

Pathophysiological principles of oxygen therapy and mechanical ventilation

MUDr. MSc. Michal Šitina, PhD.

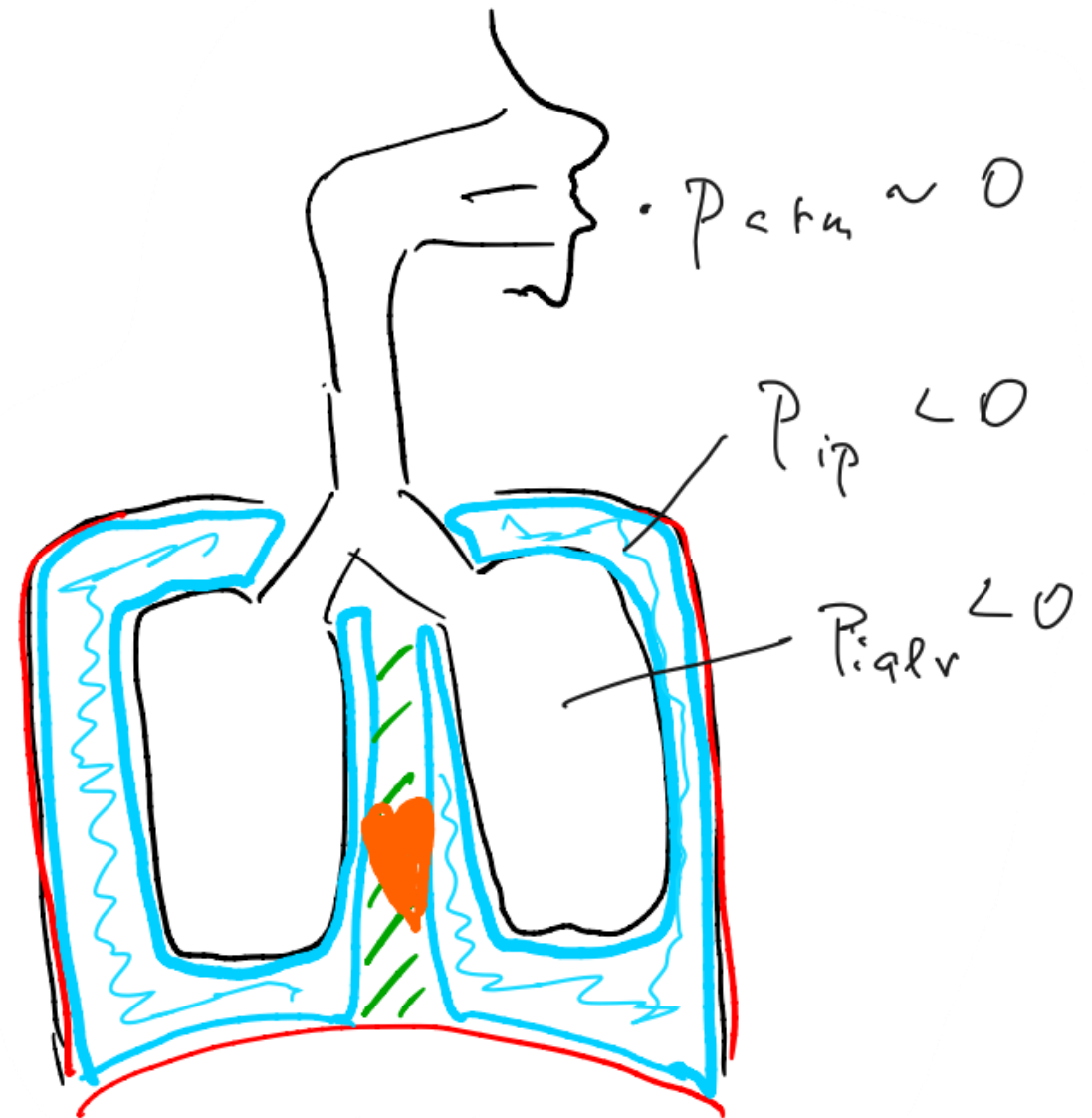
Department of pathological physiology, MUNI
Department of anaesthesia and intensive care medicine, FNUSA
Biostatistics, ICRC-FNUSA

Content

1. Basics of respiratory (patho)physiology
2. Oxygen therapy
3. Mechanical ventilation
4. Non-invasive ventilation (NIV)
5. High-flow nasal oxygen (HFNO)
6. Extracorporeal membrane oxygenation (ECMO)
7. Apnoic ventilation

Mechanics of spontaneous breathing

- pressure in area of lips approx. 0
- active inspiration
 - diaphragm, intercostal muscles
 - negative intrapleural pressure
- spontaneous expiration
 - positive intrapleural pressure



Important quantities and terms

- FiO_2 (21 %)
- PaO_2 (> 80 mmHg, hypoxemia vs. hypoxia)
- PaCO_2 (35-45 mmHg, hypo/normo/hypercapnia)
- tidal volume (\approx 500 ml)
- respiratory rate (\approx 12-16/min)
- anatomic dead space (150 ml)

6 l
↓
4 l

Respiratory insufficiency

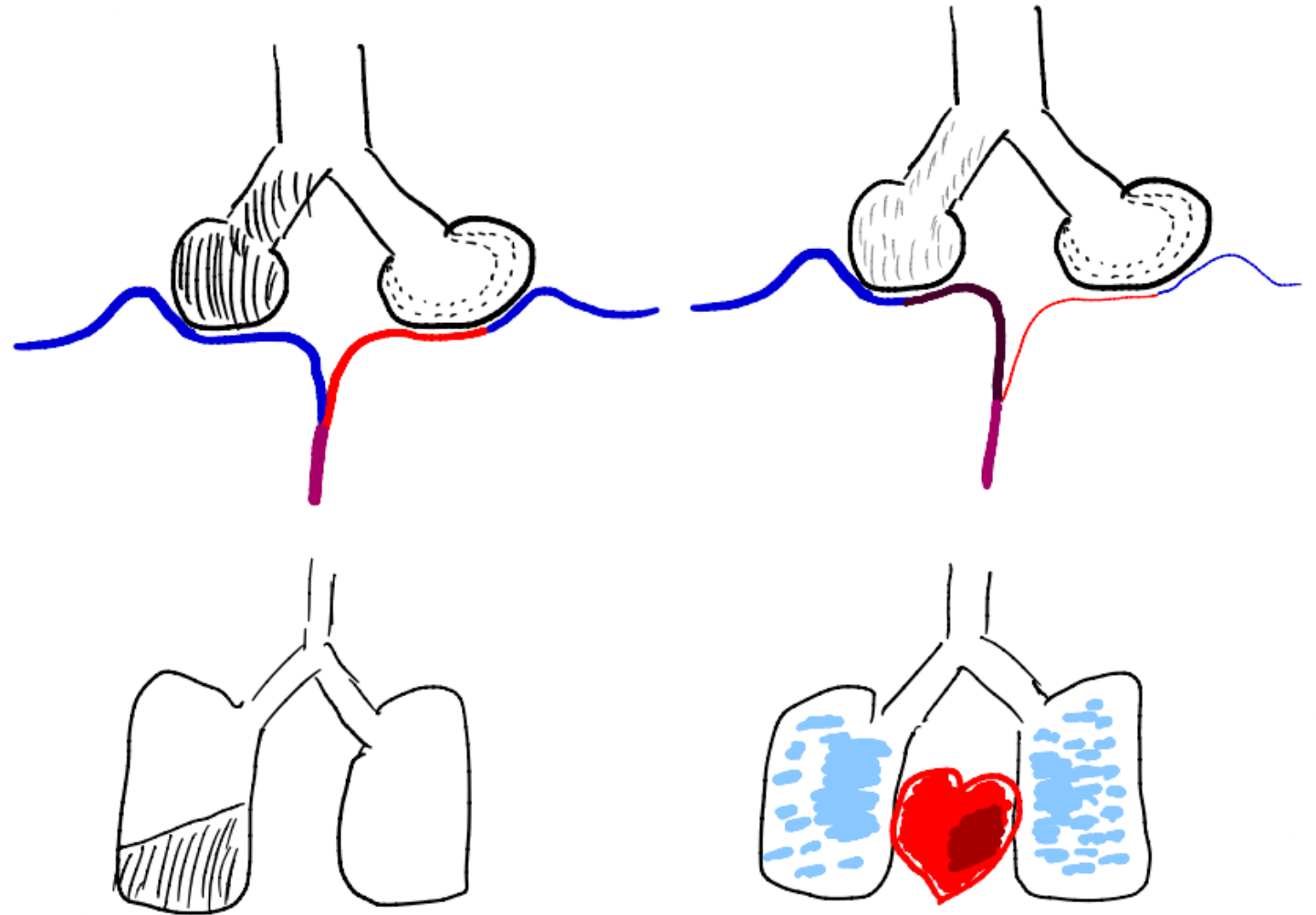
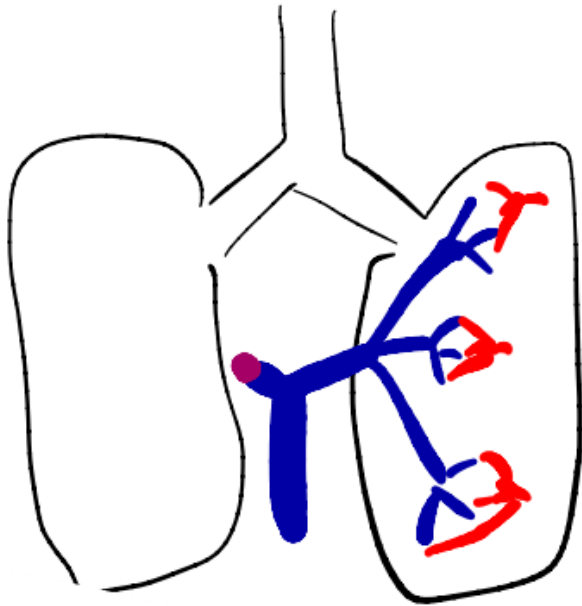
- type 1 – oxygenation dysfunction - hypoxemia without hypercapnia
- type 2 – ventilation dysfunction – hypercapnia with hypoxemia

