afterload contractility

- sympath. activation (seconds - minutes)
  - ↪ pale sweaty hands
  - ↑ Pr, ↑ Af, ↑ CO, ↑ HR
  - vasoconstriction everywhere except for brain and ♡
    - ↪ redistribution to vital organs
- activation of RAA (hours)
  - fluid retention → ↑ Pr
- secretion of vasopressin (= ADH)
  - vasoconstriction → ↑ Af
- fluids transfer extra → intravascular
  - ↓ capillary pressure → filtration into capillaries
- secretion of corticosteroids
  - permissive effect on catecholamines
  - hyperglycemia
  - stress metabolism → proteolysis
- ↑ oxygen extraction (↑ E)

Pathogenesis of hemorrhagic shock

bleeding
↓
hypovolemia
(+ anemia)
↓
↓ CO → tissue ischemia → ↑ lactate → acidosis
↓
↓ BP
↓ coronary perfusion
↓ renal kidney failure → oligoanuria
↓ sympath. activity
altered consciousness (coma)

diffuse damage of tissues
↓
systemic inflamatory response (SIRS)
↓
multiorgan failure (MODS)

- ARDS
- capillary leak syndrom
- DIC (↓ Trc)
  ↳ dysfunction of microcirculation
- intrarenal kidney failure
- encephalopathy
- GIT dysfunction
- vasoplegia

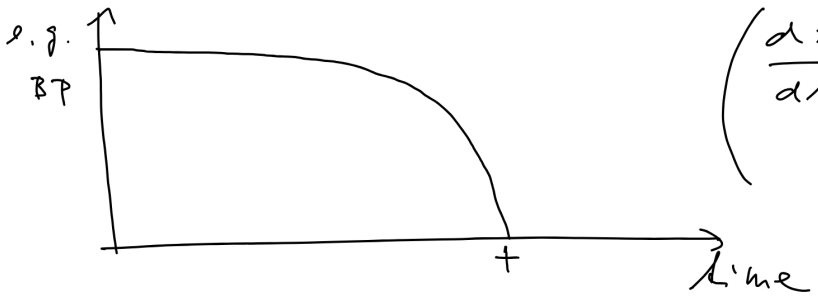
→ = positive feedback

- develop more and more with progression of shock and further accelerate its progression

→ degree of shock accelerator

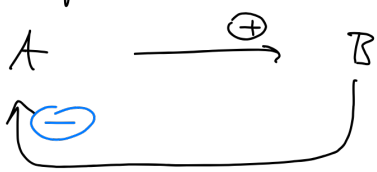
$\left(\frac{dx}{dt} = k \cdot x \right)$

$\hookrightarrow x = e^{kt}$



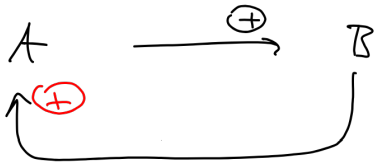
- terminal phase of each shock is cardiogen shock

