

- 1 úvod, respirační insuficience**
- 2 spirometrické dělení, restriční**
- 3 a obstrukční poruchy**
- 4 poměr ventilace/ k perfúzi, poruchy**
- kardiopulmonální a další doplňky**

Vybrané ventilační (spirometrické) parametry

-Dechová frekvence, [1/min] normální 12-16 dechů / min

-Minutová ventilace [volume/min] norma: 6-8 L/ min

-(F)VC - Forced vital capacity (usilovná/ forsírovaná VC)

ženy: $[21.7 - (0.101 \times \text{věk})] \times (\text{cm}) = [\text{mL}]$

muži: $[27.63 - (0.112 \times \text{věk})] \times (\text{cm}) = [\text{mL}]$

Hodnoty v mezích 80 - 120 % náležité hodnoty se považují za normální

-MVV (Vmax) = Maximal voluntary ventilation

Objem dosažený během (20-ti sekundové) maximální ventilace

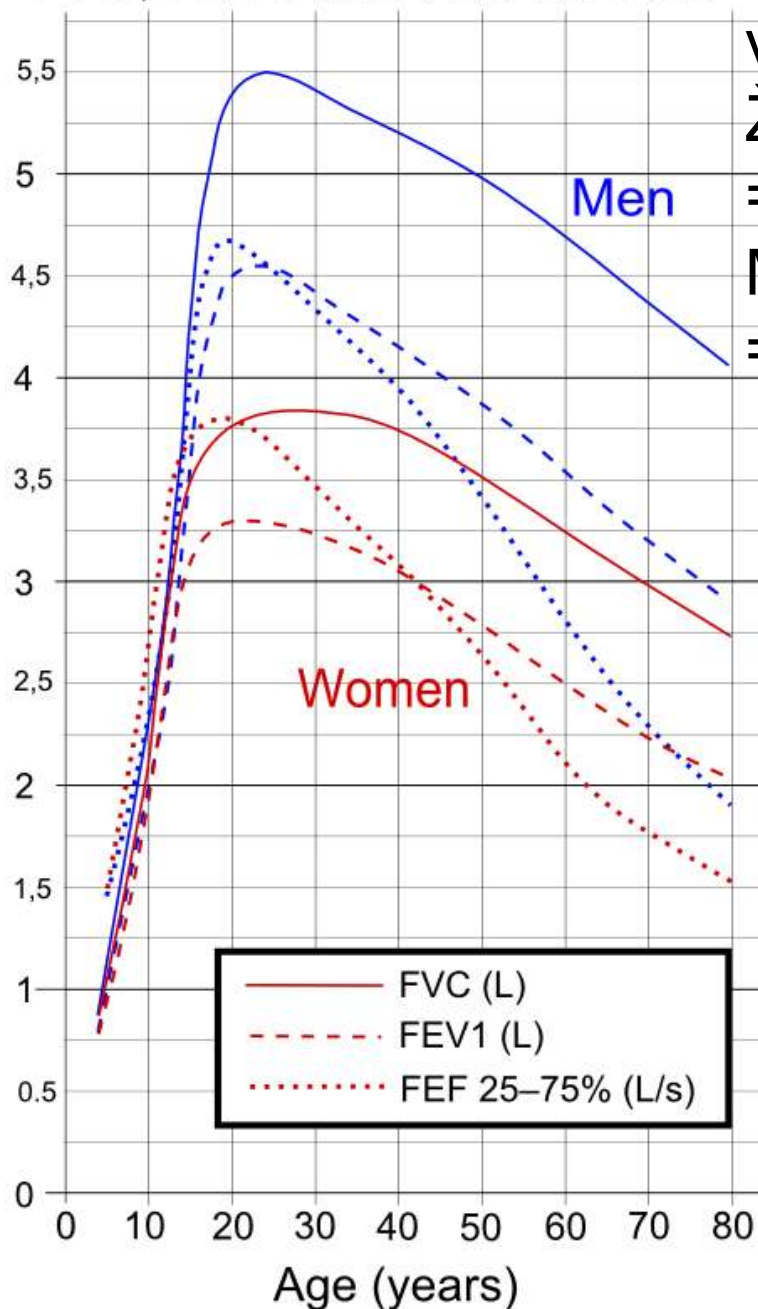
Maximální dechový objem (TV) X maximální ventilace

Po dobu 10 – 30 sec > 40 L/min

- Ventilační rezerva:

minutová ventilace / MVV > 1 : 7 (> 1:5)
= 1 : 2

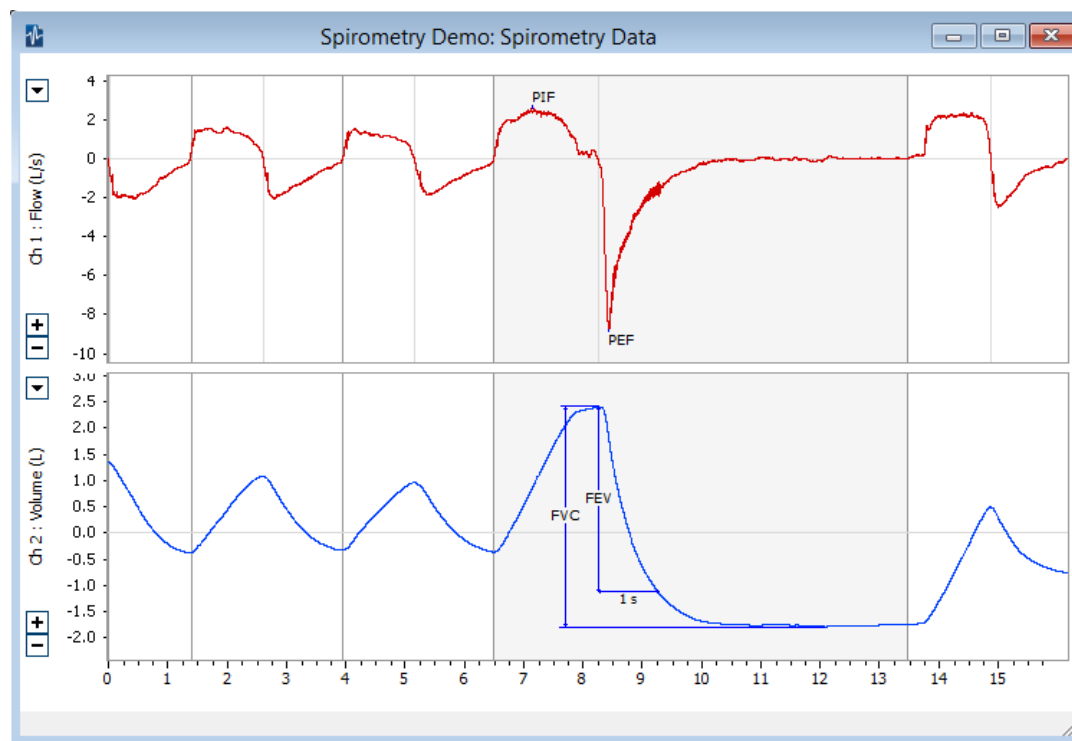
Normal values for
FVC, FEV1 and FEV 25-75%



Regresní rovnice pro určení náležité
vitální kapacity:

Ženy: $[21.7 - (0.101 \times \text{age}(y))] \times h \text{ (cm)}$
= (mL)

Muži: $[27.63 - (0.112 \times \text{age}(y))] \times h \text{ (cm)}$
= (mL)



Restrictive diseases

anatomical and/or functional loss of surface for gas exchange

resection

atelectasis

lung edema

lung fibrosis

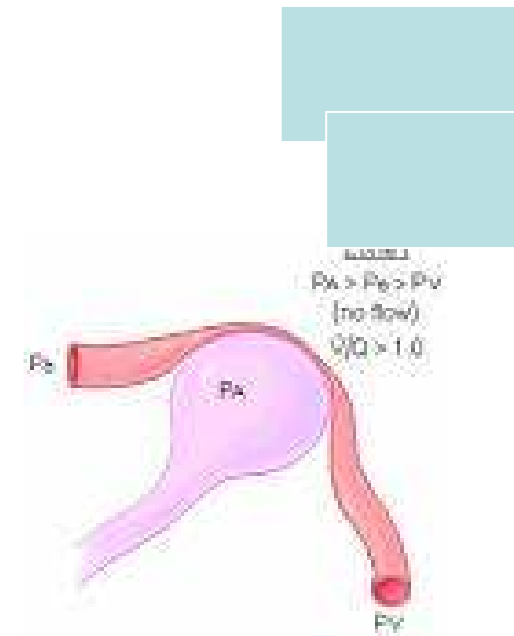
thoracic deformities / impaired breathing movements

pneumonia

pneumothorax

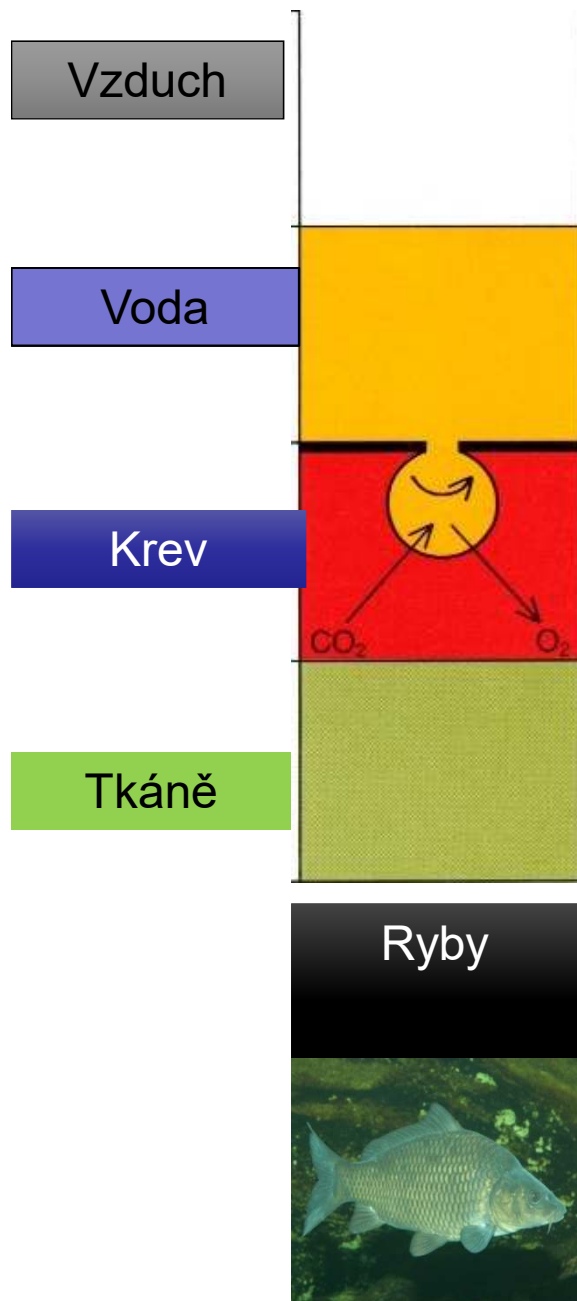
Možné poruchy funkce dýchacího systému

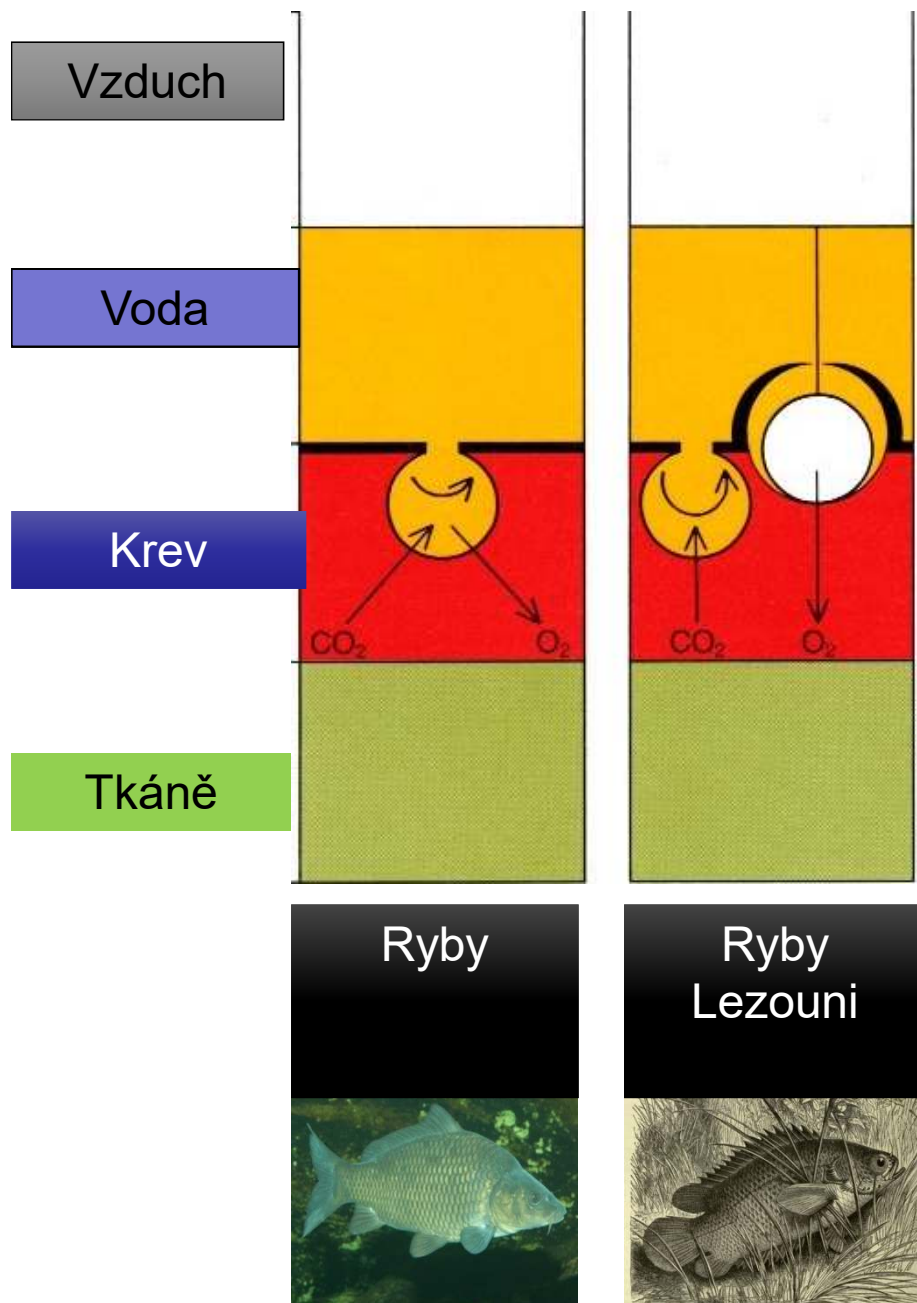
- Porucha **ventilace**
- Porucha **perfúze**
- Porucha **distribuce**
ventilace a perfúze
= ventilačně perfúzní
nerovnováha
- Porucha **difúze**

