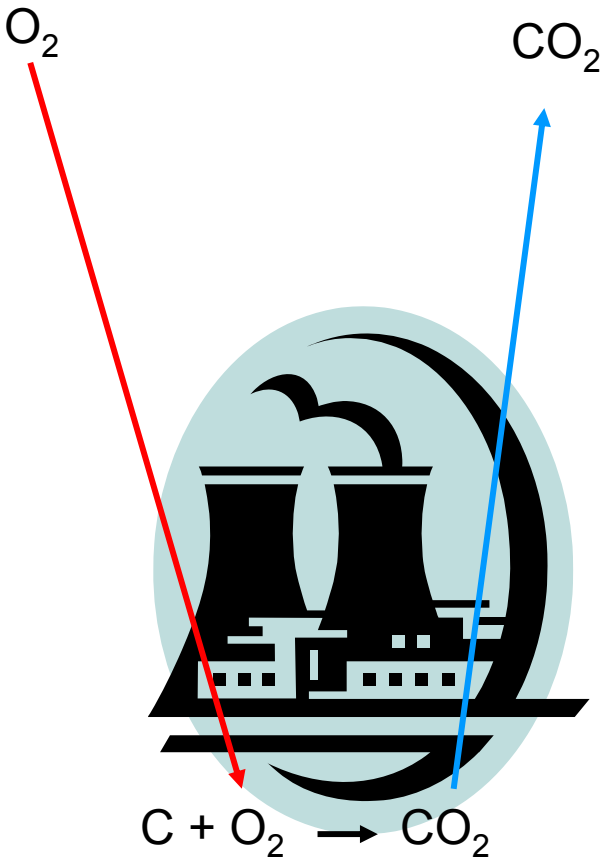
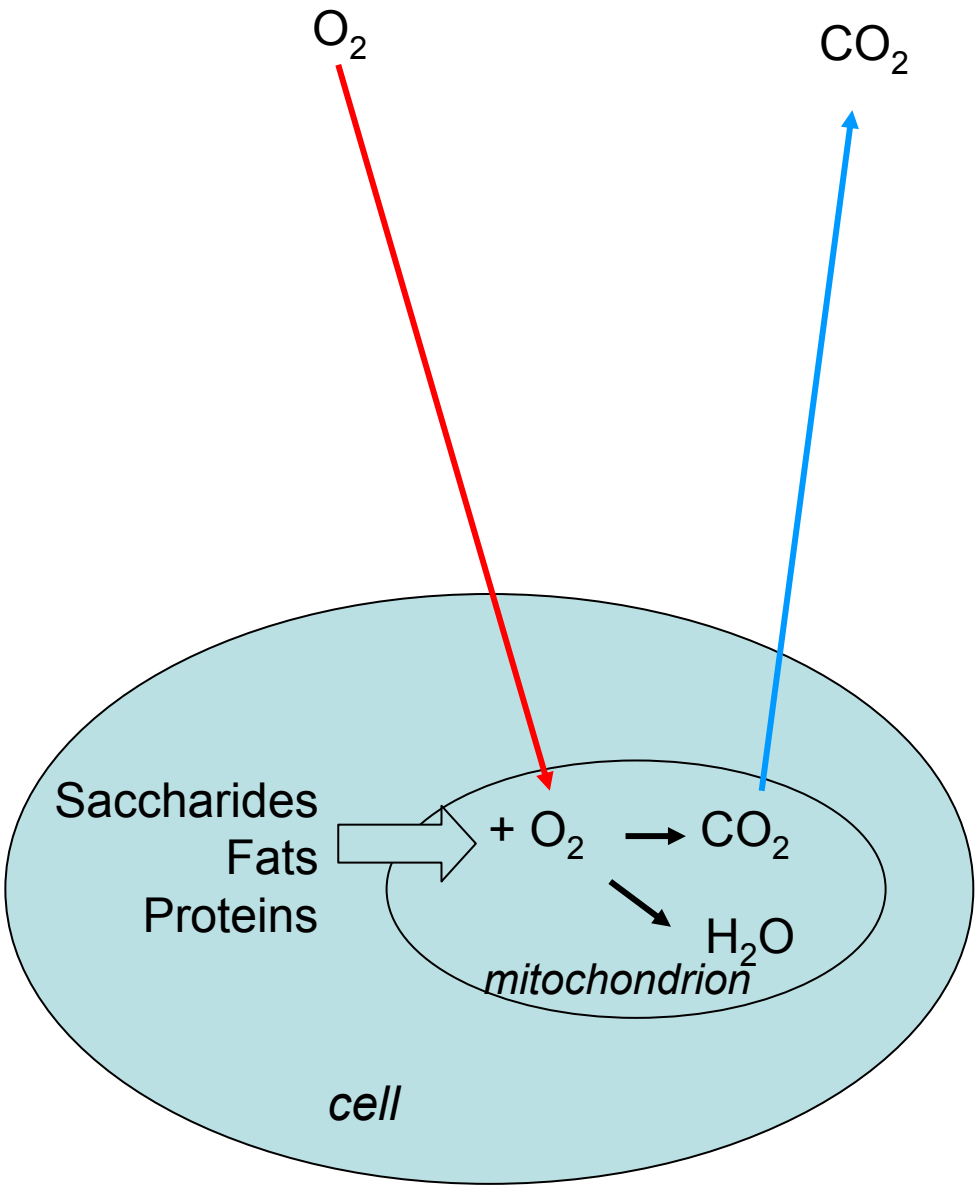