- negative feedback stabilizer



→ physiology

- positive feedback destabilizer

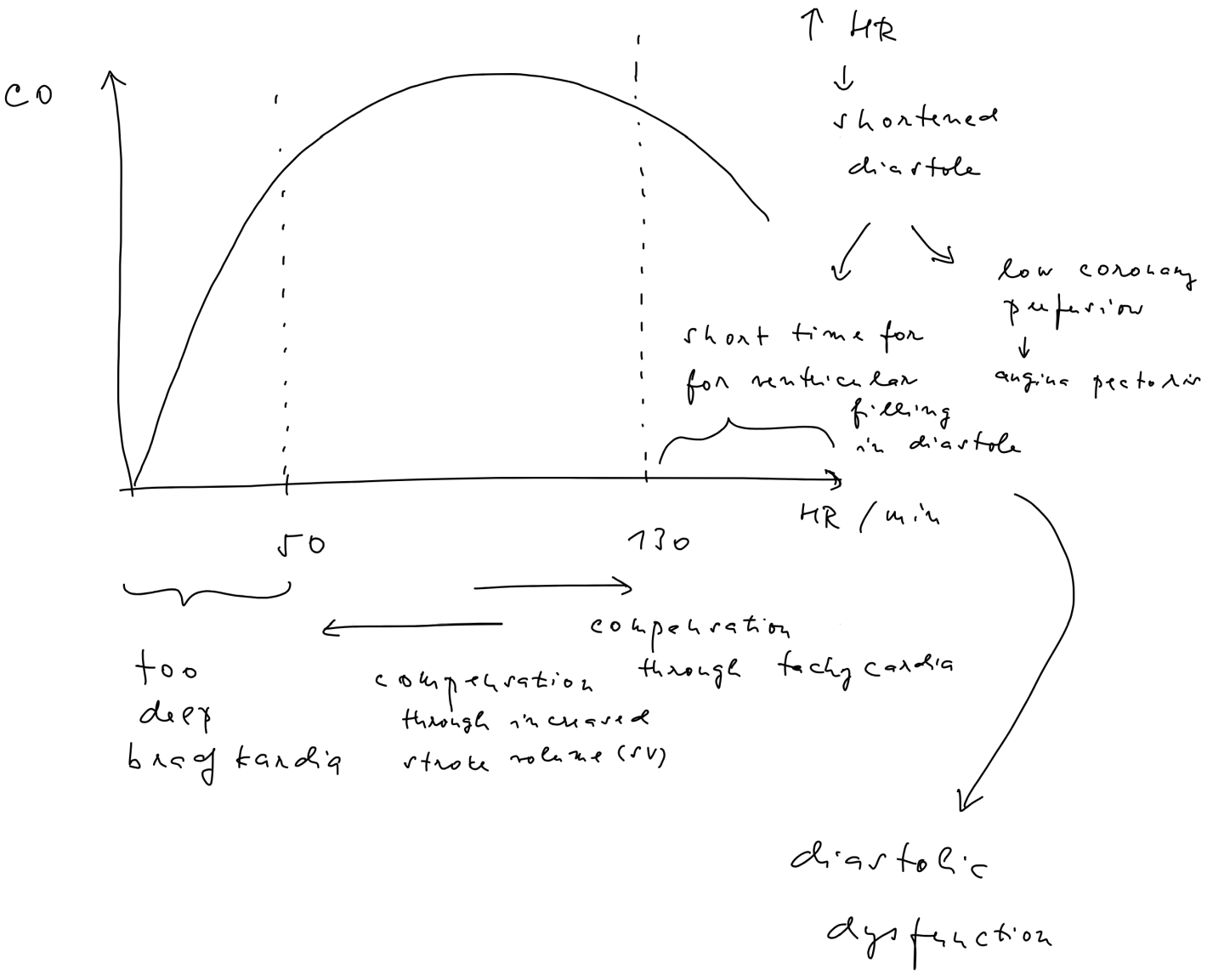


→ pathophysiology

= circulus vitiosus

heart rate and CO

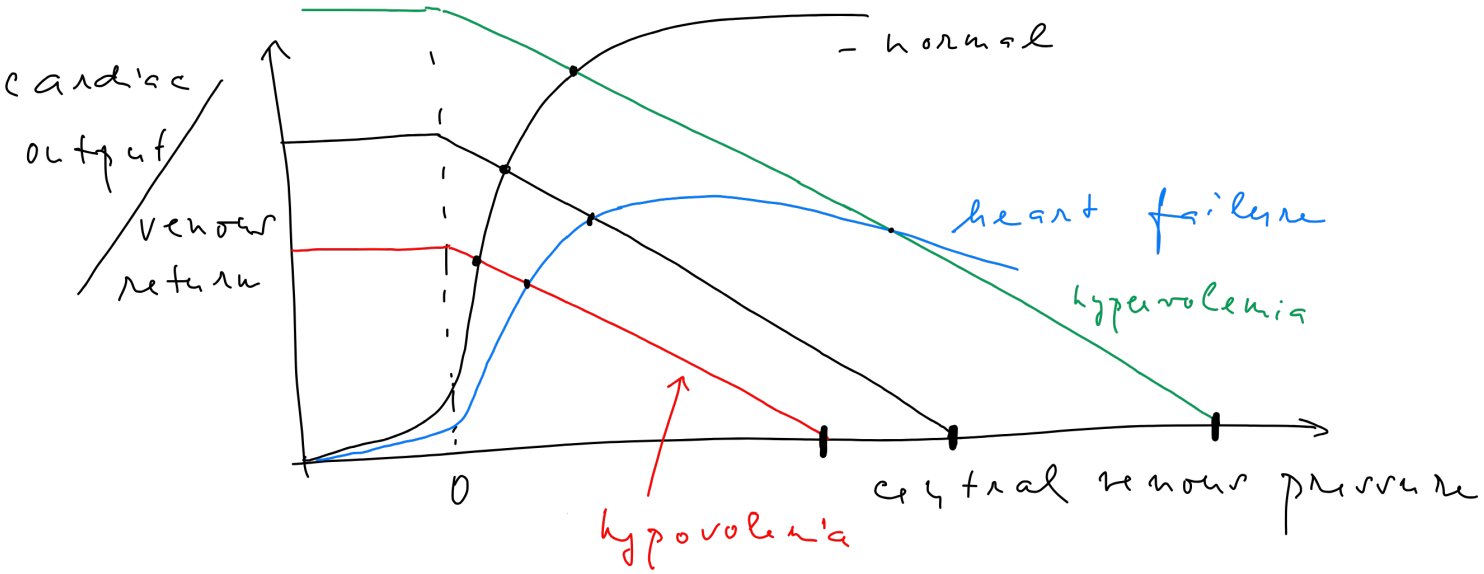
$$CO = TO \cdot TF$$



determination of cardiac output

- under physiological situation, CO is not determined by heart, but by venous return, resp. tissue needs