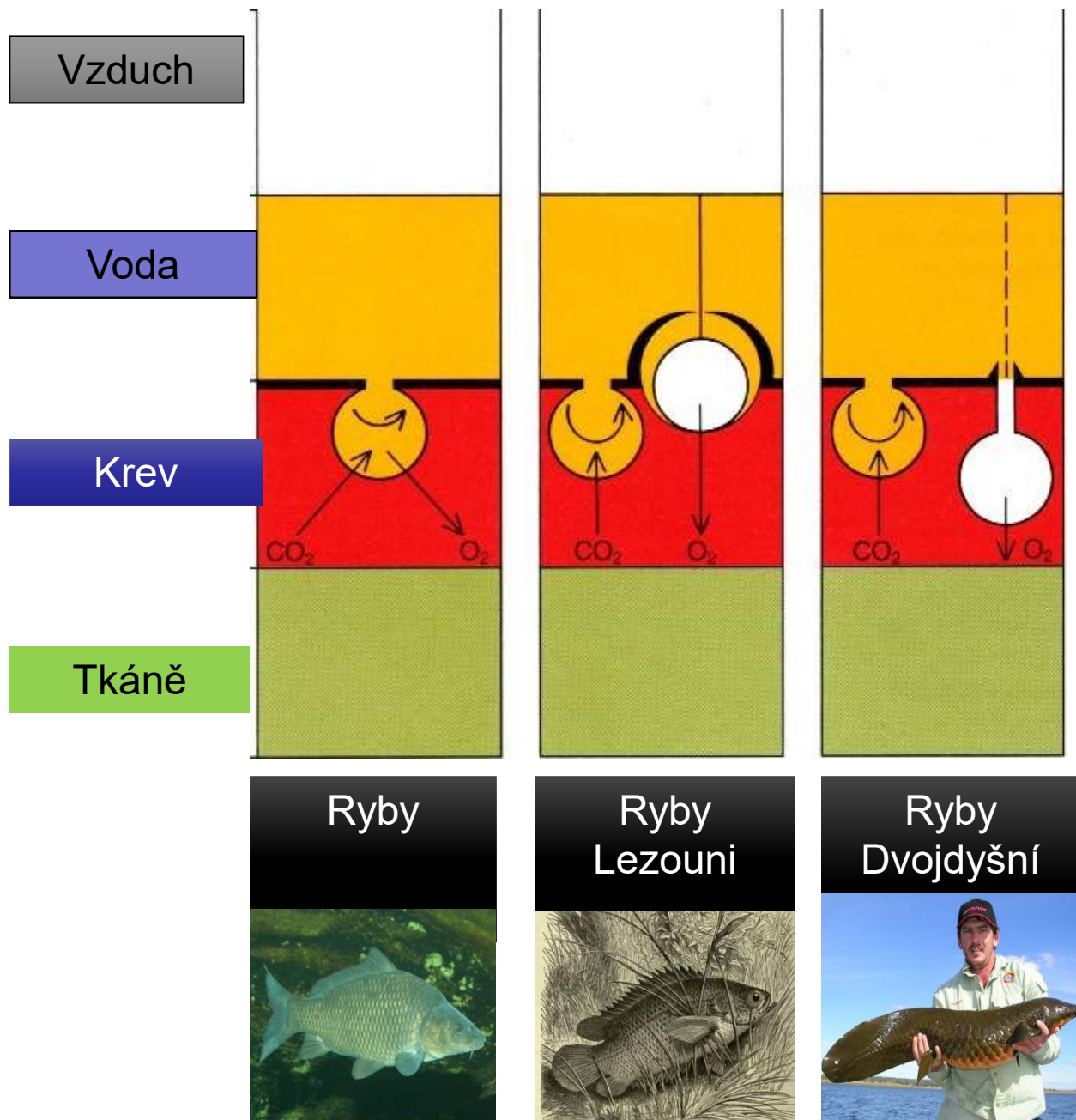


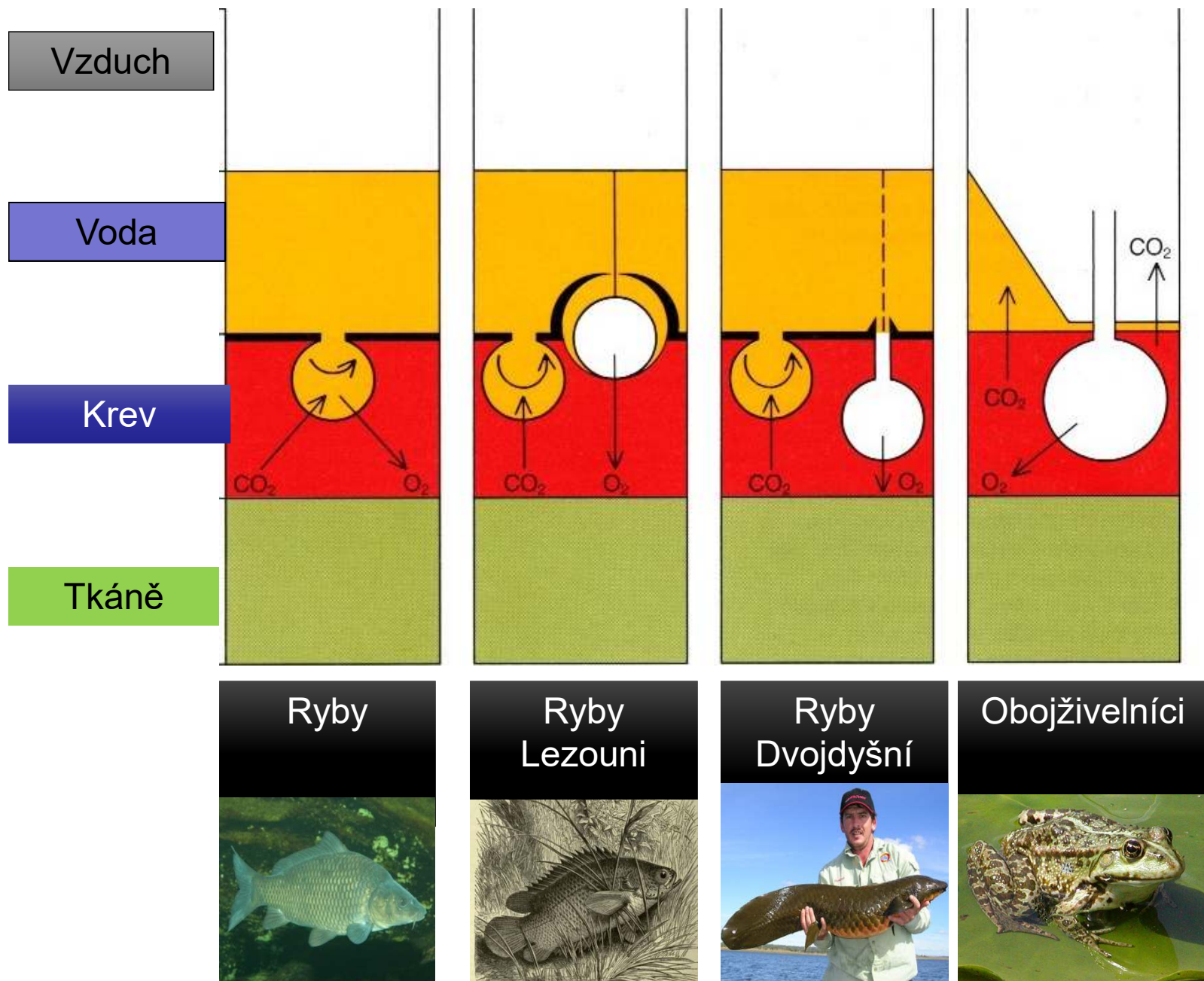
Osnova/ Shrnutí

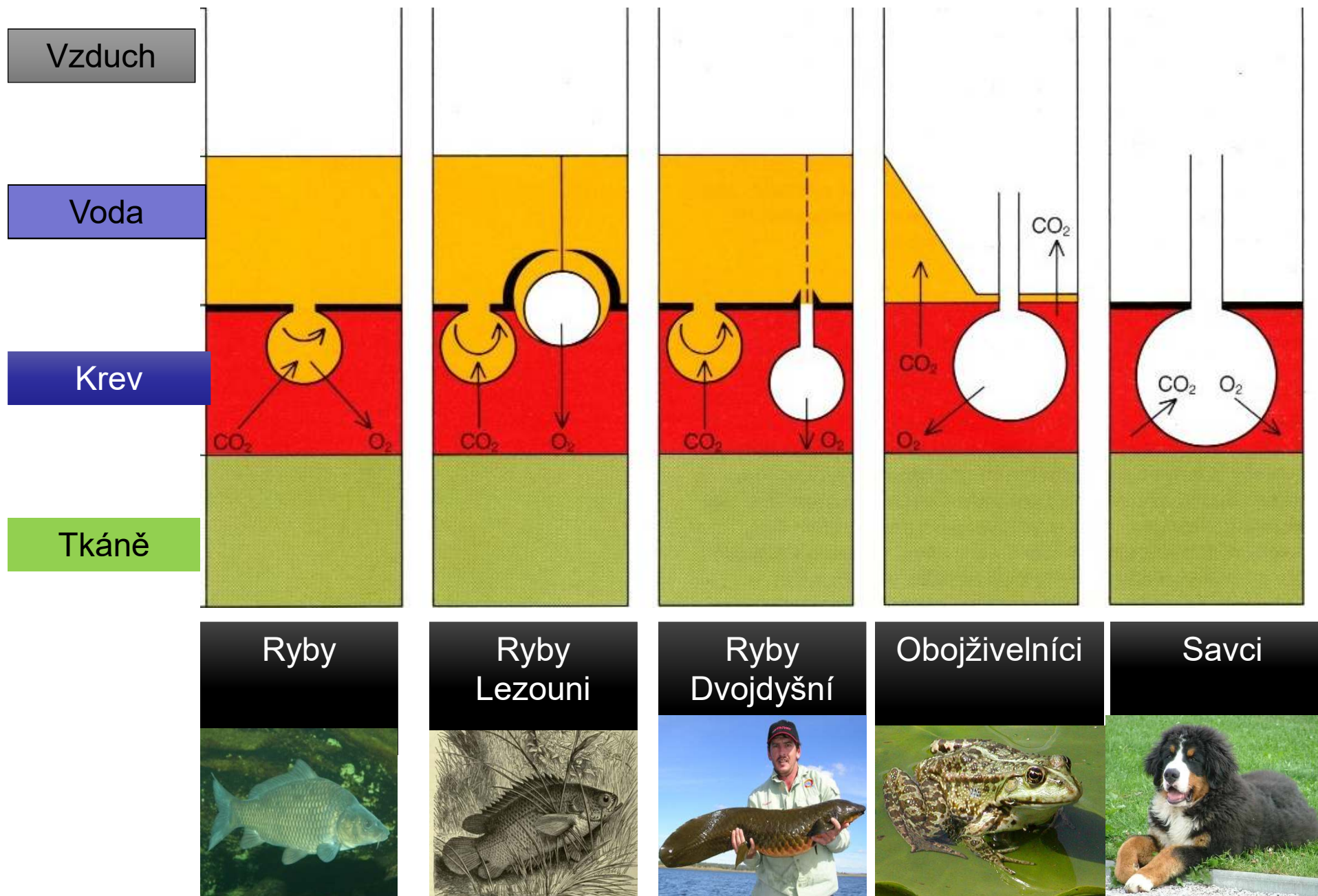
- **Plicní poruchy podle cesty dýchacích plynů do krve –**
poruchy 1) ventilace, 2) difúze
3) perfúze/ plus ventilačně perfusní nehomogeneity,
4) ostatní a mimoplicní poruchy
- **Statické parametry** – intrapleurální tlak, surfaktant,
restrikční poruchy
- **Dynamické parametry** – **obstrukční poruchy**, viz další
přednáška
- **Typické restriční poruchy** – **edém plic, ARDS/**
atelektáza, plicní fibróza, hrudní deformity,
pneumothorax, atd
- **Posouzení ventilační funkce = spirometrie,**
pletysmografie – budou podrobněji na vyšetřovacím
semináři další týden

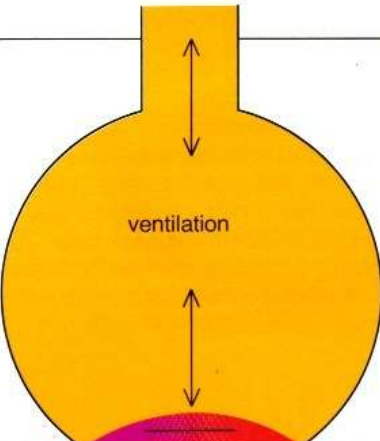
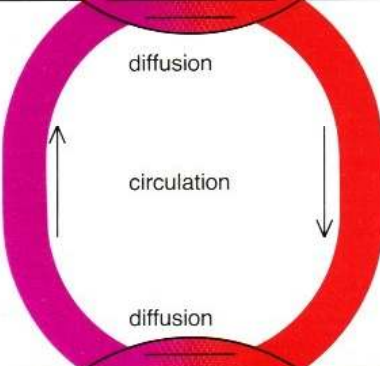
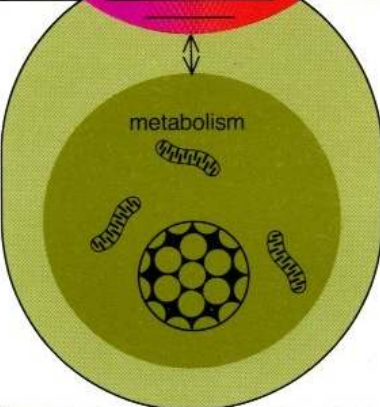


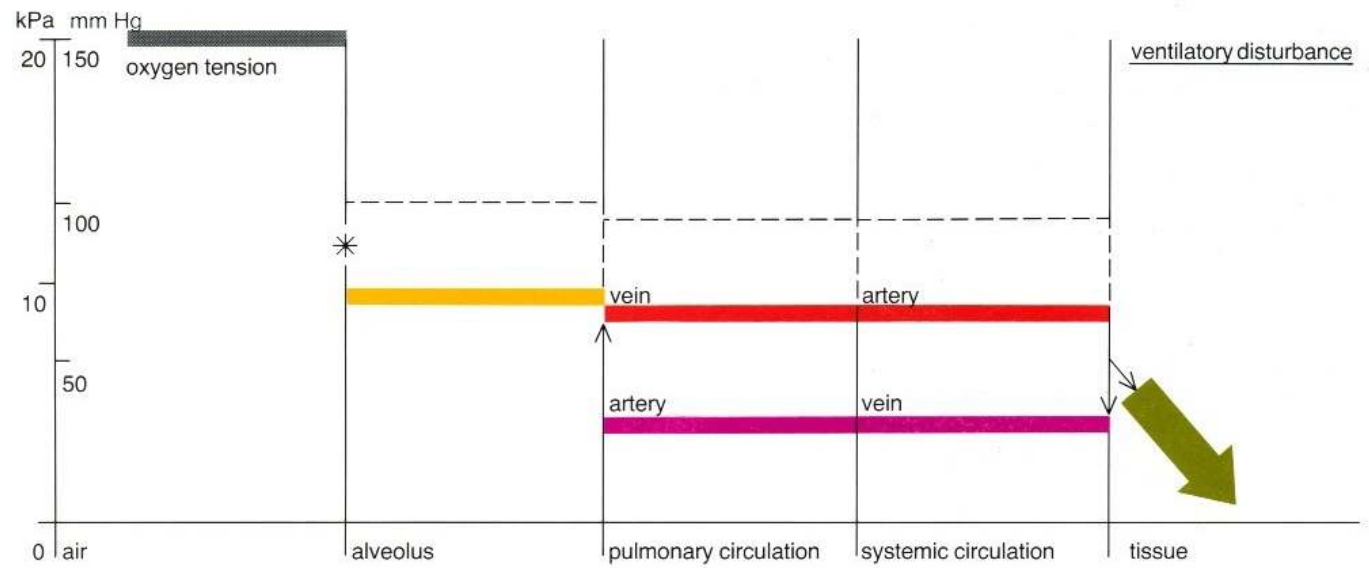
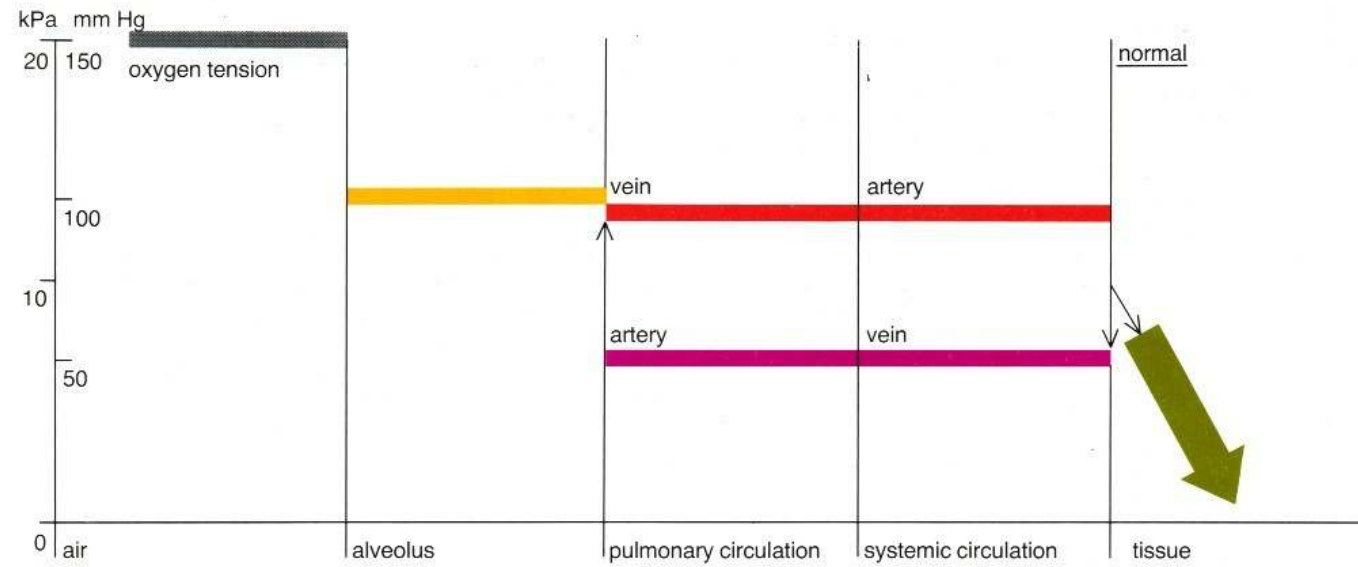




