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# Pathophysiological principles of respiratory insufficiency, oxygen therapy and mechanical ventilation

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# Contents

1. Basics of respiratory (patho)physiology
2. Principles of mechanical ventilation
3. Applied pathophysiology of MV in clinical cases
4. *Exact analysis of mechanisms of respiratory insufficiency*

# Mechanisms of respiratory insufficiency

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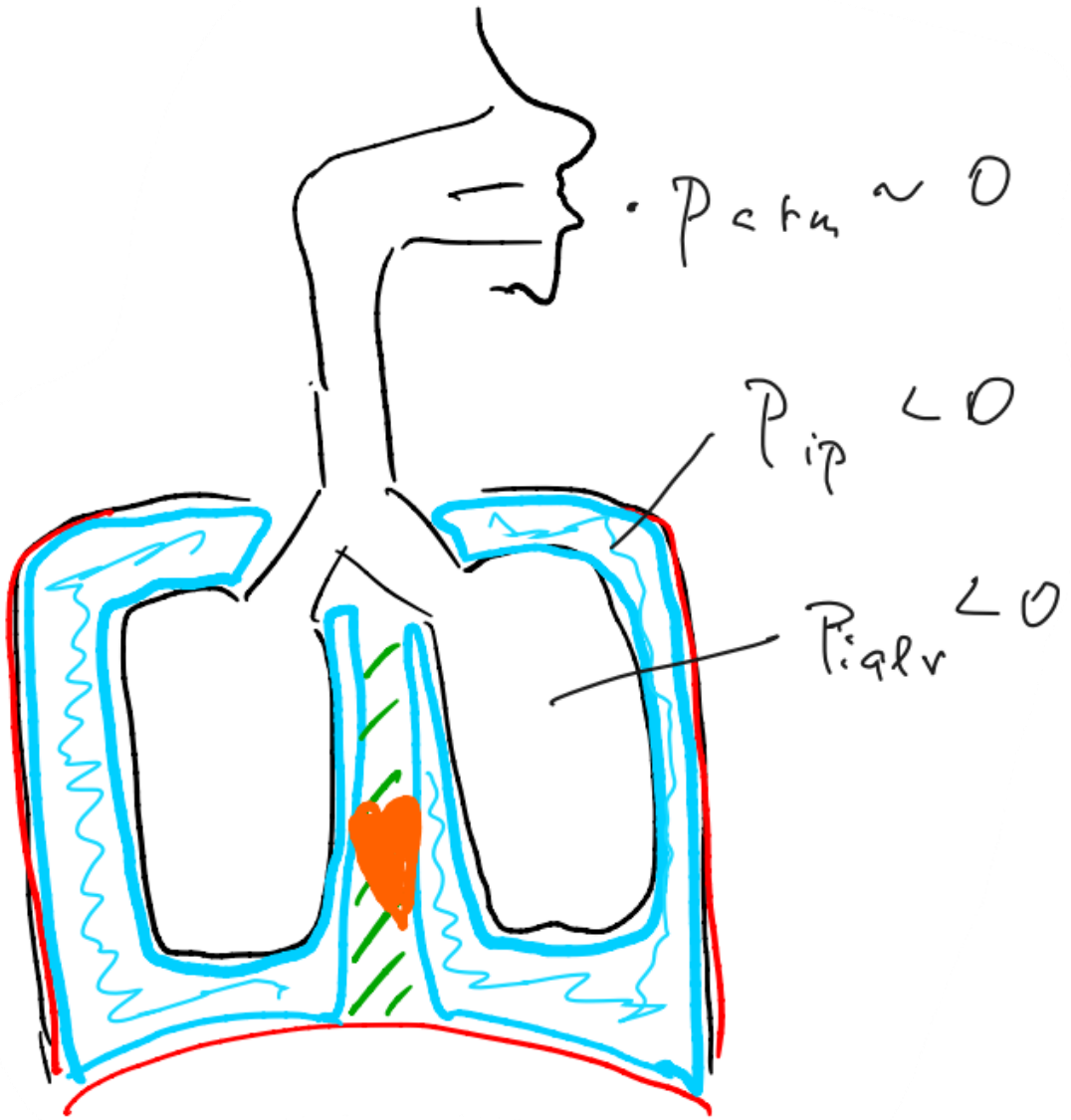
## Mechanics of spontaneous breathing

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



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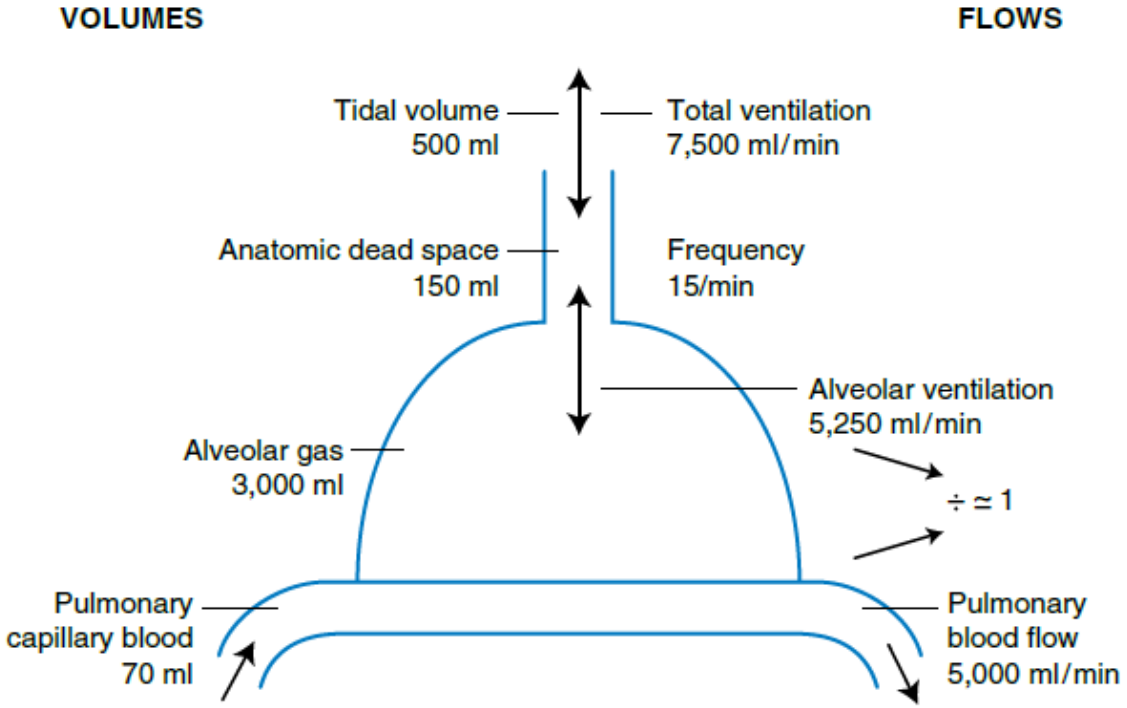
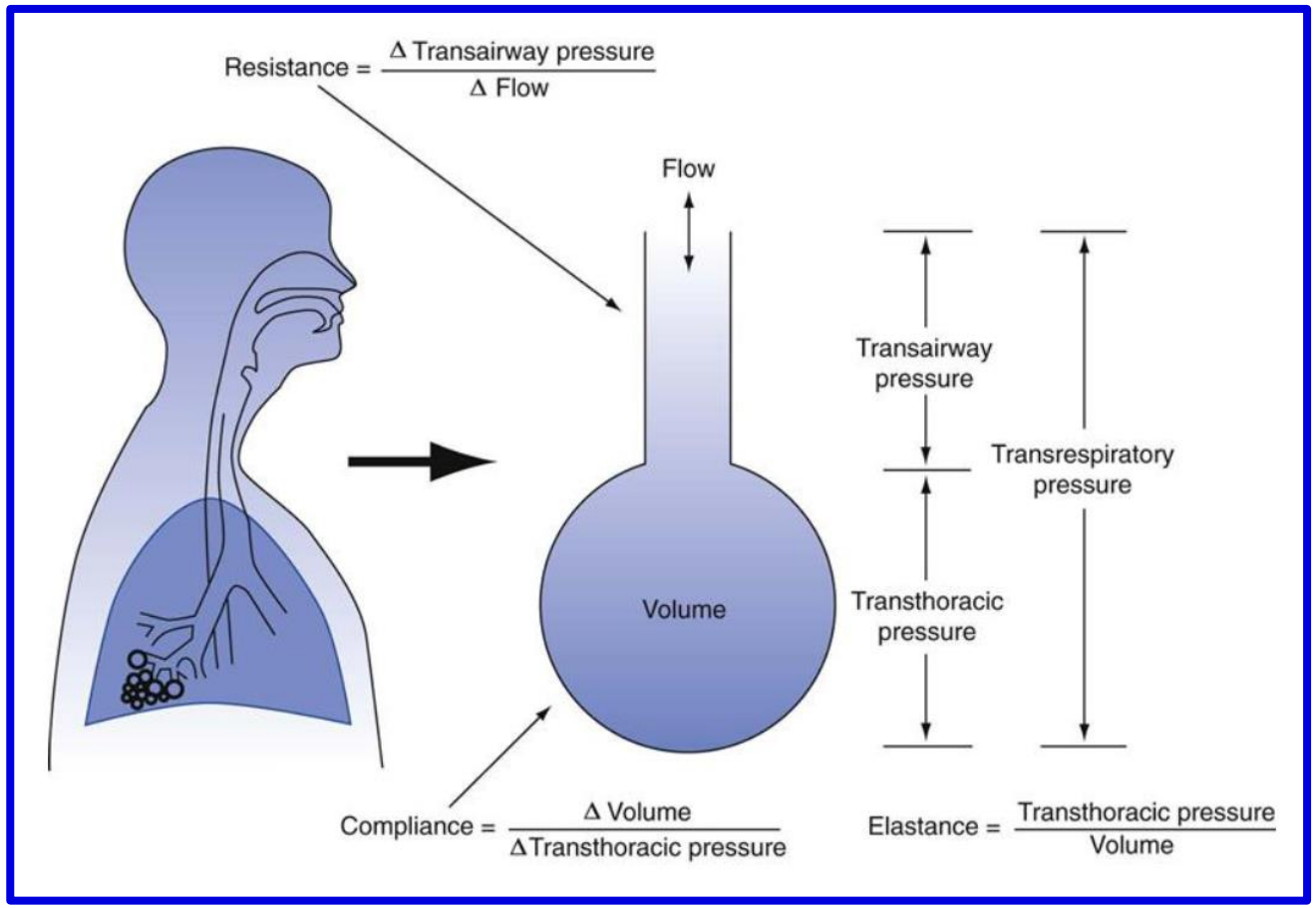
# Important quantities and terms

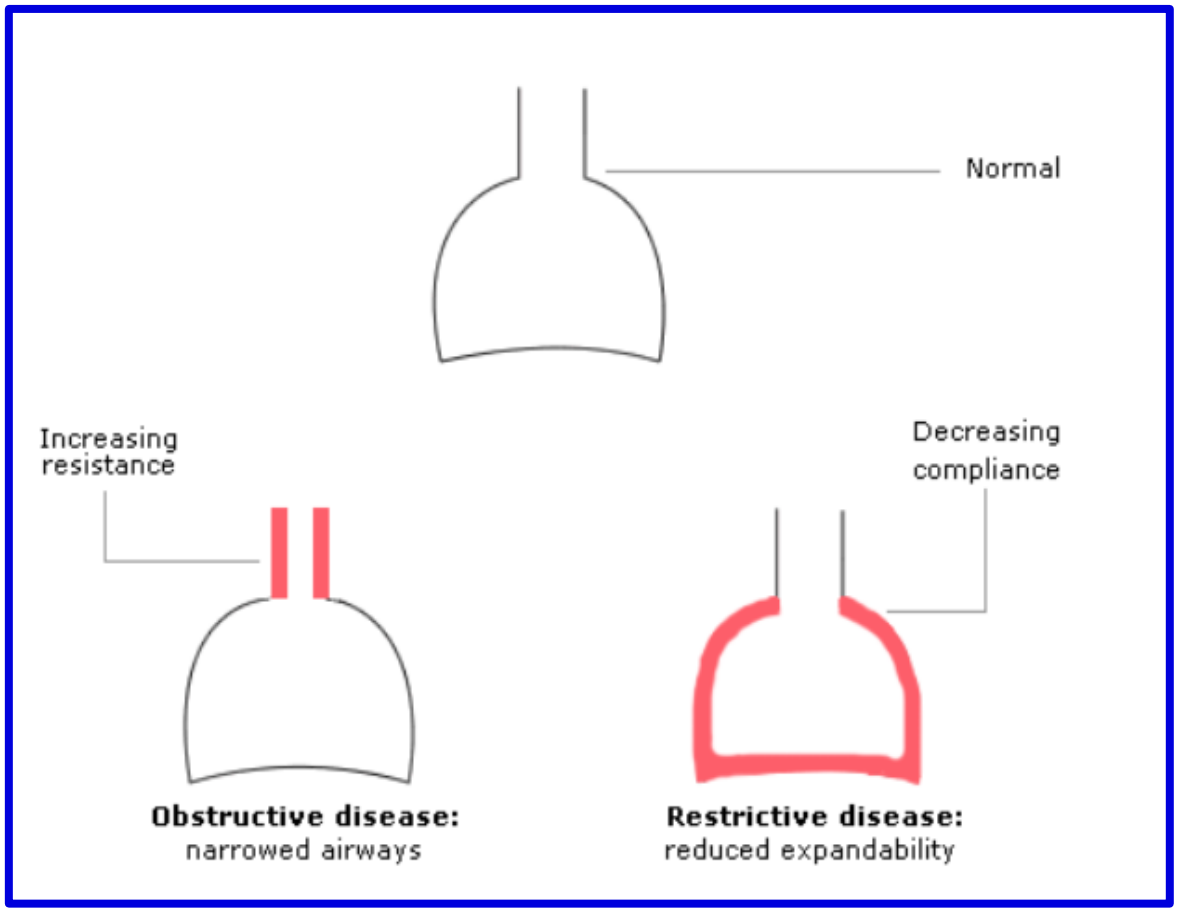



- $FiO_2$ (21 %)
- $PaO_2$ (> 80 mmHg, hypoxemia vs. hypoxia)
- $PaCO_2$ (35-45 mmHg, hypo/normo/hypercapnia)
- tidal volume (≈500 ml)
- respiratory rate (≈ 12-16/min)
- anatomic dead space (150 ml)

# Physical principles

- pressure vs. pressure difference
- compliance
- resistance



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## Respiratory insufficiency

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

## Mechanisms of respiratory insufficiency

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

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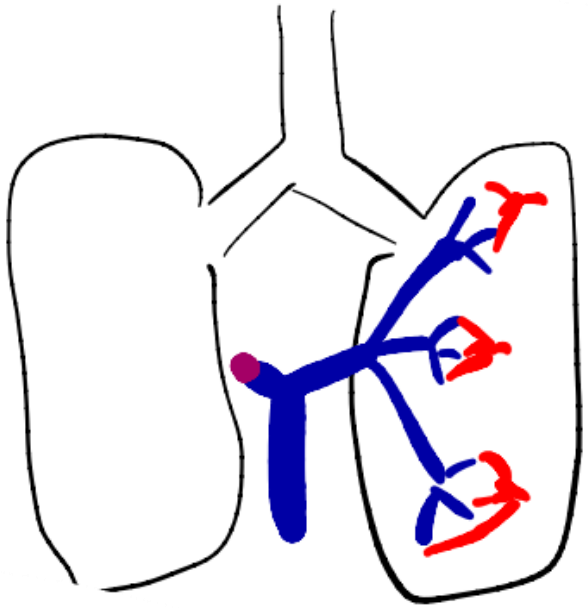
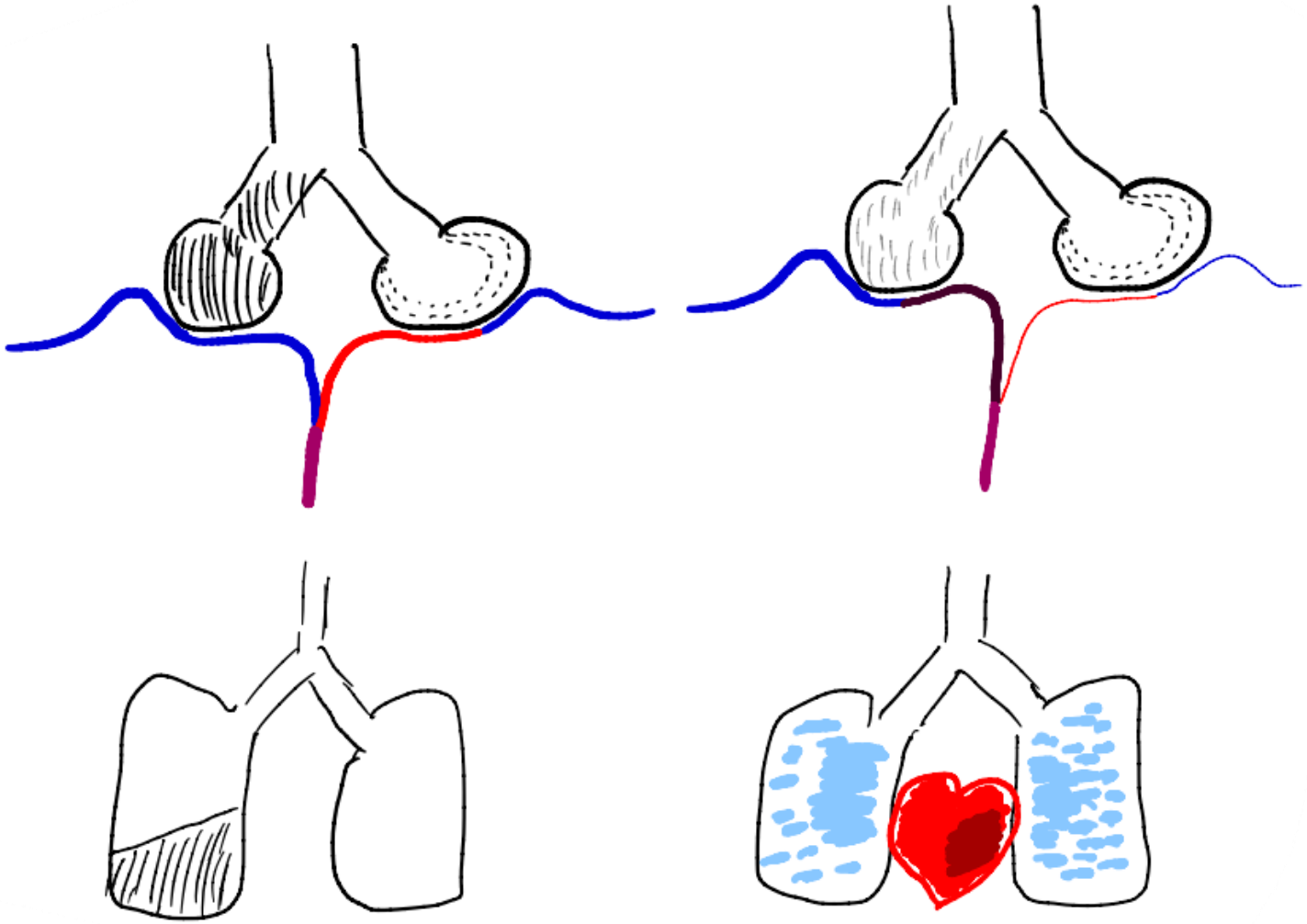
# Mechanisms of respiratory insufficiency

- hypoventilation, diffusion, dead space, shunt, V/Q mismatch





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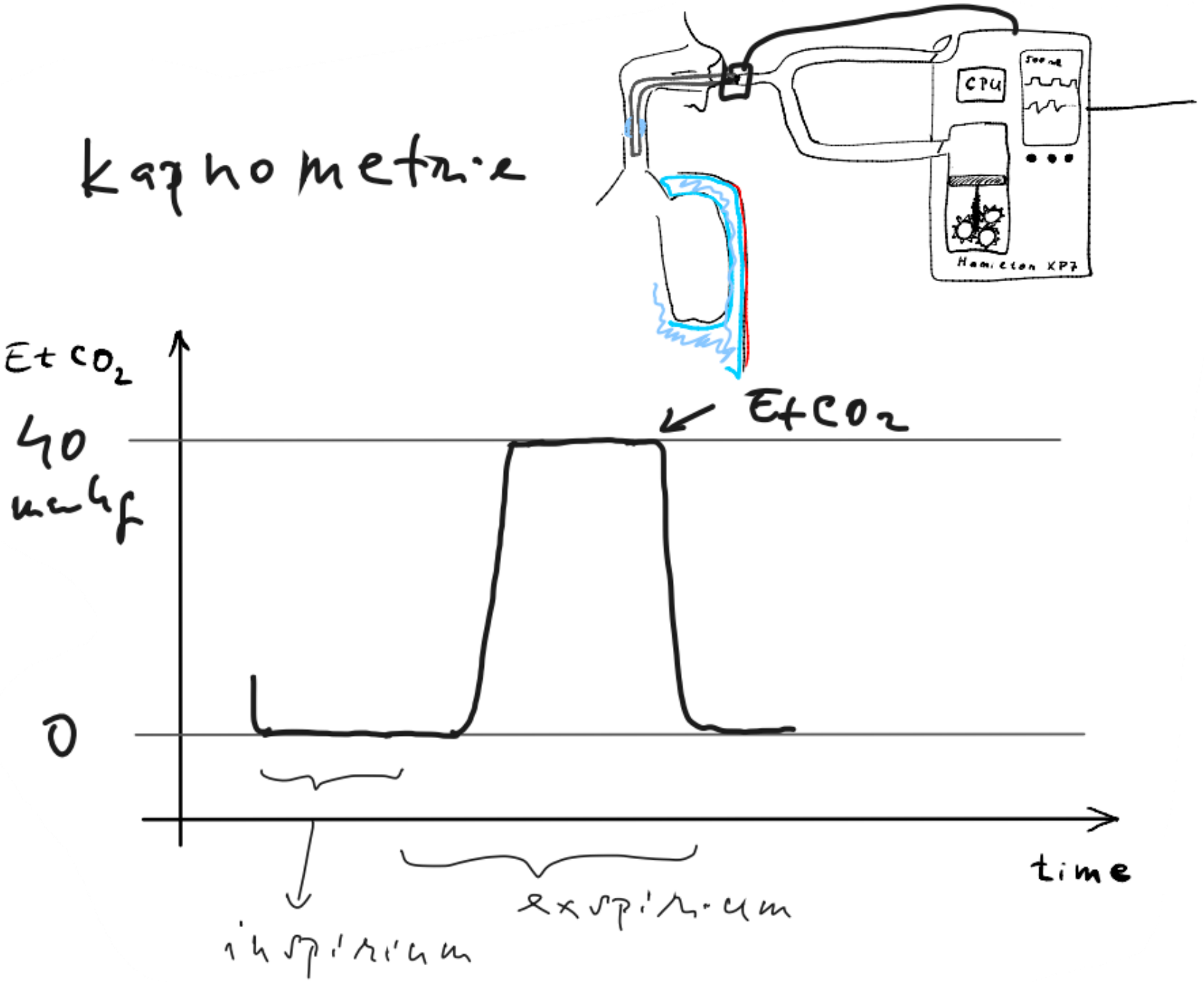
# Capnometry