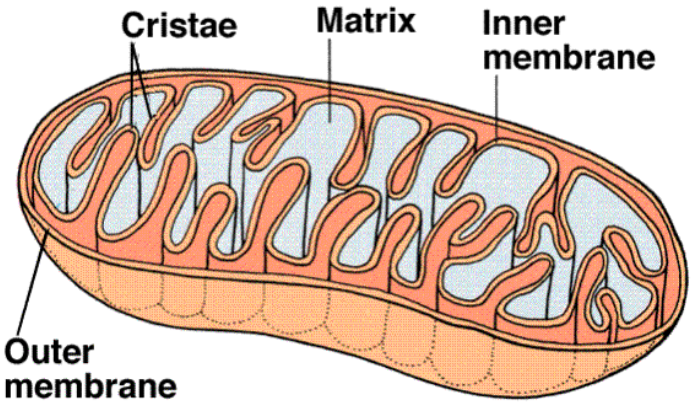
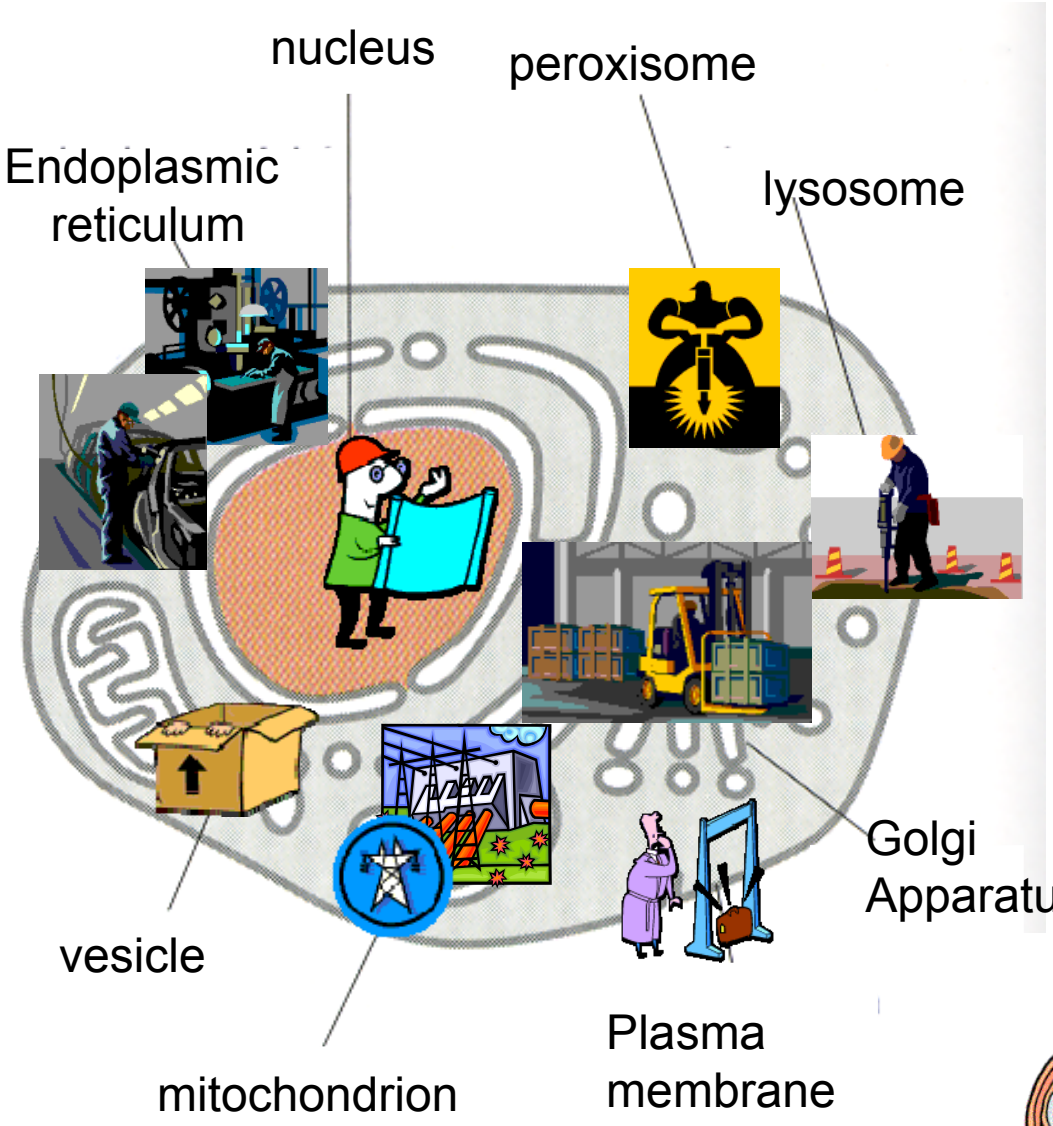