Mechanisms of respiratory insufficiency

- alveolar hypoventilation
- impaired diffusion across alveolocapillary membrane
- intrapulmonary (or extrapulmonary) shunt
- ventilation-perfusion (V/Q) mismatch

Mechanisms of respiratory insufficiency

- hypoventilation, diffusion, shunt, V/Q mismatch



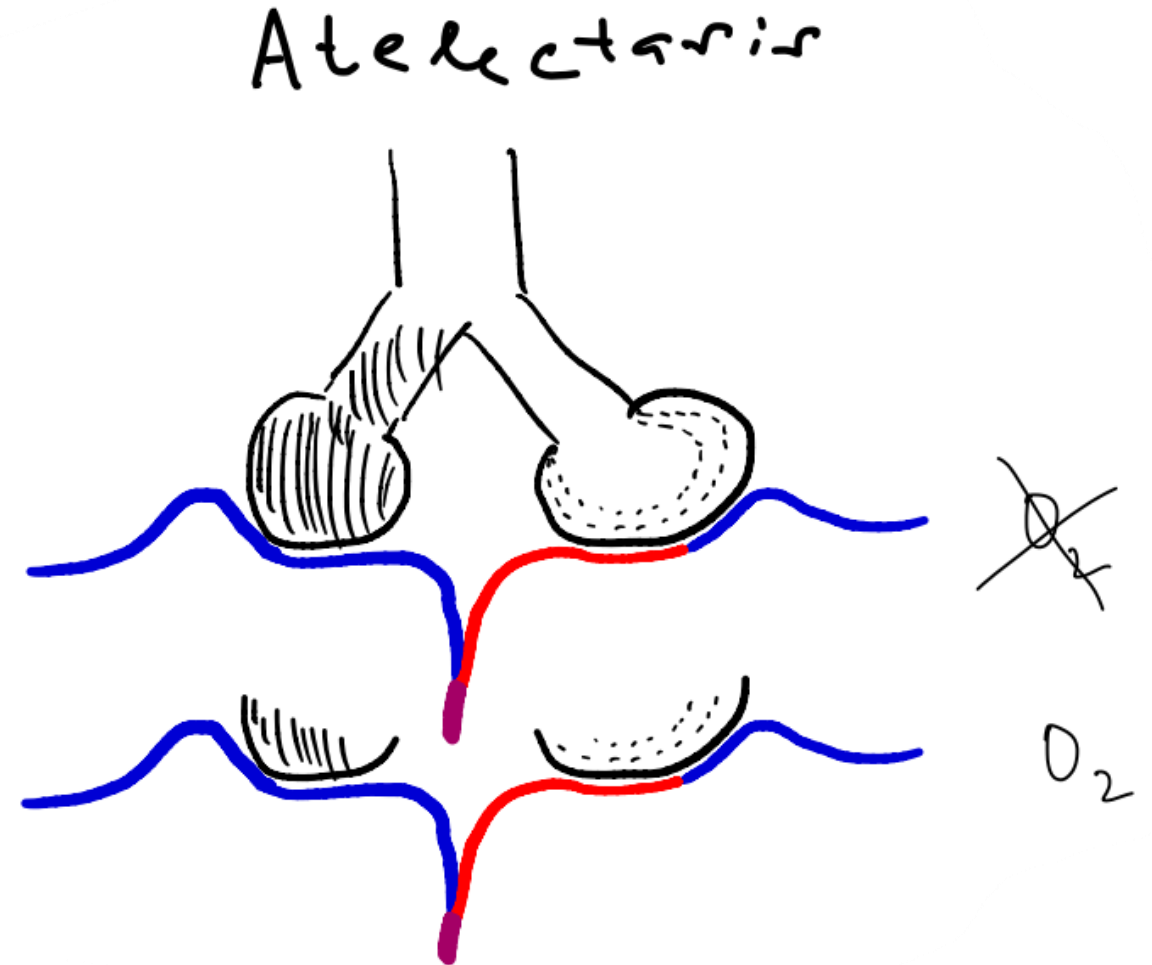
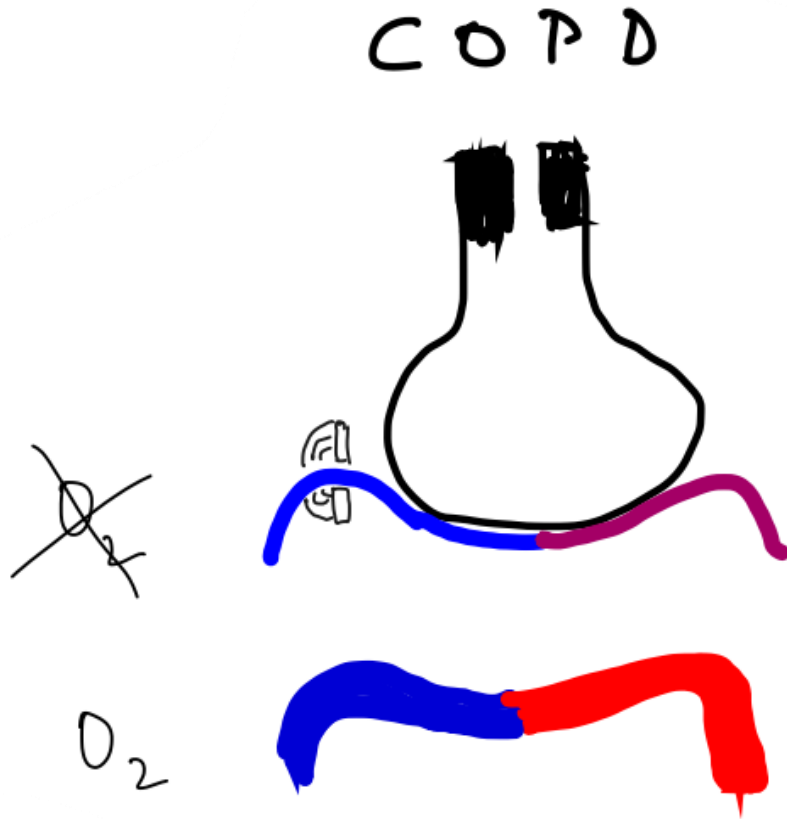
Oxygen therapy

- oxygen delivery
- principle: increased FiO_2
- corrects hypoxemia
- no correction or impairment of hypercapnia
- sometimes almost no effect



Oxygen therapy

- Why impairment of hypercapnia?
- Why sometimes almost no effect?