- monitoring of breathing
- 100 % verification of correct tracheal intubation



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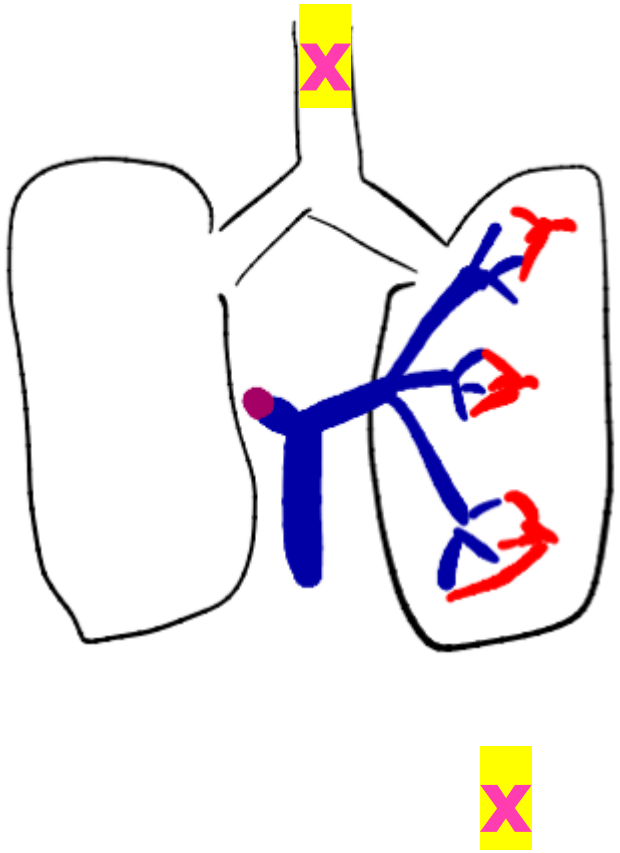
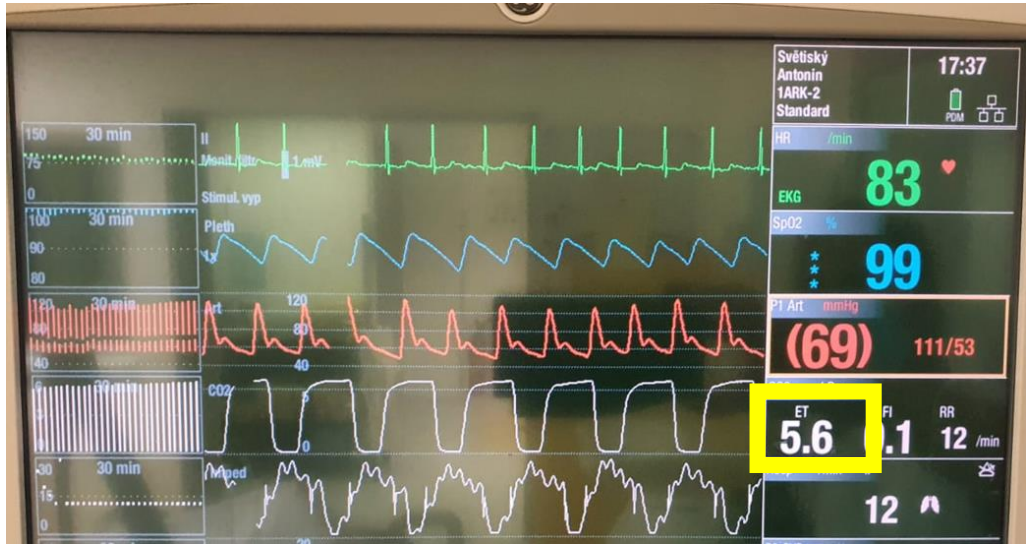
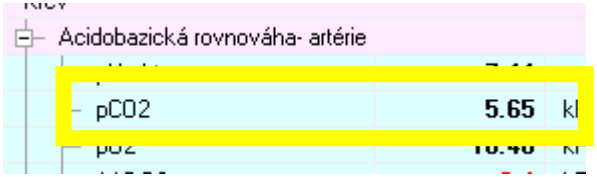
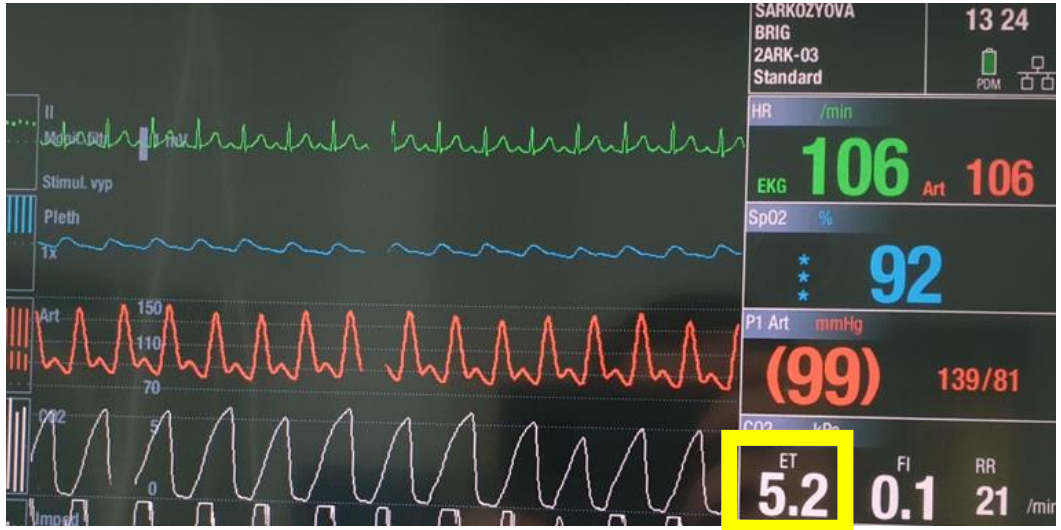
# $PaCO_2$ x $EtCO_2$ = $CO_2$-gap

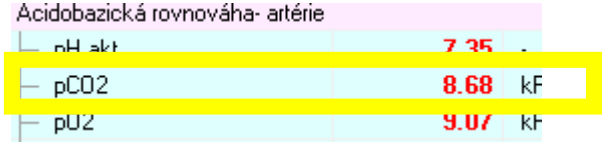
- increased with pulmonary pathology



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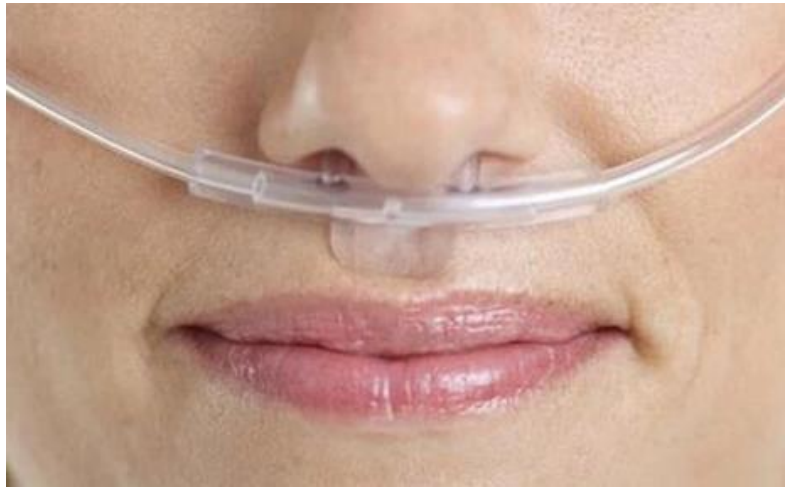
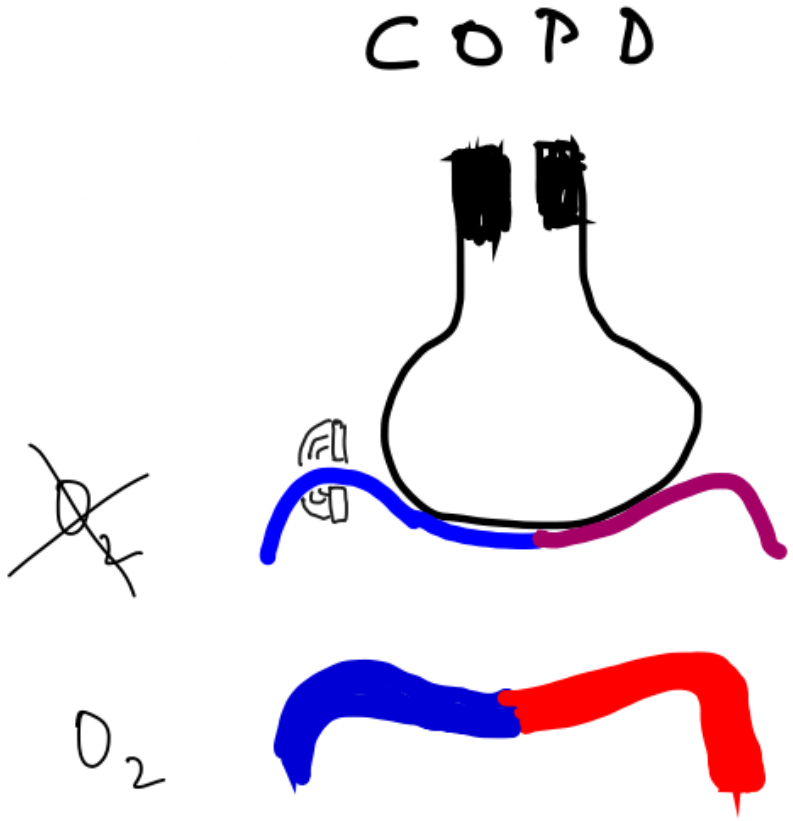


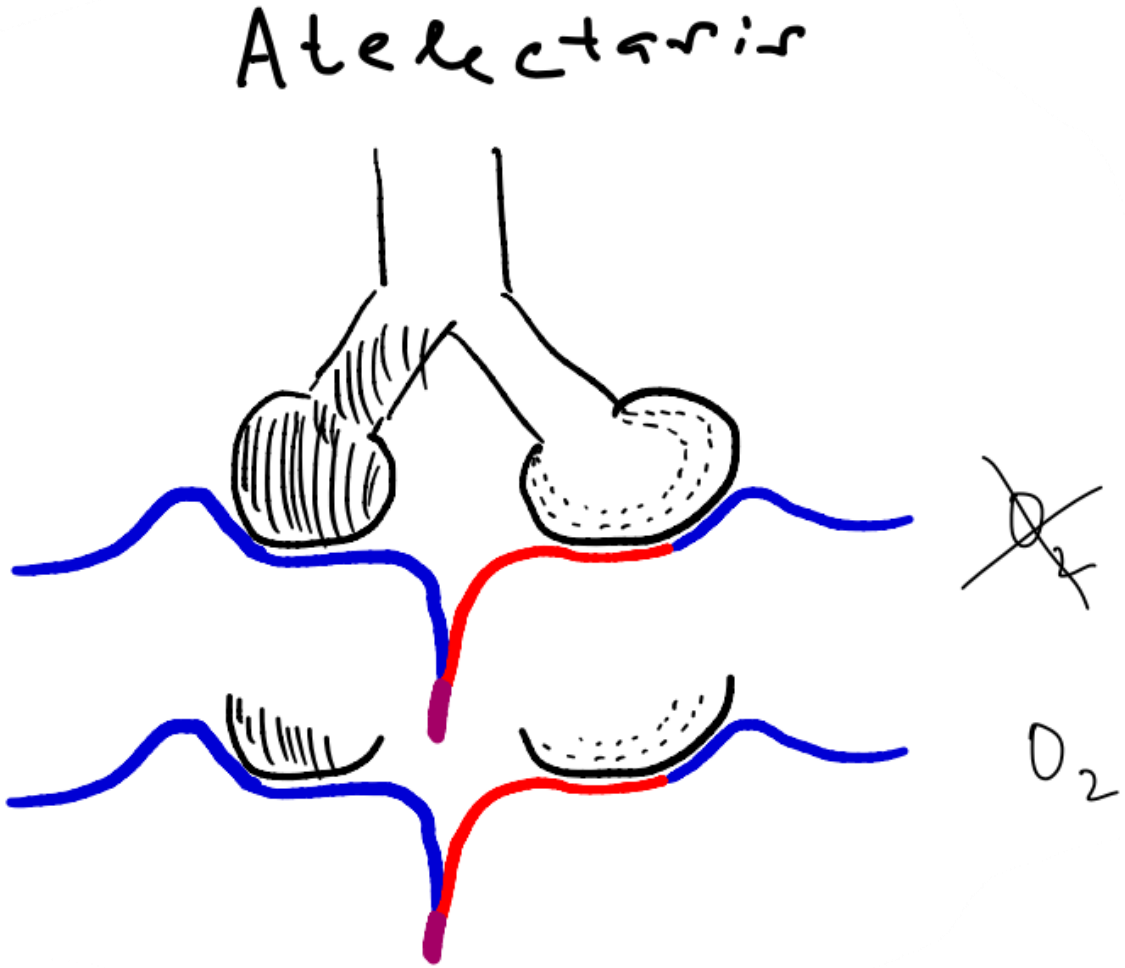
## Oxygen therapy

- 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?



# Principles of mechanical ventilation

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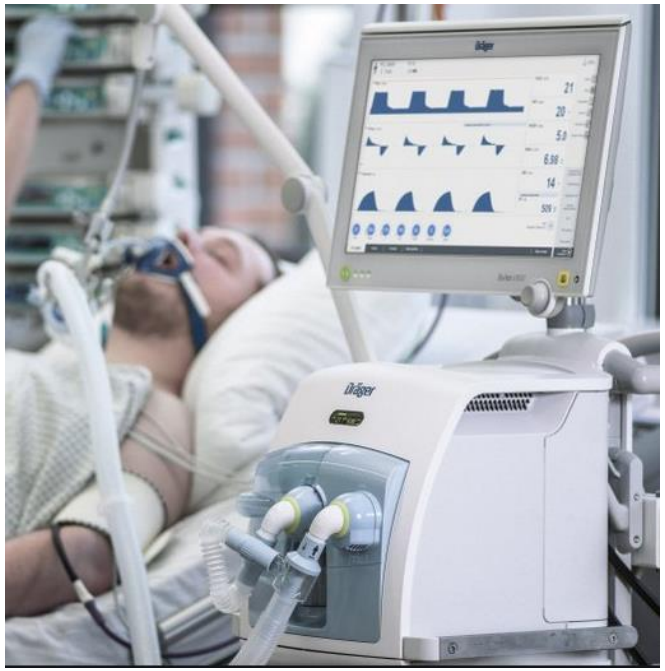
# Mechanical ventilation

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



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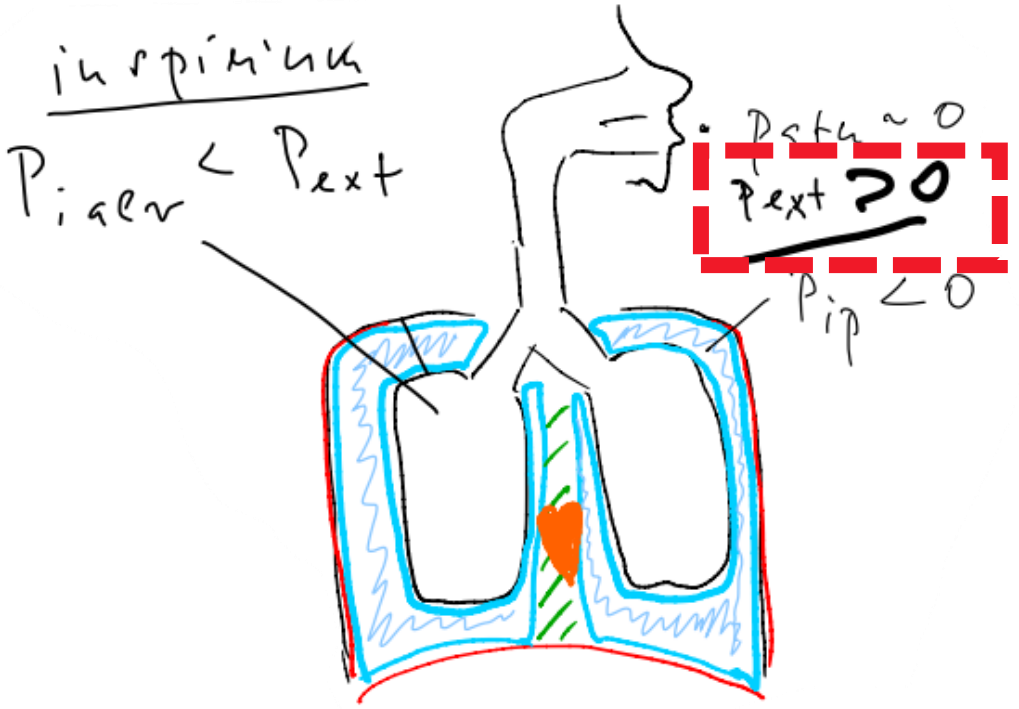
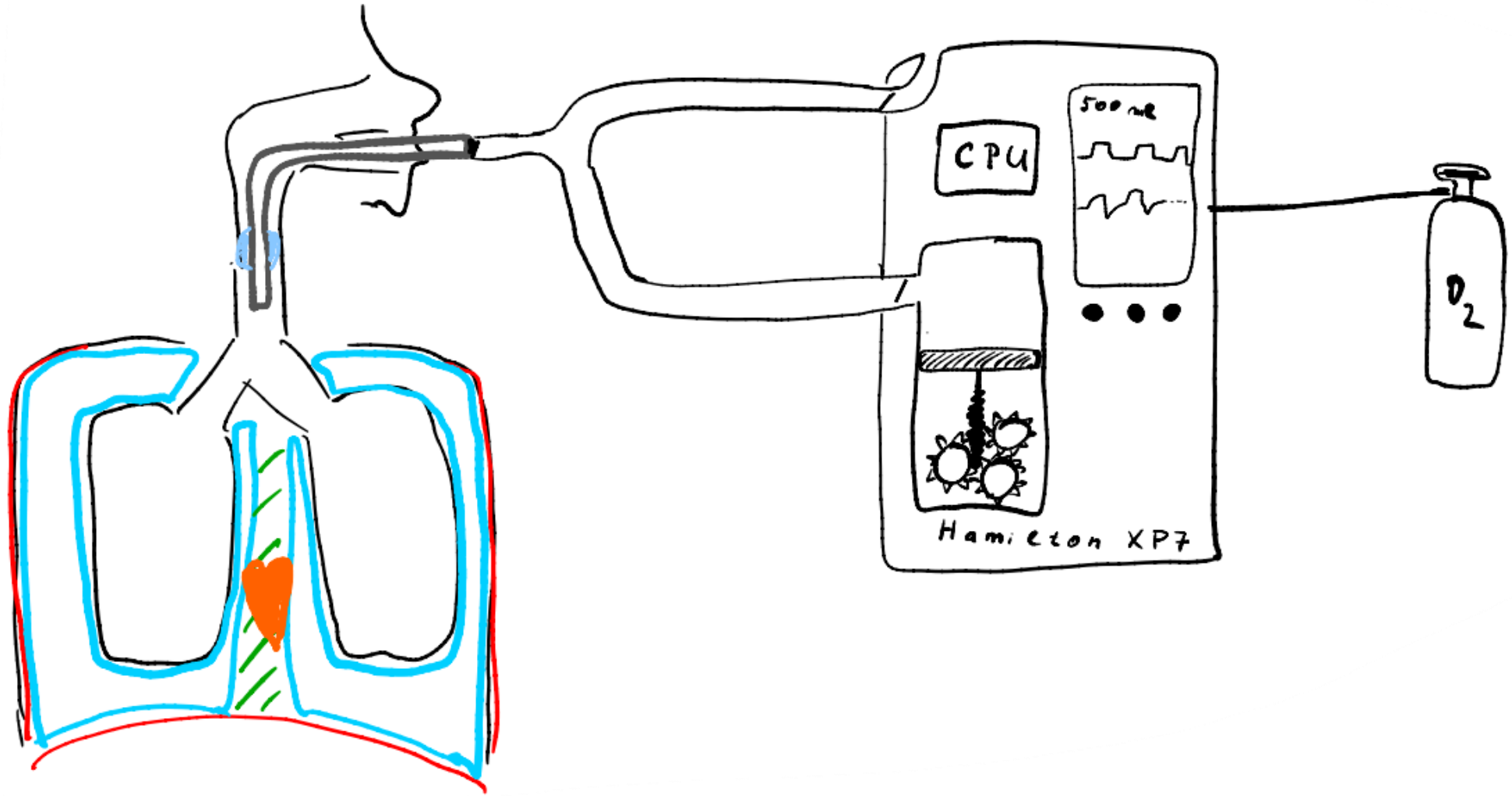
# Principle of mechanical ventilation

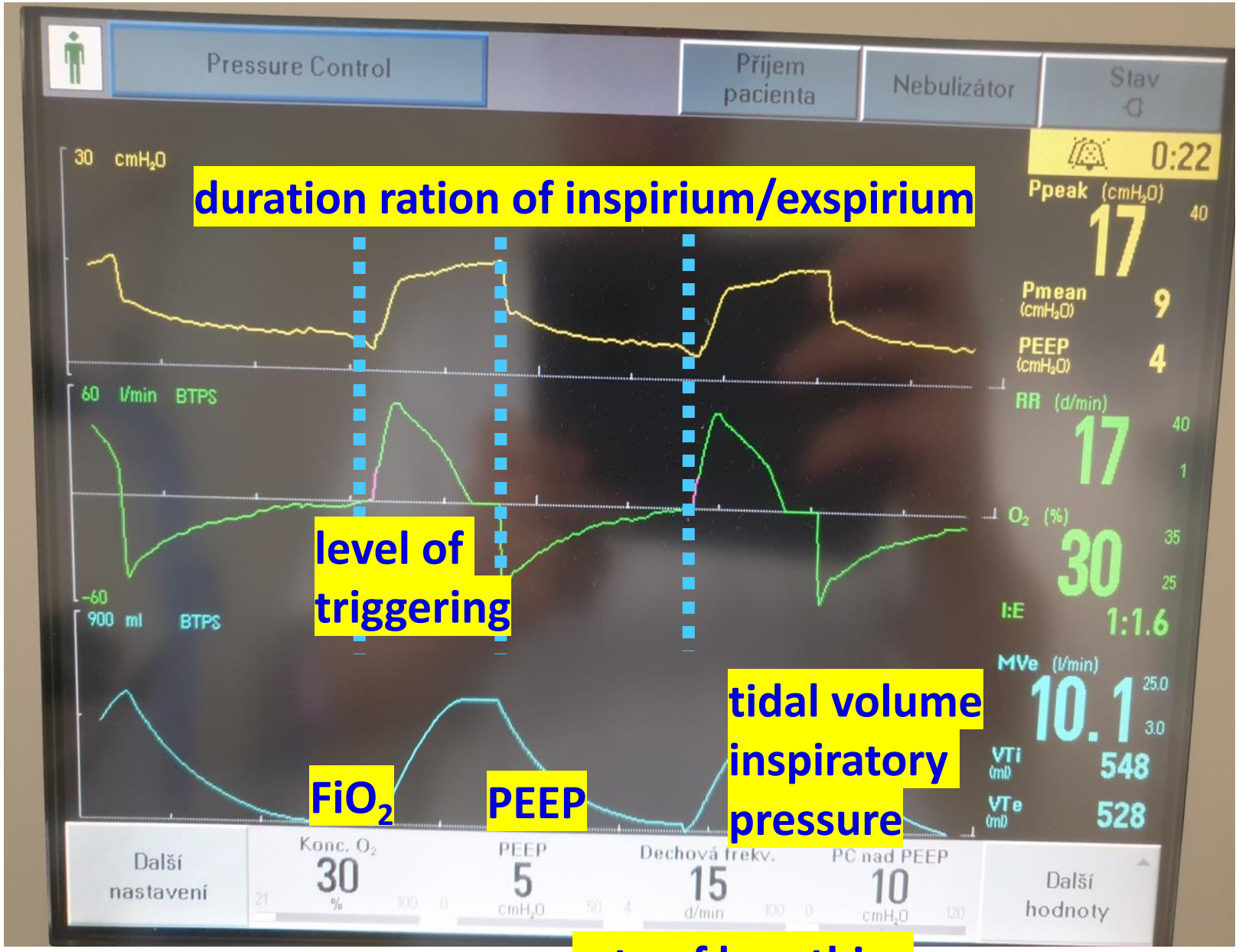



- active inspiration
  - **positive pressure in airways created by ventilator**
- passive expiration
  - just as in spontaneous breathing