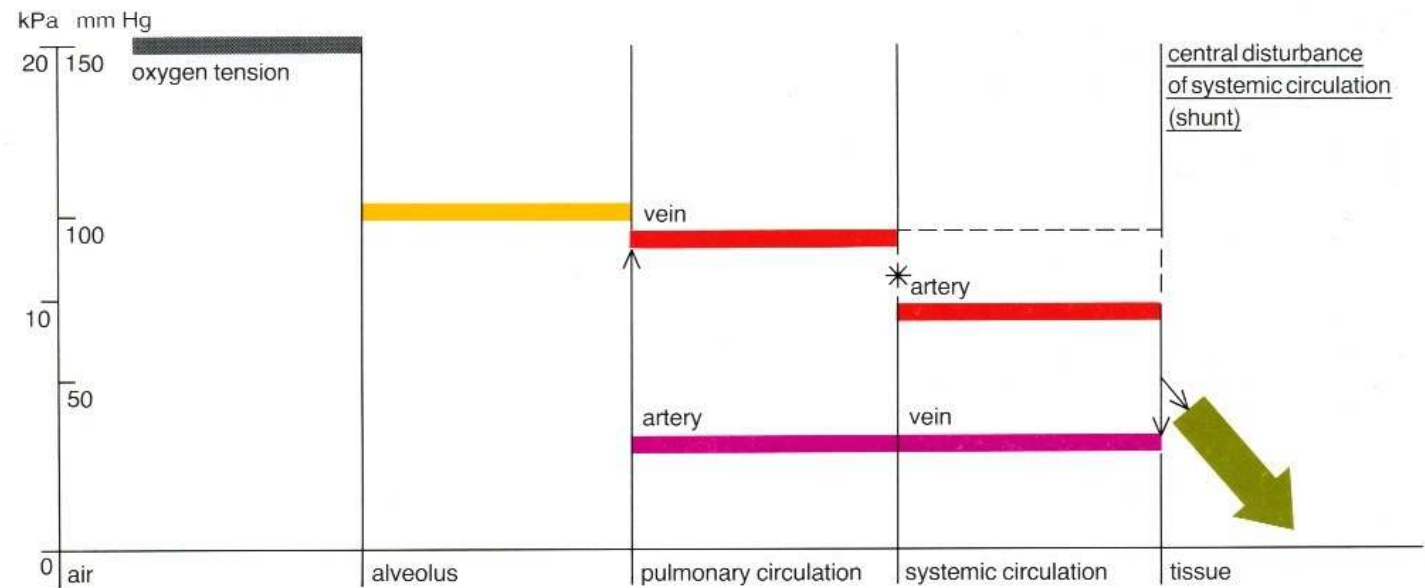
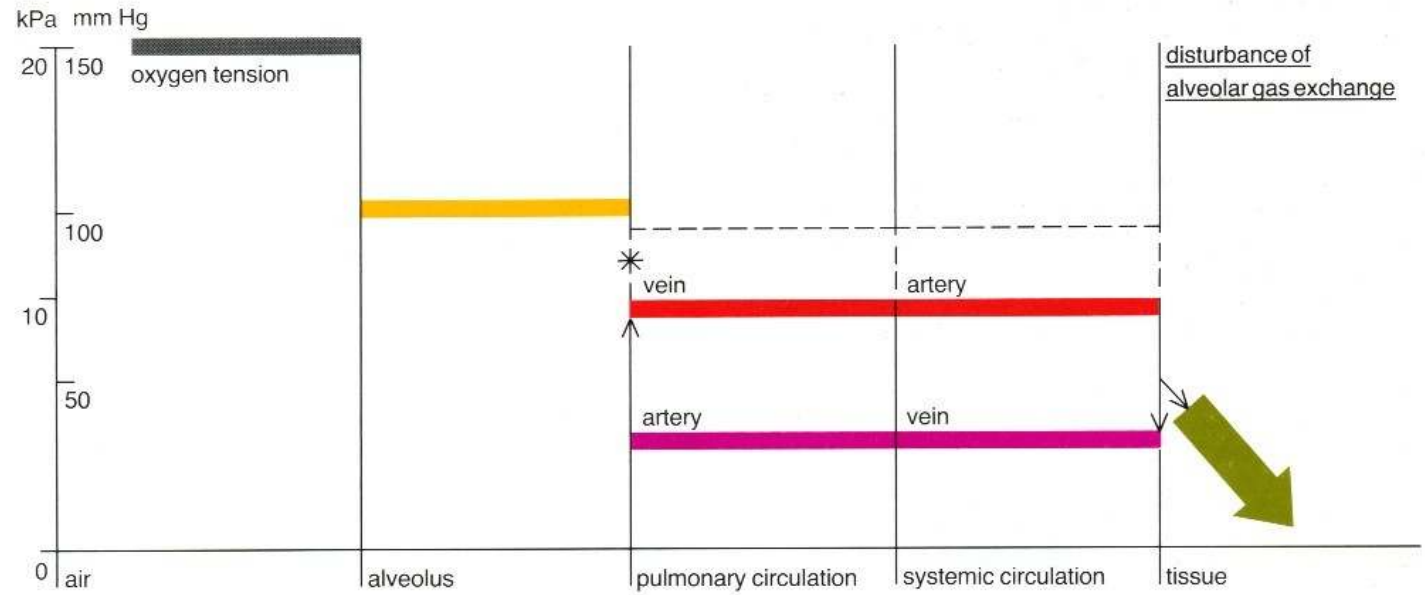




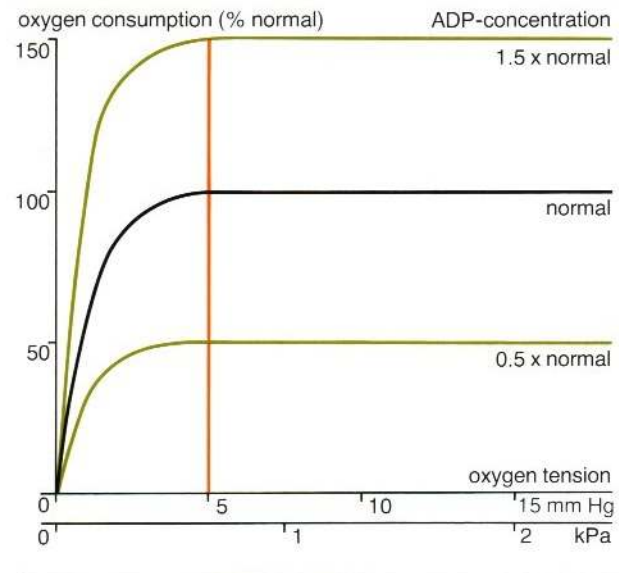
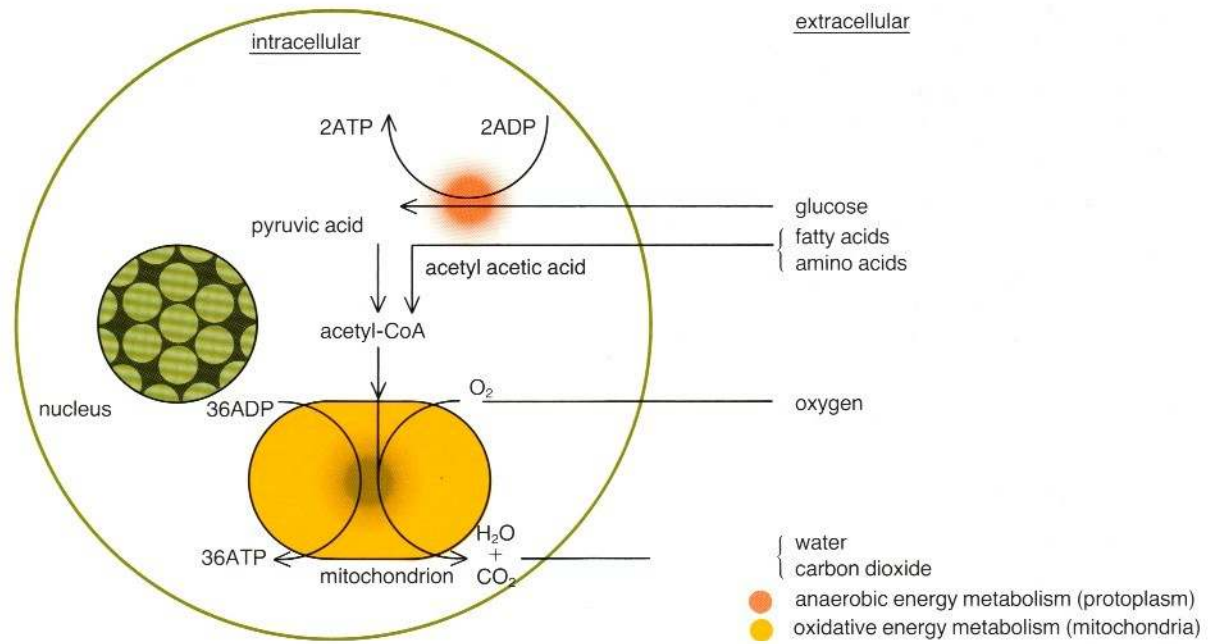
process	compartment	$\dot{V}O_2$ l/min	$\dot{V}O_2$ l	P_{O_2} mm Hg	$\dot{V}CO_2$ l/min	$\dot{V}CO_2$ l	P_{CO_2} mm Hg
	air	0.25	∞	150	0.20	≈ 0	≈ 0
	airway	0.25	0.03	—	0.20	0.02	—
	alveolus		0.40	100		0.17	40
	arterial	0.25	0.80	90–100	0.20	2.70	40
	capillary			35–45			45
	venous						
	extracellular	0.25	0.30	40	0.20	3.30	45
	intracellular			<5			>45
	mitochondria						
		0.25	1.53		0.20	6.19	



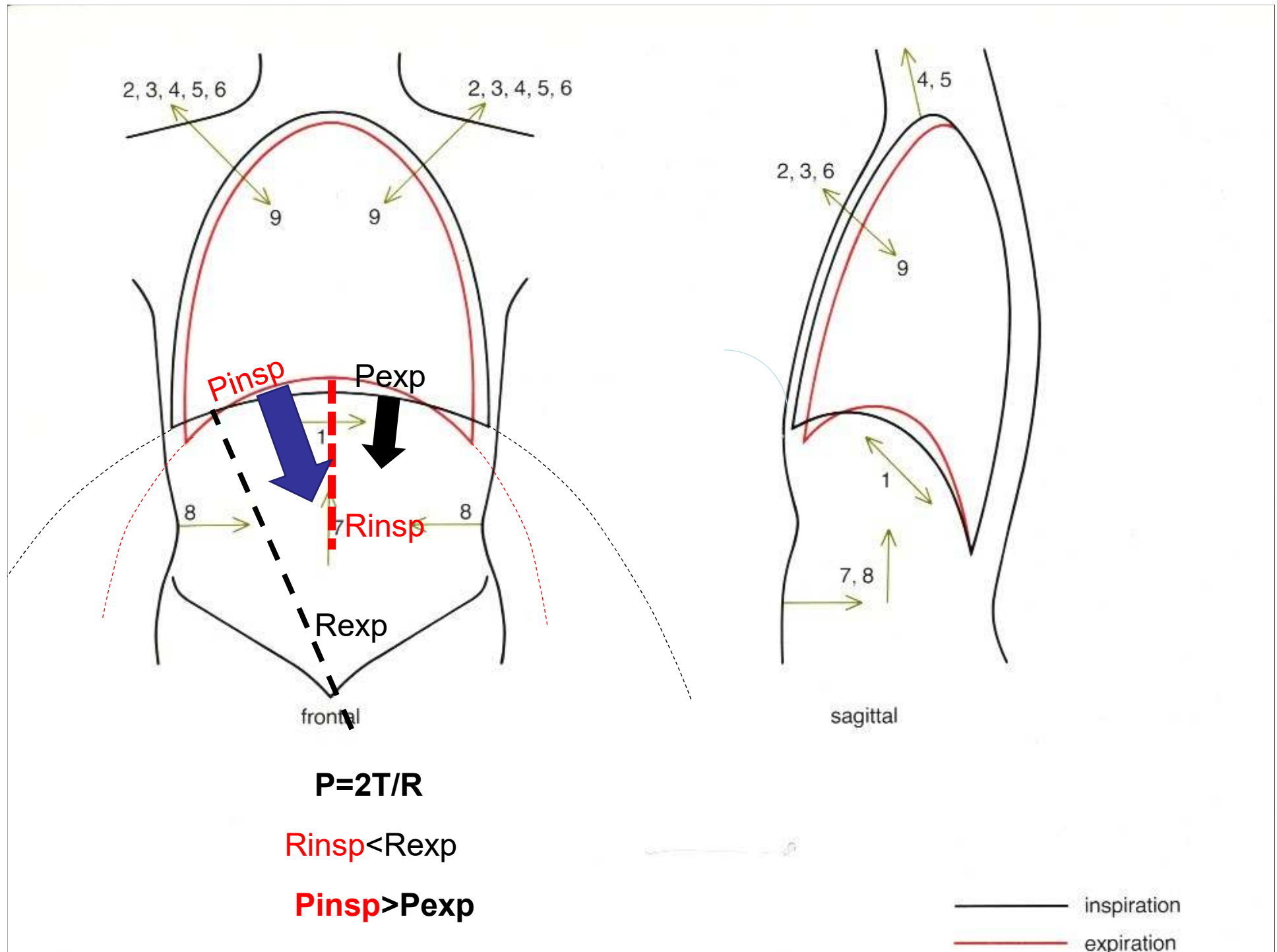
* disturbance
 1 mm Hg = 0.133 kPa (see A3)

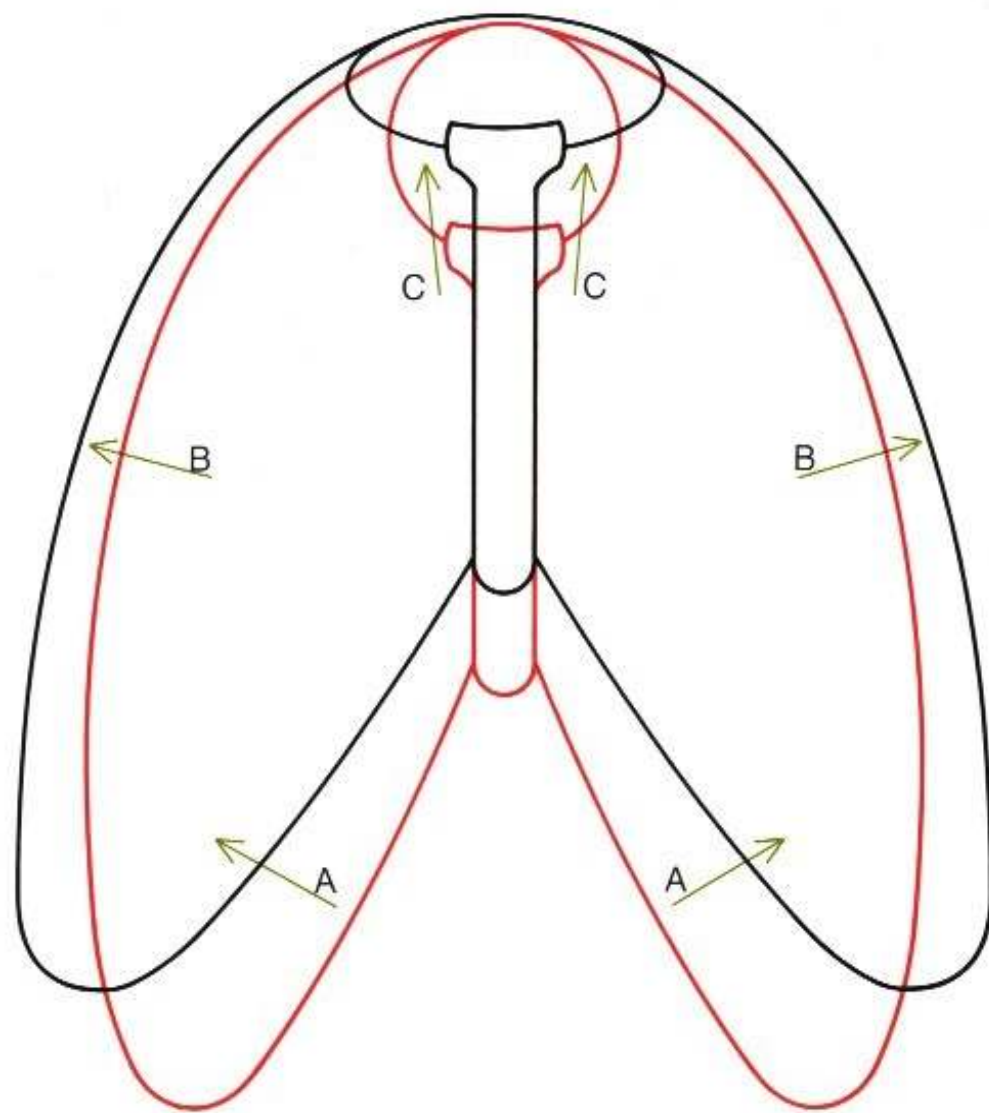


* disturbance
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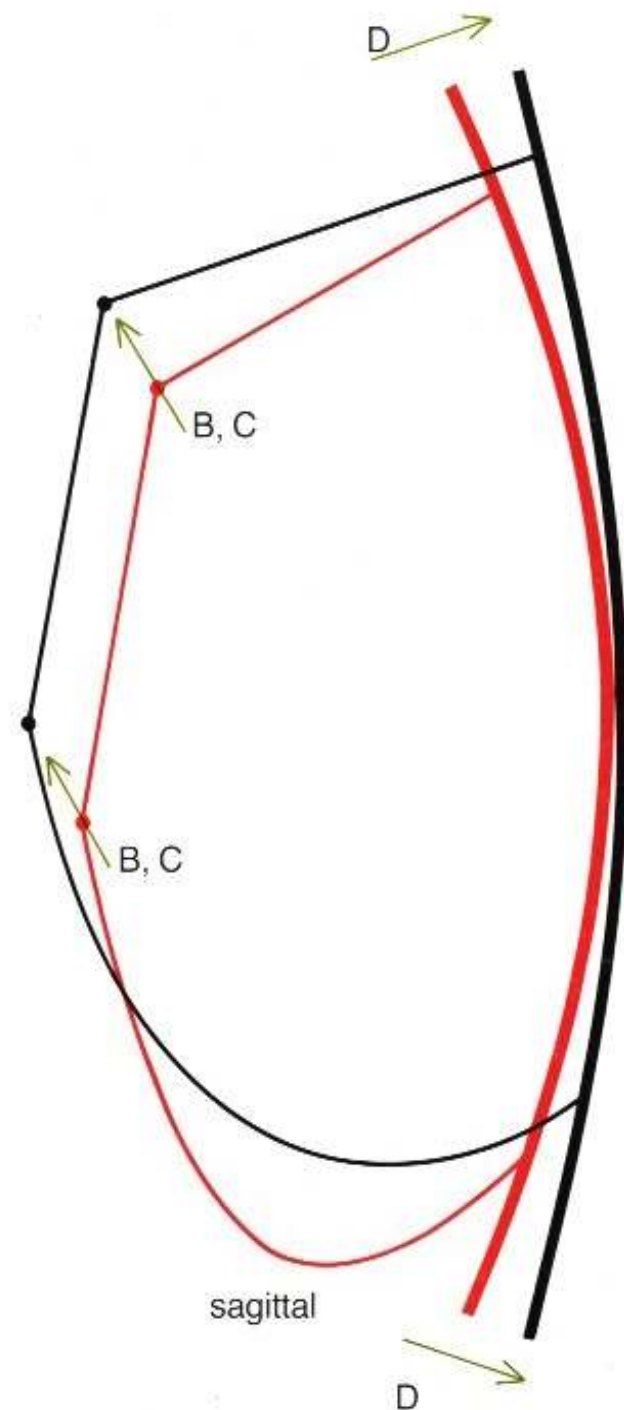