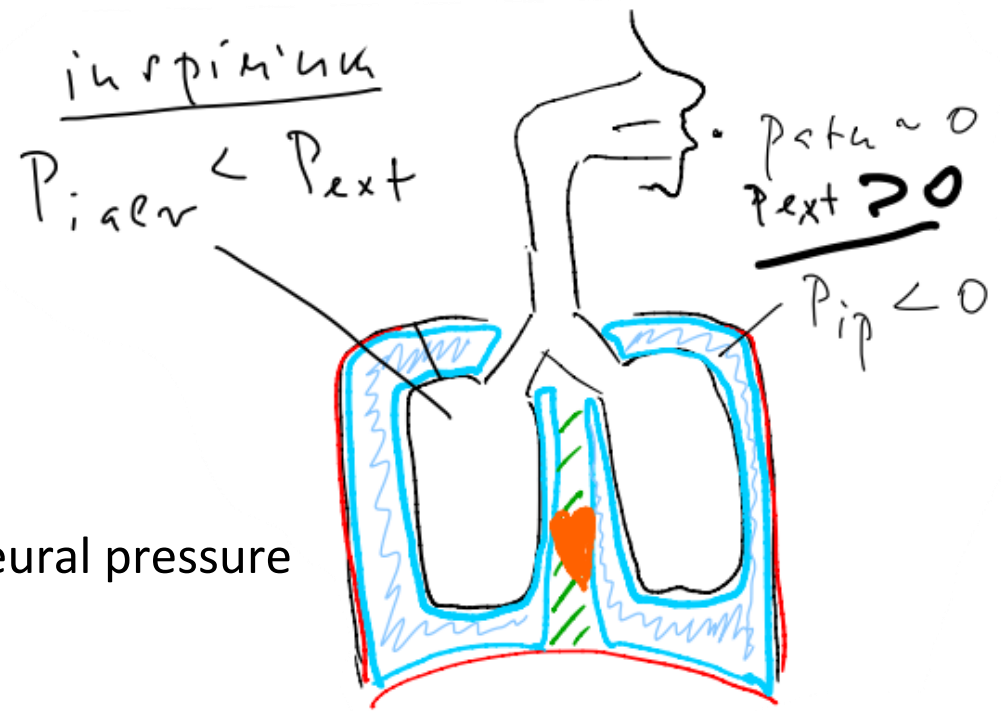
Mechanical ventilation

- ventilation using ventilator replacing a part or a whole work of breathing of a patient
- therapeutic goals
 - correction of oxygenation
 - correction of hypercapnia
 - decrease in work of breathing
 - hemodynamic stabilization
 - airways protection
 - performance of an operation
 - ...

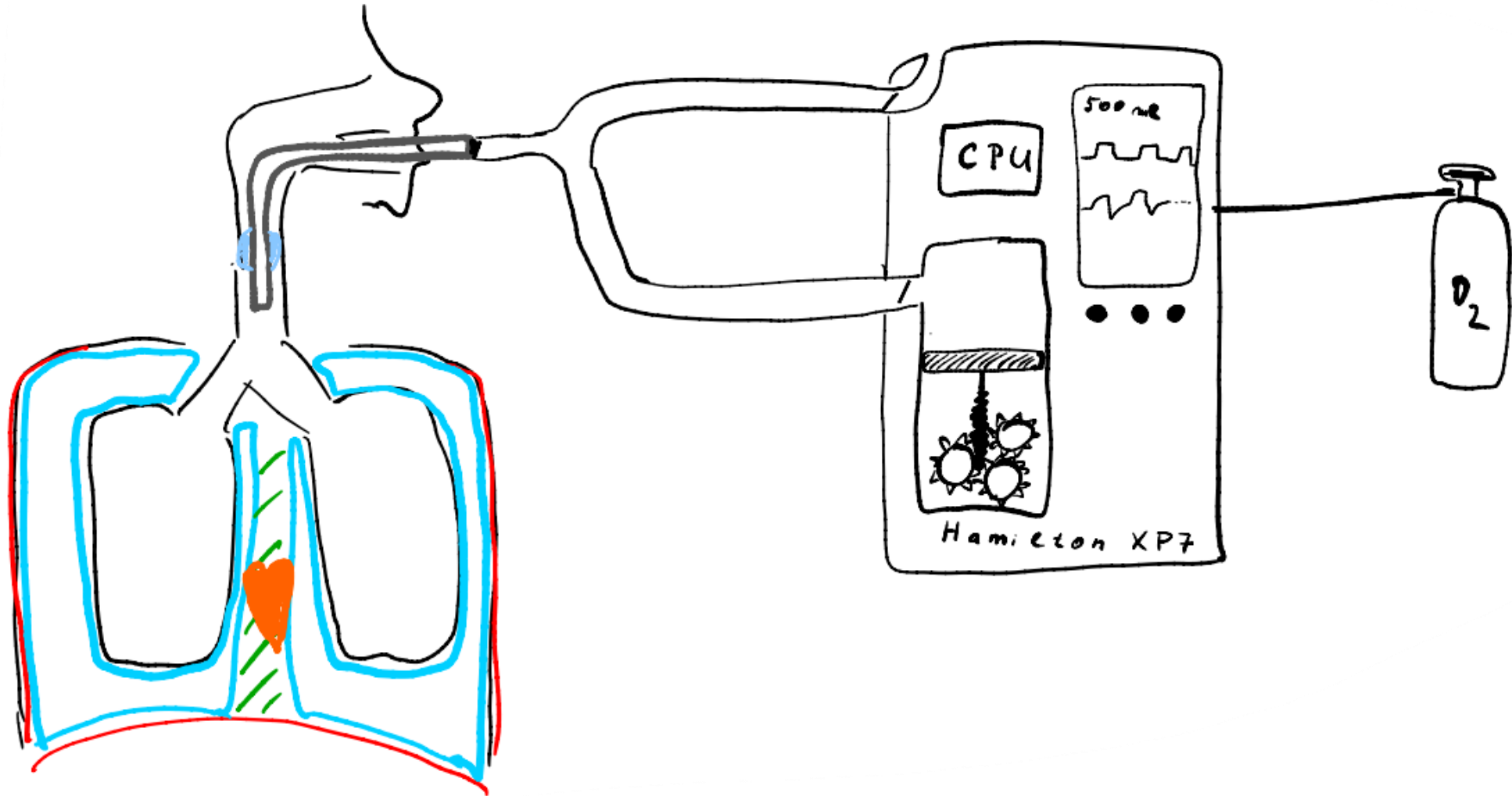


Principle of mechanical ventilation

- active inspiration
 - positive pressure in airways, higher than the intrapleural pressure
- passive expiration
 - just as spontaneous breathing
- there are many ways (called modes) of ventilation (volume controlled, pressure controlled, supported, triggered ...)
- inappropriate ventilation can result in significant lung damage (VILI, VALI, SILI)