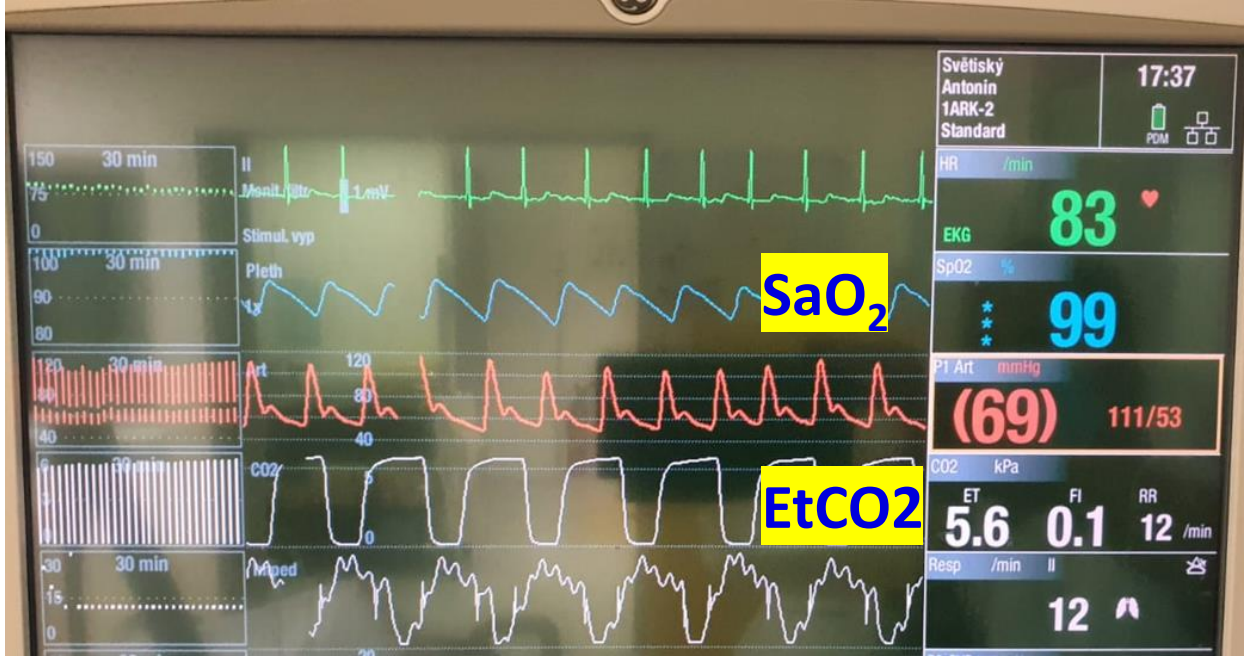
# Principle of mechanical ventilation

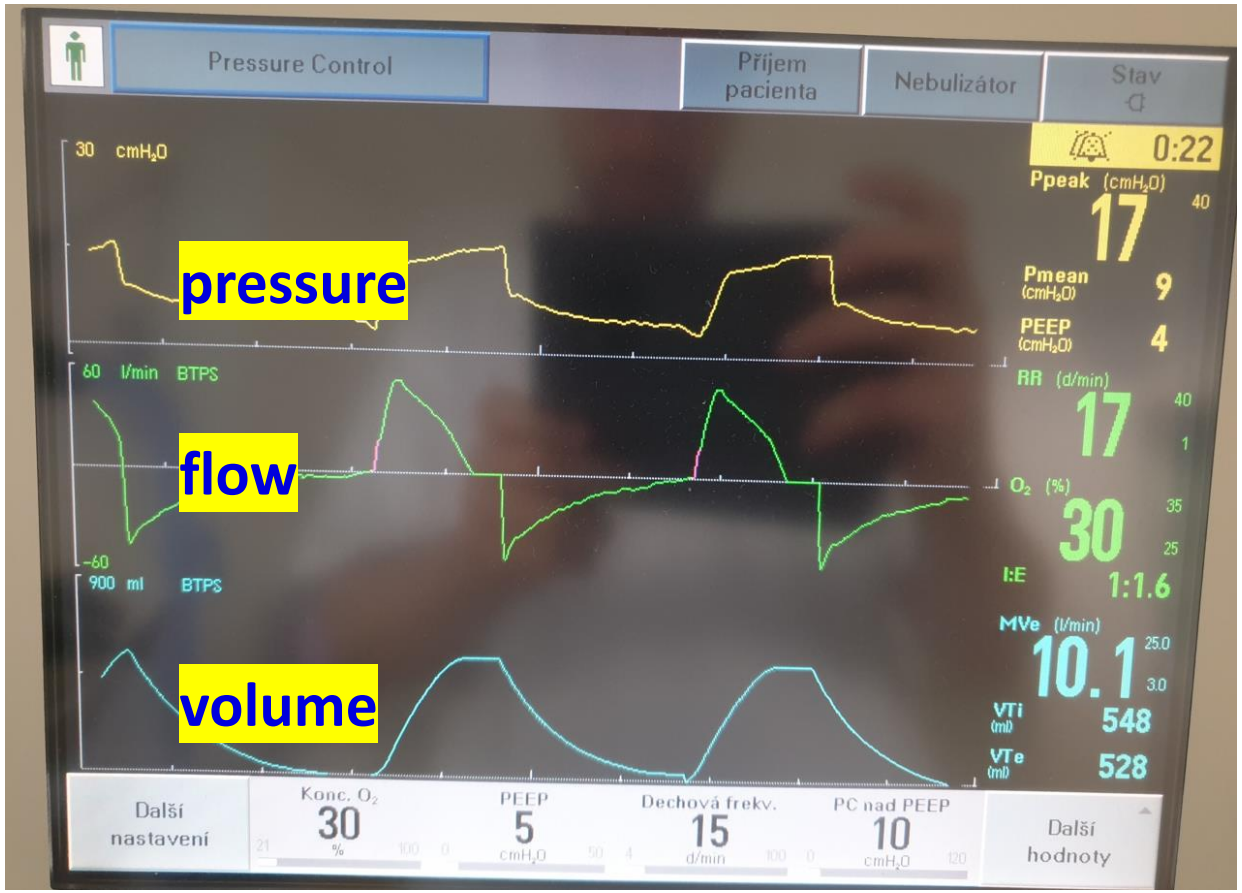


# What can we set on the ventilator?



# What do we monitor?

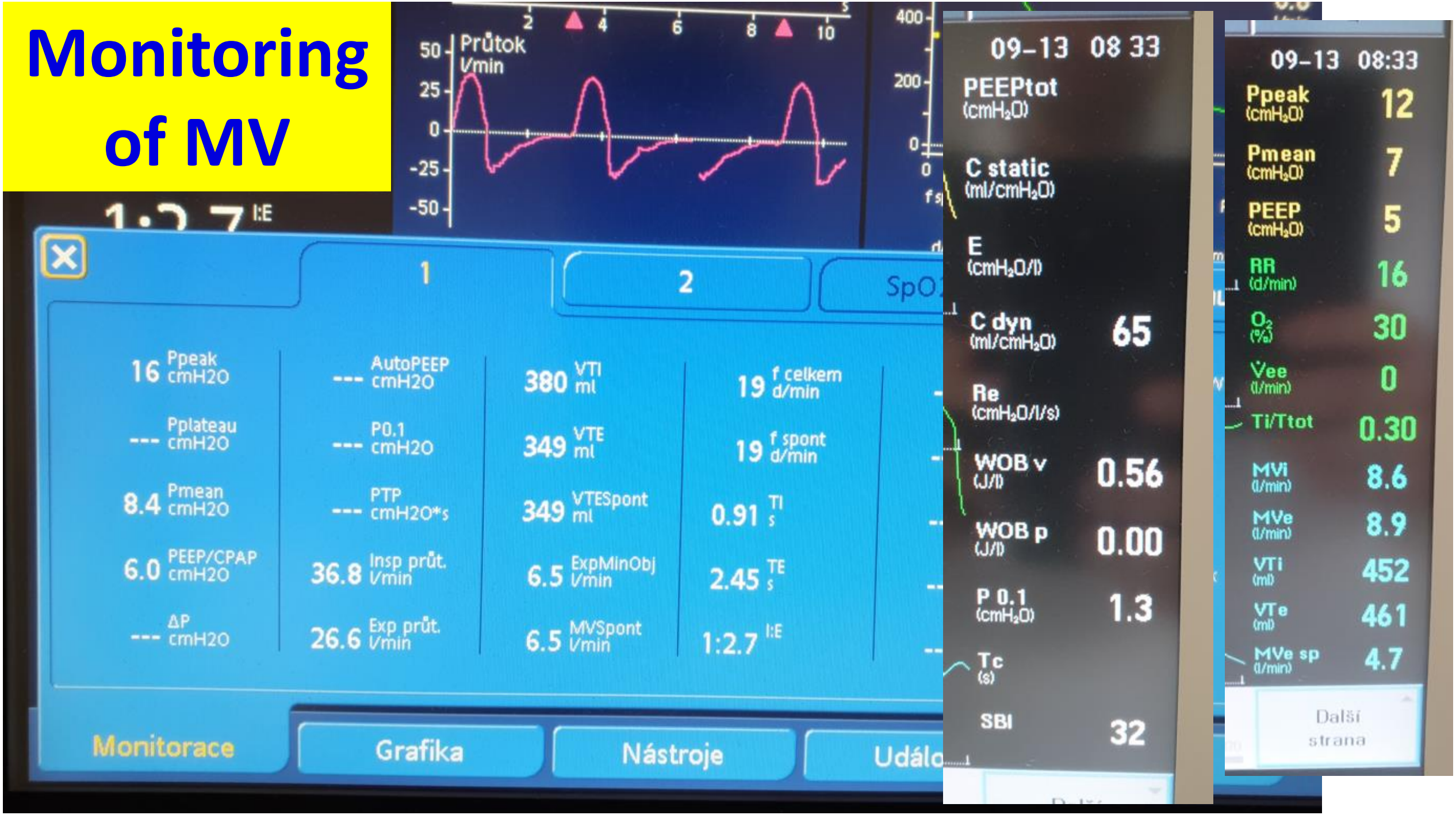



| Krev | | |
|---|---|---|
| Acidobazická rovnováha- artérie | | |
| pH akt. | 7.47 | - |
| pCO2 | 5.06 | kPa |
| pO2 | 10.00 | kPa |
| AADO2 | 12.3 | kPa |
| Base excess | 3.9 | mmol/l |
| Hydrogenkarbonáty aktuální | 27.7 | mmol/l |
| Hydrogenkarbonáty stand... | 27.8 | mmol/l |
| Saturace kyslíkem | 95.9 | % |
| pCO2 - korigované na tepl... | 5.03 | kPa |
| pO2 - korigované na teplotu | 9.97 | kPa |
| Hematokrit | 0.30 | |
| Methemoglobin | 1.0 | % |
| Karbonylhemoglobin | 0.9 | % |
| Hemoglobin | 99 | g/l |
| Acidobazická rovnováha | | |
| Inspirační frakce kyslíku | 30 | % |

**blood gas analysis**

# Monitoring of MV



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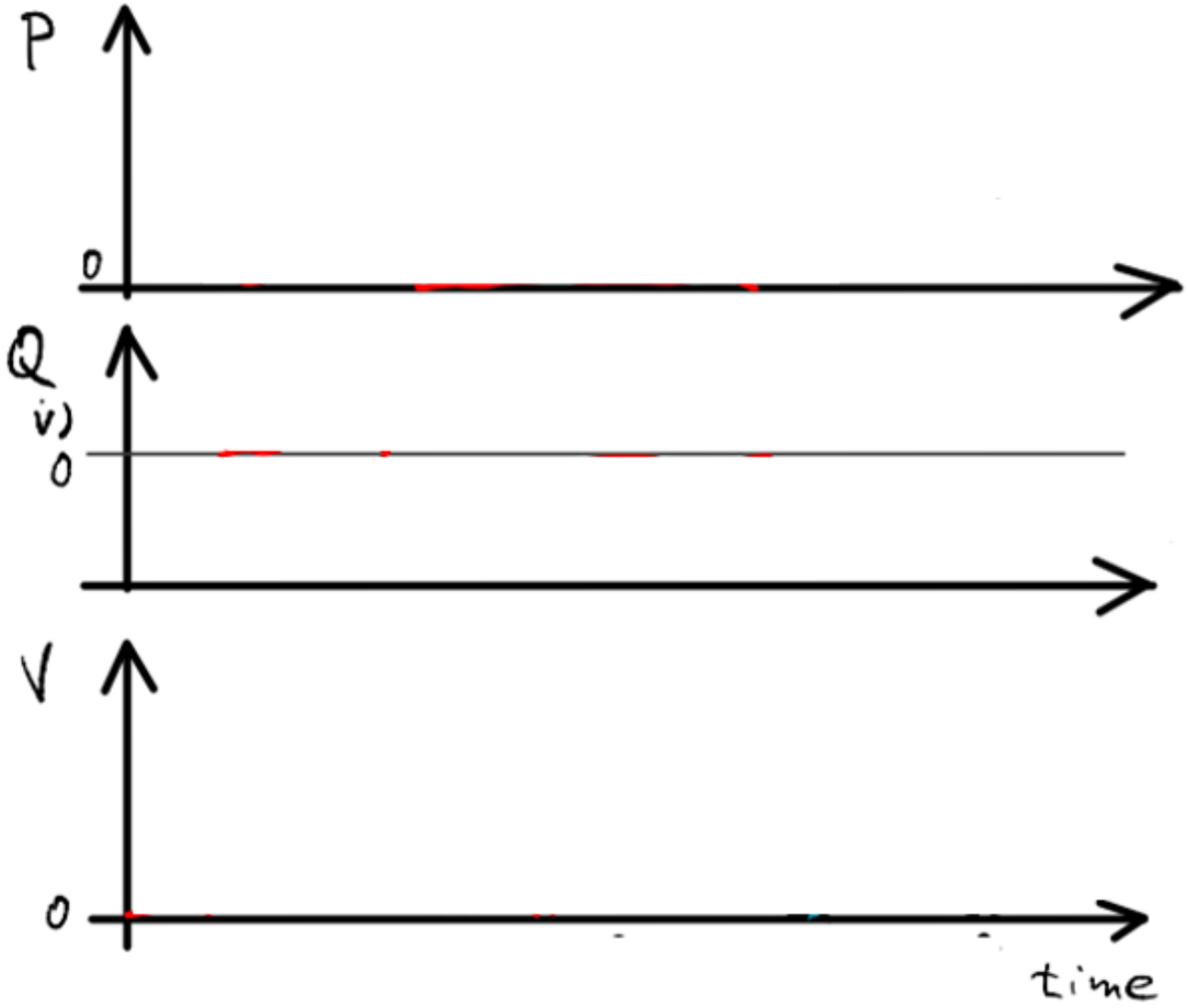
# Analysis of MV curves

- **modes of MV**
  - volume controlled
  - pressure controlled
  - pressure support
  - SIMV
  - …

# volume controlled ventilation (CMV)

- **we set**
  - breath rate
  - tidal volume
  - length of inspirium
  - length of inspiratory pause



# volume controlled ventilation (CMV)

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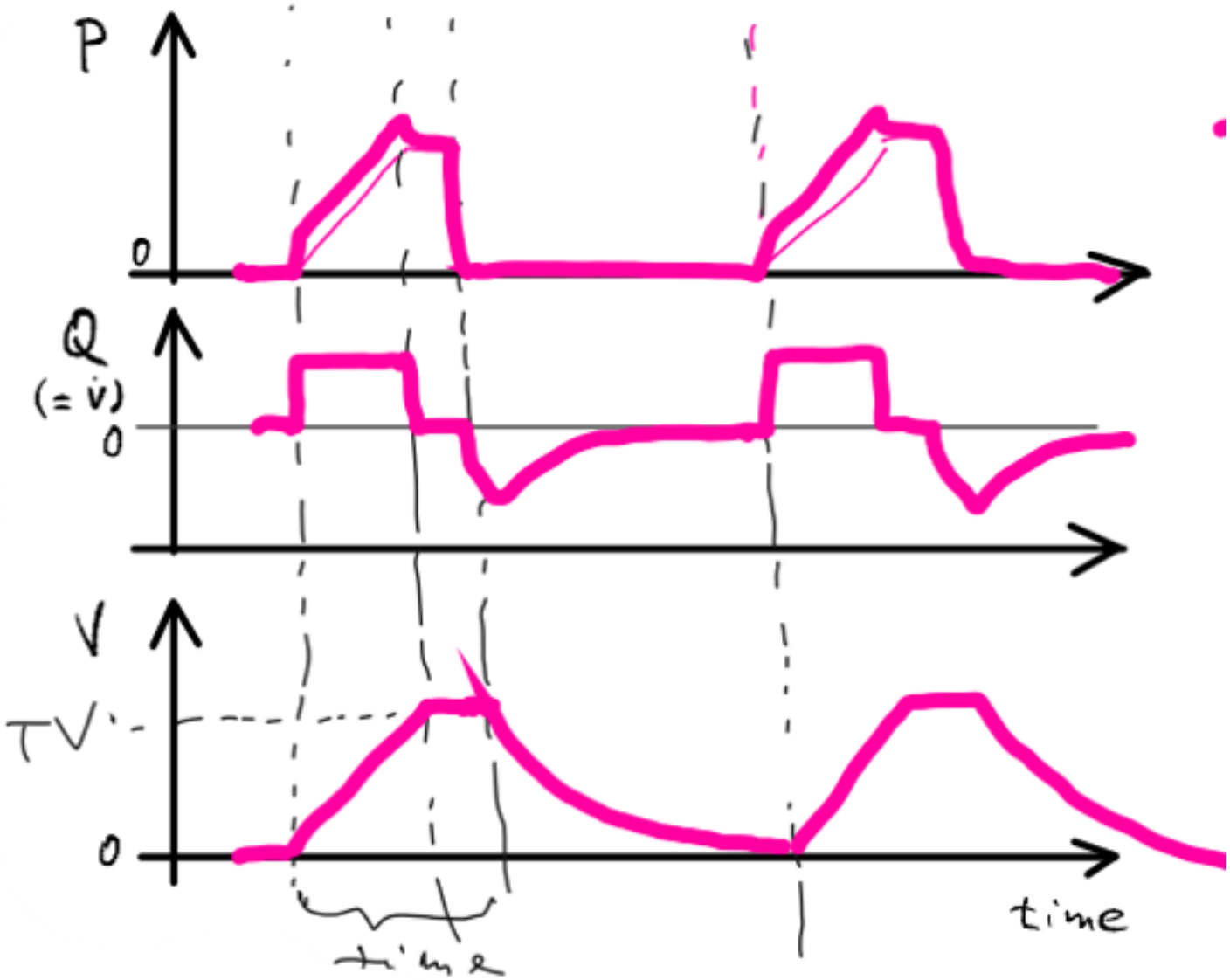
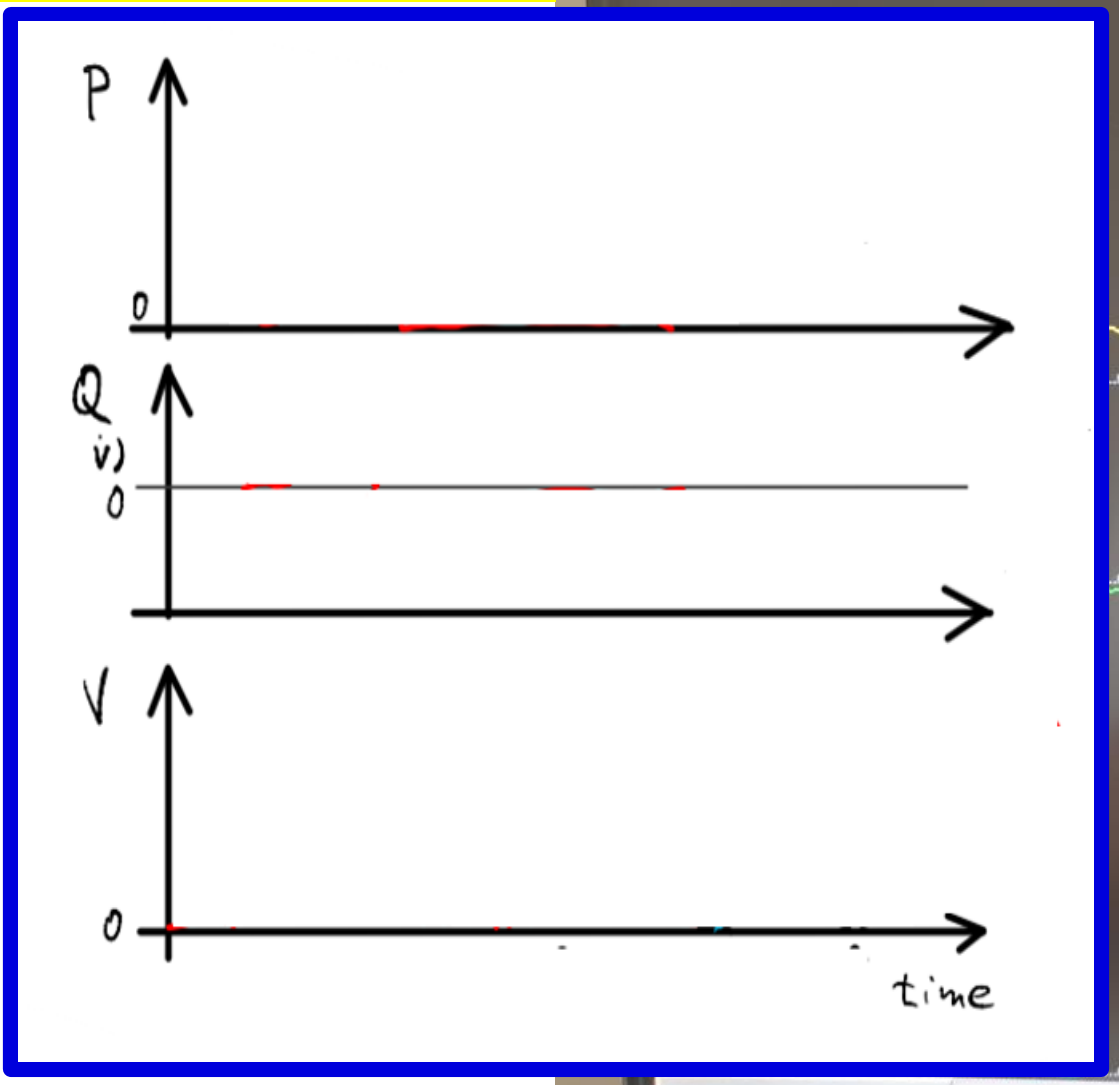
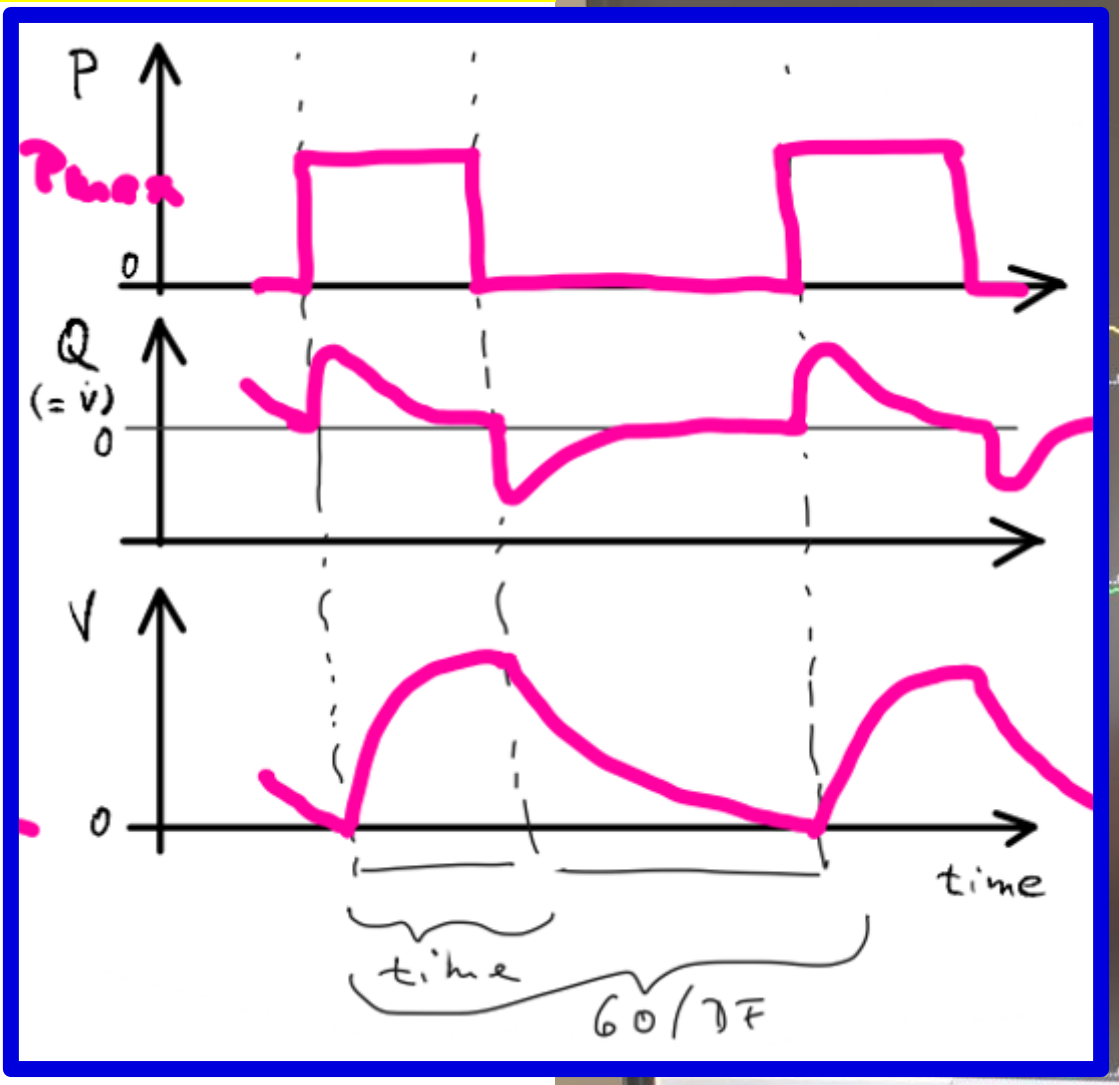