ADP = adenosine diphosphate
ATP = adenosine triphosphate





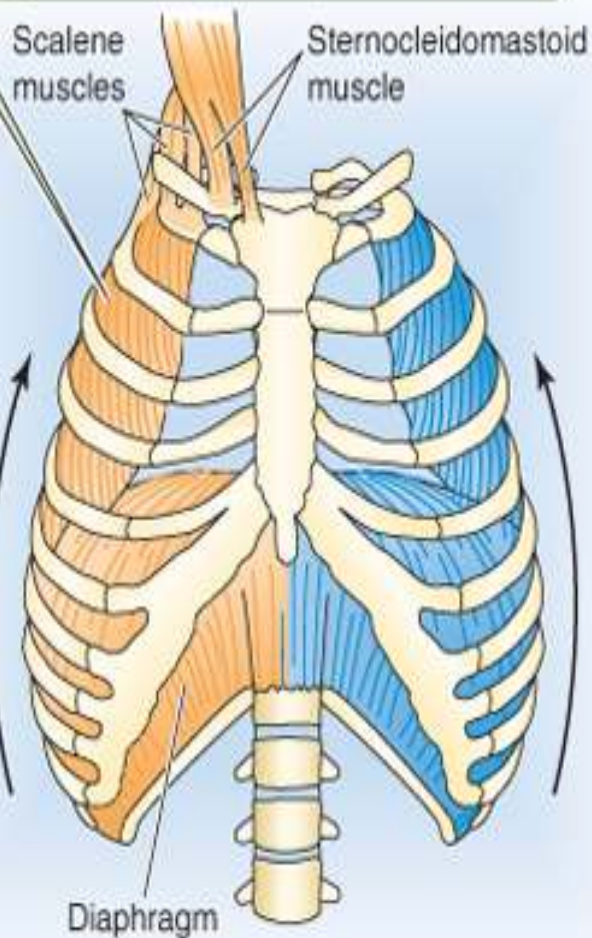
frontal



sagittal

A INSPIRATION

External intercostal muscles slope obliquely between ribs, *forward* and downward. Because the attachment to the lower rib is farther forward from the axis of rotation, contraction raises the lower rib more than it depresses the upper rib.

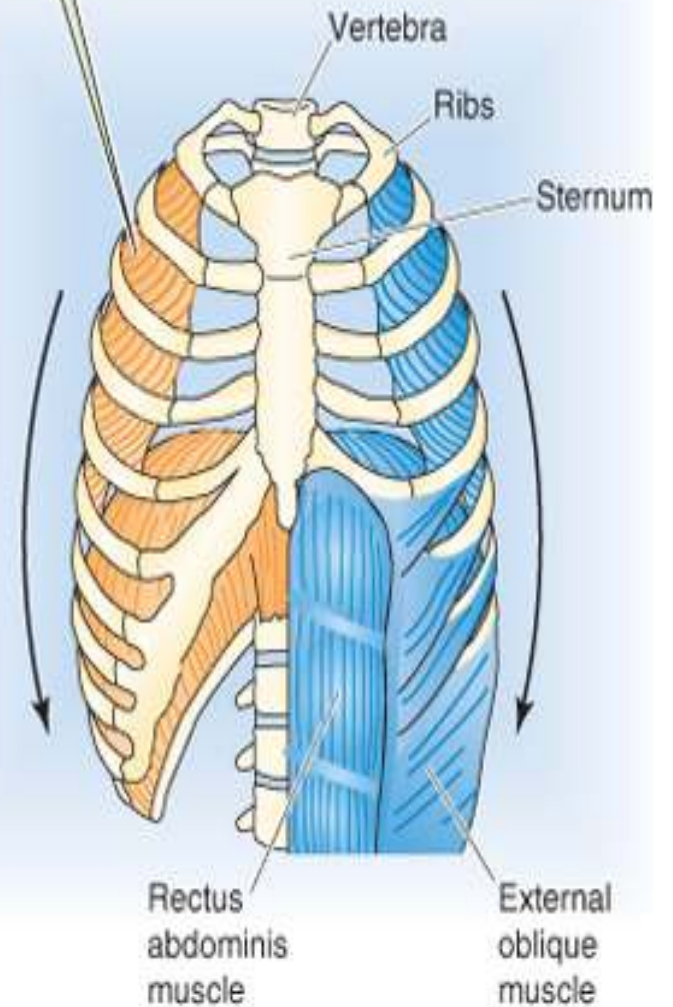


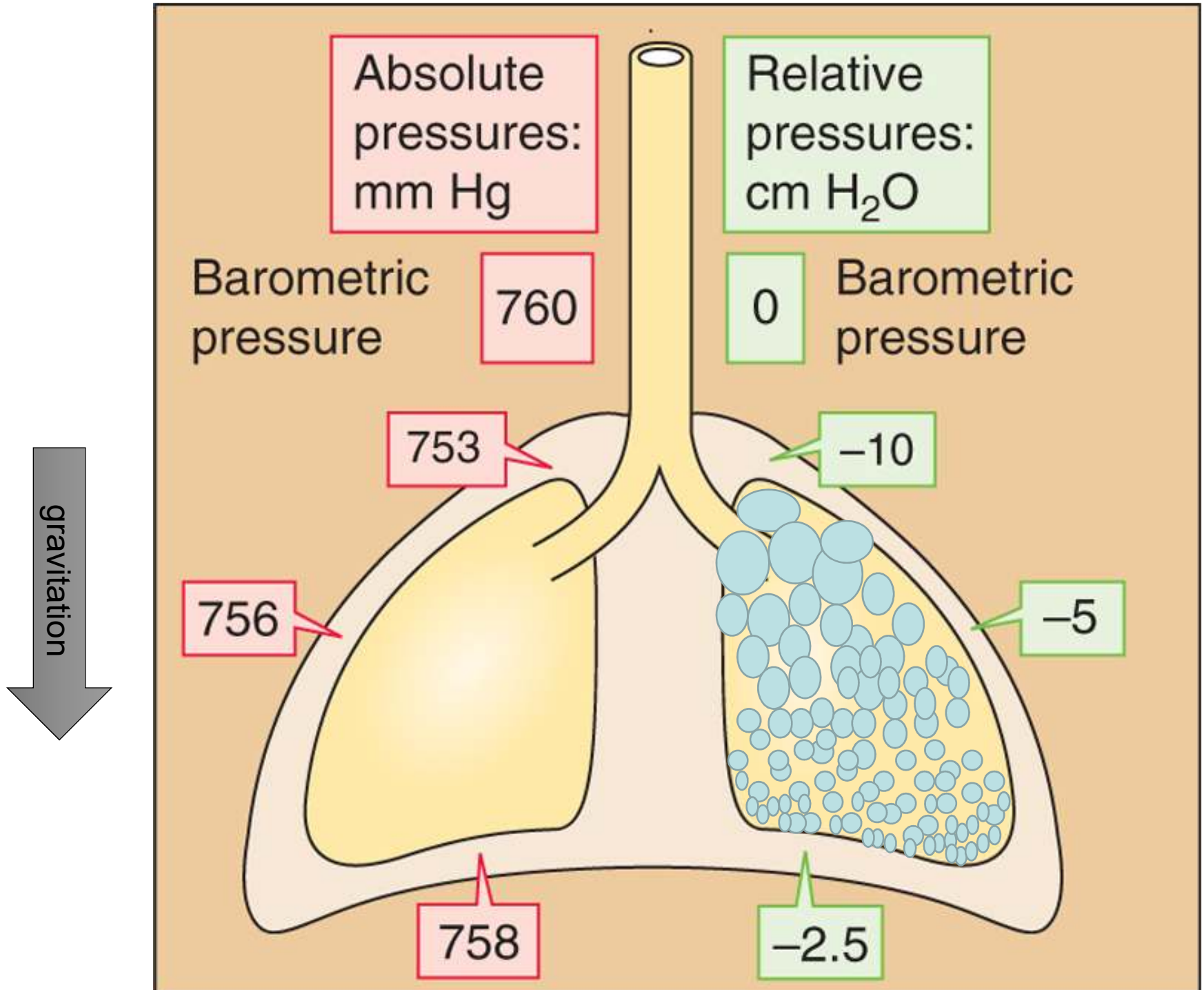
B BUCKET-HANDLE AND WATER-PUMP-HANDLE EFFECTS

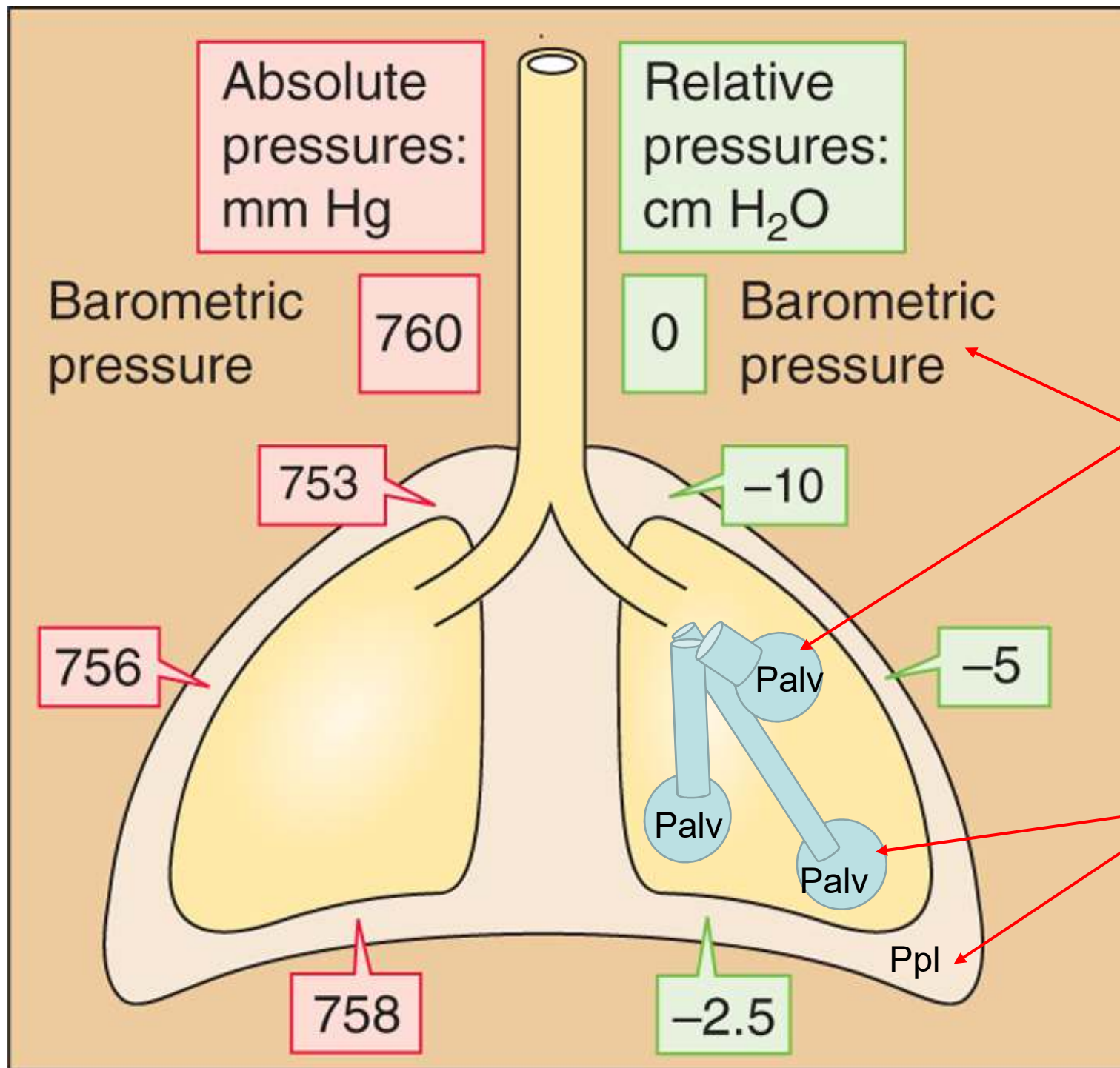


C EXPIRATION

Internal intercostal muscles slope obliquely between ribs, *backward* and downward, depressing the upper rib more than raising the lower rib.