Principle of mechanical ventilation



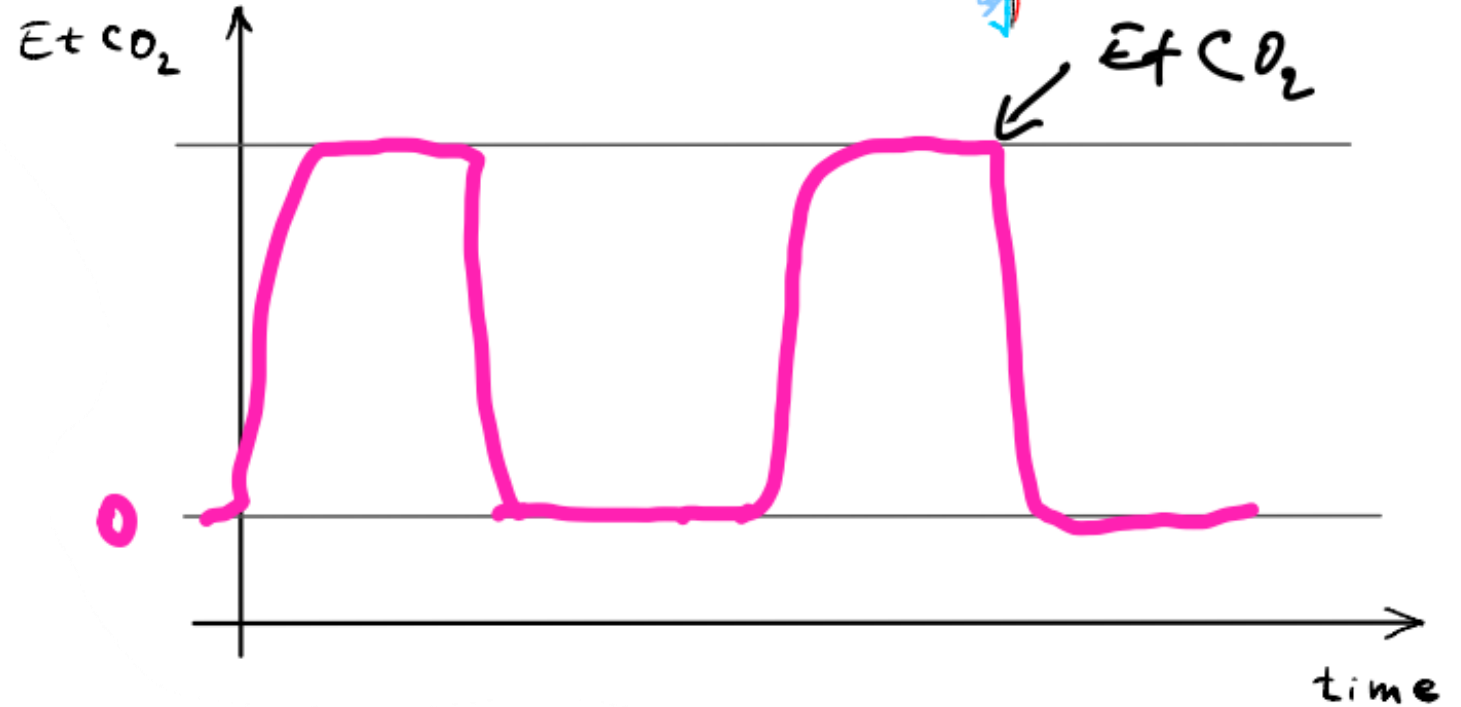
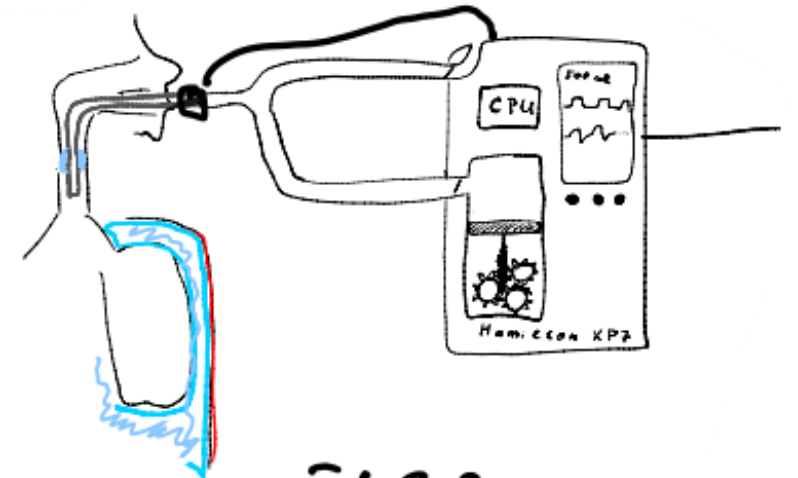
What can we set on the ventilator?

- FiO_2
- PEEP (positive end-expiratory pressure)
- tidal volume
- peak pressure
- respiratory rate
- ratio of duration of inspiration/expiration
- trigger level
- ...

What do we monitor?

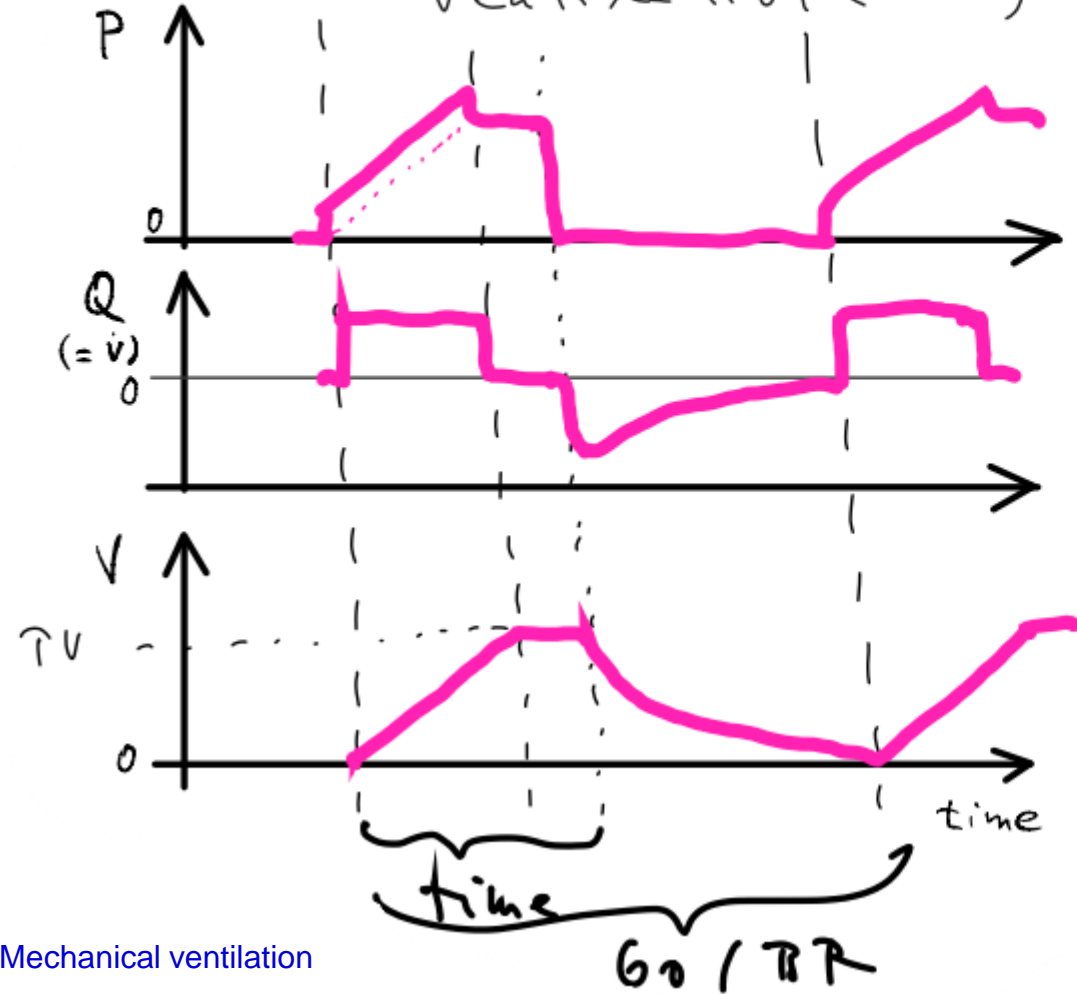
- SaO_2
- blood gases
- $EtCO_2$ (end-tidal CO_2)
- pressures
- volumes
- flows
- corresponding curves
- ...