# PCV


P
0
Q
V̇
0
V
0
time

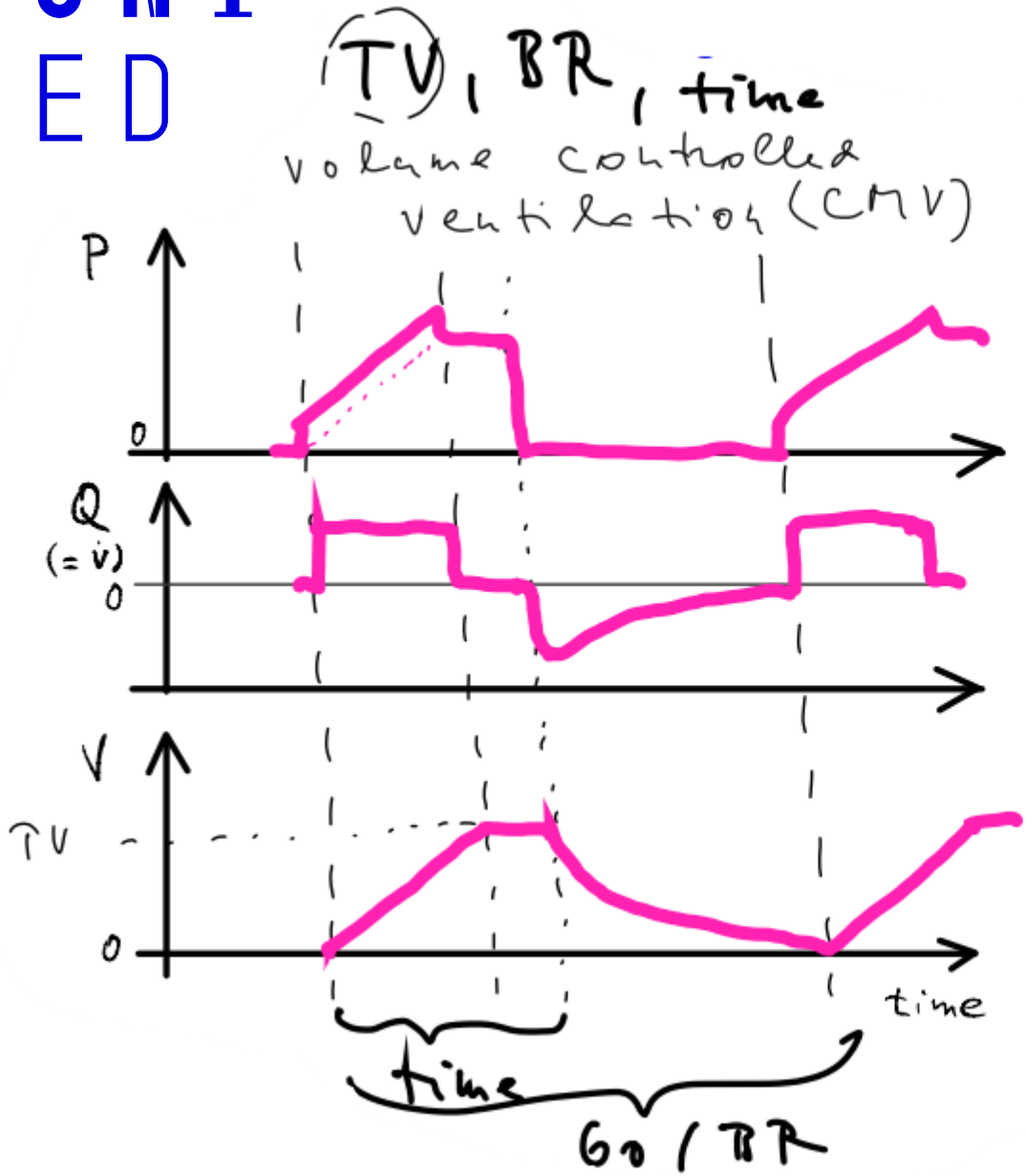

Pressure Control
Příjem pacienta
Nebulizátor
Stav
0:22
Ppeak (cmH2O)
17
40
Pmean (cmH2O)
9
PEEP (cmH2O)
4
RR (d/min)
17
40
1
O2 (%)
30
35
25
I:E
1:1.6
MVe (l/min)
10.1
25.0
3.0
VTi (ml)
548
VTe (ml)
528
Další nastavení
Konc. O2
30
%
PEEP
5
cmH2O
Dechová frekv.
15
d/min
PC nad PEEP
10
cmH2O
Další hodnoty

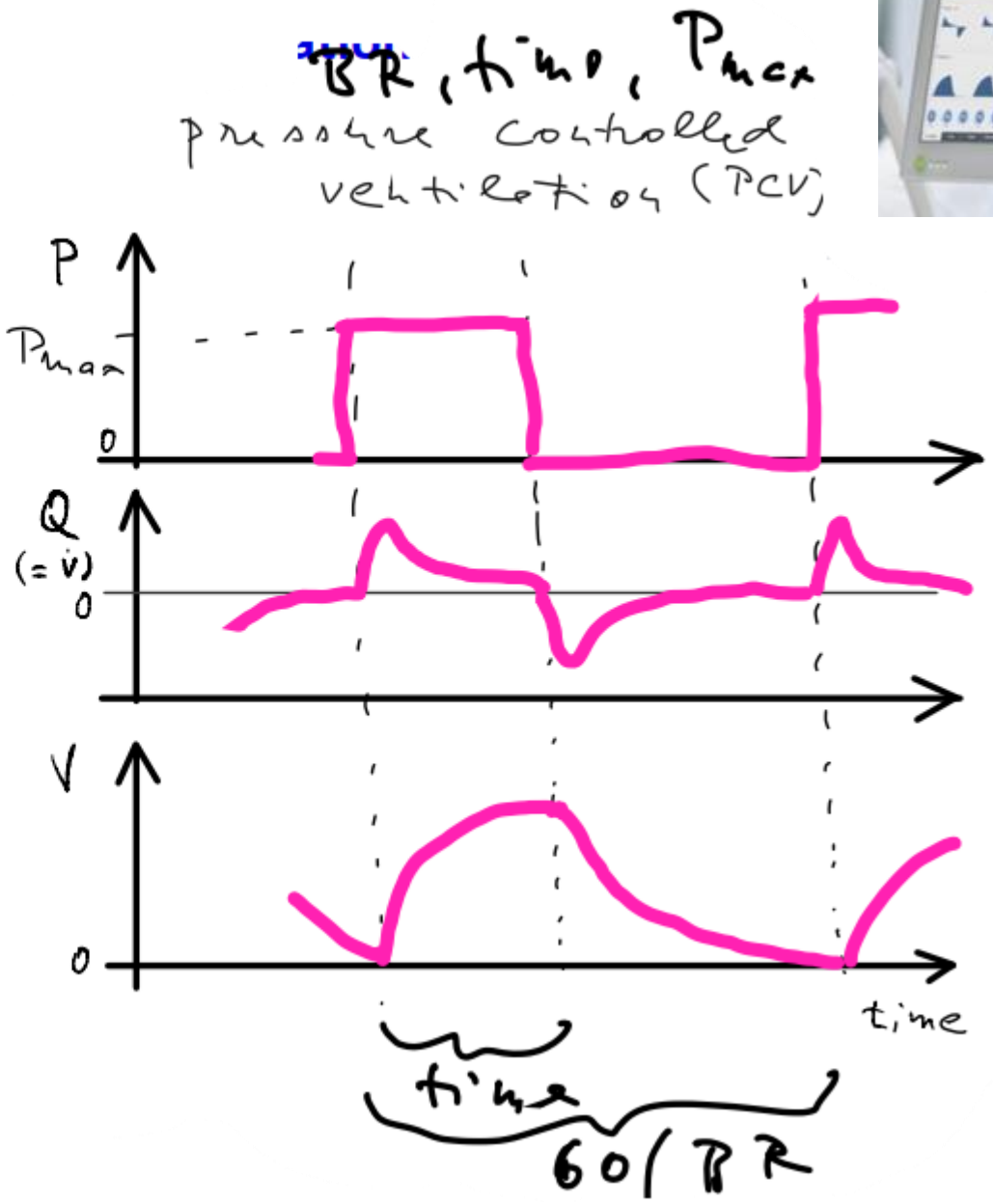
# PCV


P
0
Q
(= V̇)
0
V
0
time
time
60/TF


Pressure Control
Příjem pacienta
Nebulizátor
Stav
0:22
Ppeak (cmH2O)
17
40
Pmean (cmH2O)
9
PEEP (cmH2O)
4
RR (d/min)
17
40
1
O2 (%)
30
35
25
I:E
1:1.6
MVe (l/min)
10.1
25.0
3.0
VTi (ml)
548
VTe (ml)
528
Další nastavení
Konc. O2
30
%
PEEP
5
cmH2O
Dechová frekv.
15
d/min
PC nad PEEP
10
cmH2O
Další hodnoty

# Curves of MV


TV, BR, time
volume controlled ventilation (CMV)
P
0
Q
(= V̇)
0
V
TV
0
time
time
60 / BR


BR, time, Pmax
pressure controlled ventilation (PCV)
P
Pmax
0
Q
(= V̇)
0
V
0
time
time
60 / BR

# CPAP/PSV


PS/CPAP
Příjem pacienta
Nebulizátor
Stav
09–13 08 32
Ppeak (cmH2O)
16
40
Pmean (cmH2O)
8
PEEP (cmH2O)
5
RR (d/min)
15
40
1
O2 (%)
30
35
25
Ti/Ttot
0.24
MVe (l/min)
8.8
25.0
3.0
VTi (ml)
654
VTe (ml)
677
PS/CPAP
Základní
I:E
Trigger
PS nad PEEP
10
0
cmH2O
120
T insp. náběhu
0.15
0.00
s
0.40
Trigg. – průtok
1
-20
0
PEEP
5
0
cmH2O
50
Ukončení insp.
30
1
%
70
Konc. O2
30
21
%
100
Další nastavení
Konc. O2
30
21
%
100
PEEP
5
0
cmH2O
50
PS nad PEEP
10
0
cmH2O
120
Další hodnoty

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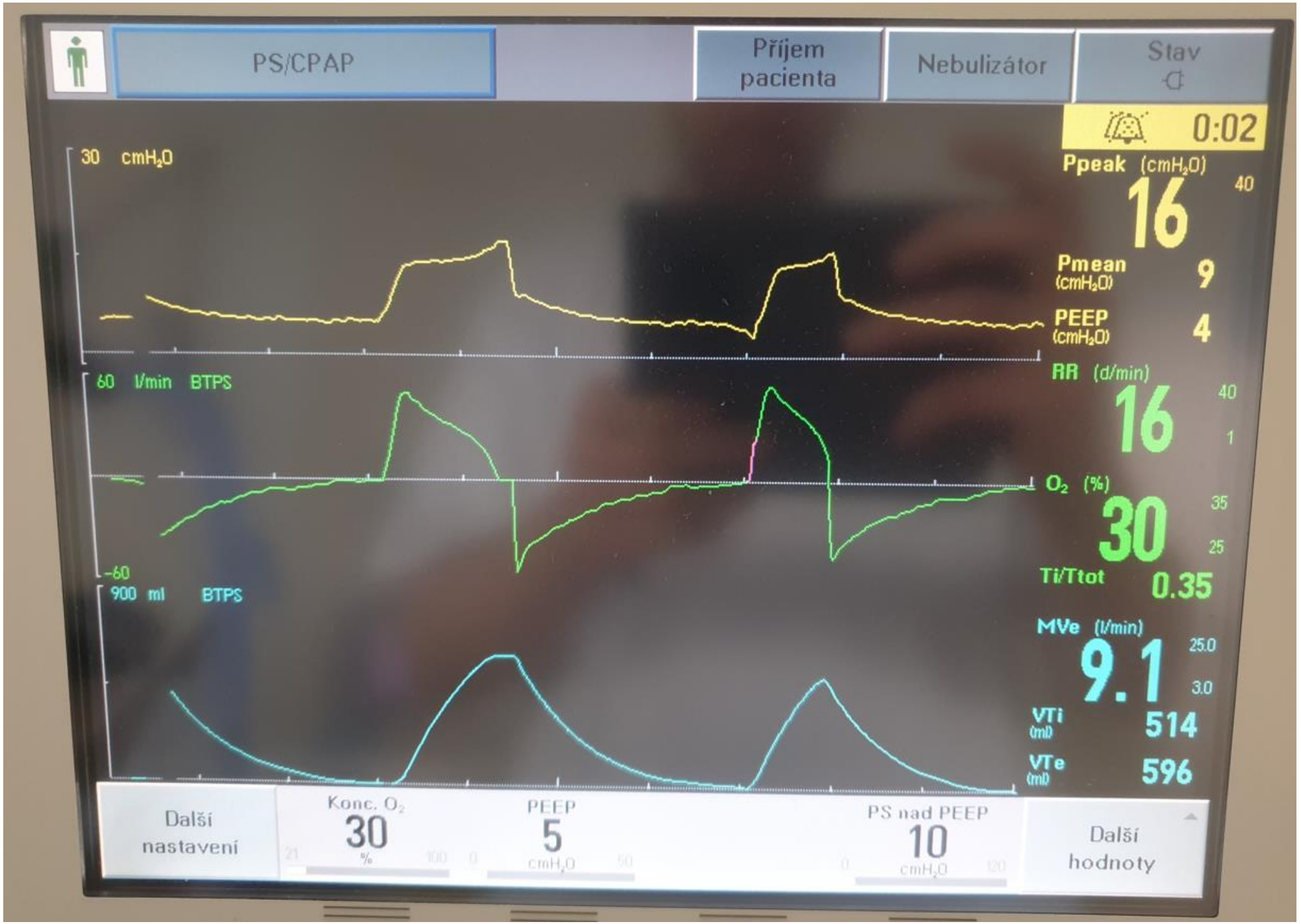
# PCV versus PSV



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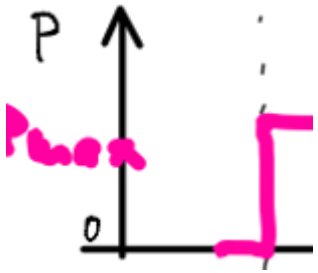


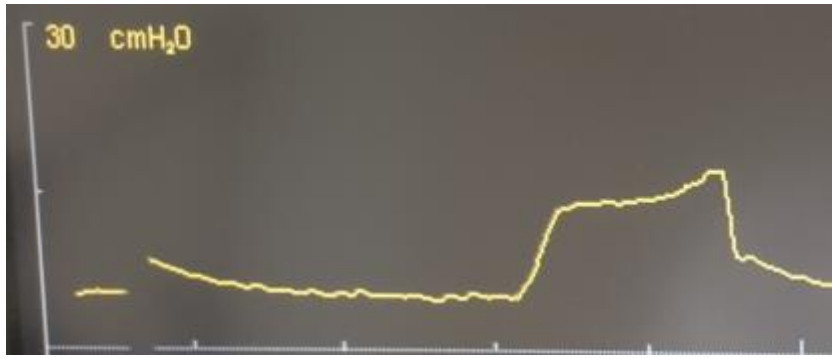


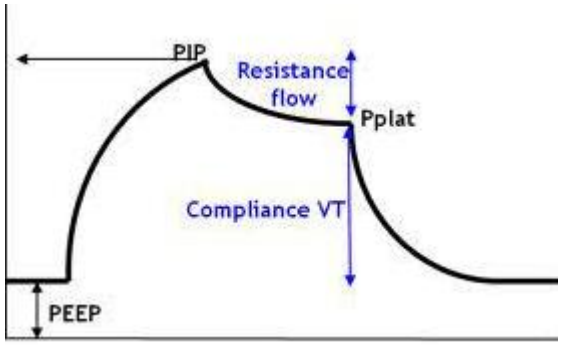


# PEEP (positive end-expiratory pressure)

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

- both too low and too high values are harmful
  - ➢ it is necessary to find the optimal value

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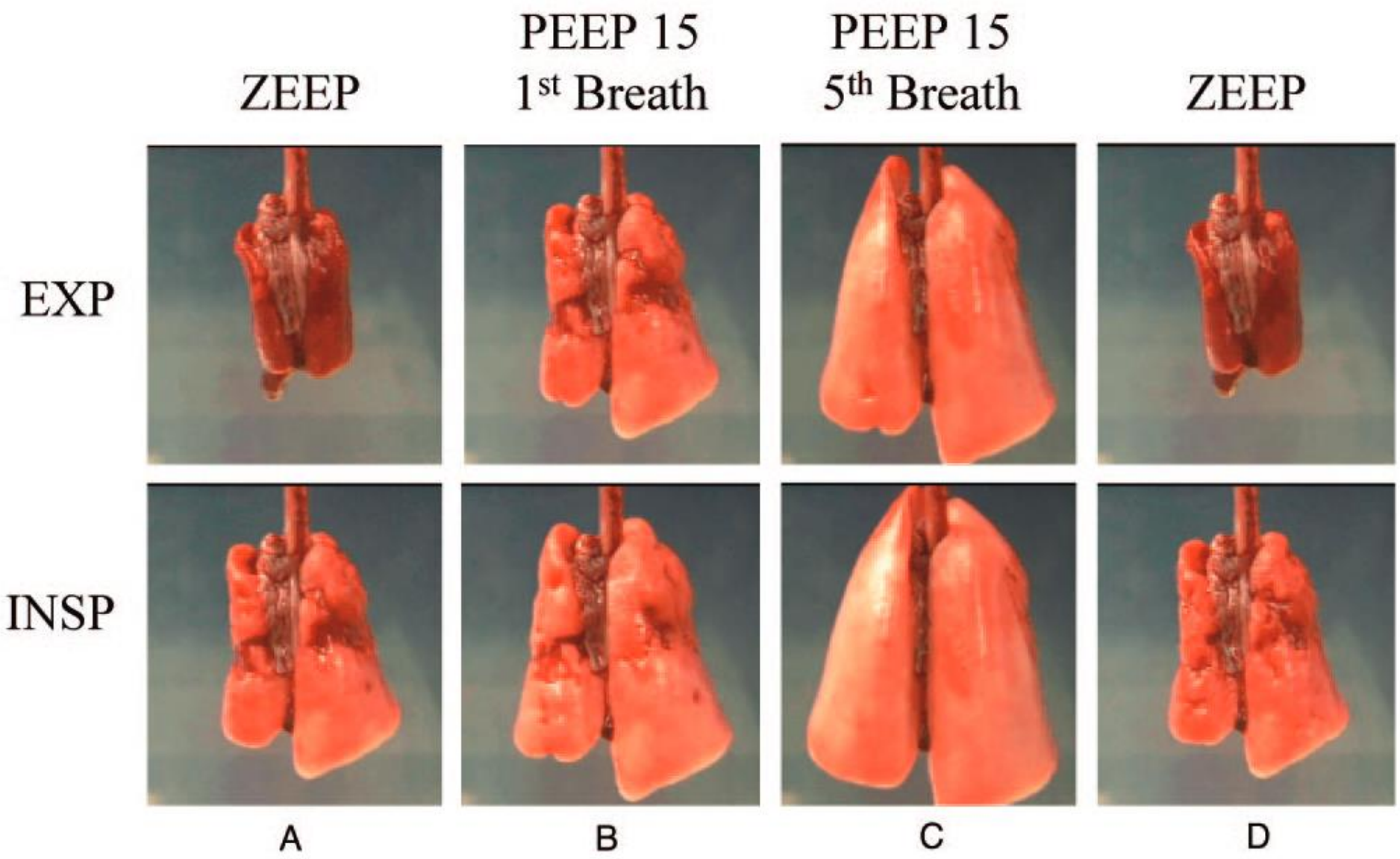
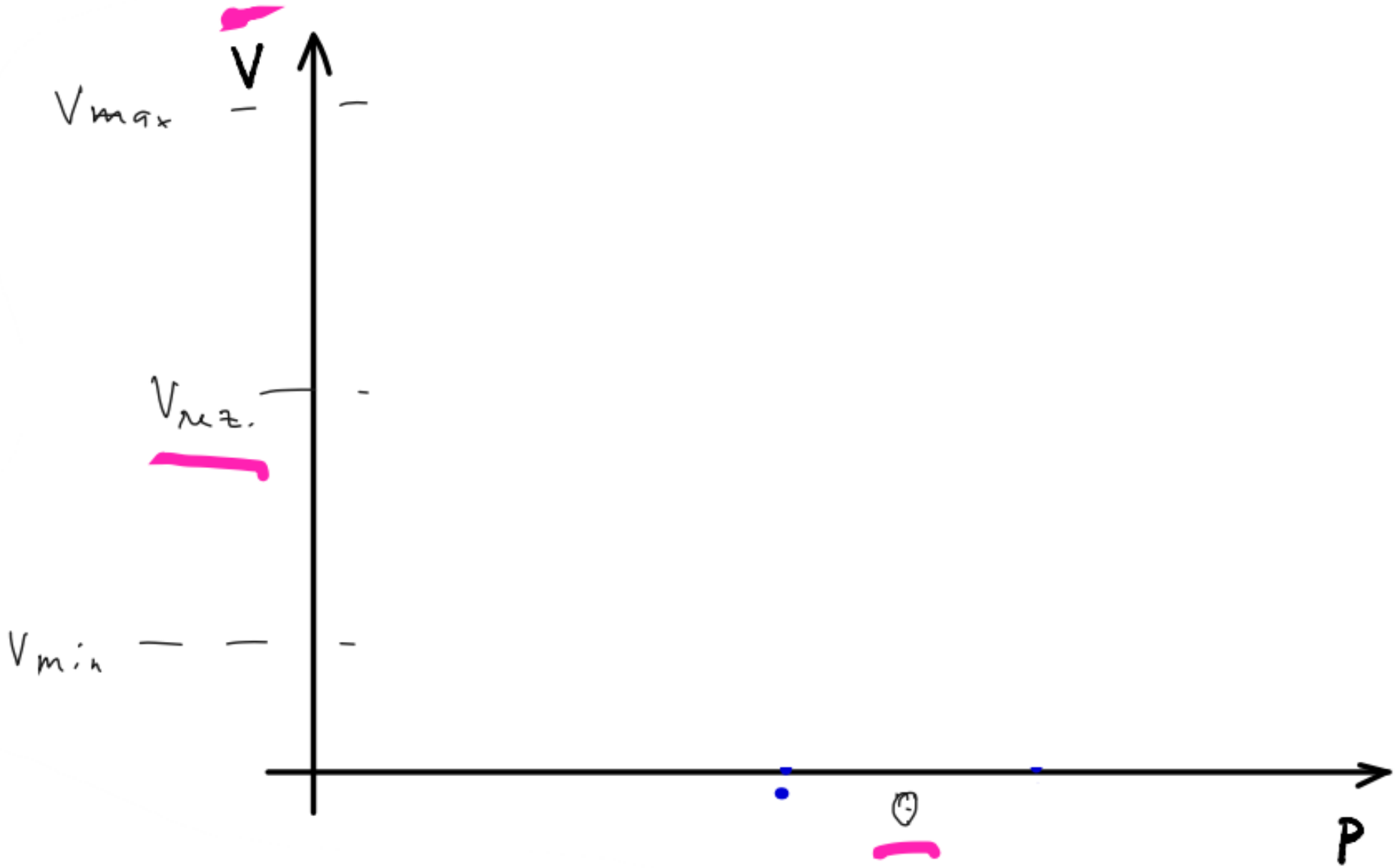
# Influence of PEEP on lung aeration