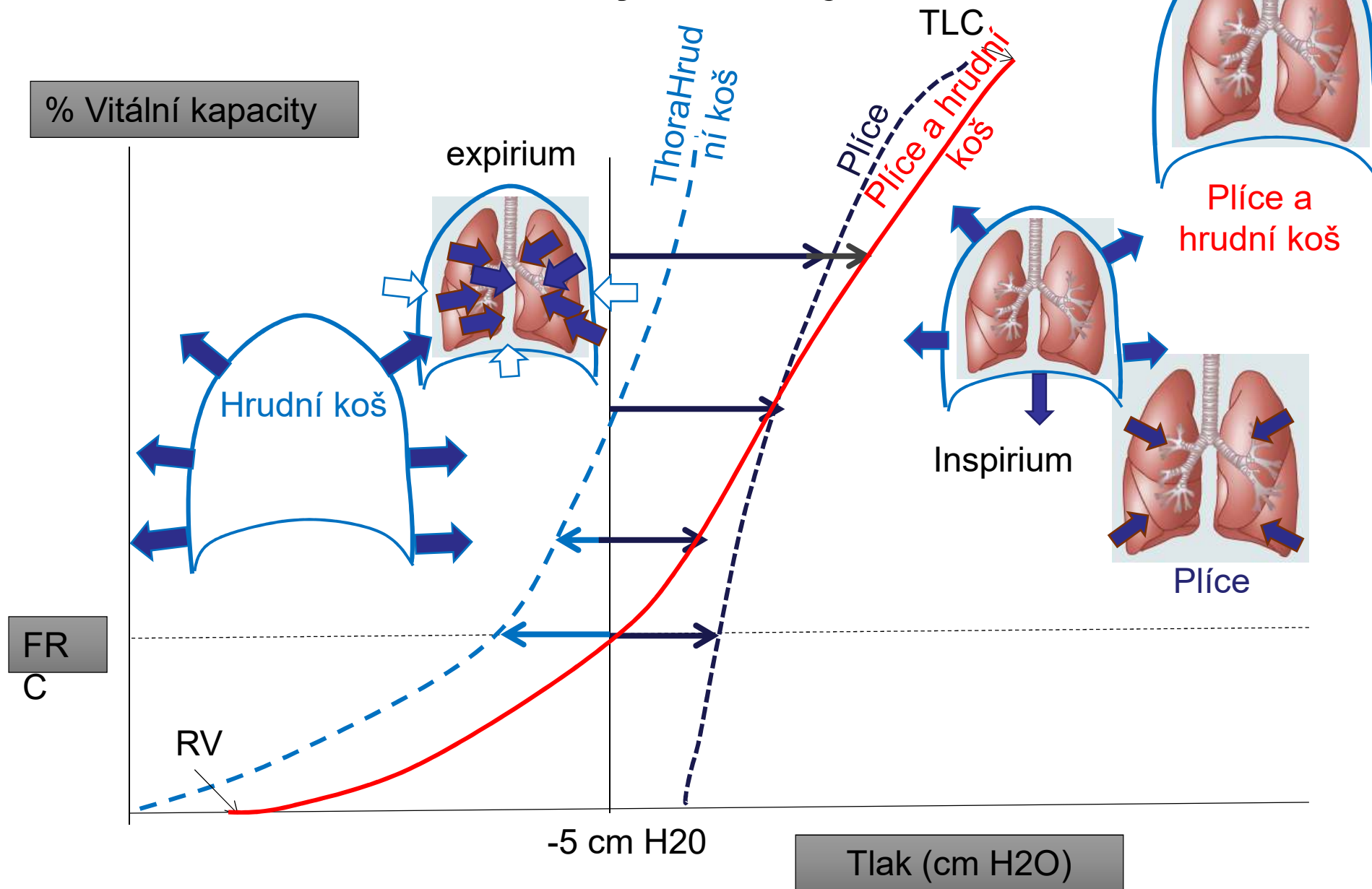




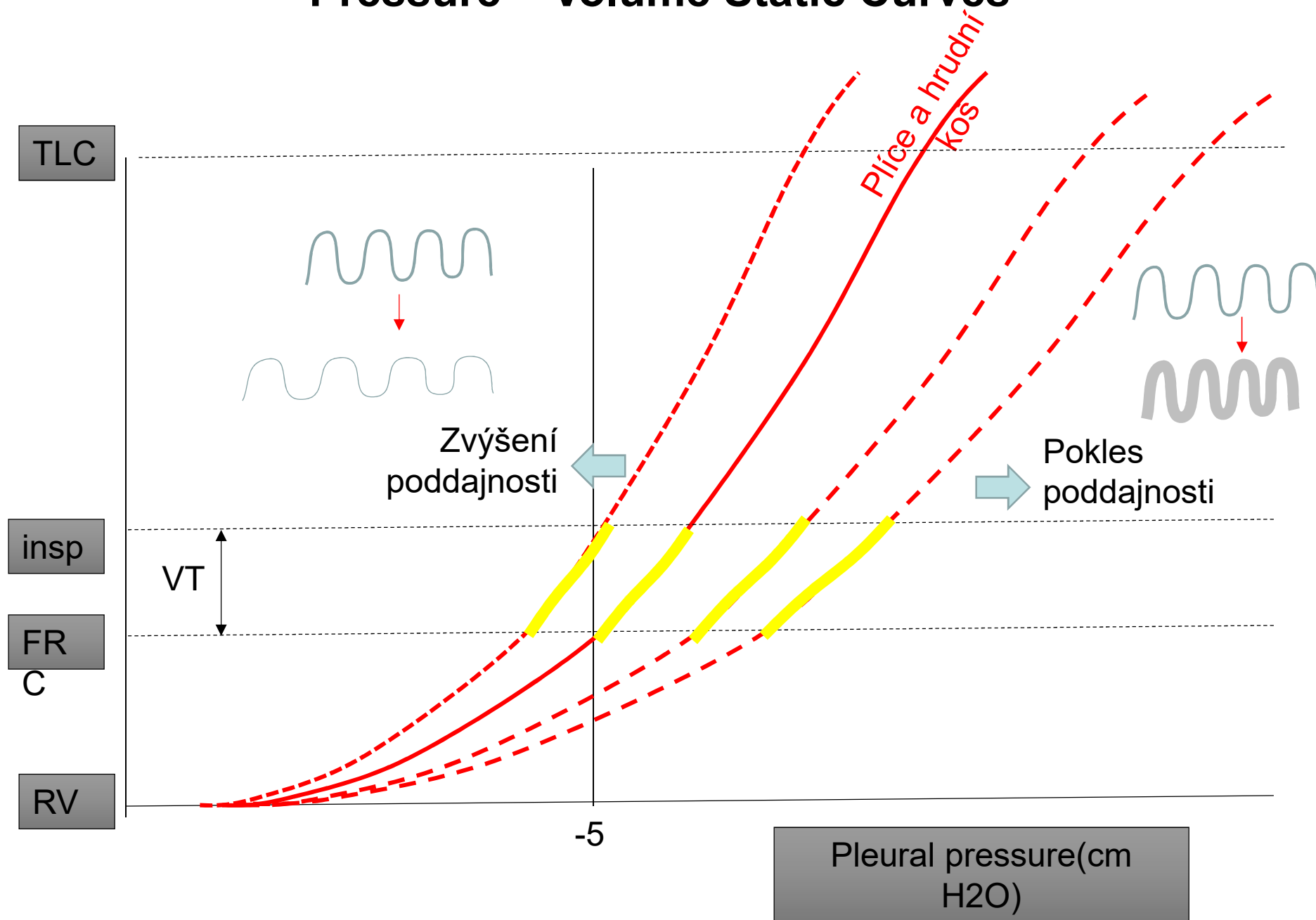
Transthorakální
tlak

Transpulmonární
tlak

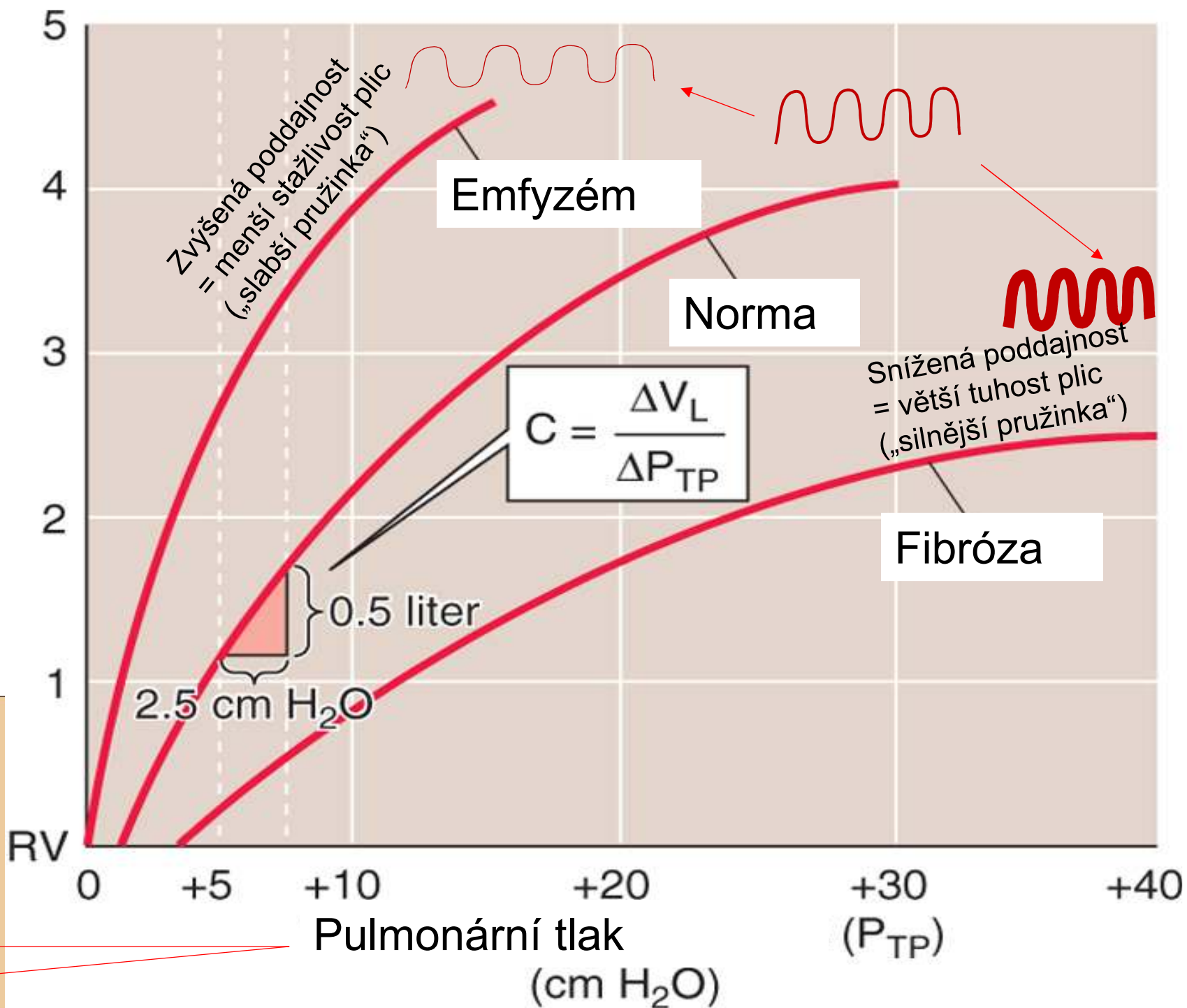
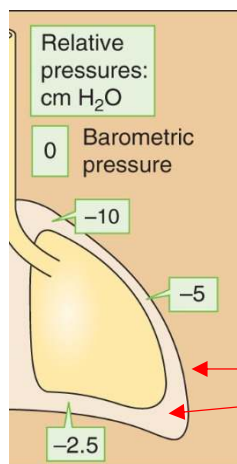
Statické křivky Tlak-Objem