capnometry

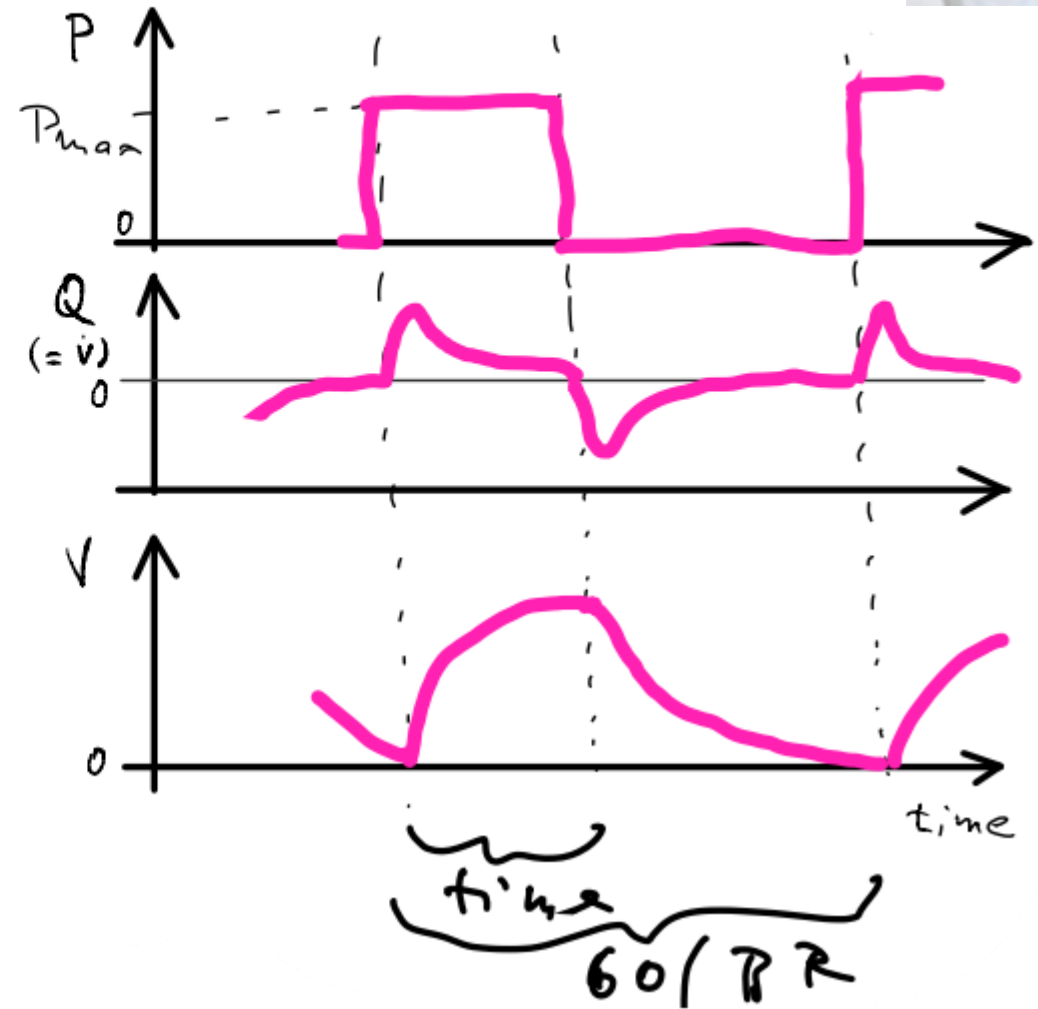


Curves of mechanical ventilation

(TV) , RR, time
volume controlled
ventilation (CMV)



RR, time, P_{max}
pressure controlled
ventilation (PCV)

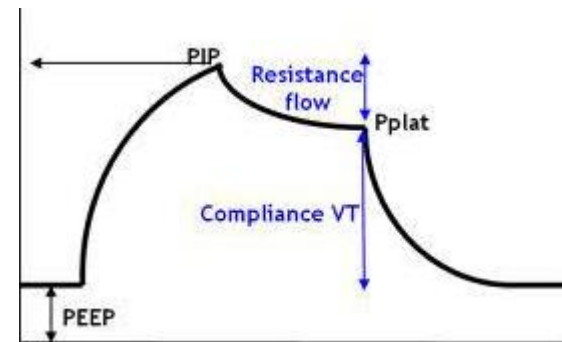


PEEP (positive end-expiratory pressure)

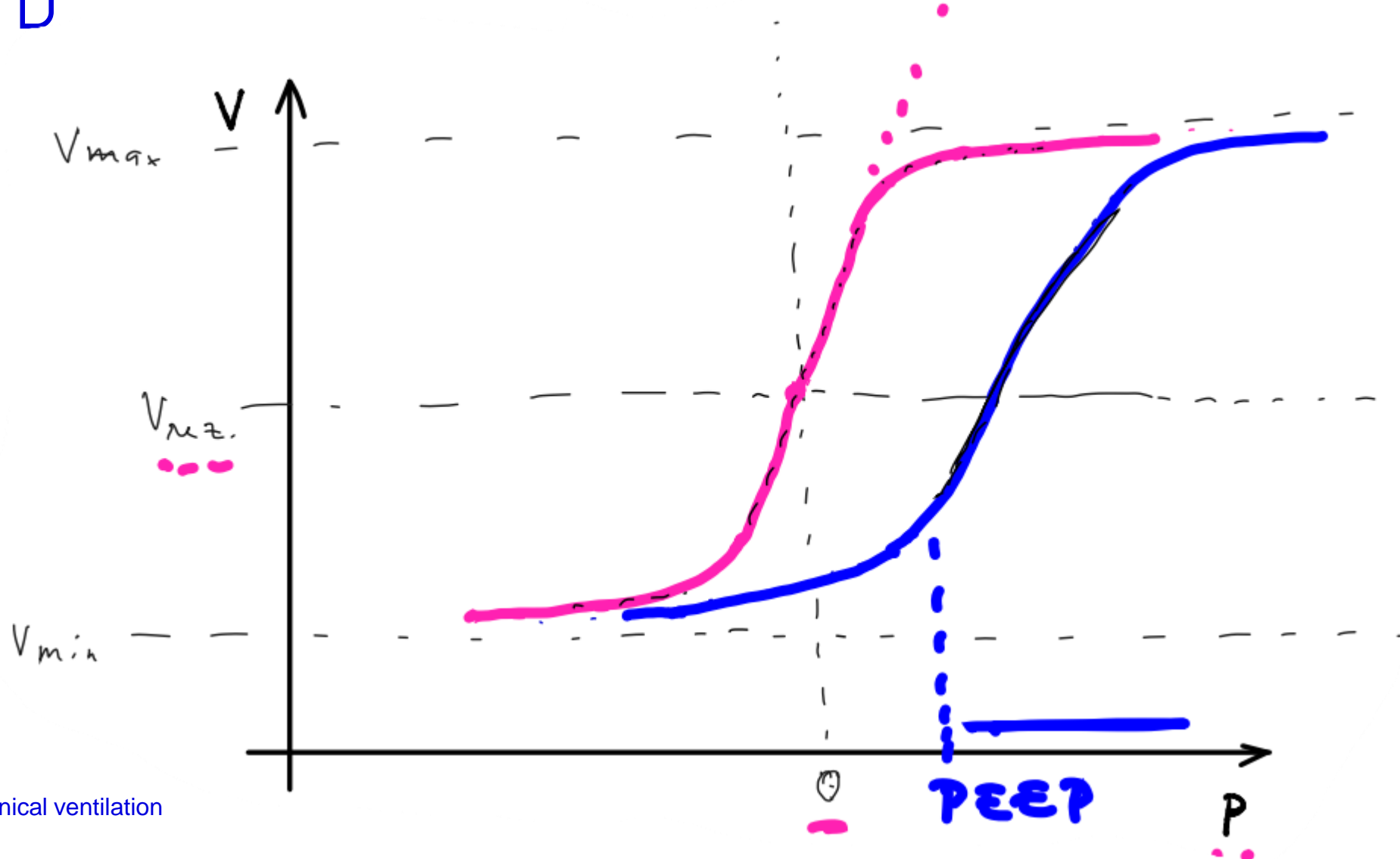
- the lowest pressure in airways
- prevents alveolar collapse (called atelectasis)
- maintain opened bronchi

- too low or too high values are harmful
- it is necessary to find the optimal value

- affects the cardiovascular system



p-V curve and PEEP



Effects on cardiovascular system

- decreases venous return and subsequently the cardiac output
- affects pulmonary hypertension and so the right ventricle function
- can help the failing left ventricle
- decreases oxygen consumption in respiratory muscles

Effects on other systems

- significant influence on acid-base balance (CO₂)
- decreases renal blood flow and so promotes fluid retention
- increases intraabdominal pressure and reduces splanchnic perfusion
- can increase intracranial pressure
- „motor“ of multiorgan failure

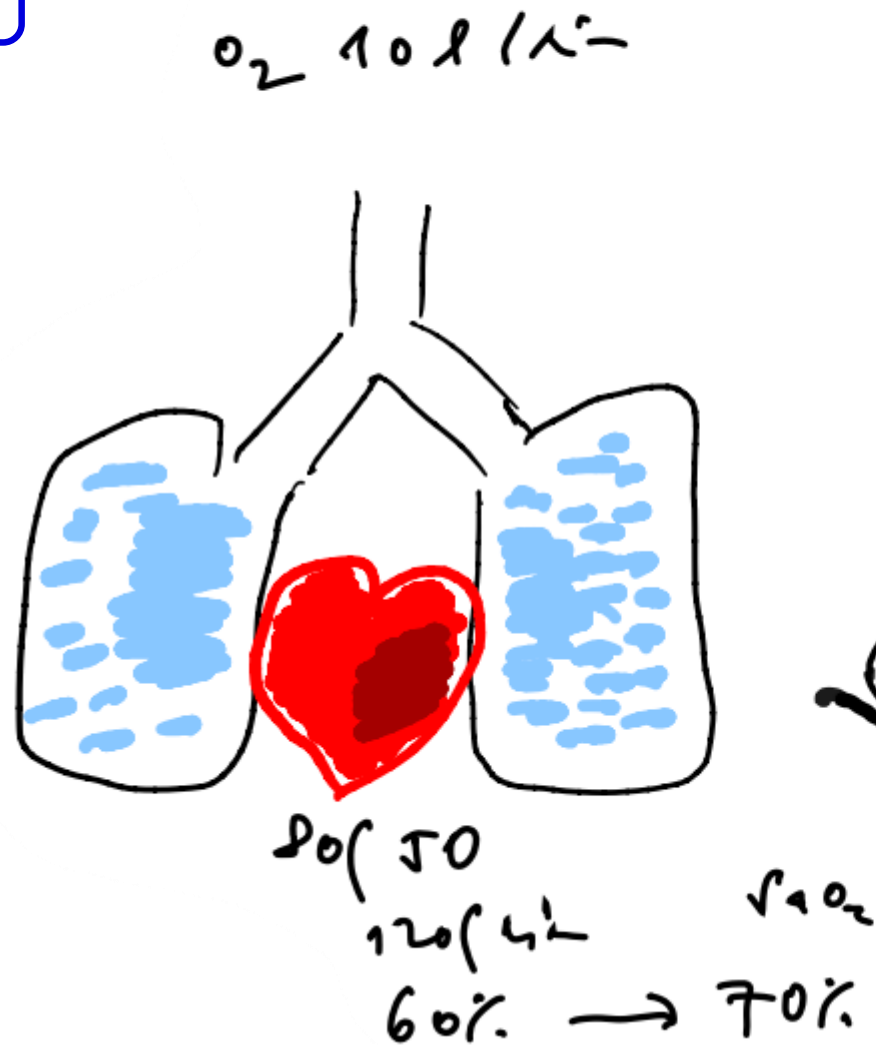
How can mechanical ventilation damage lungs (VILI, VALI, SILI)?