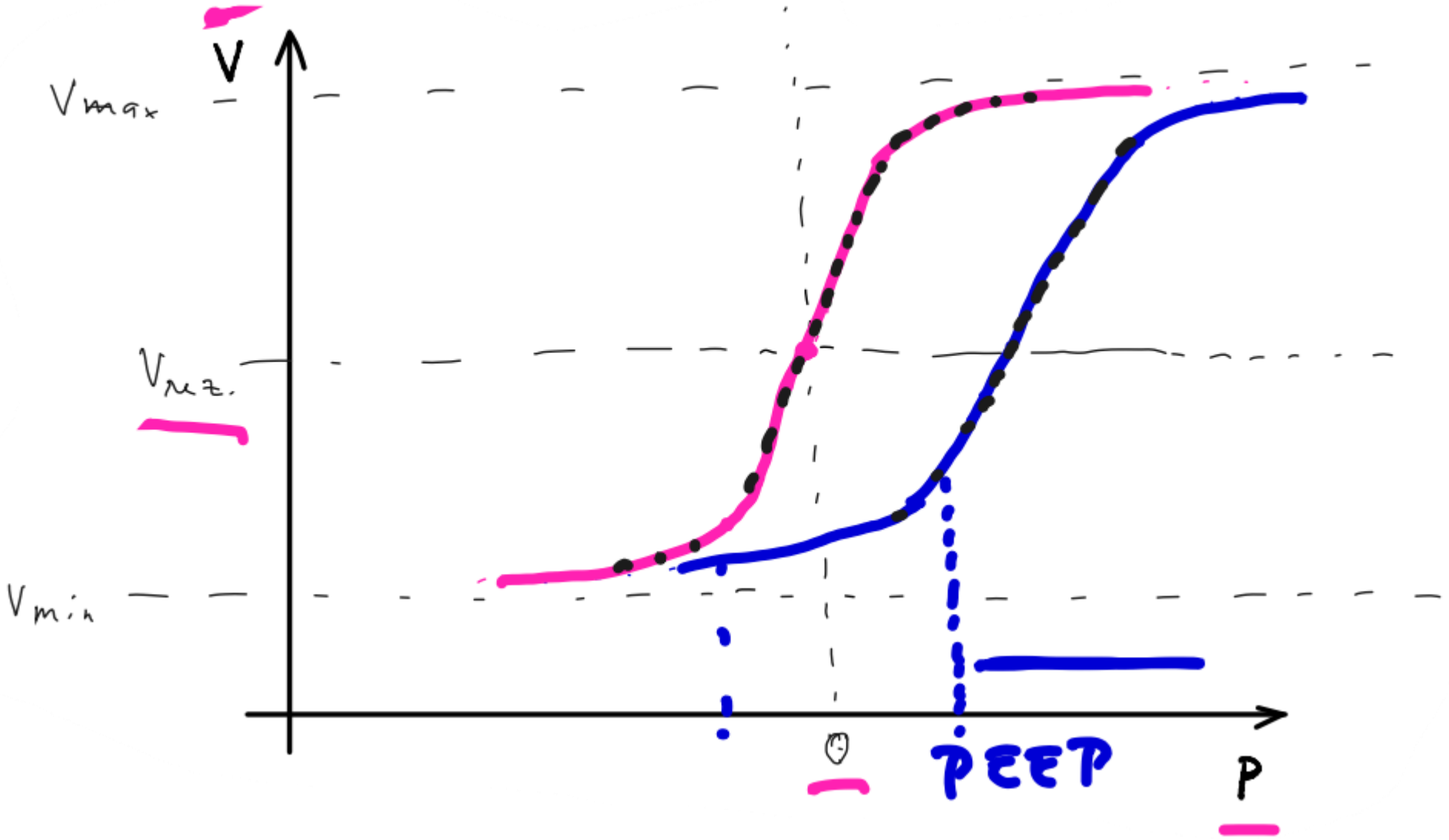



RESPIRATORY CARE • MAY 2005 VOL 50 NO 5

# p-V curve and PEEP



# p-V curve and PEEP



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# Effects of MV on other systems

## 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 ($CO_2$)
- decreases renal blood flow and so promotes fluid retention
- increases intraabdominal pressure and reduces splanchnic perfusion
- can increase intracranial pressure
- „motor“ of multiorgan failure

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# Ventilator induced lung injury (VILI)

!!! inappropriate ventilation can damage lungs

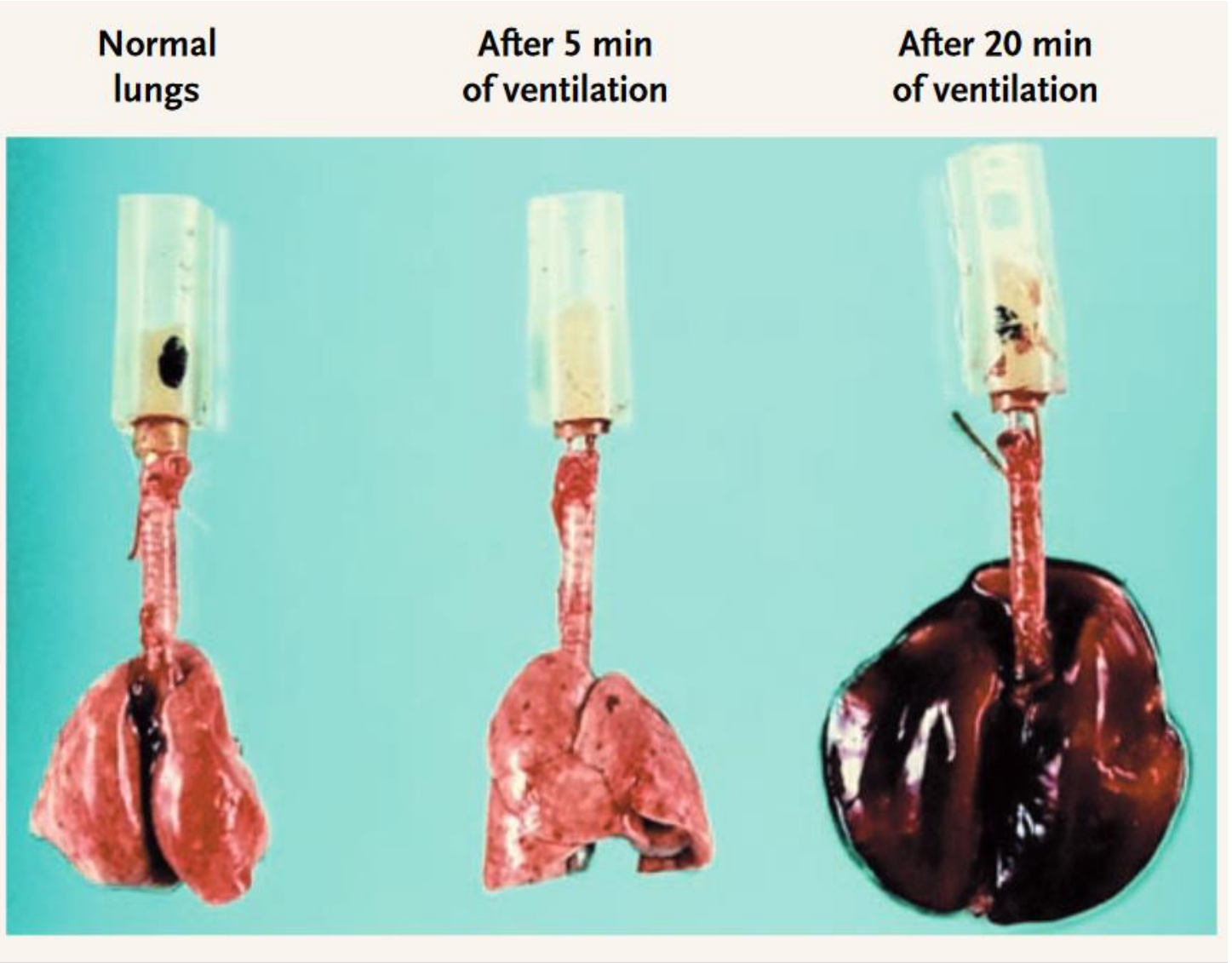


Figure 1. Normal Rat Lungs and Rat Lungs after Receiving High-Pressure Mechanical Ventilation at a Peak Airway Pressure of 45 cm of Water.

6. Webb HH, Tierney DF: **Experimental pulmonary edema due to intermittent positive pressure ventilation with high inflation pressures. Protection by positive end-expiratory pressure.** *Am Rev Respir Dis* 1974, **110:** 556–565.

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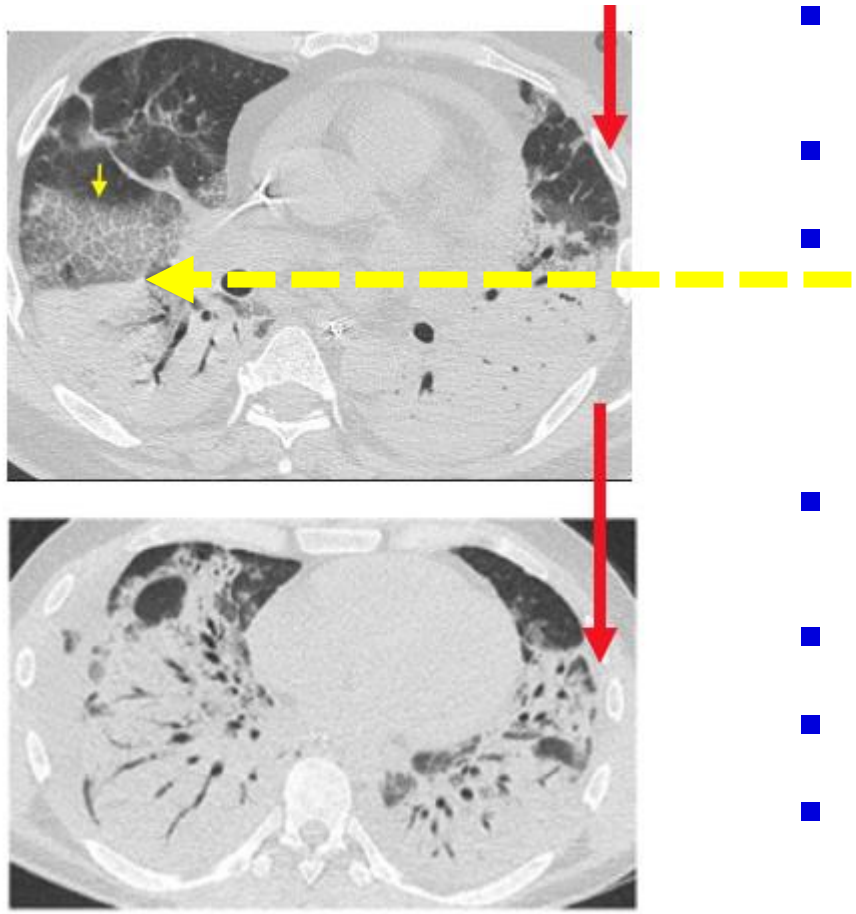
# How can MV damage lungs?



- large distension tears pulmonary structures
  - econdary inflammatory reaction and fibrosis
- danger of lung perforation in thin areas -> pneumothorax
- shear forces on the boundary of ventilated and non-ventilated areas of lungs

- elimination of natural immune barriers
  - ventilator-associated pneumonia
- risks of intubation and airway management
- promotion of muscle weakness of critically ill patients
- necessary sedation

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# Non-invasive ventilation

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

# PSV at NIV


MAQUET v.č.35641
HM000015820
(35641)
Ventilátor plicní Servo-s
NIV PS
Příjem pacienta
Stav
30 cmH2O
19/09 13:25
Ppeak (cmH2O) 18
Pmean (cmH2O) 10
PEEP (cmH2O) 8
70 l/min BTPS Komp.
RR (d/min) 21
O2 (%) 41
Ti/Ttot 0,23
-70
800 ml BTPS Komp.
MVi (l/min) 11,8
MVe (l/min) 11,8
VTi (ml) 613
VTe (ml) 510
Únik (%) 20
Další nastavení
Konc. O2 40 %
PEEP 8 cmH2O
PS nad PEEP 10 cmH2O
Další strana
Profil alarmu
Trendy
Rychlý přístup
Menu
Hlavní obraz.

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# Administration of prostacyclin

- prostacyclin causes dilatation of pulmonary arterioles and thus reduces pulmonary hypertension
- can be inhaled or administered intravenously
- in patients with respiratory insufficiency, just one of both routes of administration improves respiratory insufficiency

**which one and why?**

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# Applied pathophysiology of MV in clinical cases

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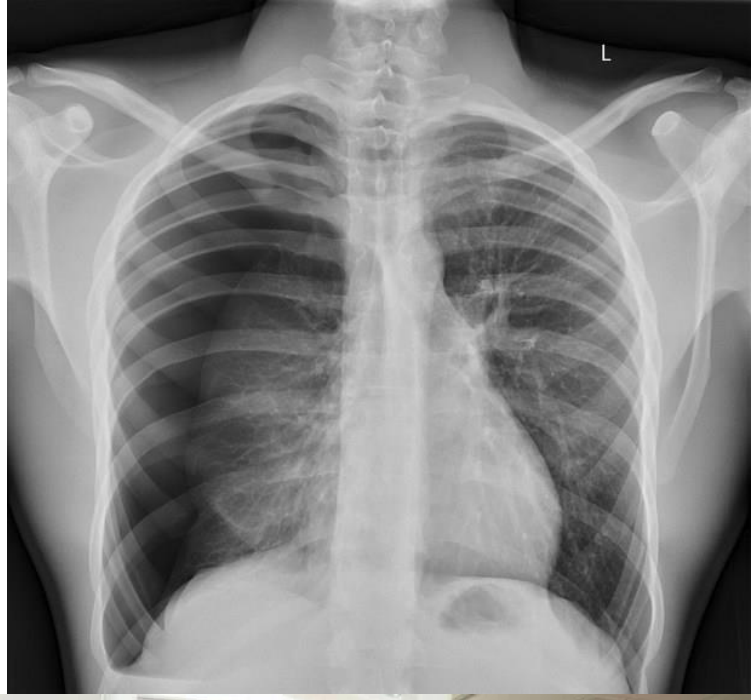
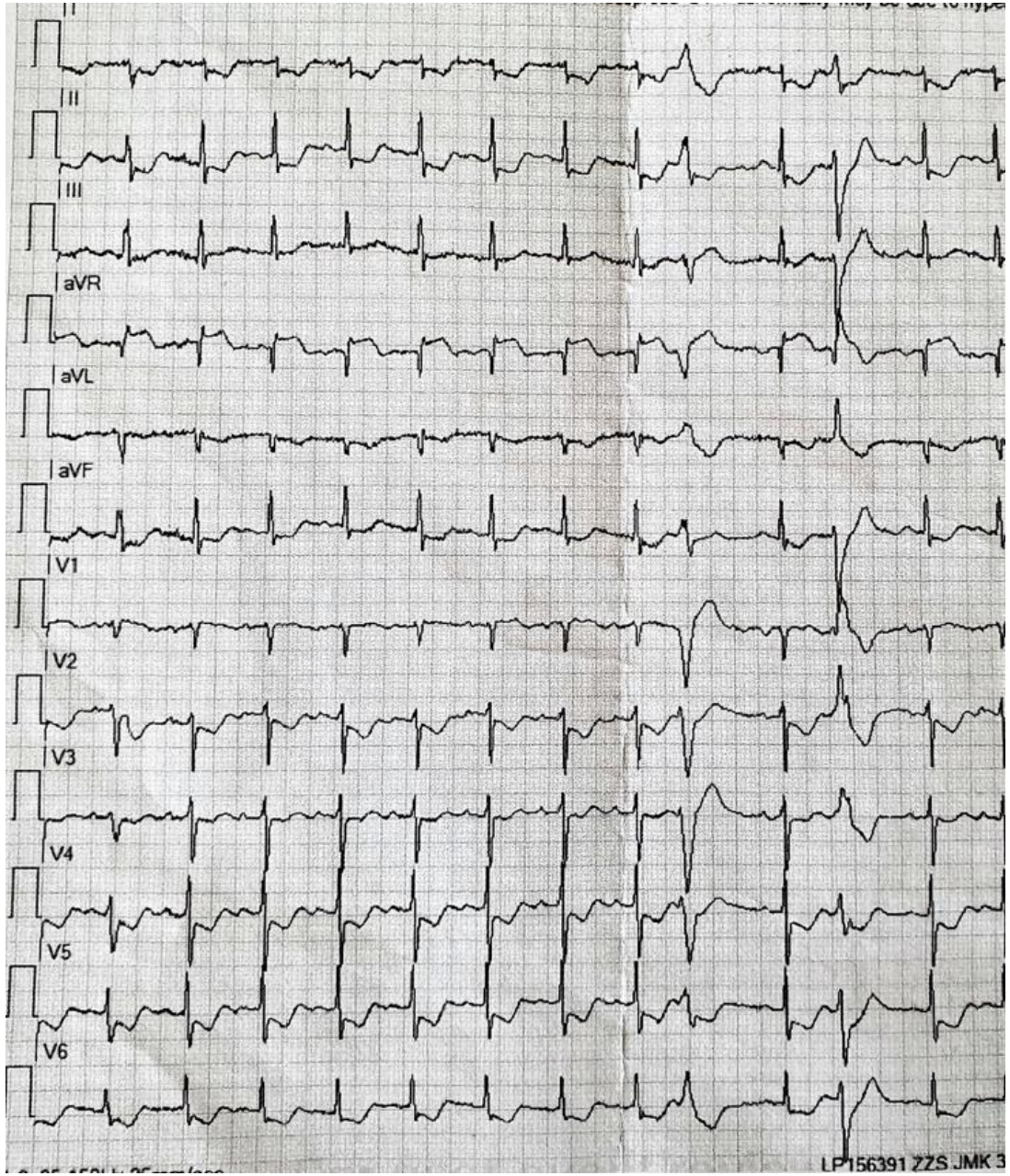
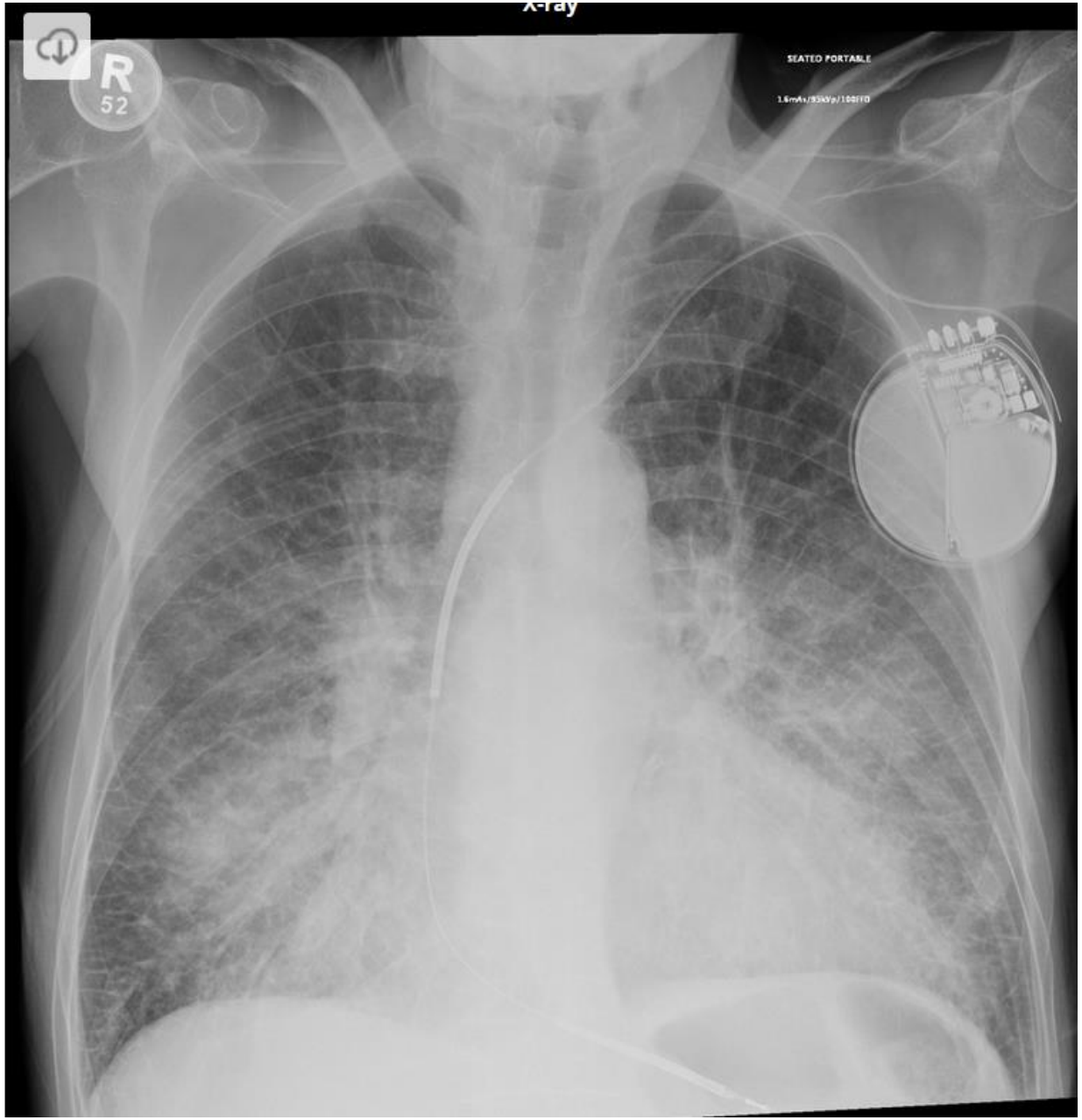
# MV in clinical cases