  = afterload - independent
  ↳ increase in BP does not decrease CO
- in heart failure, the determining factor is heart itself
  = afterload - dependent
  ↳ decrease in BP increases CO

guyton's diagram



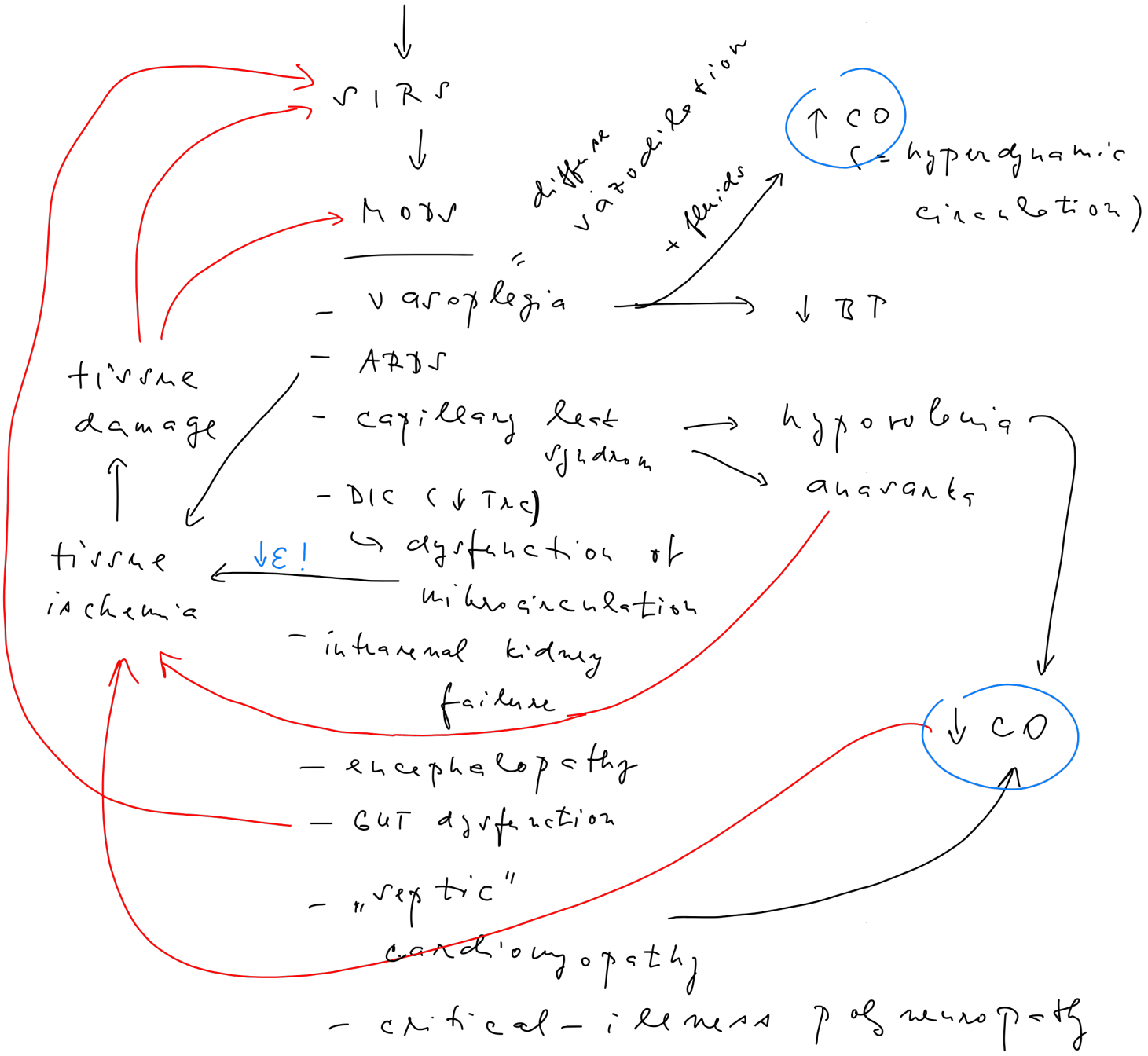
• - working point | = mean circulatory filling pressure (≈ venous pressure)