- large distension tears pulmonary structures
 - secondary inflammatory reaction, support of fibrotization
 - increased permeability for bacteria
 - motor of multiorgan dysfunction
 - baby lung concept - ARDS
- danger of lung perforation in a thin area
 - pneumothorax, pneumomediastinum
- shearing forces in the boundary of ventilated and non-ventilated areas of lungs
- elimination of natural immune barriers
 - ventilator-associated pneumonia (VAP vs. HAP vs. CAP)
- risks of intubation and airway management
- promotion of muscle weakness of critically ill patients
- necessary sedation

Examples of use of MV in clinical situations

- Pulmonary edema by acute myocardial infarction
- COPD exacerbation
- Intubation and MV by polytrauma
- Massive pulmonary embolism
- ARDS – COVID-19 pneumonia

Pulmonary edema by acute myocardial infarction



PEEP 15
CH₂H₂O

$NOB = 0$
25% O_2

↑ $\sqrt{100}$
stabilization

Intubation and MV by polytrauma



OTI, MV \Rightarrow

75/40

140/min

↑ lactate \rightarrow acidosis

hyperventilate
40/min

\downarrow
pH = 7.1

$\downarrow \downarrow$ CO

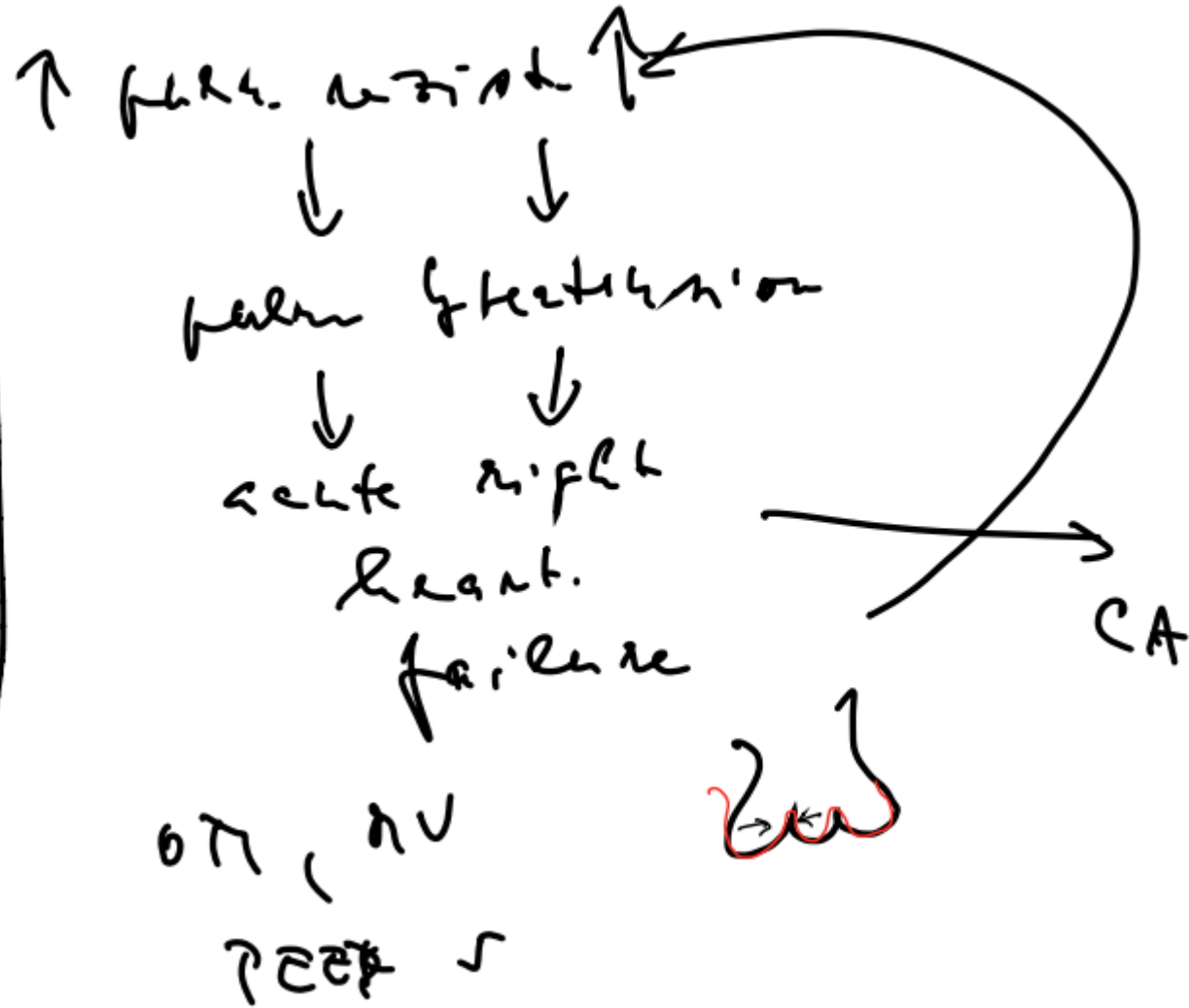
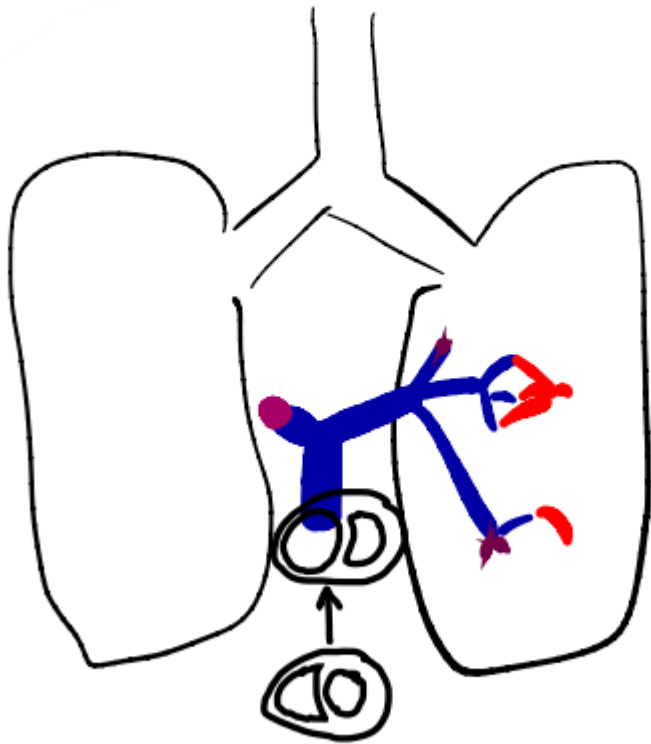
$\downarrow \downarrow$ BP (50/20)