- 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



# Interstitial vs. alveolar pulmonary edema

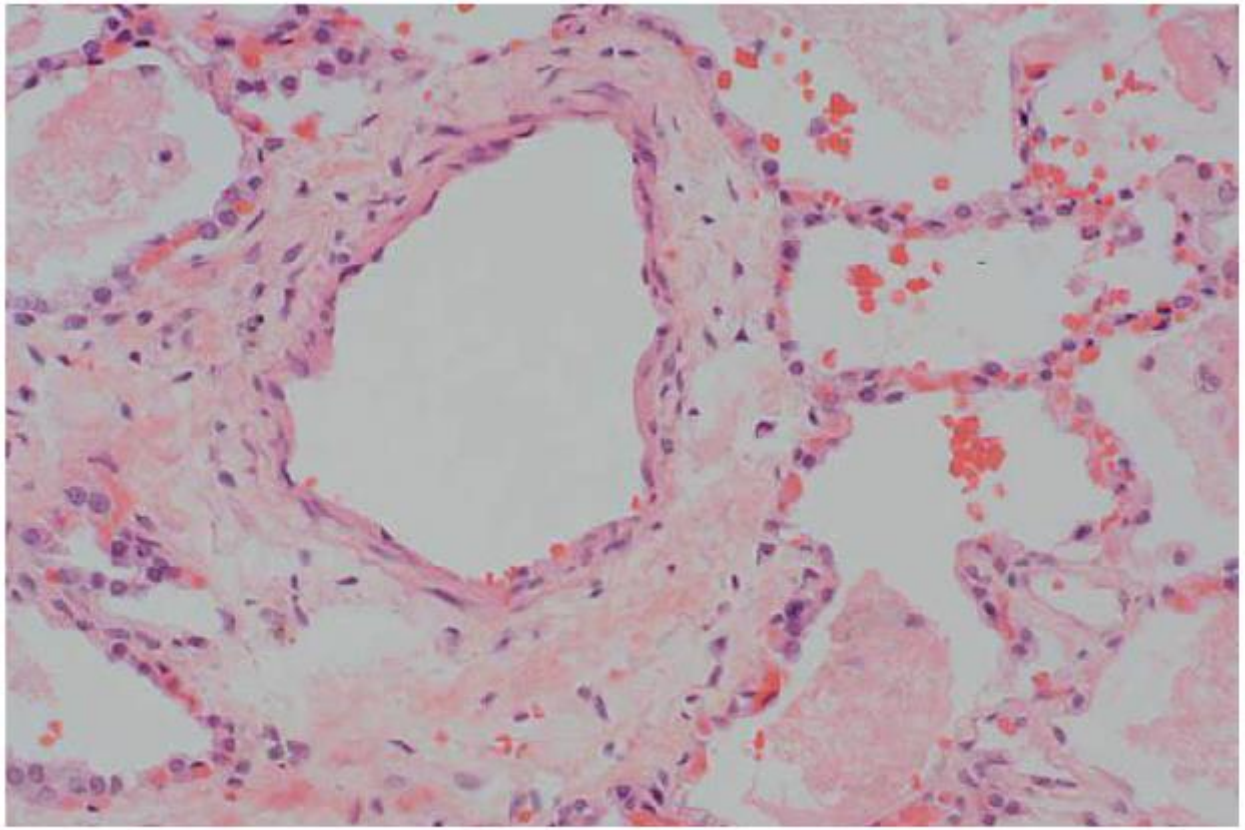


Figure 6.2. **Example of engorgement of the perivascular space of a small pulmonary blood vessel by interstitial edema.** Some alveolar edema is also present. (Image courtesy of Edward Klatt, MD.)

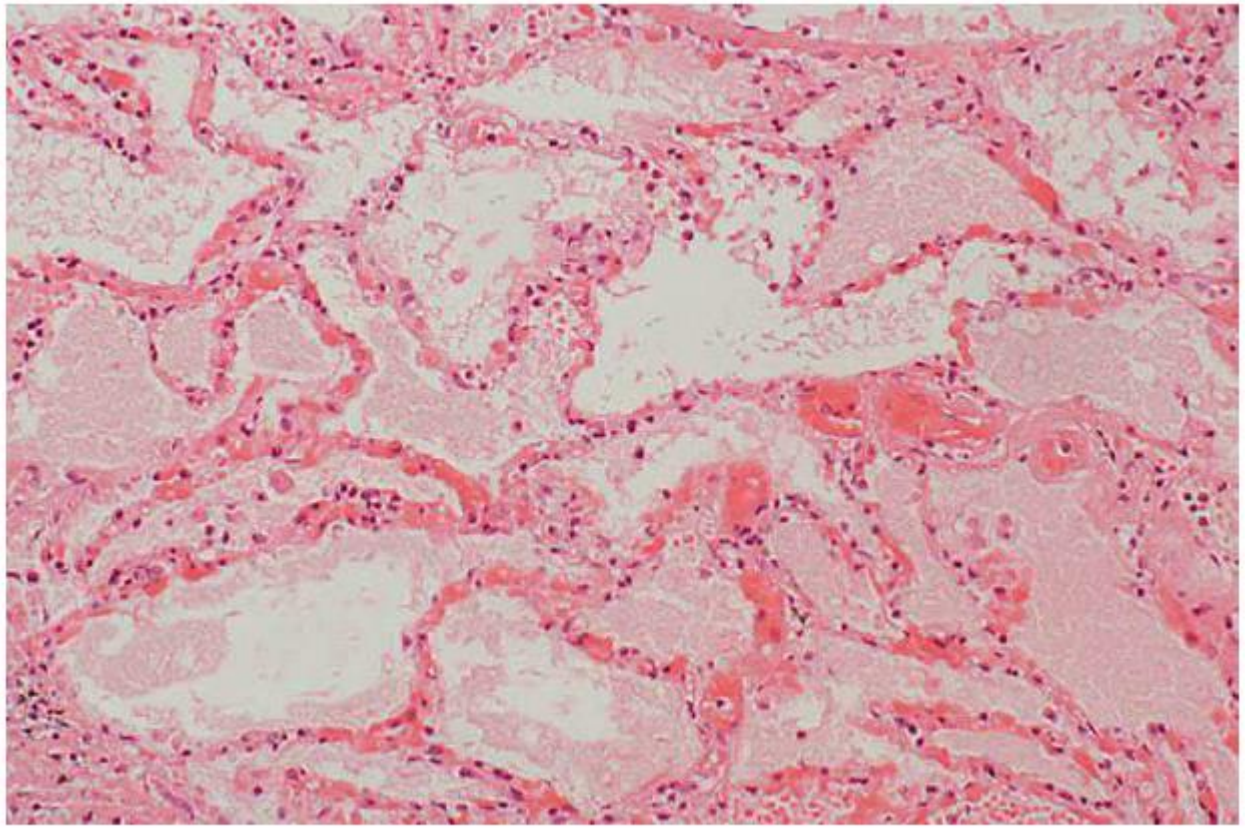


Figure 6.3. **Section of human lung showing alveolar edema.** (Image courtesy of Edward Klatt, MD.)



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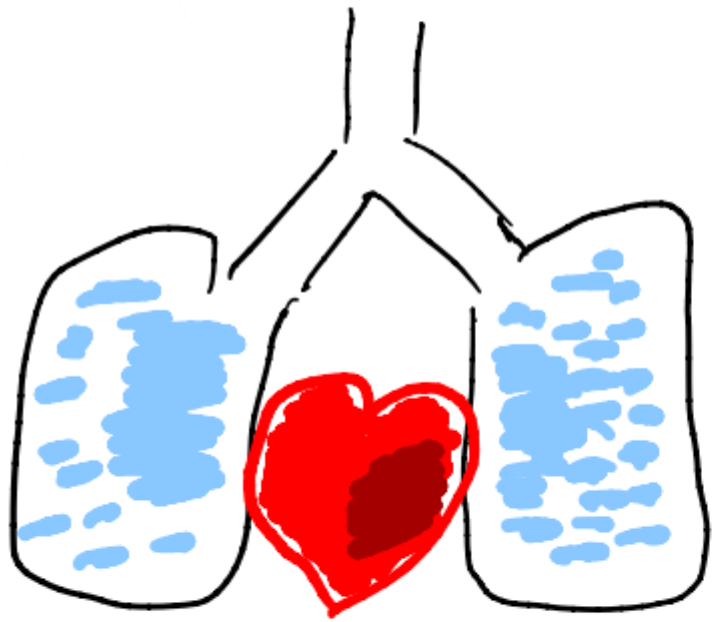
# Pulmonary edema by acute myocardial infarction



$O_2$ 10 l/min

OTI, NV

sedation

PEEP 15

cm $H_2O$

NOB = 0

(25% $O_2$)