Disorders of ventilation to perfusion matching in lungs

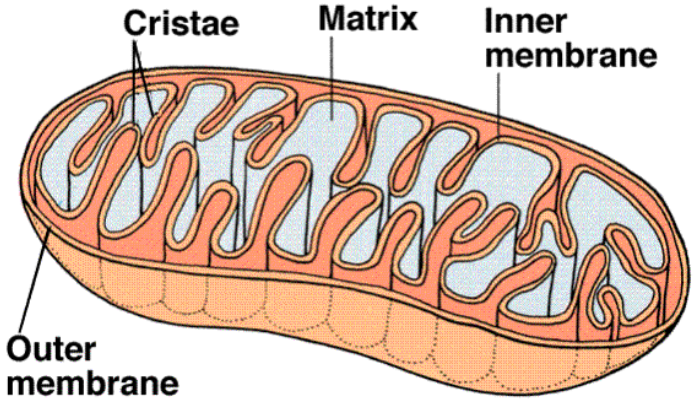

O_2
CO_2
$C + O_2 \rightarrow CO_2$



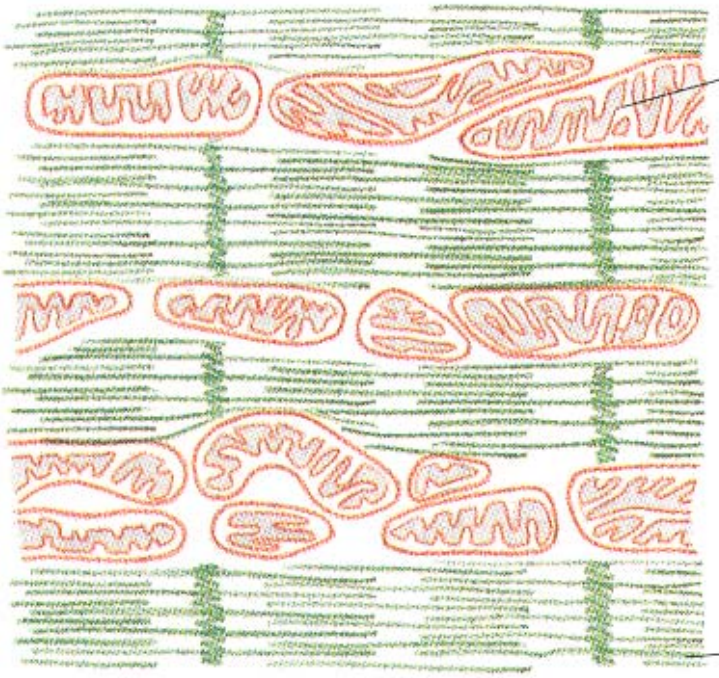