20/min

\downarrow
pH = 6.8

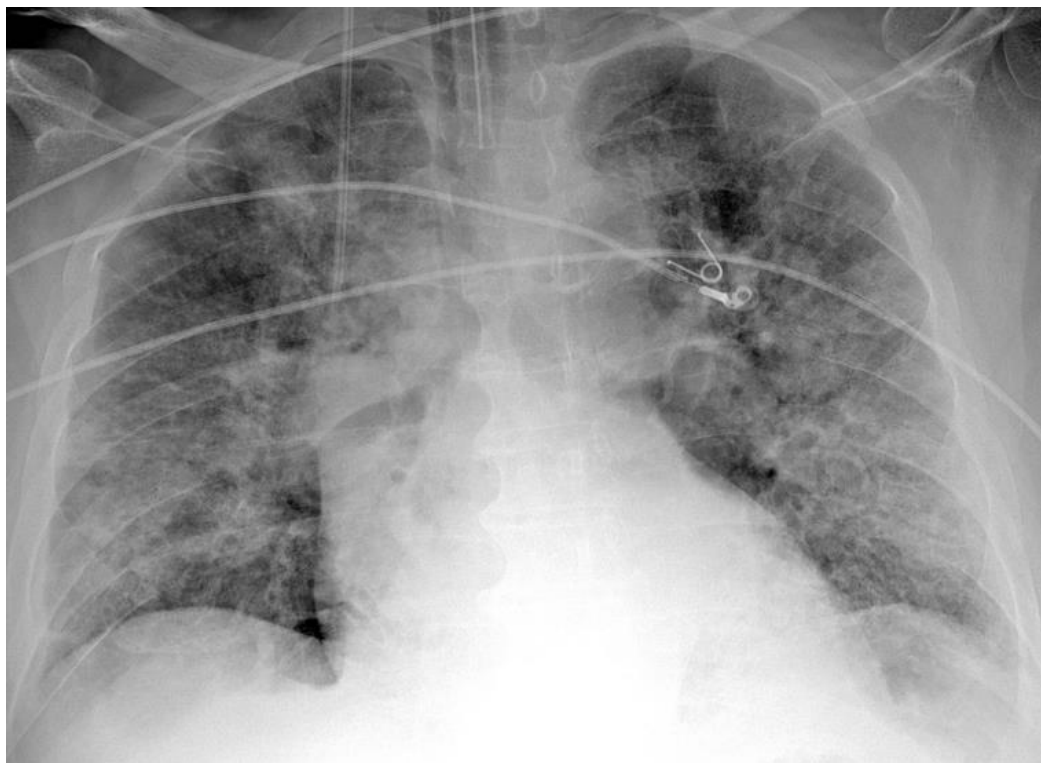
cardiac
arrest
 \downarrow
CPR

Massive pulmonary embolism

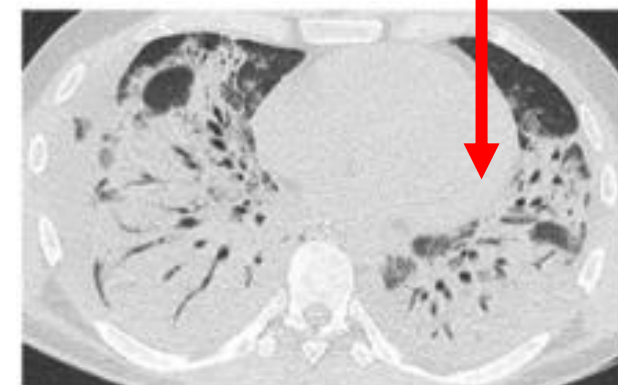
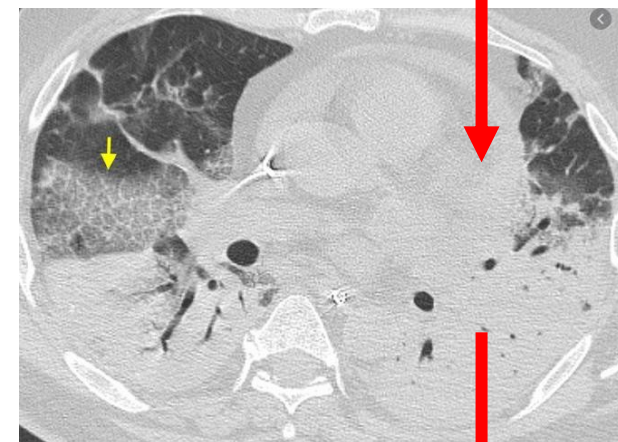


ARDS – COVID-19 pneumonia

- protective ventilation
- permissive hypercapnia (pH >7.2)
- prone position



↓ TV } ↑ pO₂
↑ PEEP }
↑ RR



Non-invasive ventilation

- just as mechanical ventilation, but
 - patient is not (deeply) sedated
 - airways are not secured
- not possible to use too high PEEP or inflation pressures
- shortterm or repeated usage
- typical indications
 - acute COPD exacerbation
 - moderate cardiogenic pulmonary edema
 - intermittent support after extubation



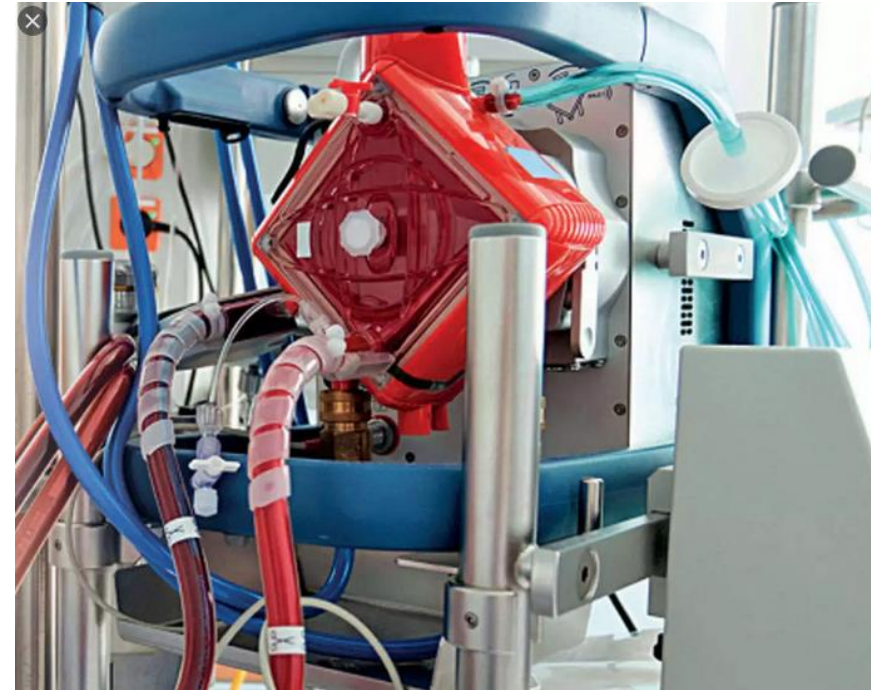
High-flow nasal oxygen (HFNO)

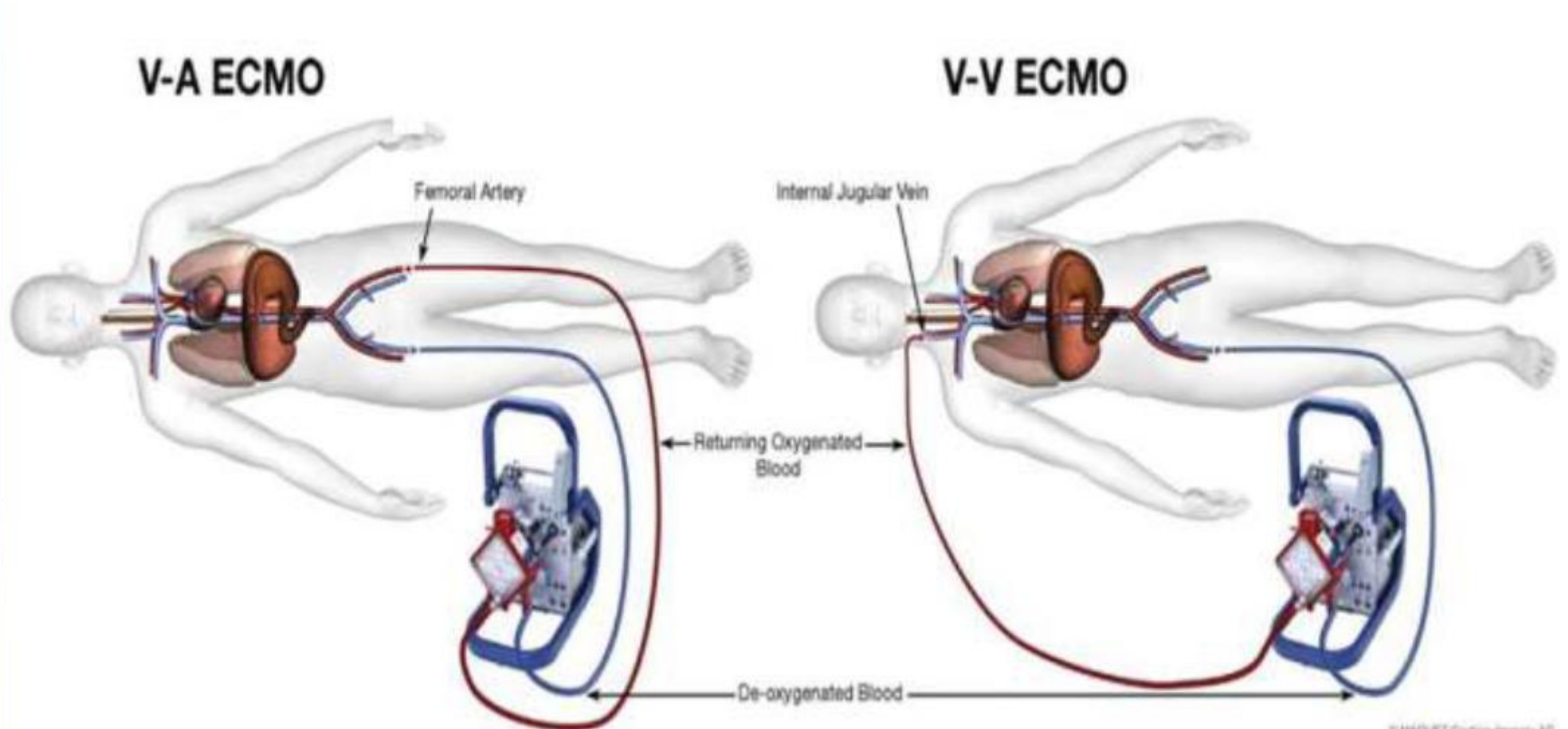
- just as nasal cannula, but
 - humidified oxygen up to 60 l/min
 - FiO₂ up to 100 %
- high flow of gases builds up excess pressure in upper airways and so the PEEP 2-4 cmH₂O
- better tolerated than NIV
- similar indications as NIV
 - moderate COVID-19 pneumonia
 - acute COPD exacerbation
 - moderate cardiogenic pulmonary edema
 - intermittent support after extubation