→ can explain in details the hemodynamics of shock

Pathogenesis of sepsis (short introduction)

sepsis = infectious antigens (= PAMPs)

SIRS = autoantigens (= DAMPs)
- shock from damaged tissues



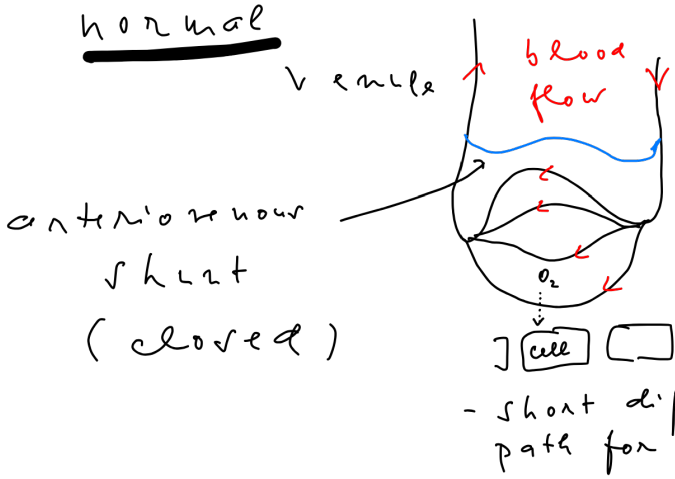
- depending on actual situation, CO can be increased or decreased

"distributive" shock

- What is the meaning of distributive ?

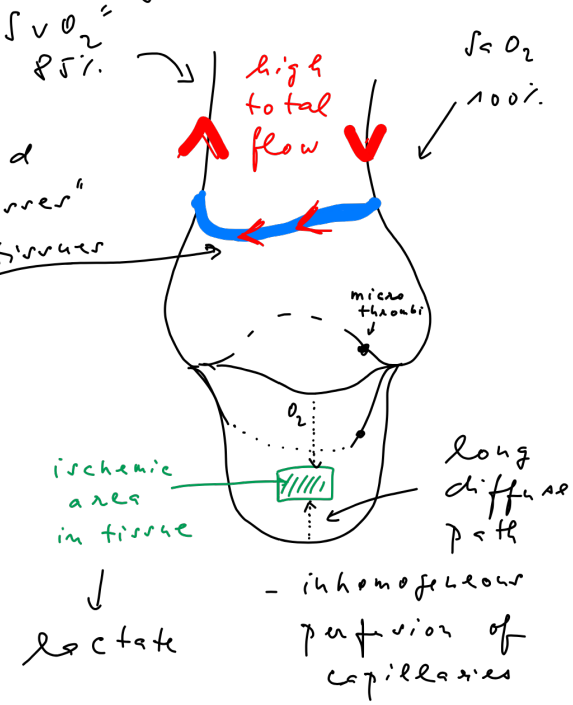
= inhomogeneous distribution of blood flow inside tissues

normal



sepsis

- diffuse vaso-dilation = "bypasses" the tissues → open AV-shunts
- microthrombi formed by thrombocyter → obstruction of capillaries
- mitochondrial dysfunction → impaired utilization of O_2



- the problem is in a low extraction of oxygen from blood, although CO is increased

high central venous saturation