mitochondrion

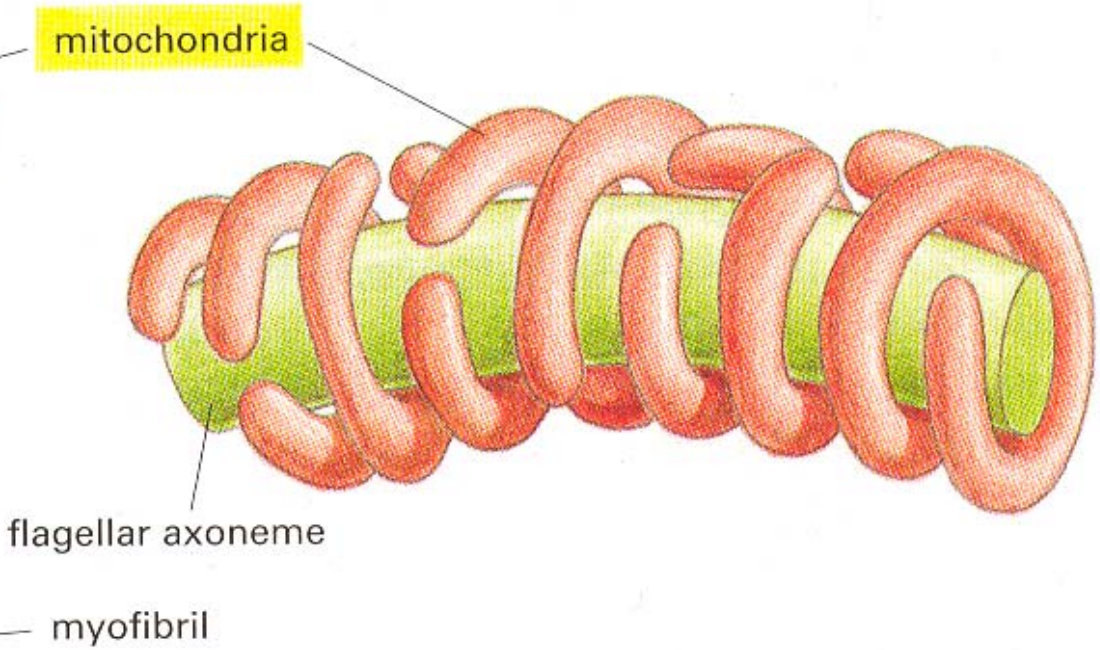