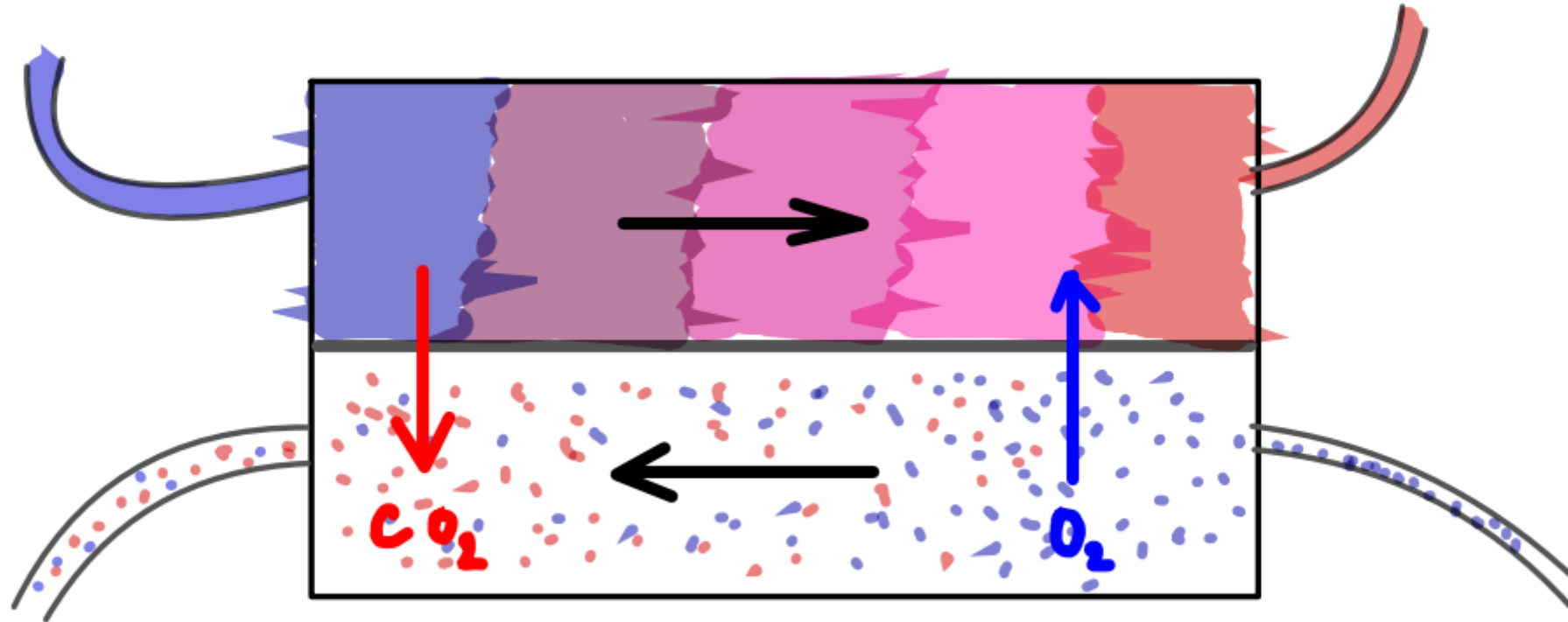
Extracorporeal membrane oxygenation (ECMO)

- extracorporeal circuit
- up to complete substitute of lungs (VV-ECMO) or heart and lungs (VA-ECMO)
- construction based on pump and oxygenator
- in oxygenator blood and air/oxygen come to contact over a membrane
- Indication
 - reasonable chance of solution of the basic problem (as e.g. cure of COVID pneumonia) or bridge-to-transplantation



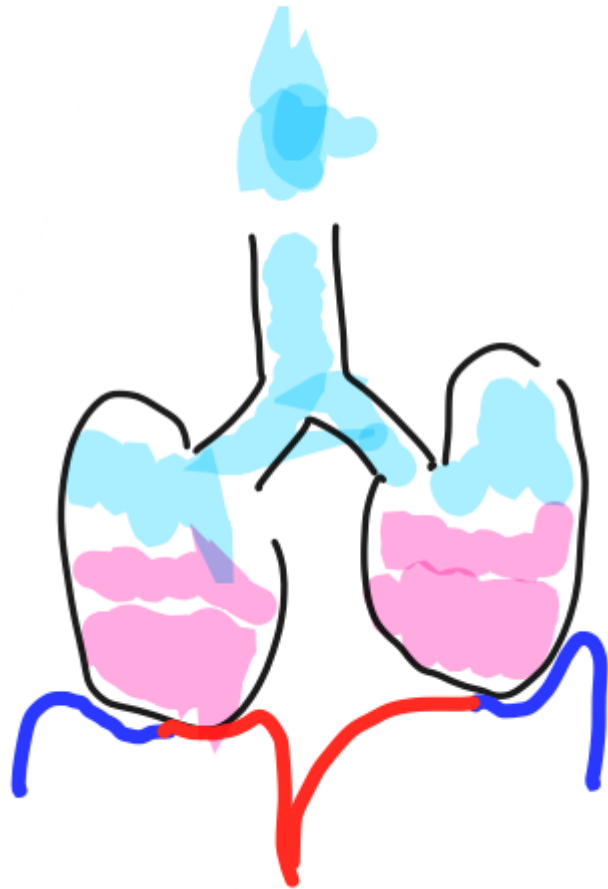


Principle of membrane oxygenator



Apnoic ventilation

- Contradiction?
- You let a patient breathe oxygen via mask, so you create an oxygen reserve in lungs, and with continuing oxygen supply the general anaesthesia and muscle relaxation will be induced, so that patient does not breathe any more, but oxygen would continue to be supplied
- oxygen reserve in lungs (5 l) with consumption of 250 ml O₂/min would be sufficient for max. 20 minutes
- How long will it take, until the oxygen saturation of this non-breathing patient starts to fall? Up to 60 minutes!!
- How is it possible?



$O_2 \rightarrow CO_2$
 $P_a = 0.8$
 $\dot{V}_R \rightarrow \dot{V}_E$

\dot{V}_R
 12ℓ

Life-threatening respiratory disorders

Cardiogenic pulmonary edema

Non-cardiogenic pulmonary edema
– ARDS

Severe pneumonia

Exacerbation of COPD/asthma

Tension pneumothorax

Upper airway obstruction

Allergic edema

Laryngitis

Epiglottitis

Aspiration

Massive pulmonary embolism

Coma with secondary asphyxia

Acute neuromuscular disorders

Myasthenia gravis

Syndrome Guillain-Barré

Thorax trauma

Lung contusion

Block rib fracture

Massive hemothorax

Massive haemoptysis