$SpO_2$ 50

120 l/min

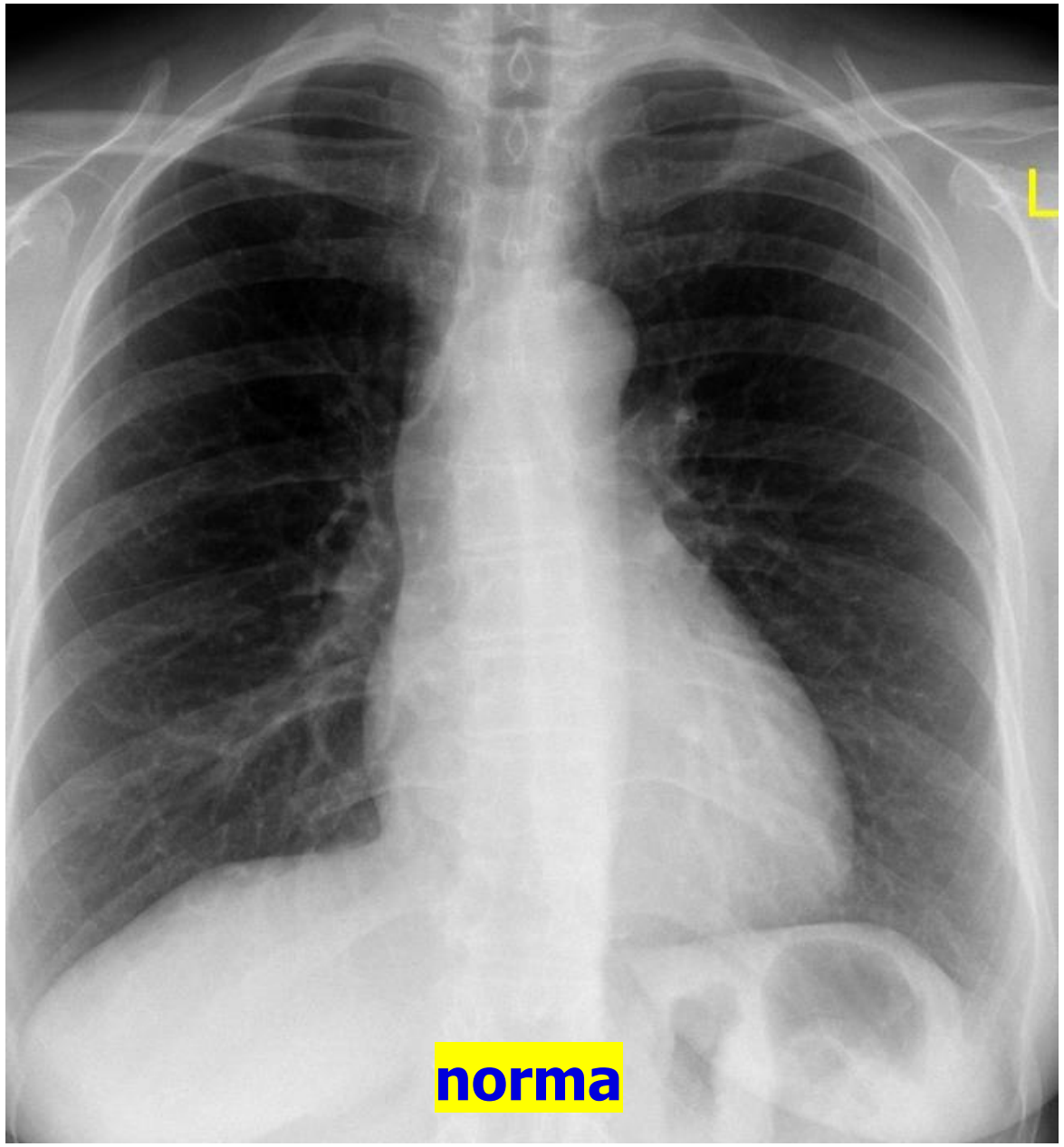
60% → 70%

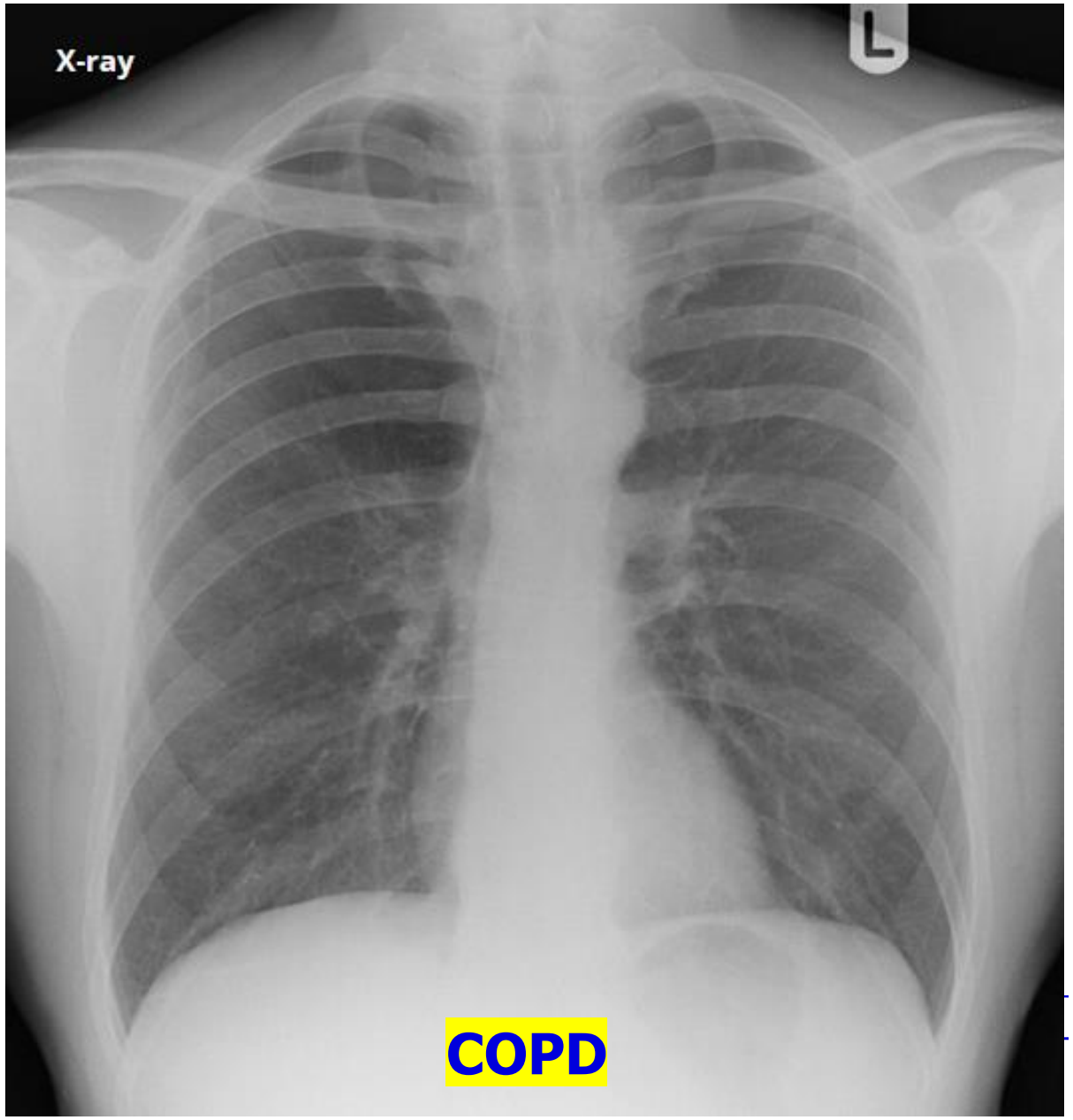
$\uparrow$ $SaO_2$

$\Rightarrow$

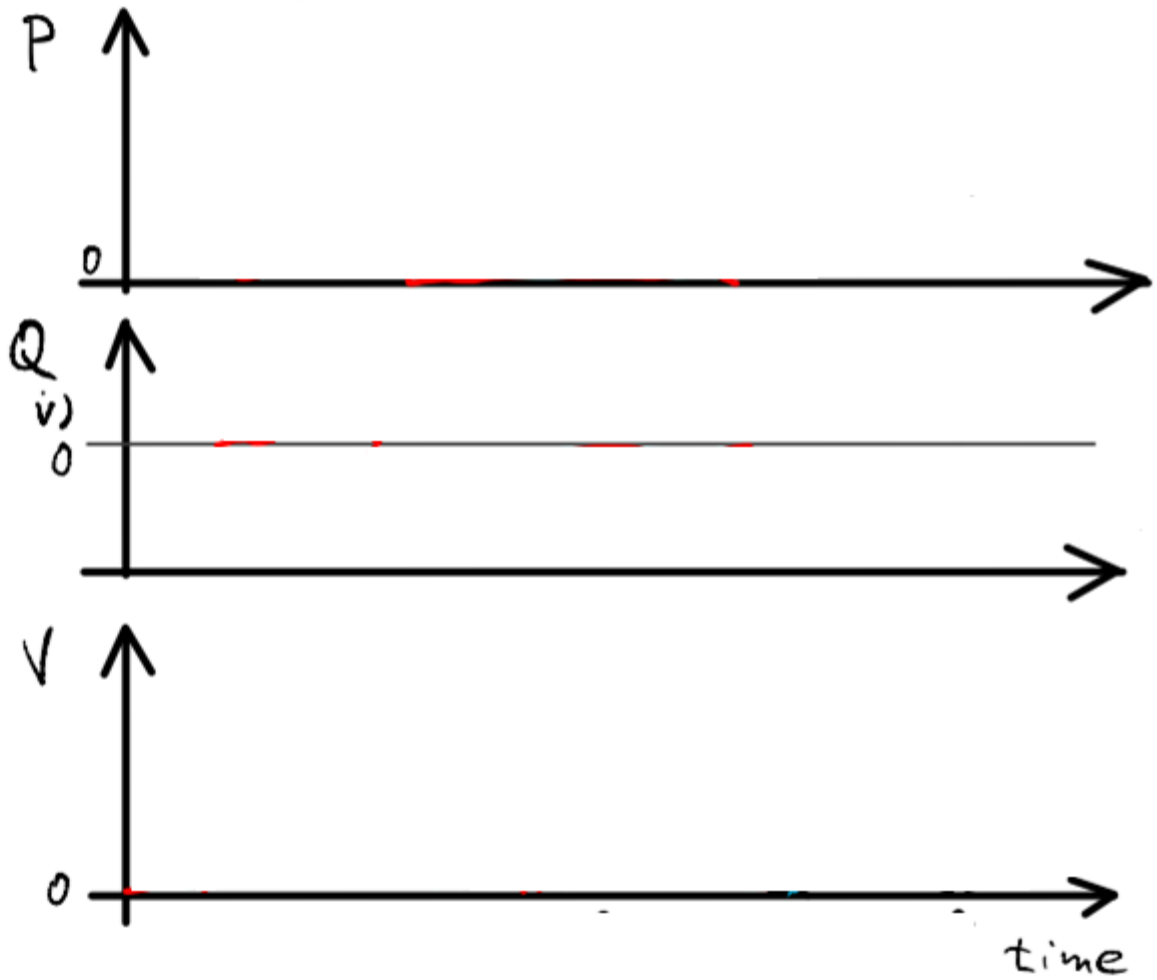
stabilization

# Acute exacerbation of COPD

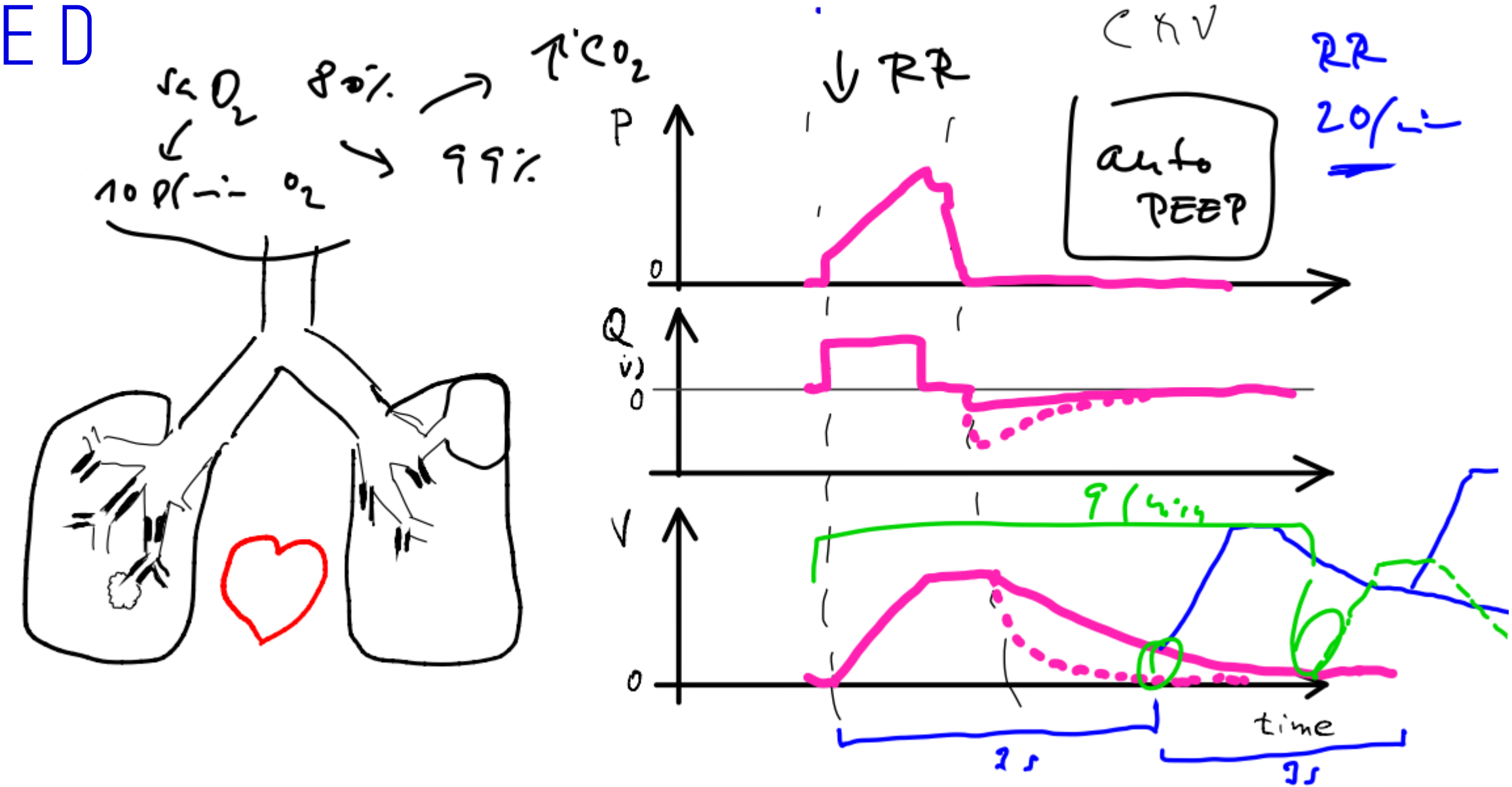


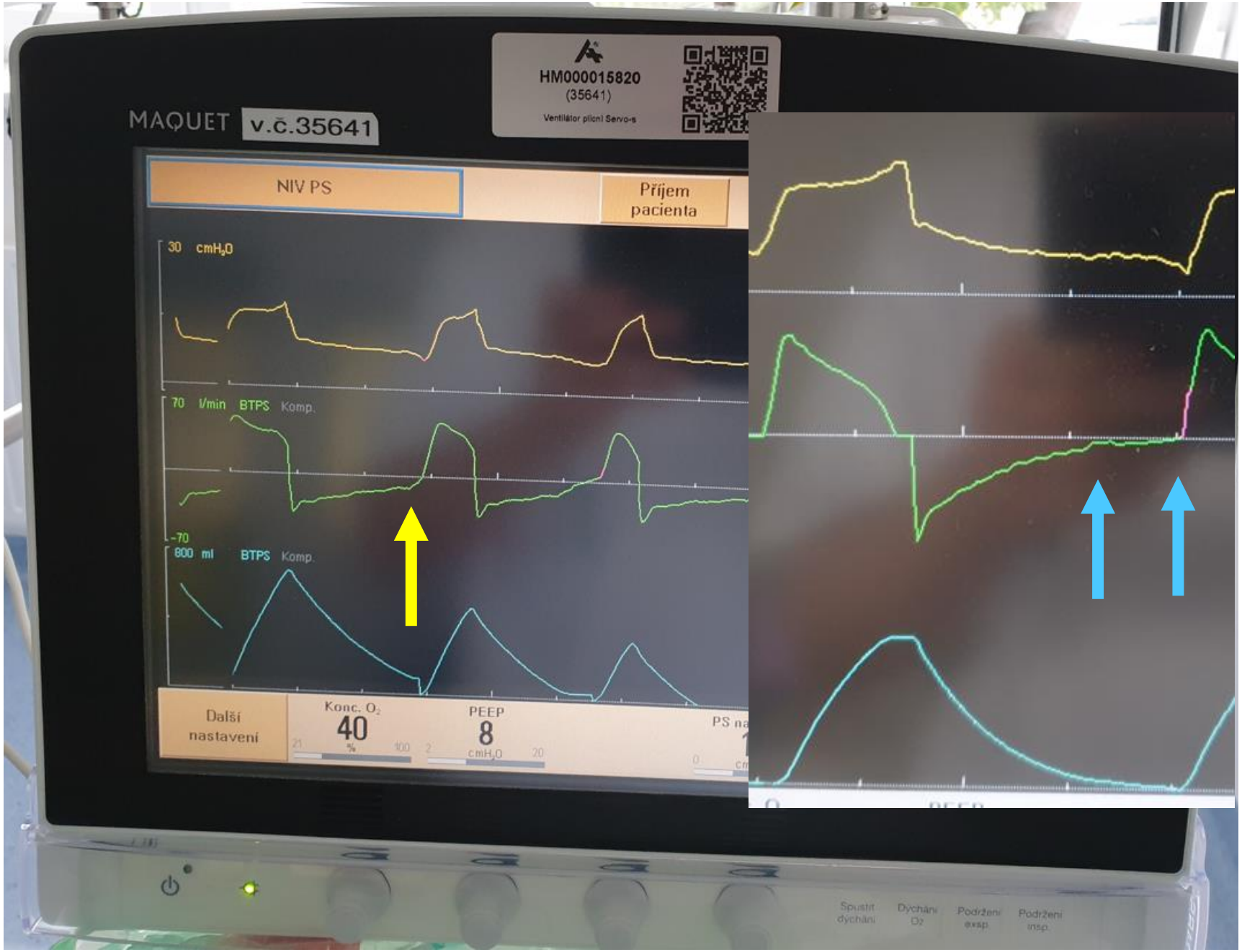
# Acute exacerbation of COPD

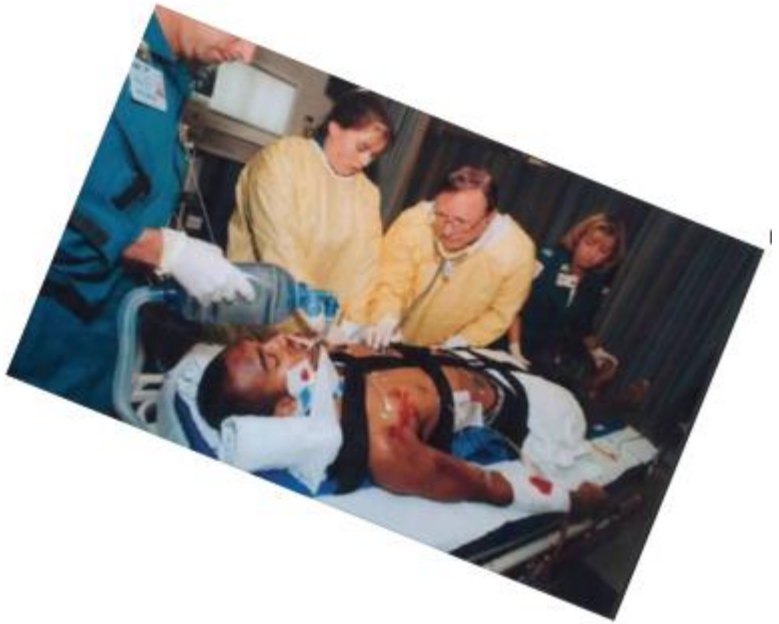
# COPD exacerbation



# Auto-PEEP


MAQUET v.č.35641
HM000015820
(35641)
Ventilátor plicní Servo-s
NIV PS
Příjem pacienta
30 cmH2O
70 l/min BTPS Komp.
-70
800 ml BTPS Komp.
Další nastavení
Konc. O2
40
%
PEEP
8
cmH2O
Spustit dýchání
Dýchání O2
Podržení exsp.
Podržení insp.

# Major trauma





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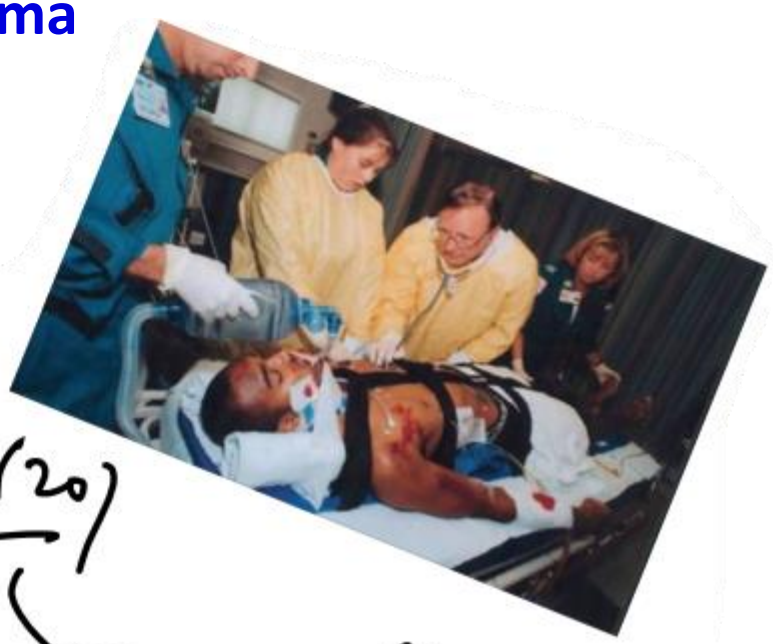
# Intubation and MV by polytrauma



OTI, MV ⇒ ↓↓ CO

↓↓ BP (50/20)

75/40

↑ HR

↑ Lactate → acidosis

Hypovolemia

↓

pH = 7.1

20 min

↓

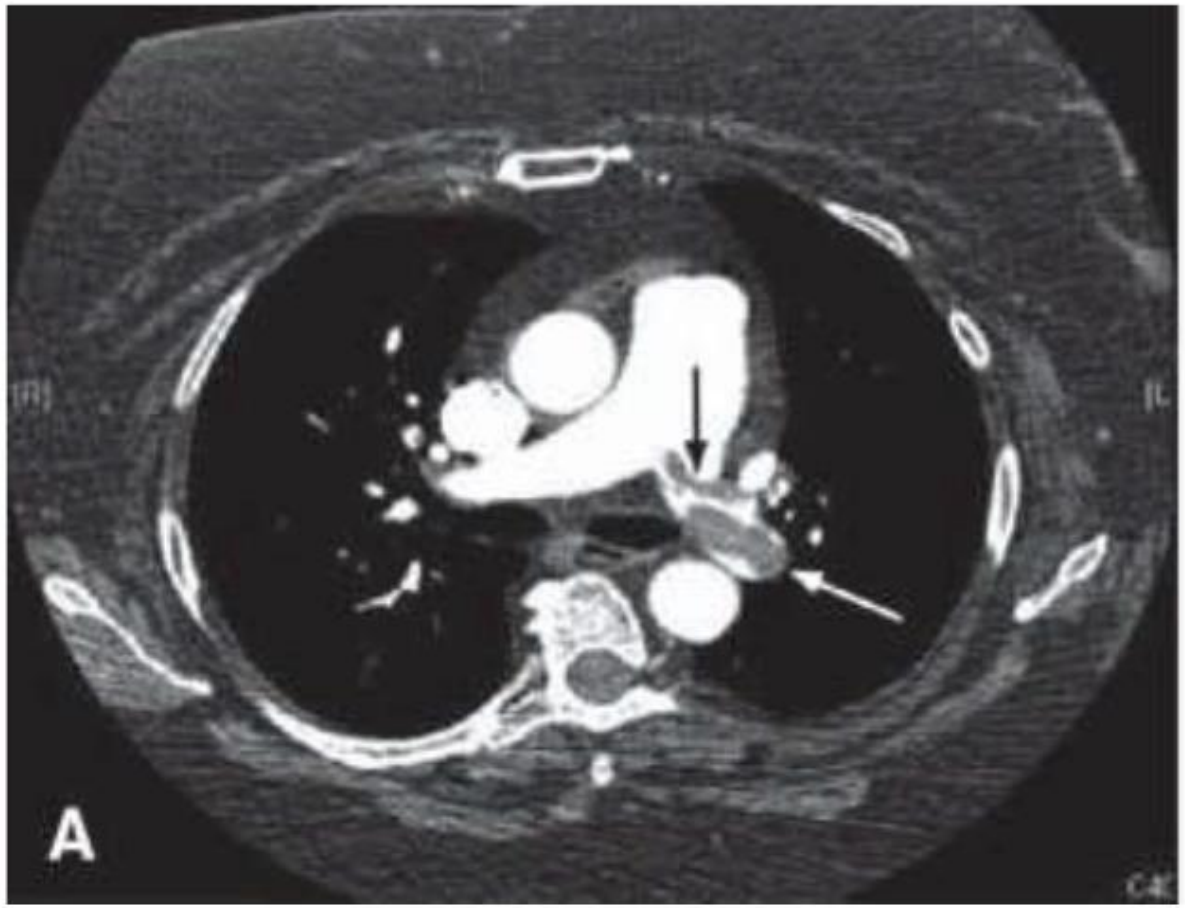
pH = 6.8

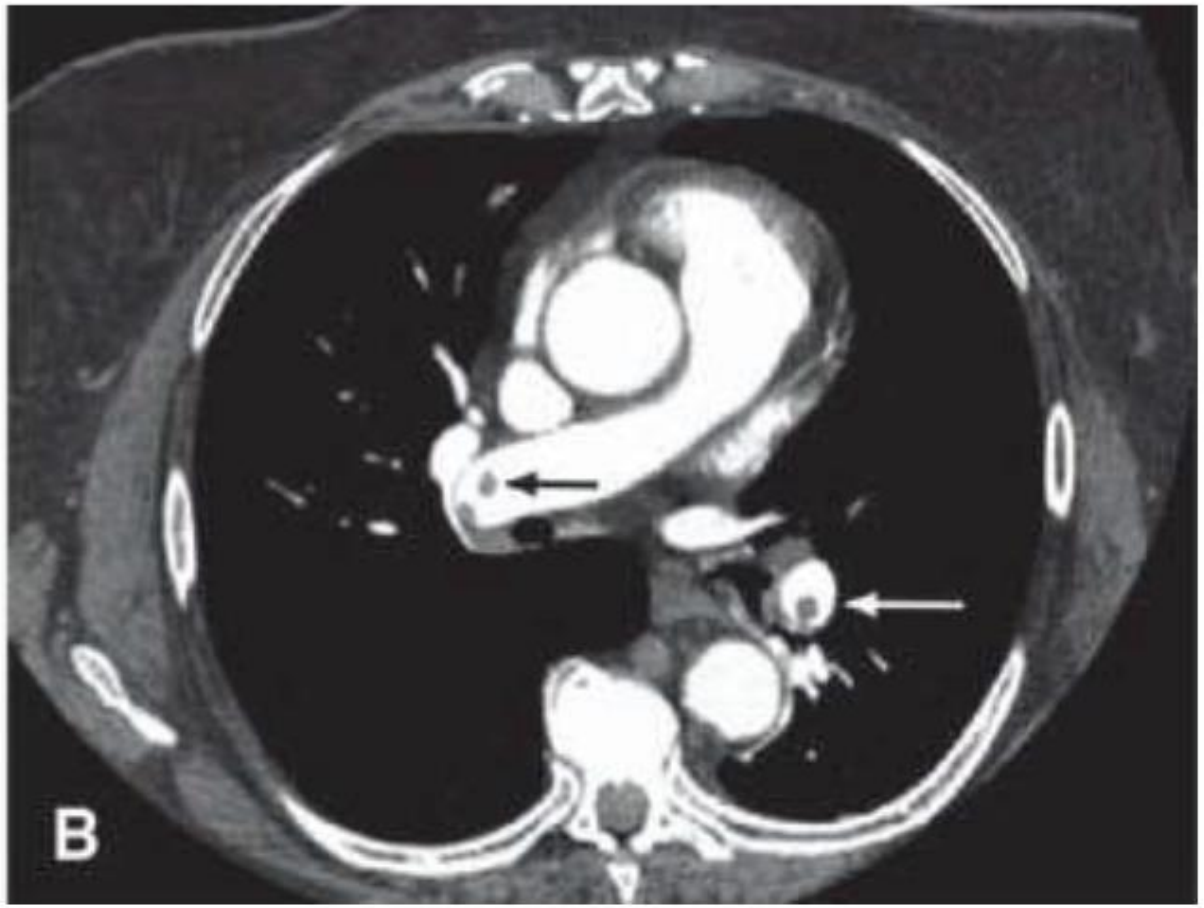
cardiac arrest

↓

CPR

# Pulmonary embolism – angio CT



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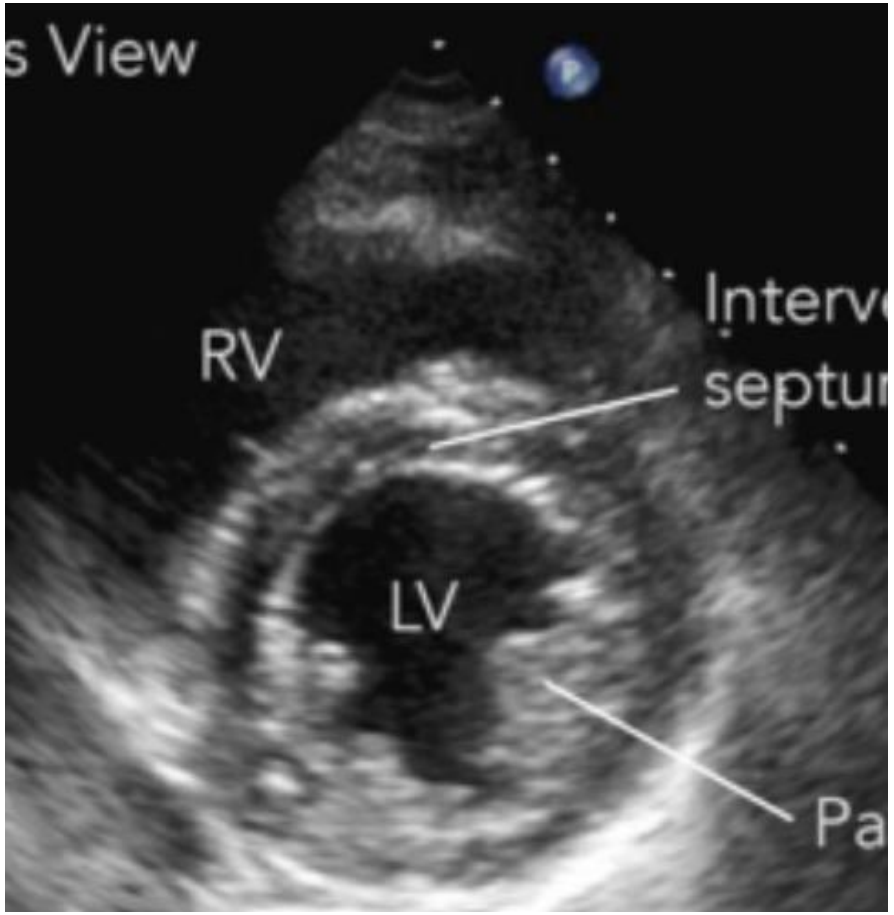
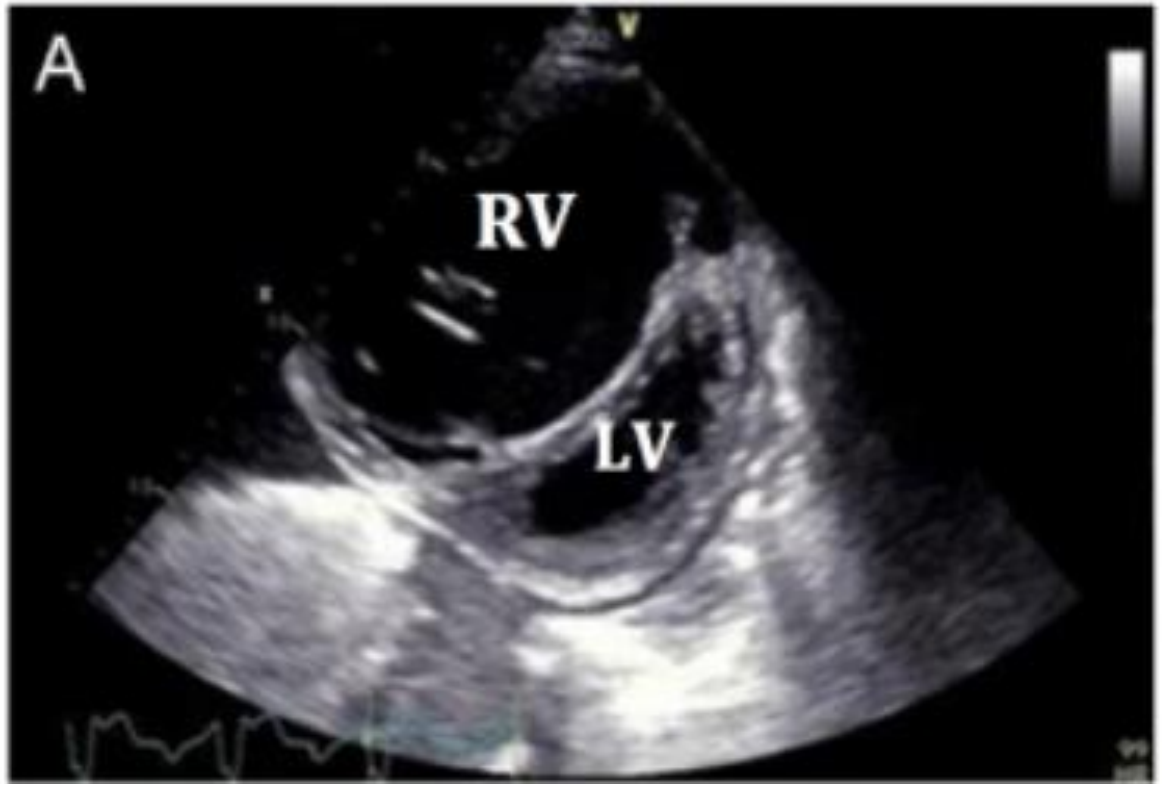
# Pulmonary embolism - echocardiography



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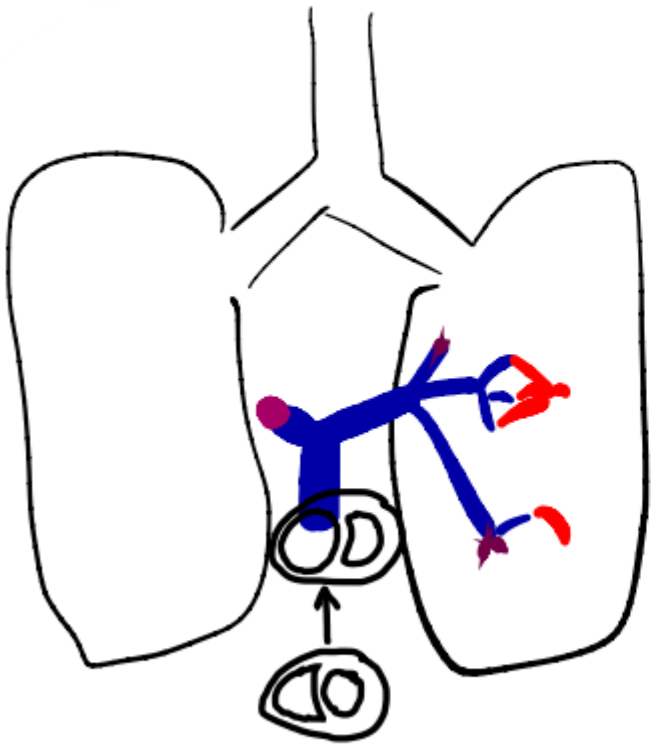
# Massive pulmonary embolism



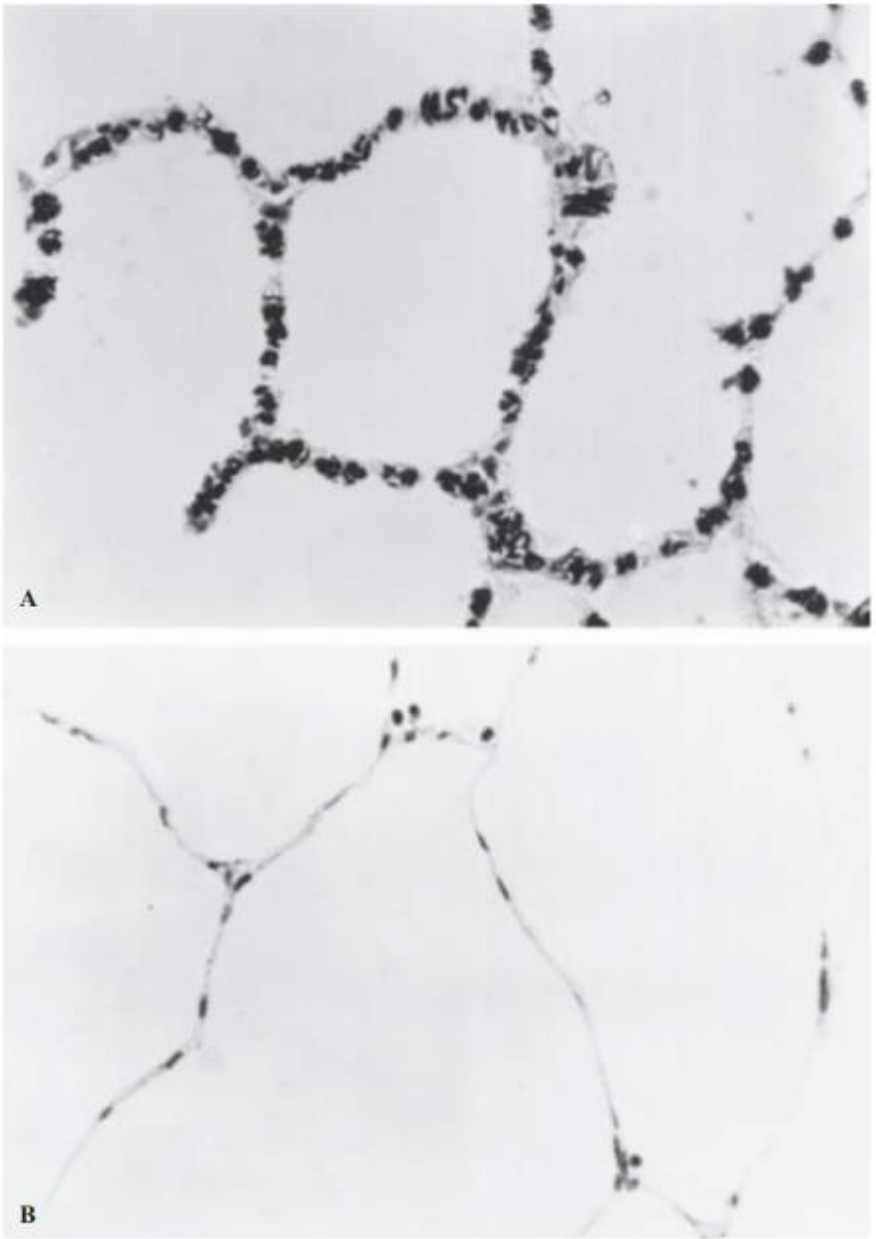



Figure 10.3. **Effect of raised airway pressure on the histologic appearance of pulmonary capillaries. A.** Normal appearance. **B.** Collapse of capillaries when alveolar pressure is raised above capillary pressure. (From Glazier JB, Hughes JMB, Maloney JE, et al. Measurements of capillary dimensions and blood volume in rapidly frozen lungs. J Appl Physiol 1969;26:65–76.)