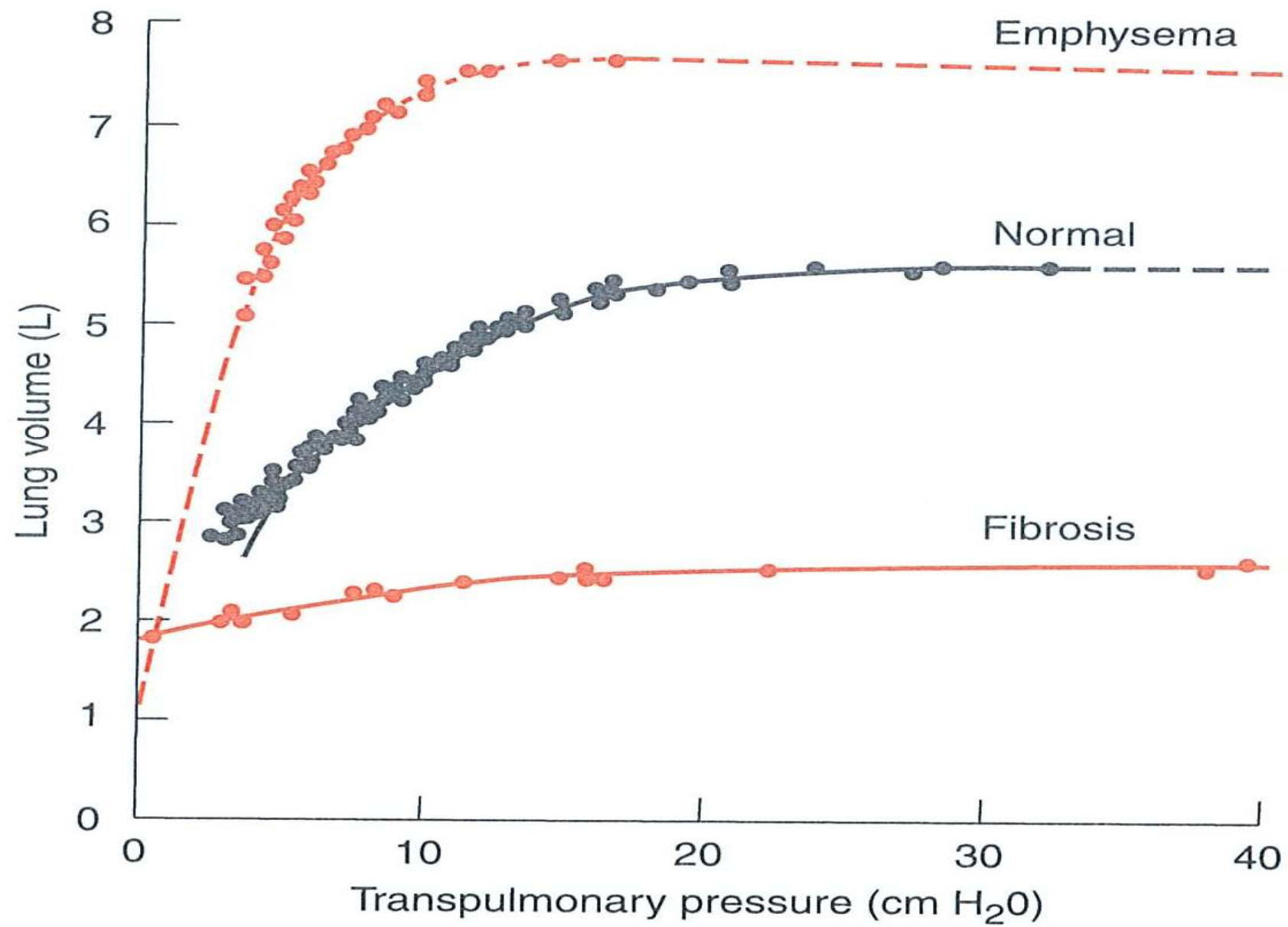


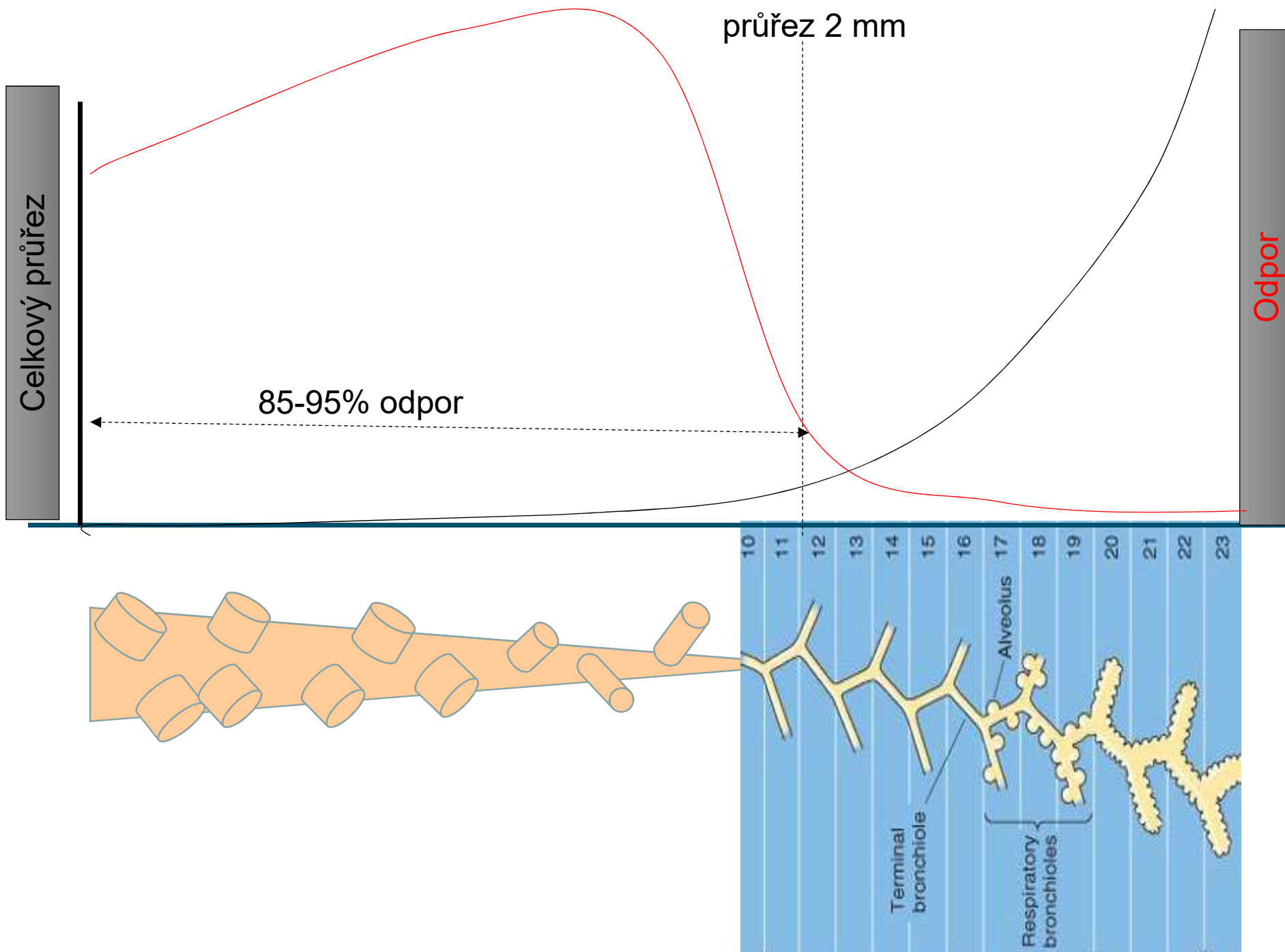
Pressure – Volume Static Curves

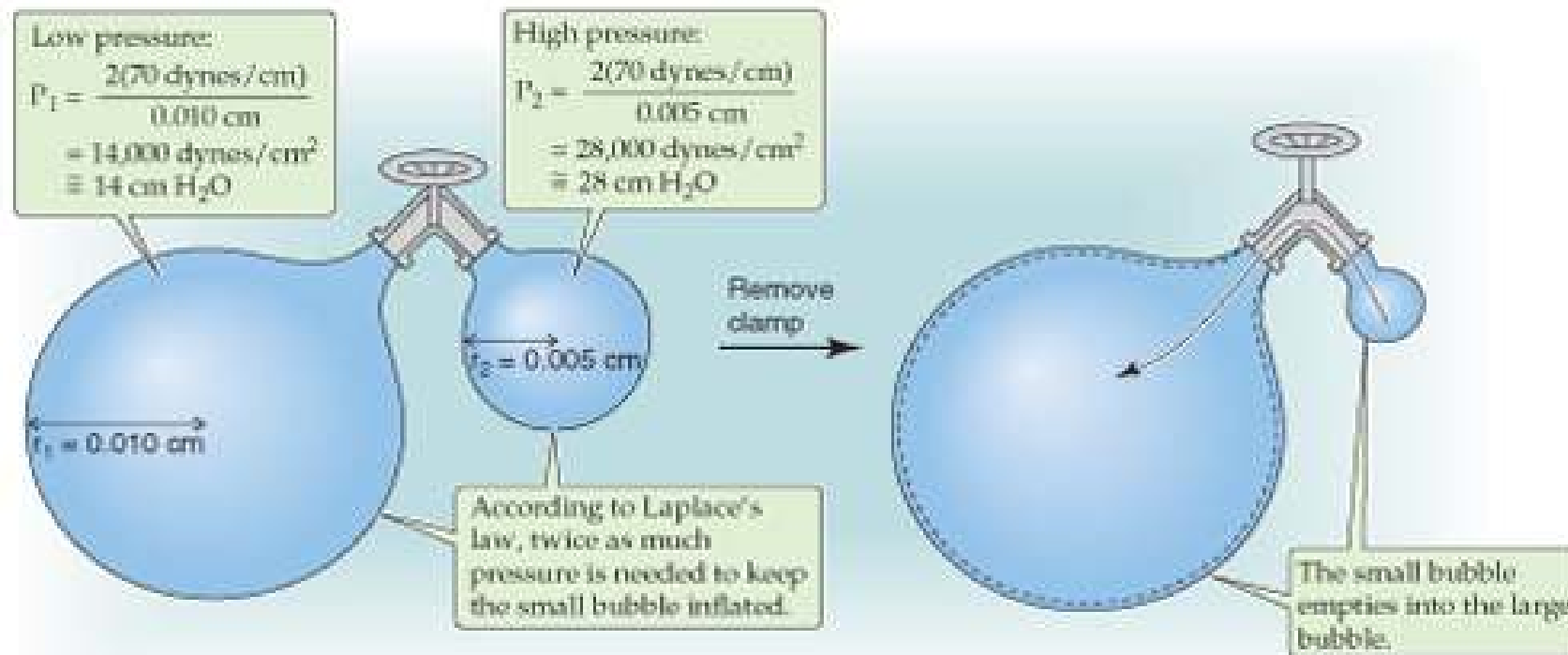


Změna
objemu
plic od
RV
(litry)

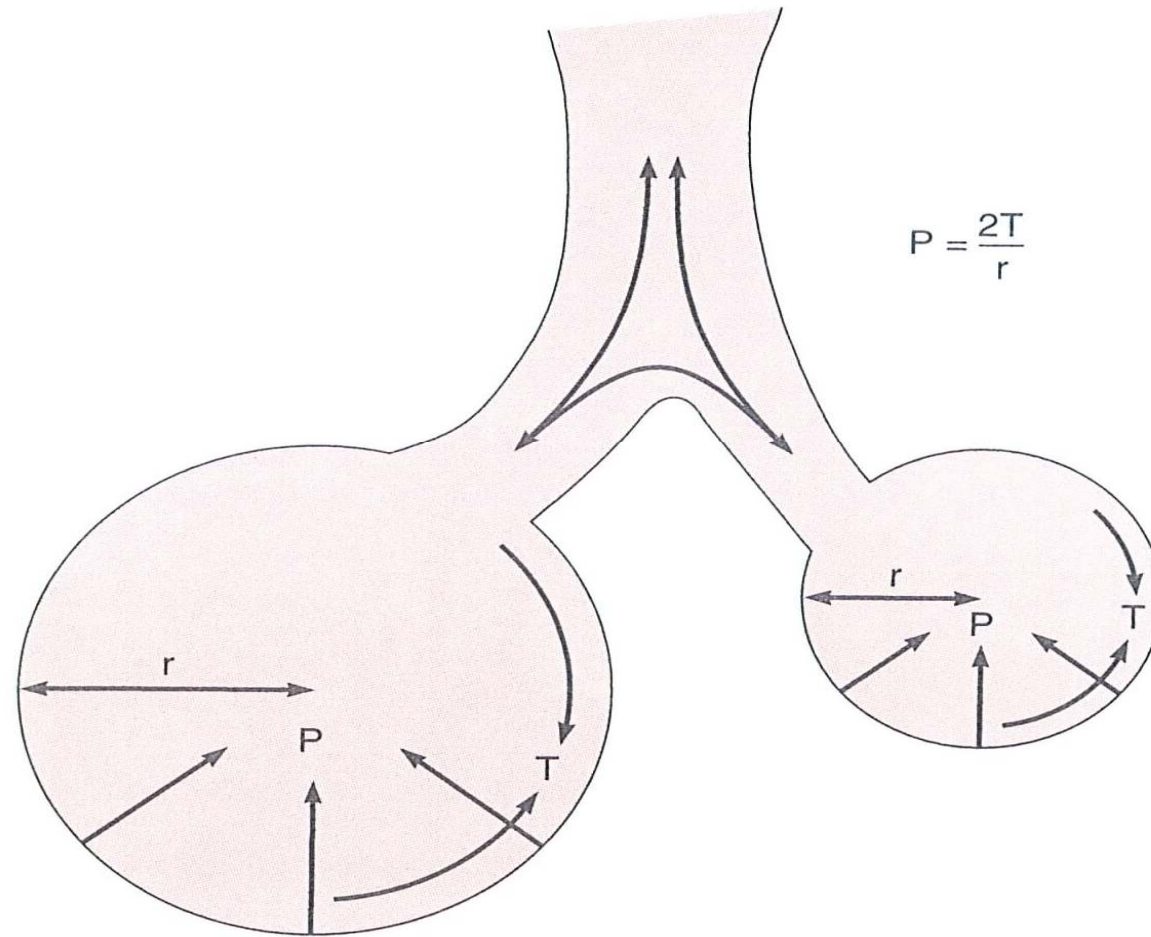




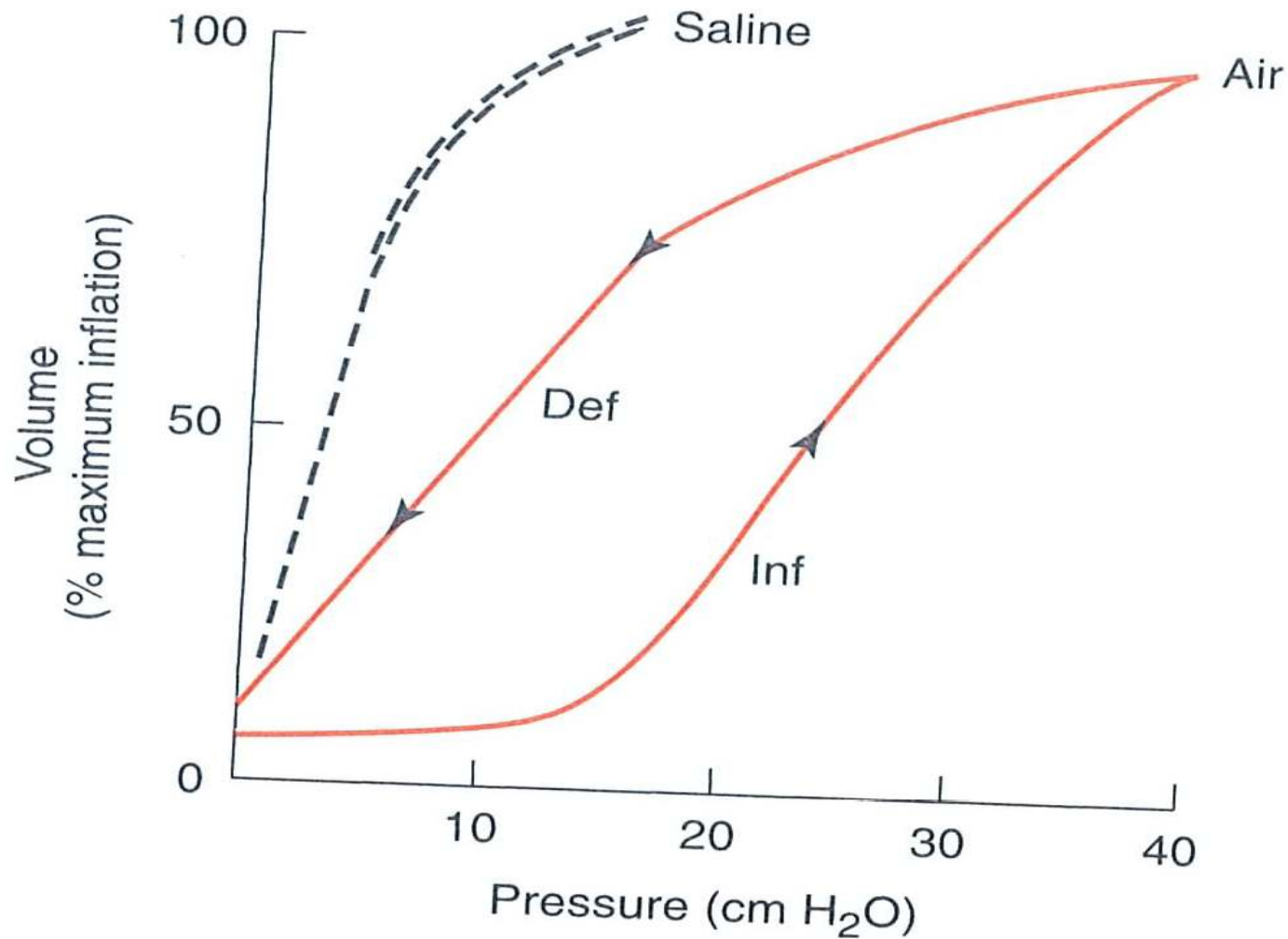




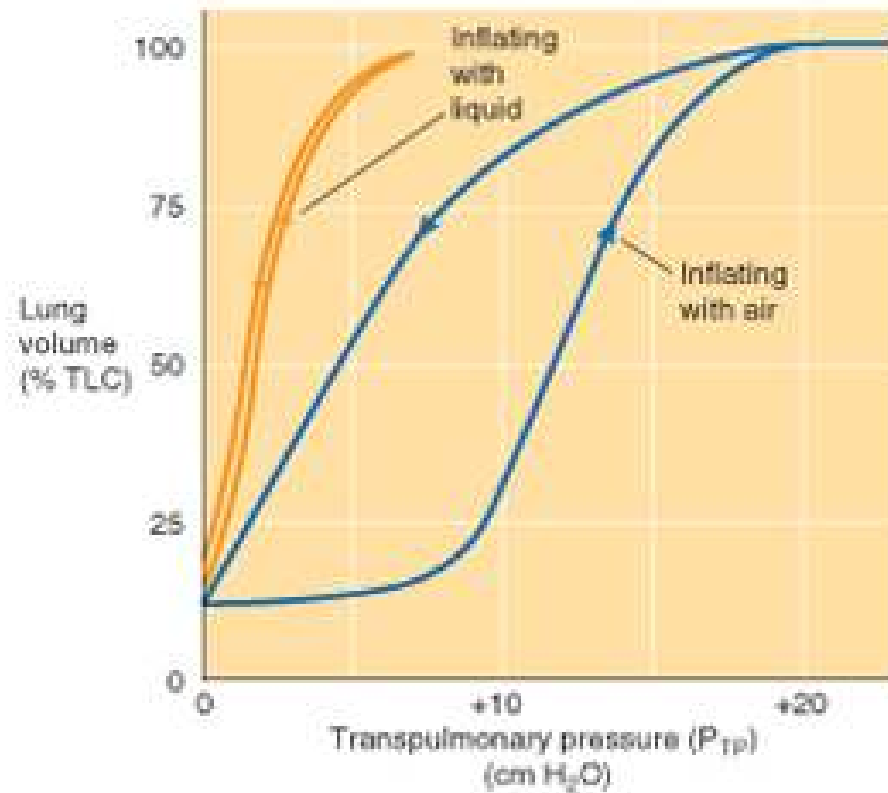
Stabilita rozvinutí alveolů, síly proti kolapsu