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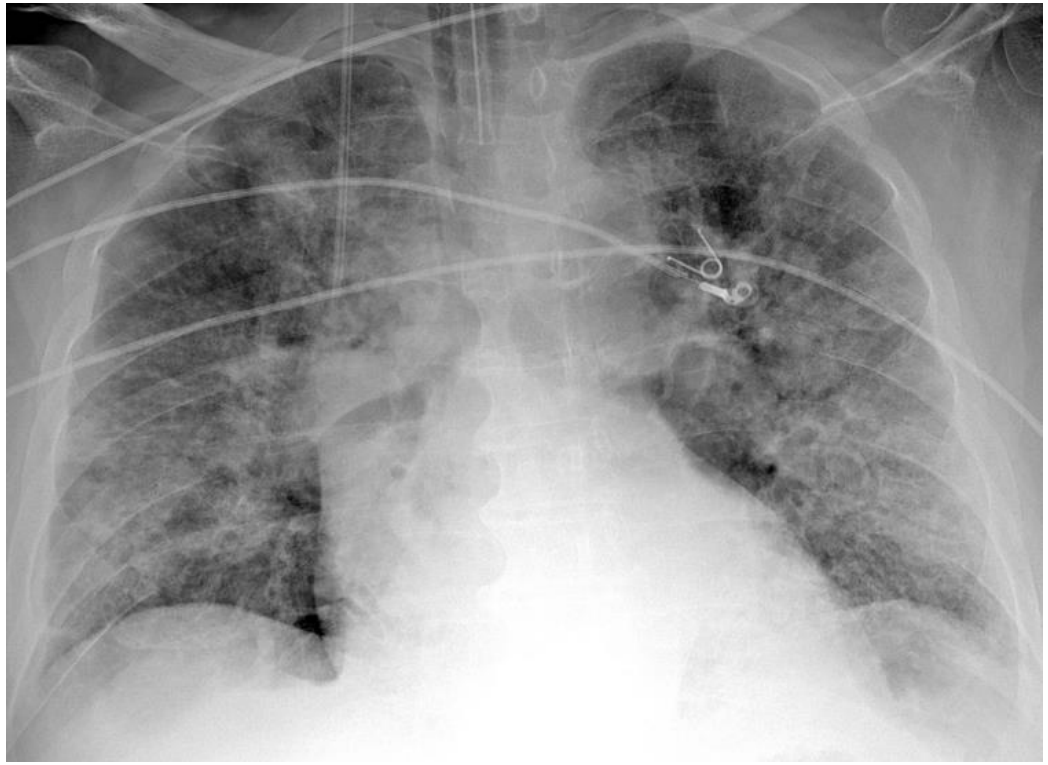
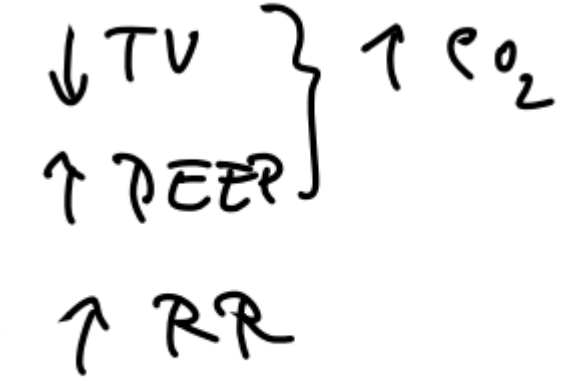
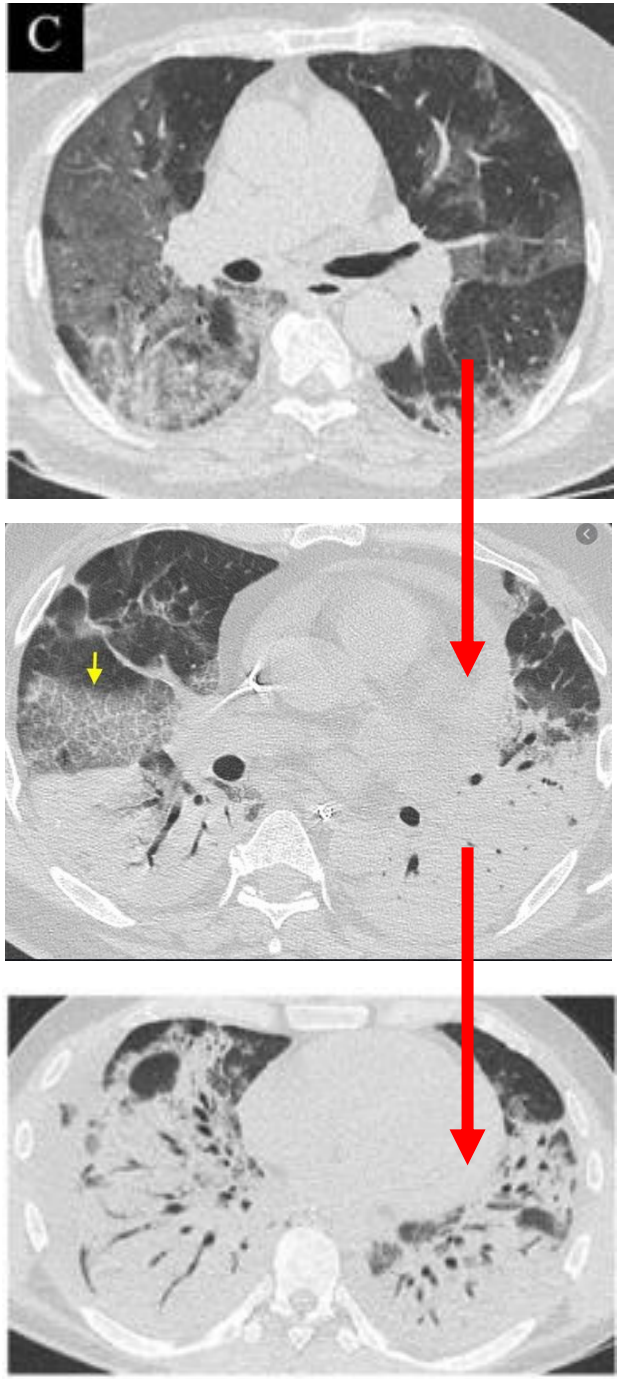
# ARDS – COVID-19 pneumonia

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





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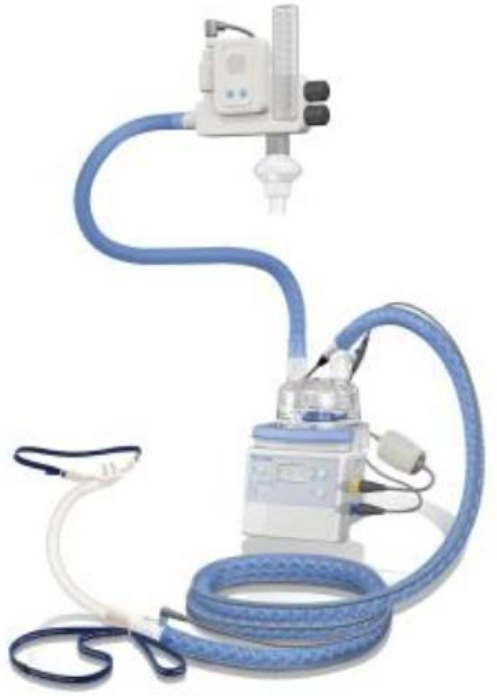
# High-flow nasal oxygen (HFNO)

- just as nasal cannula, but
  - humidified oxygen up to 60 l/min
  - $FiO_2$ up to 100 %
- high flow of gases builds up excess pressure in upper airways and so the PEEP 2-4 $cmH_2O$
- better tolerated than NIV
- similar indications as NIV
  - moderate COVID-19 pneumonia
  - acute COPD exacerbation
  - moderate cardiogenic pulmonary edema
  - intermittent support after extubation



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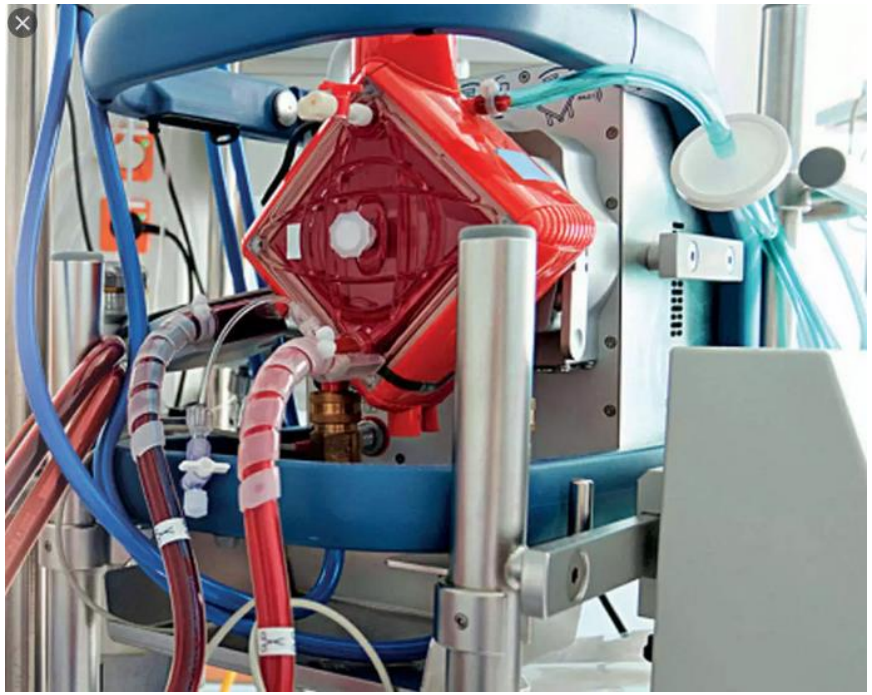
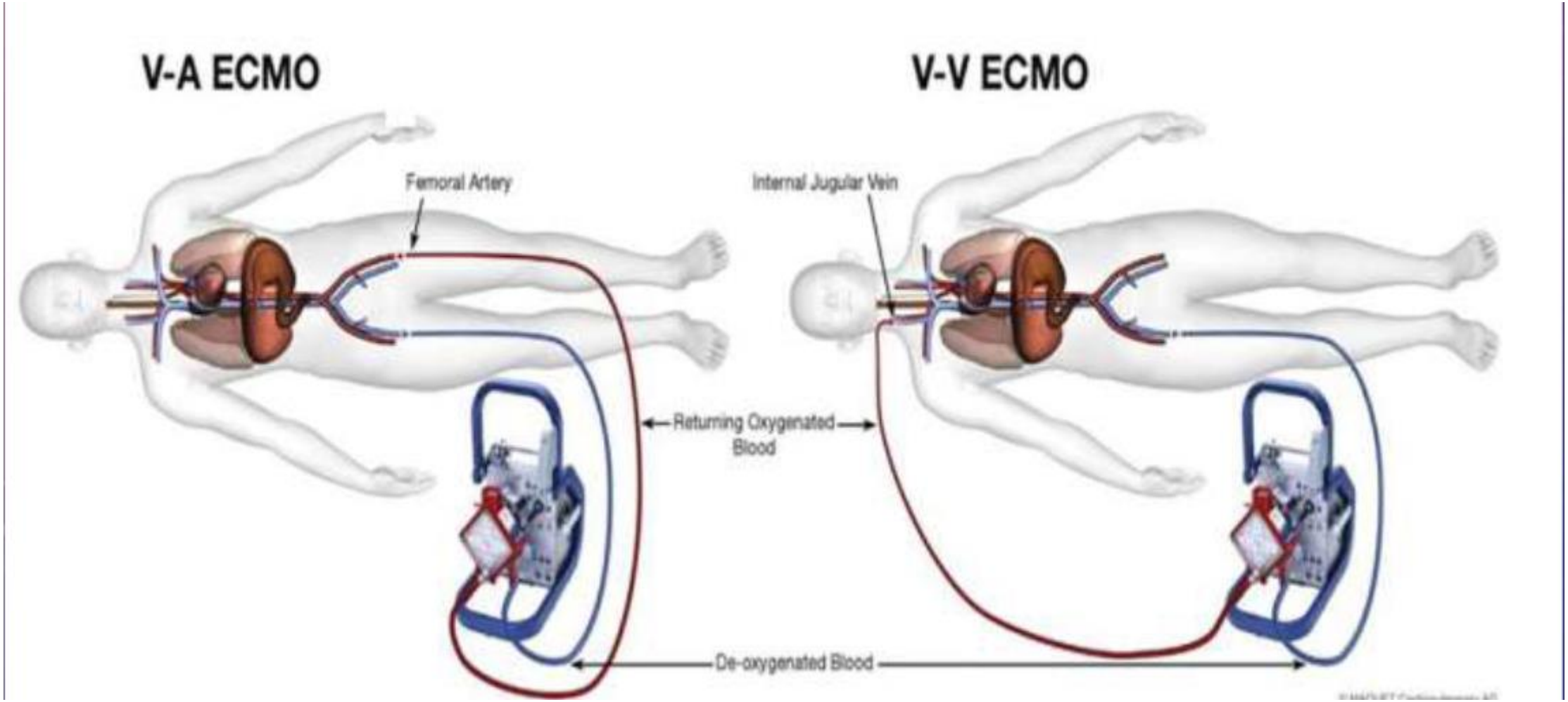
# Extracorporeal membrane oxygenation (ECMO)



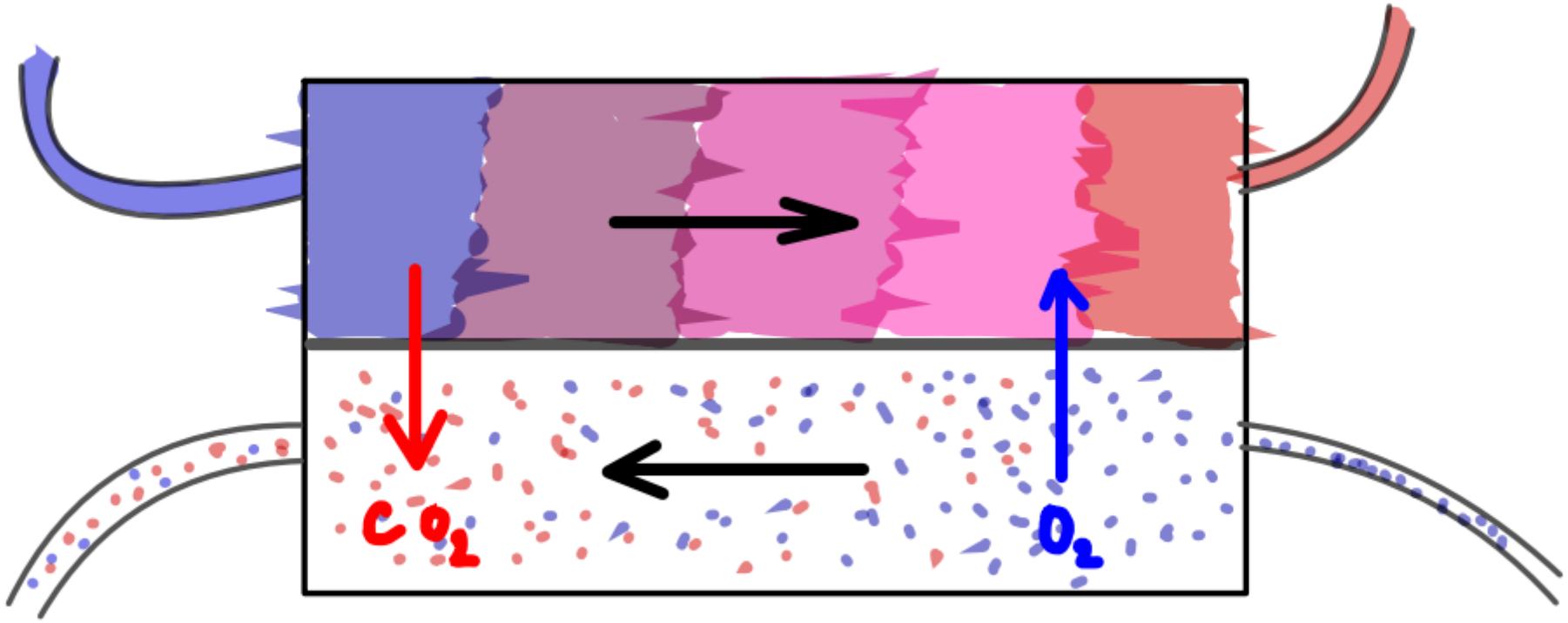
- extracorporeal circuit
- up to complete substitute of lungs (VV-ECMO) or heart and lungs (VA-ECMO)
- construction based on pump and oxygenator
- in oxygenator blood and air/oxygen come to contact over a membrane

- Indication
  - reasonable chance of solution of the basic problem (as e.g. cure of COVID pneumonia) or bridge-to-transplantation

# Extracorporeal membrane oxygenation (ECMO) variants



# Principle of membrane oxygenator



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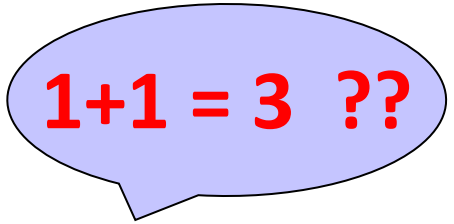
# Apneic ventilation




- apneic test of brain death

1. the patient breathes $O_2$ through the mask, creating an oxygen reserve in the lungs
2. general anaesthesia and muscle relaxation
3. the patient is not breathing, but we continue to administer $O_2$

How long does it take for the patient to desaturate?
The lung $O_2$ reserve (5 l) would be sufficient for a maximum of 20 min at 250 ml $O_2$ /min.

**Up to 60 minutes!!**

# Apnoic ventilation



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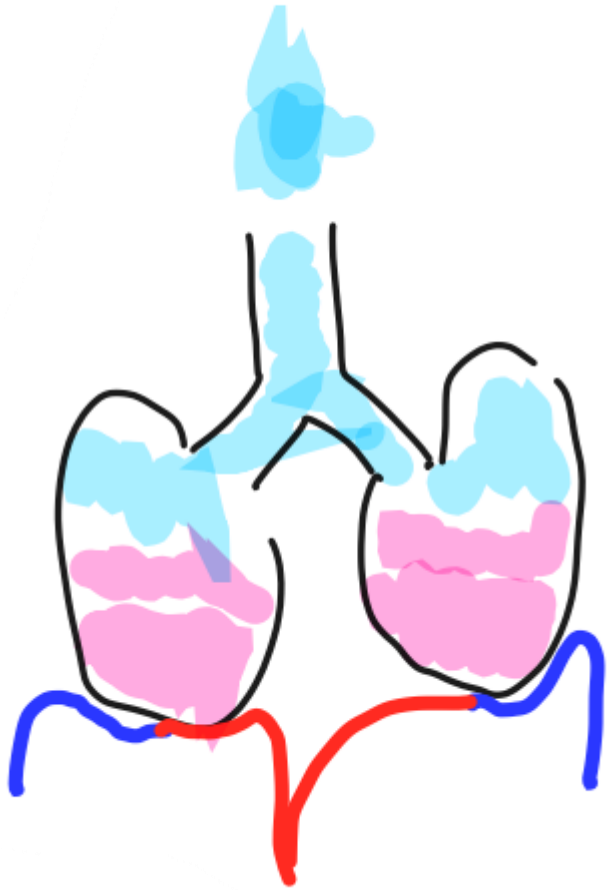

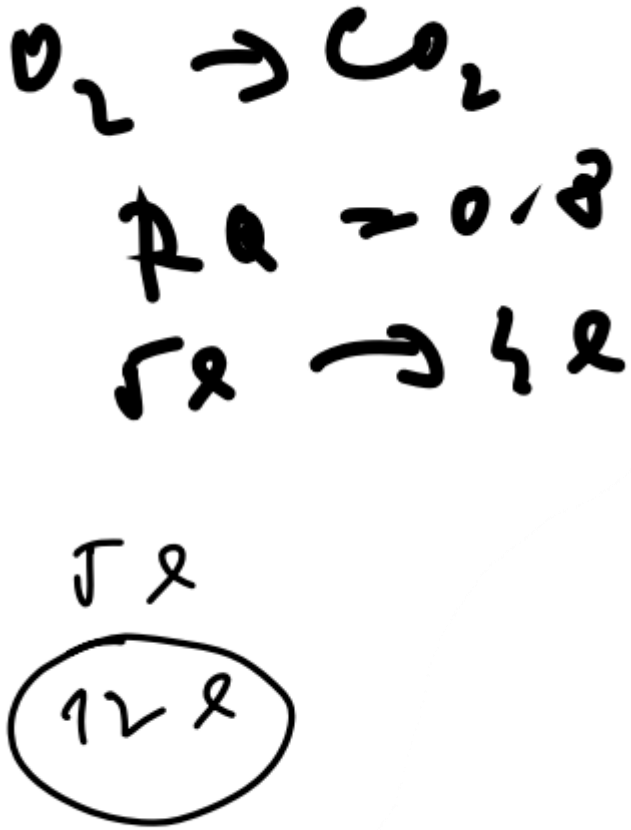
$O_2 \rightarrow CO_2$
RQ = 0,8
5 l
5 l
7 l
12 l

# 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
  - Epiglotitis
- Aspiration
- Massive pulmonary embolism
- Coma with secondary asphyxia
- Acute neuromuscular disorders
  - Myasthenia gravis
  - Syndrome Guillain-Barré
- Thorax trauma
  - Lung contusion
  - Block rip fracture
  - Massive hemothorax
- Massive haemoptysis