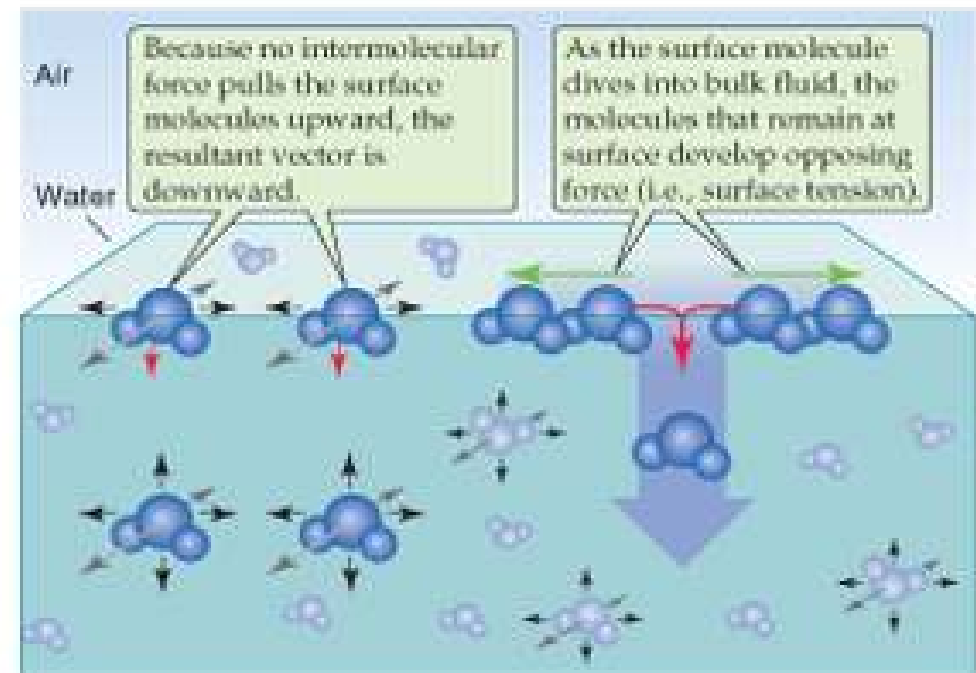
Compliance – effect of surfactant



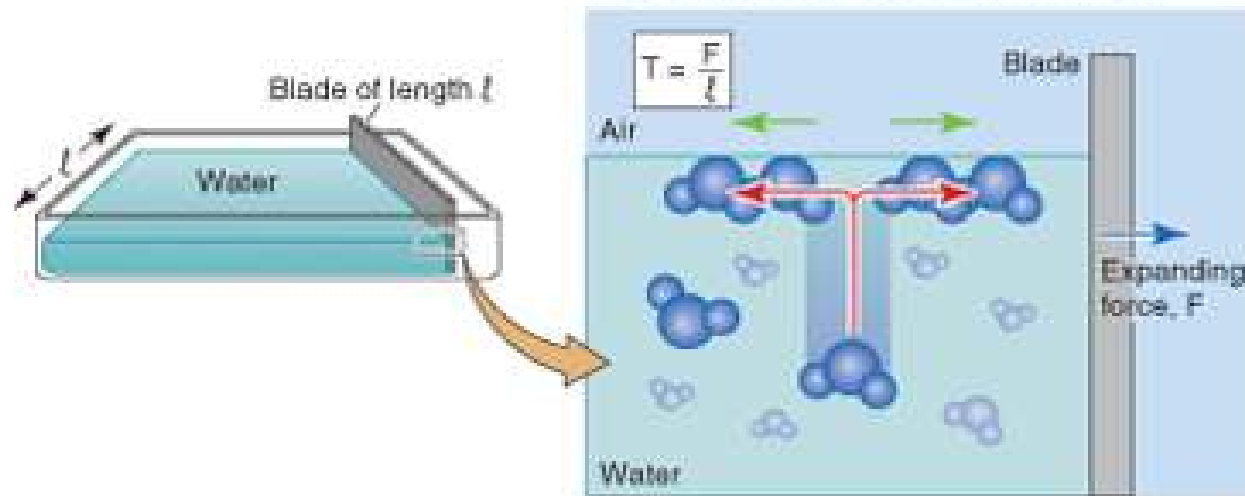
A EFFECT OF SURFACE TENSION ON COMPLIANCE



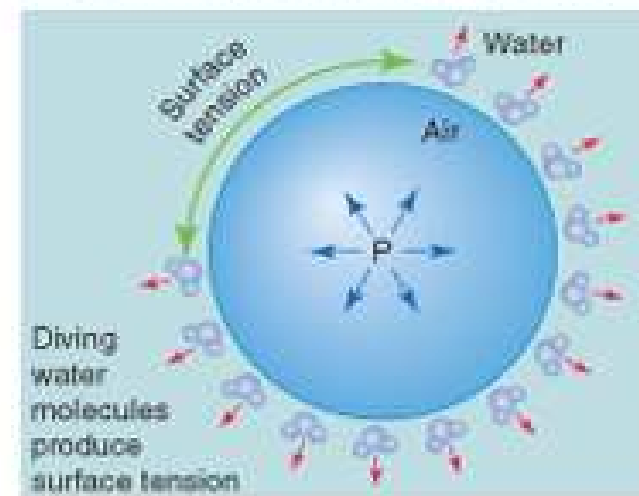
B FLAT AIR-WATER INTERFACE



C DEFINITION OF SURFACE TENSION



D SPHERICAL AIR-WATER INTERFACE



alveolar space
alveolar surface

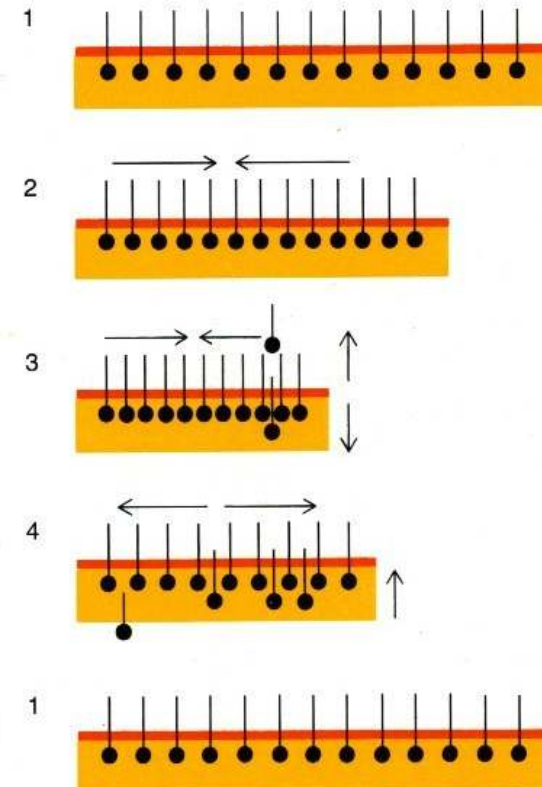
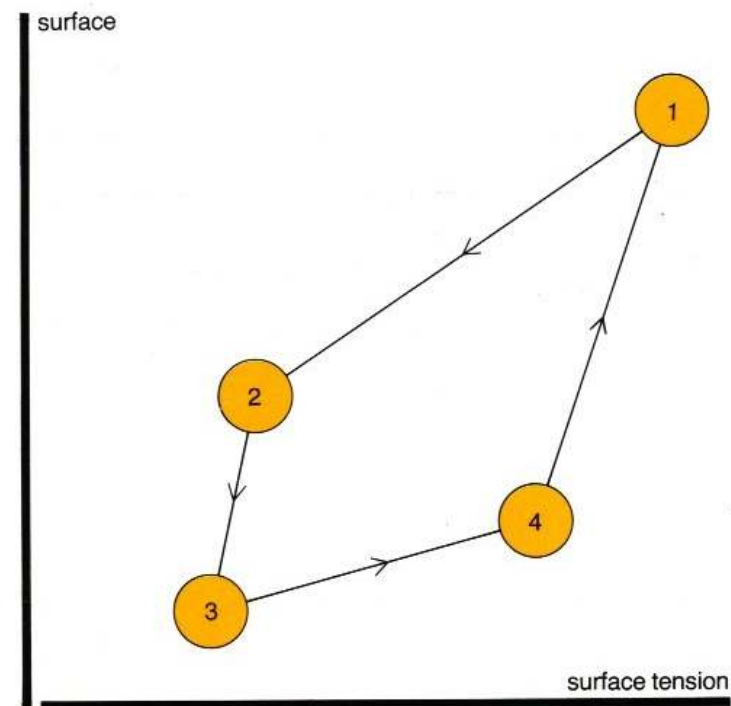
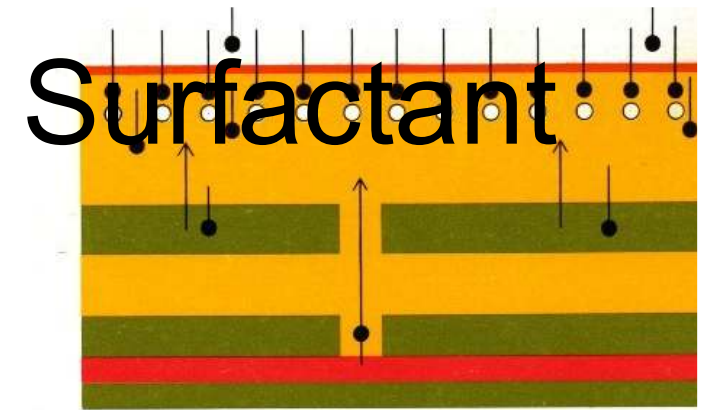
hypophase

granular pneumocyte

alveolar interstitium

endothelial cell

blood capillary



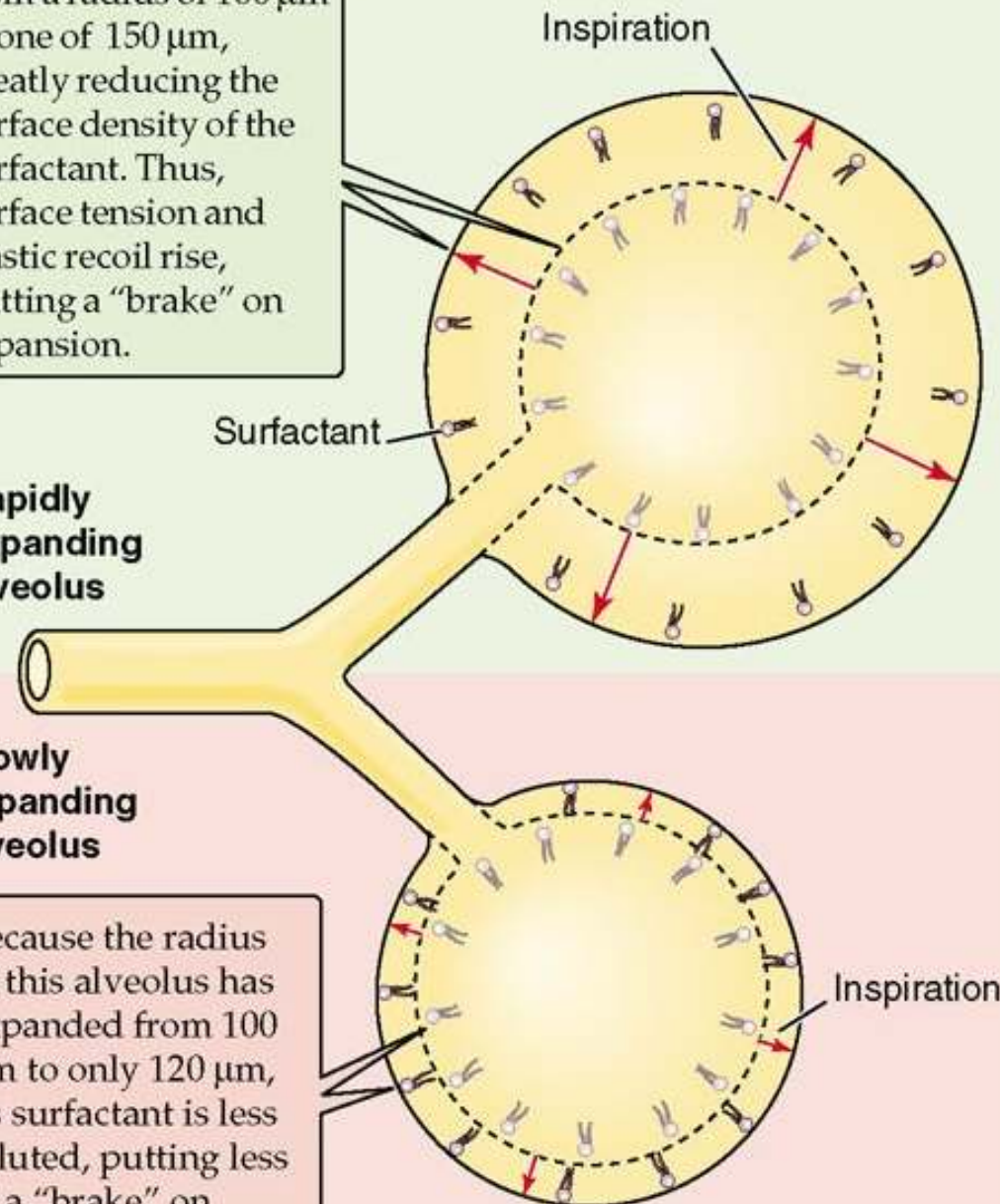
During inflation, the alveolus has expanded from a radius of 100 μm to one of 150 μm , greatly reducing the surface density of the surfactant. Thus, surface tension and elastic recoil rise, putting a "brake" on expansion.

$$P = T / (2R)$$

Rapidly expanding alveolus

Slowly expanding alveolus

Because the radius of this alveolus has expanded from 100 μm to only 120 μm , its surfactant is less diluted, putting less of a "brake" on expansion.



bez surfaktantu:

$\uparrow R$

$$P = T / (2R)$$

se surfaktantem:

$\uparrow T \quad \uparrow R$

$$P = T / (2R)$$

bez surfaktantu:

$\downarrow R$

$$P = T / (2R)$$

se surfaktantem:

$\downarrow T \quad \downarrow R$

$$P = T / (2R)$$

The „most important“ measure of respiratory system function

- pO_2 & pCO_2 in arterial blood - („Astrup“)
- O_2 solubility in water is low => need of Hemoglobin
- **$pO_2 = 13,3 \text{ kPa} = 100 \text{ Torr}$**
- Conversion: $1 \text{ Atm} = 10 \text{ m H}_2\text{O} = 100 \text{ kPa} = 760 \text{ Torr} = 760 \text{ mmHg}$
..... $1 \text{ kPa} = 10 \text{ cm H}_2\text{O} = 7,6 \text{ Torr}$
- **$pCO_2 = 40 \text{ Torr} = 5,3 \text{ kPa}$**

Alveolární ventilace

$$V_{CO_2} = F_A CO_2 * V_A$$

$$V_A = V_{CO_2} / F_A CO_2$$

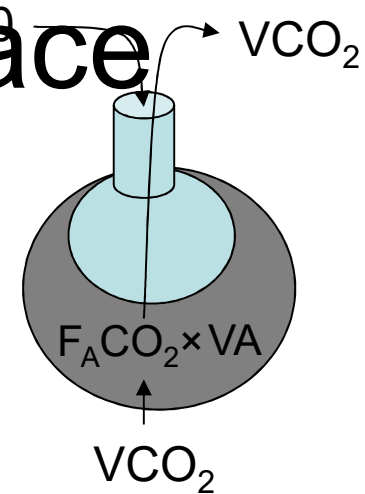
$$P_A CO_2 = F_A CO_2 \times \text{Barometrický tlak}$$

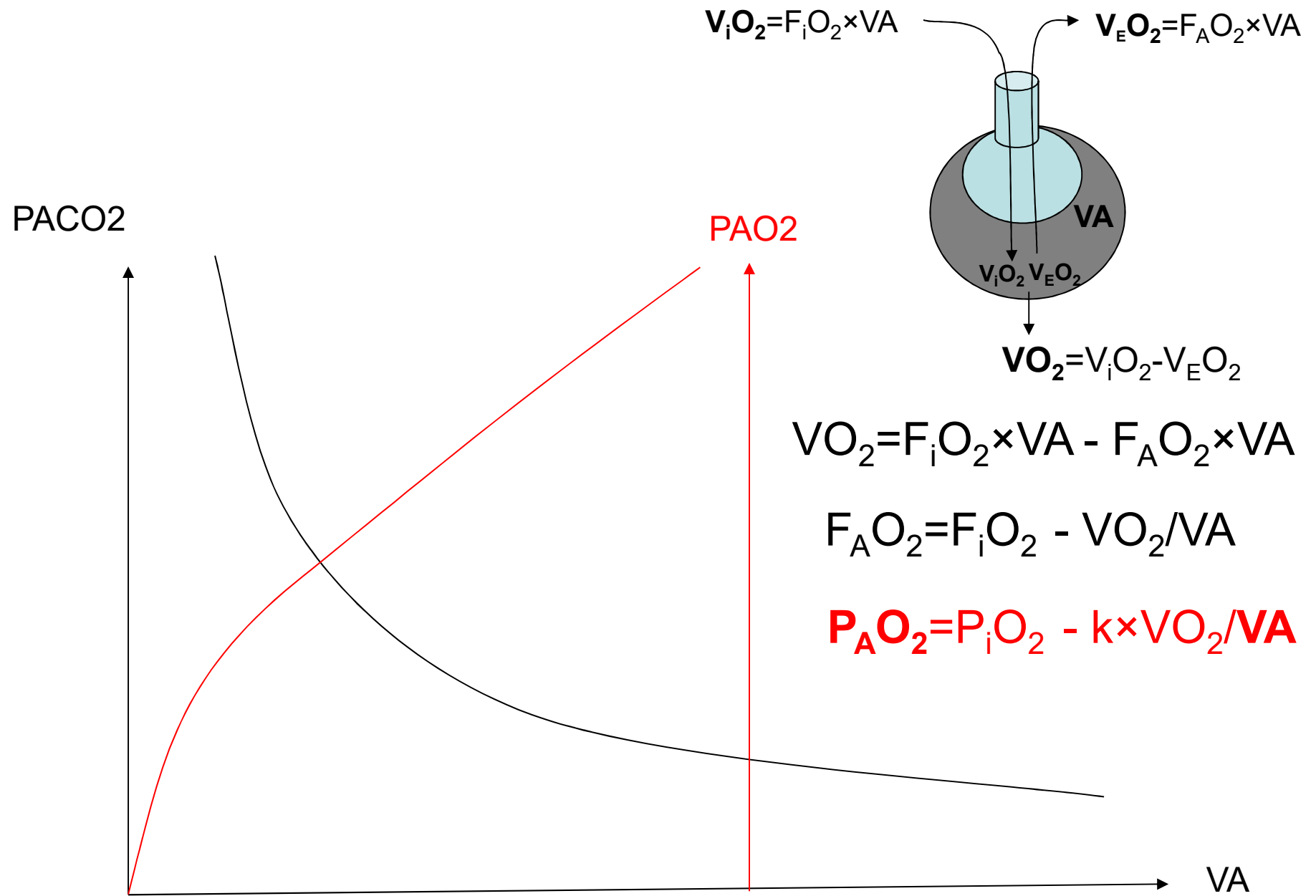
$$V_A = k_1 \times V_{CO_2} / P_A CO_2$$

$$P_A CO_2 = k_2 \times \underset{\substack{\uparrow \\ \text{STPD}}}{V_{CO_2}} / \underset{\substack{\uparrow \\ \text{BTPS}}}{V_A}$$

$\frac{P \times V}{T} = R$	$\frac{(P - P_{H_2O}) \times V_{BTPS}}{273 + t^{\circ} \text{patient}} = \frac{760 \times V_{BTPS}}{273}$
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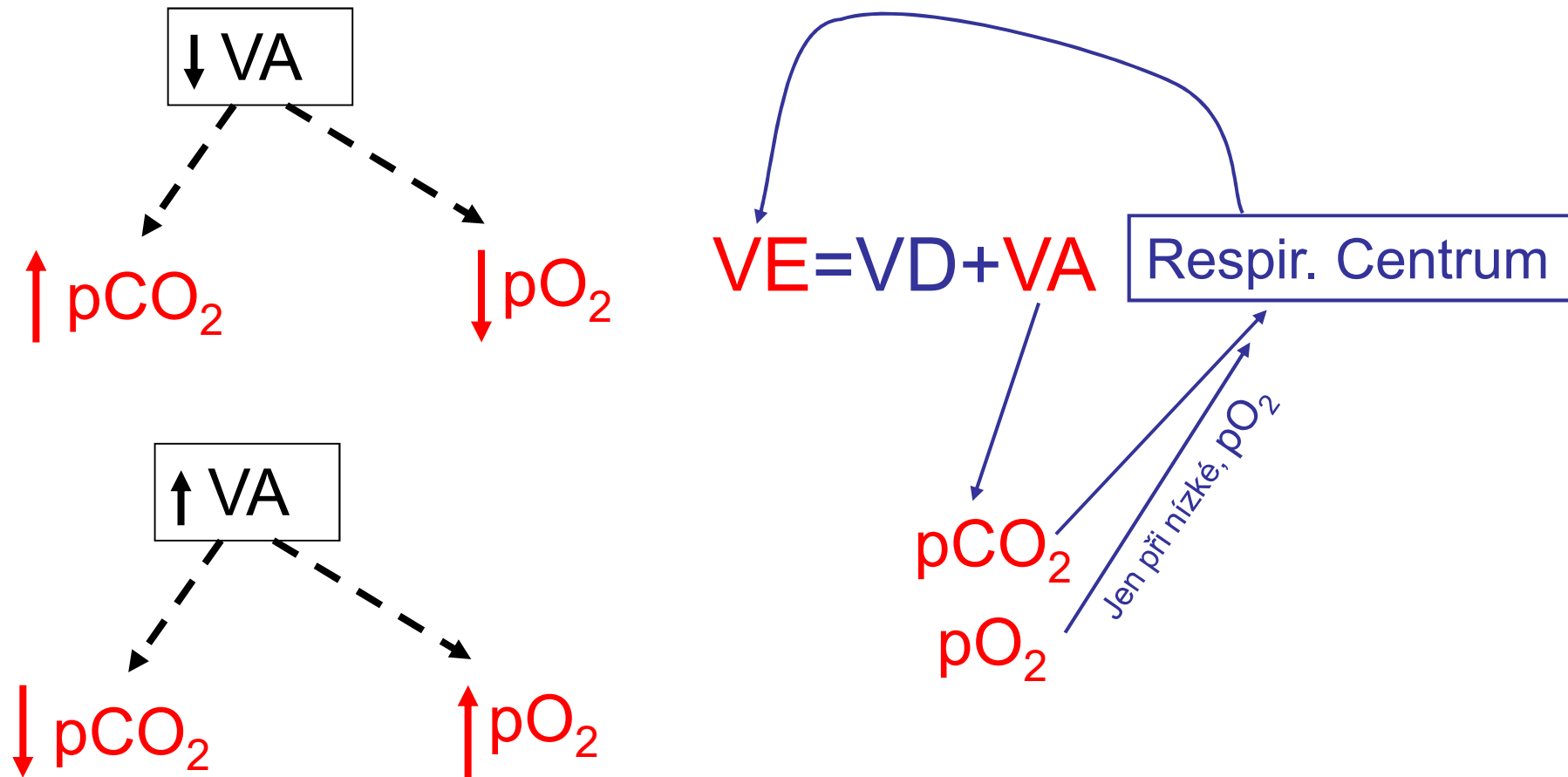
$$P_A CO_2 [\text{torr}] = 0,863 * V_{CO_2} [\text{ml/min STPD}] / V_A [\text{l/min BTPS}]$$





$$P_ACO_2[\text{torr}] = 0,863 \times VCO_2[\text{ml/min STPD}] / VA[\text{l/min BTPS}]$$

Chemorecepční řízení ventilace



Ventilation disorders

- **Lung impairment (mechanical properties change)**
 - **Obstructive disease** - $\uparrow$ increased resistance R of airways (R = “dynamic lung resistance”)
 - **Restrictive disease** – $\downarrow$ decreased lung compliance C ($\uparrow$ static resistance” `; $C = 1/$ static lung resistance)
- **Chest wall impairment**
 - $\downarrow$ decreased C of chest wall – severe scoliosis, extensive fibrosis, serial fractures
- **Insufficient activity of respiratory muscles** ($//$ innervation or $//$ muscle strength , $//$ of CNS) – E.g..
Respiratory centre suppression in barbiturate poisoning, myasthenia gravis

Lung fibrosis = Interstitial lung disease (ILD)

- = diffuse parenchymal lung disease
- Inflammation in alveolar wall leads to scarring and collagen deposition
- Chest X-ray, pulmonary function testing, (lung biopsy)
- Affect the alveolar wall or the interstice of the lung (alveolar epithelium, capillary endothelium, basal membrane, interstice and perilymphatic tissue)
- Fibrosis may be a late sign

Causes of lung fibrosis/ ILD

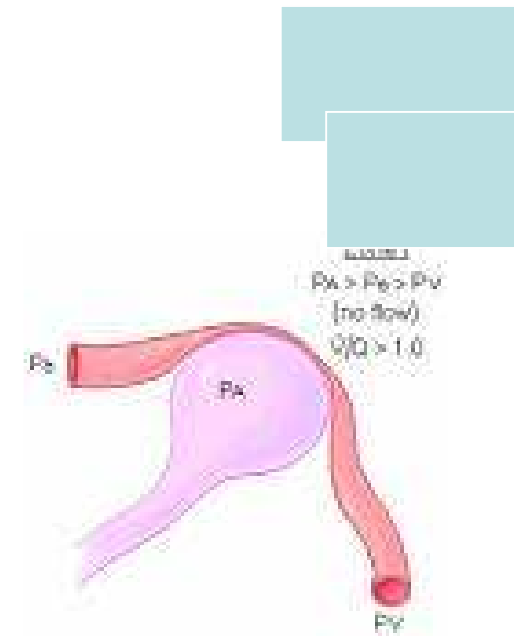
- Inhalation of particles
 - 1. Asbestosis, silicosis, pneumoconiosis, = **Pneumokonioser**
 - 2. hypersensitivity (farmer's lung) = **Alveolitis allergica**
- Drug induced (Abio, chemo, Antiarrhythmic) =
- Connective tissue disease: Systemic sclerosis, Dermatomyositis, SLE, RA
- **Infection:** Atypical pneumonia, pneumocystis, TBC
- Lymphangitic carcinoma = **maligne sygdrome**
Idiopathic: **Sarcoidosis**
- Idiopathic pulmonary fibrosis = **Alveolitis fibrosa**

Fibrosing alveolitis= Idiopathic pulmonary fibrosis

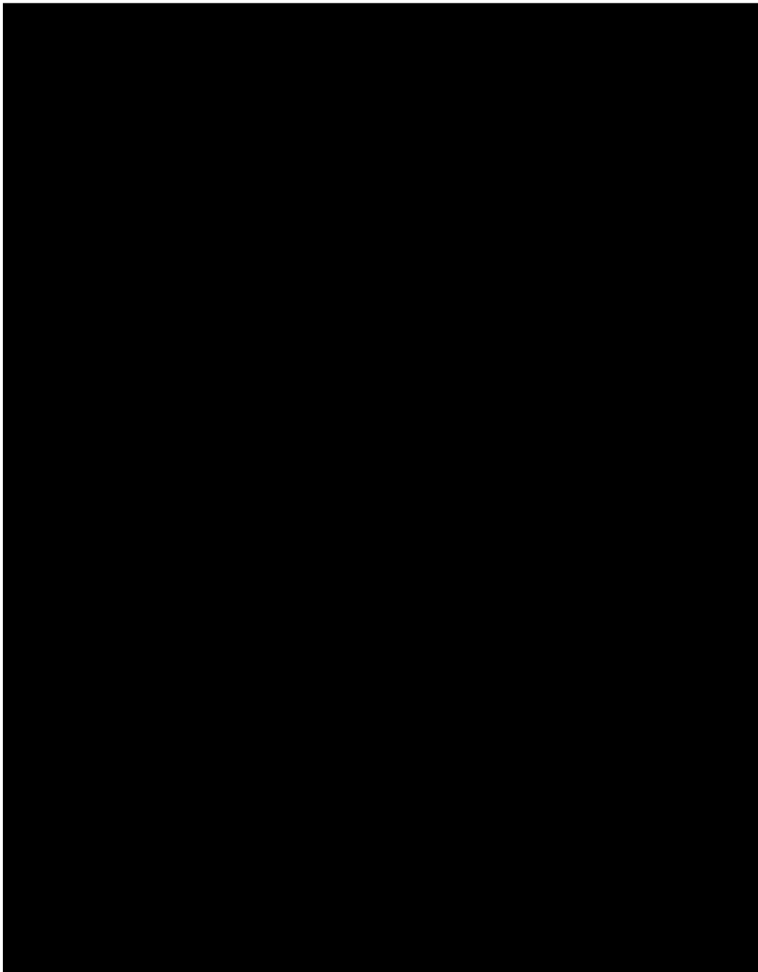
- Unknown cause – Autoimmunity?
- People after 50 / 3 years life expectancy
- Symptoms:
 - Tachypnoe, cyanosis, finger clubbing
- Dg:
 - Chest X-ray
 - HRCT
 - Spirometry/whole body pletysmography
 - Blood gas analysis
 - Inflammatory and autoimmunity testing
- Biopsy and histology: Histology: Usual interstitial pneumonia

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- Porucha **perfúze**
- Porucha **distribuce**
ventilace a perfúze
= ventilačně perfúzní
nerovnováha
- Porucha **difúze**



Ventilace

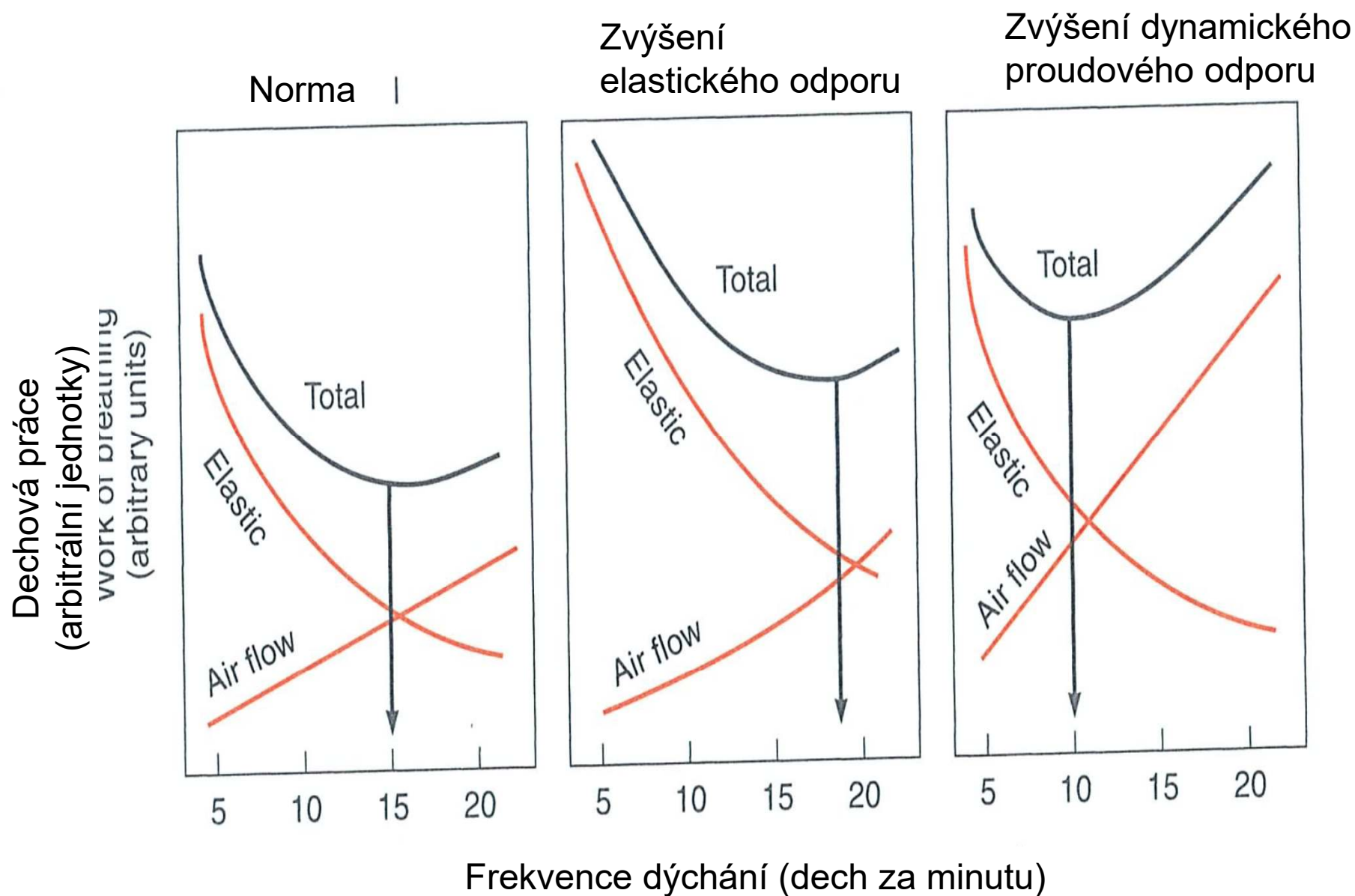


- Je uskutečňována respiračními svaly a bránicí, které mění nitrohrudní objem.
- Objemové změny způsobují změny tlaků
- Změna tlaků vedou k průtoku vzduchu
- ($\uparrow$ tlak – expirium;
- $\downarrow$ tlak – inspirium) podle Ohmova zákona

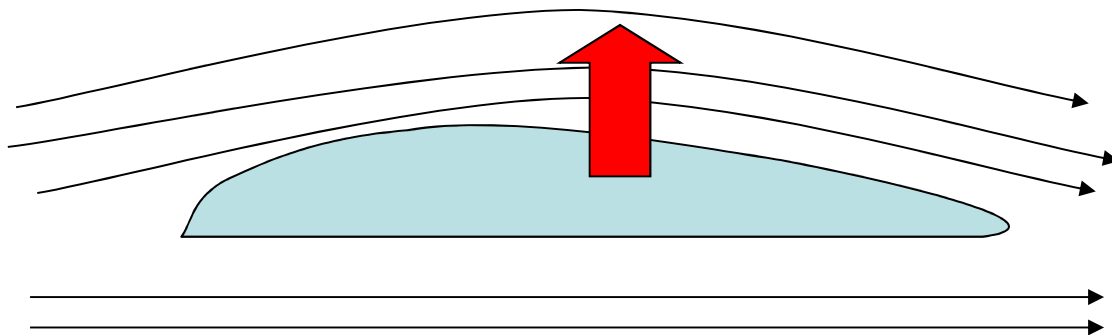
Poruchy ventilace

- **Zhoršení mechanických vlastností plic**
 - **Obstrukční choroby** - zvyšuje se odpor R dýchacích cest vůči proudění (odpor R = dynamický odpor plic)
 - **Restrikční poruchy** – snižuje se poddajnost plic C (poddajnost $C = 1 / \text{statický odpor plic}$)
- **Nedostatečná aktivita dýchacích svalů**
(poruchy inervace či síly svalů, poruchy řízení ze strany CNS) – Př. Útlum dechového centra při otravě barbituráty, myasthenia gravis

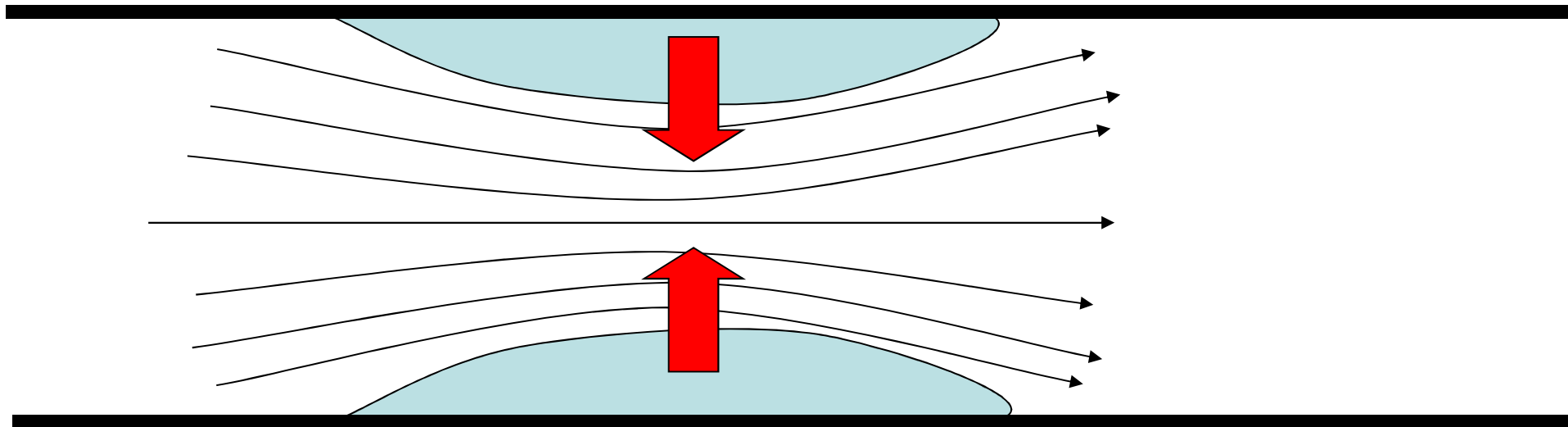
Norma Restrikční Obstrukční



Proč létá letadlo?



Zúžení bronchiolu (bronchokonstrikce, hlen...)



Shrnutí

- **Plicní poruchy podle cesty dýchacích plynů do krve –**
poruchy 1) ventilace, 2) difúze
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4) ostatní a mimoplicní poruchy
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pletysmografie – budou podrobněji na vyšetřovacím
semináři další týden

Děkuji

Vám za pozornost

- Toto je v jakékoliv formě
(PDF, PPT, PPTX atd.)
neoficiální výukový materiál
- pro interní potřebu
- nešířit
- pro dotazy kontaktujte:
Petr.Marsalek@LF1.CUNI.CZ

Nadpis...