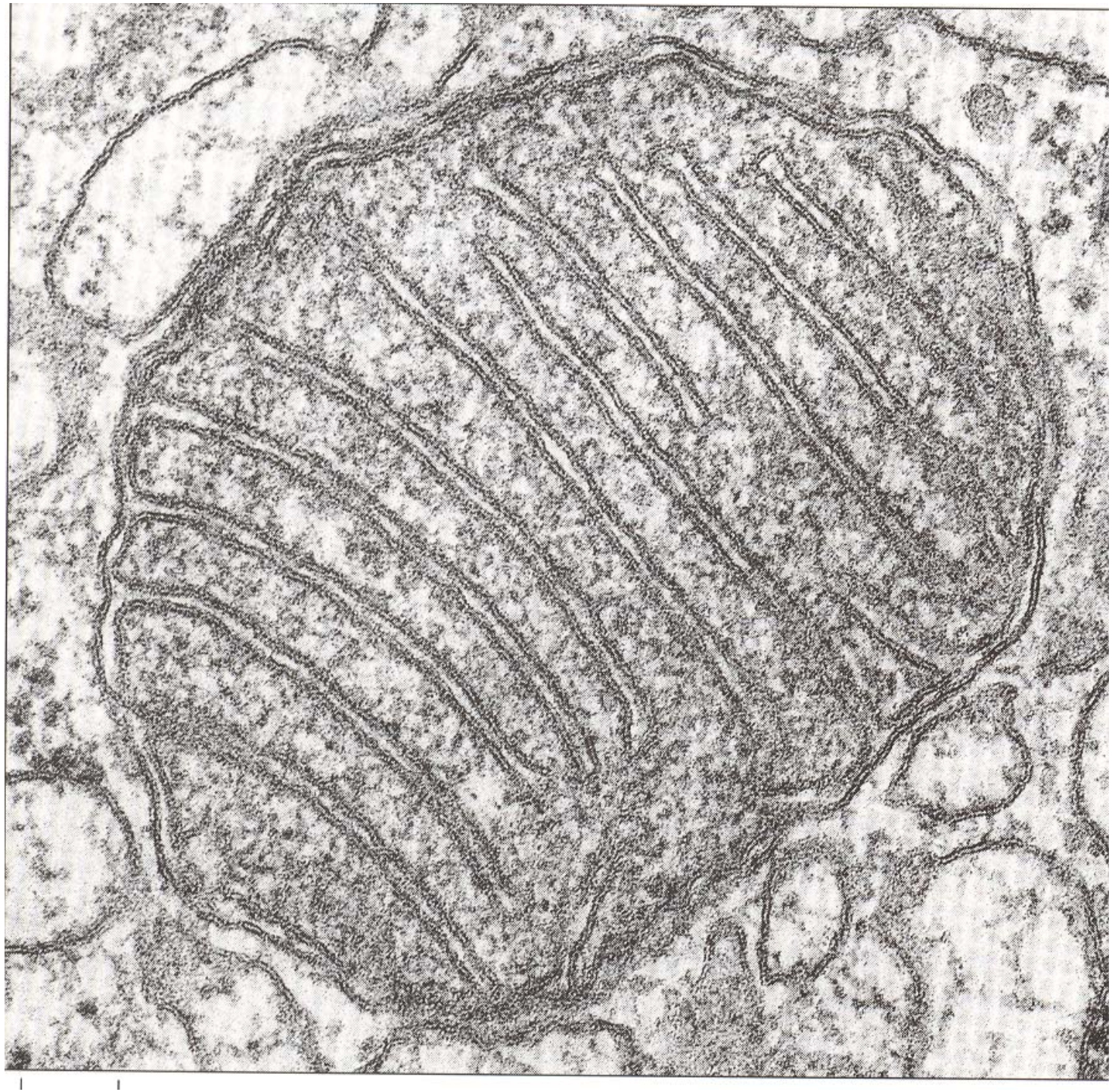
mitochondrion

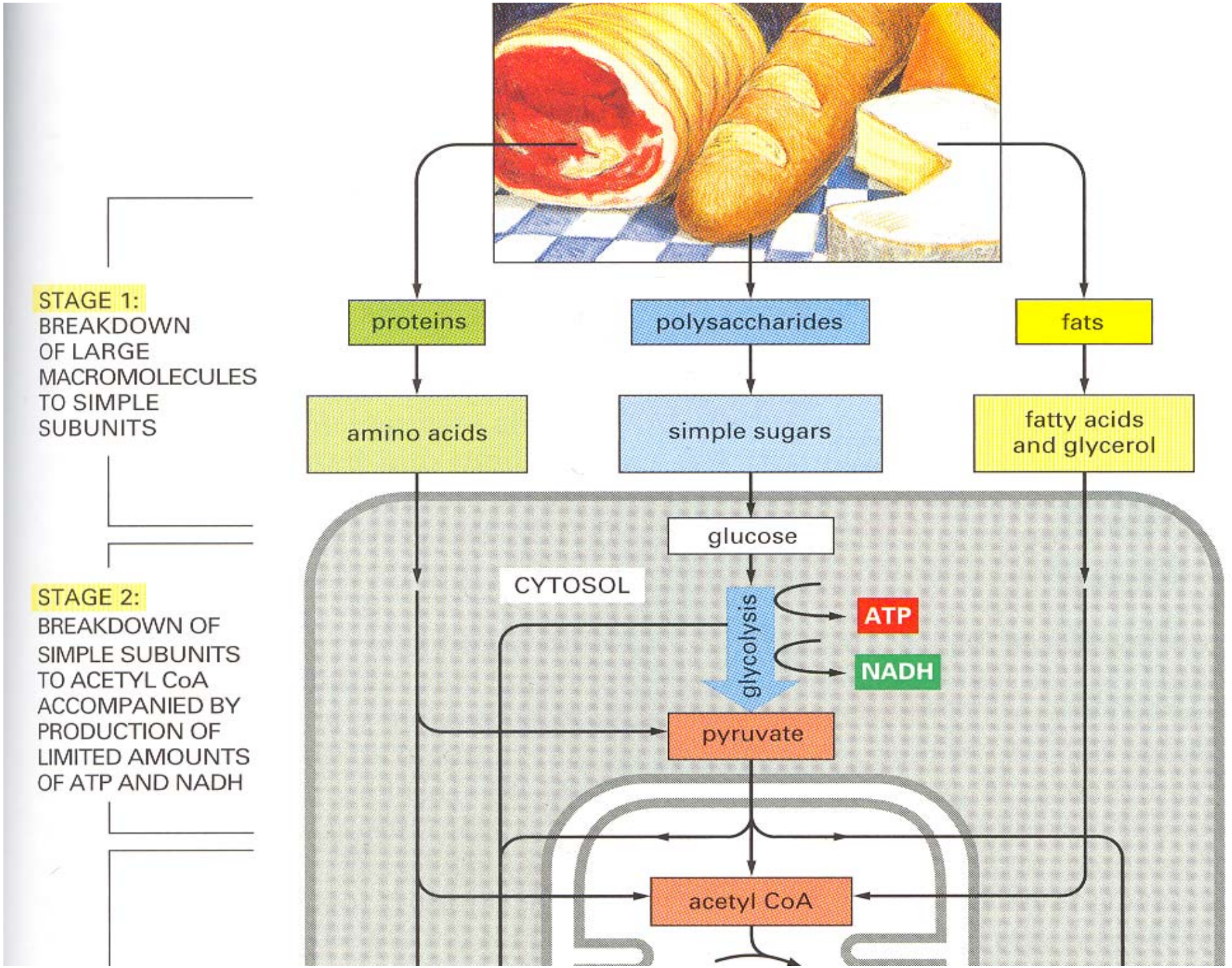


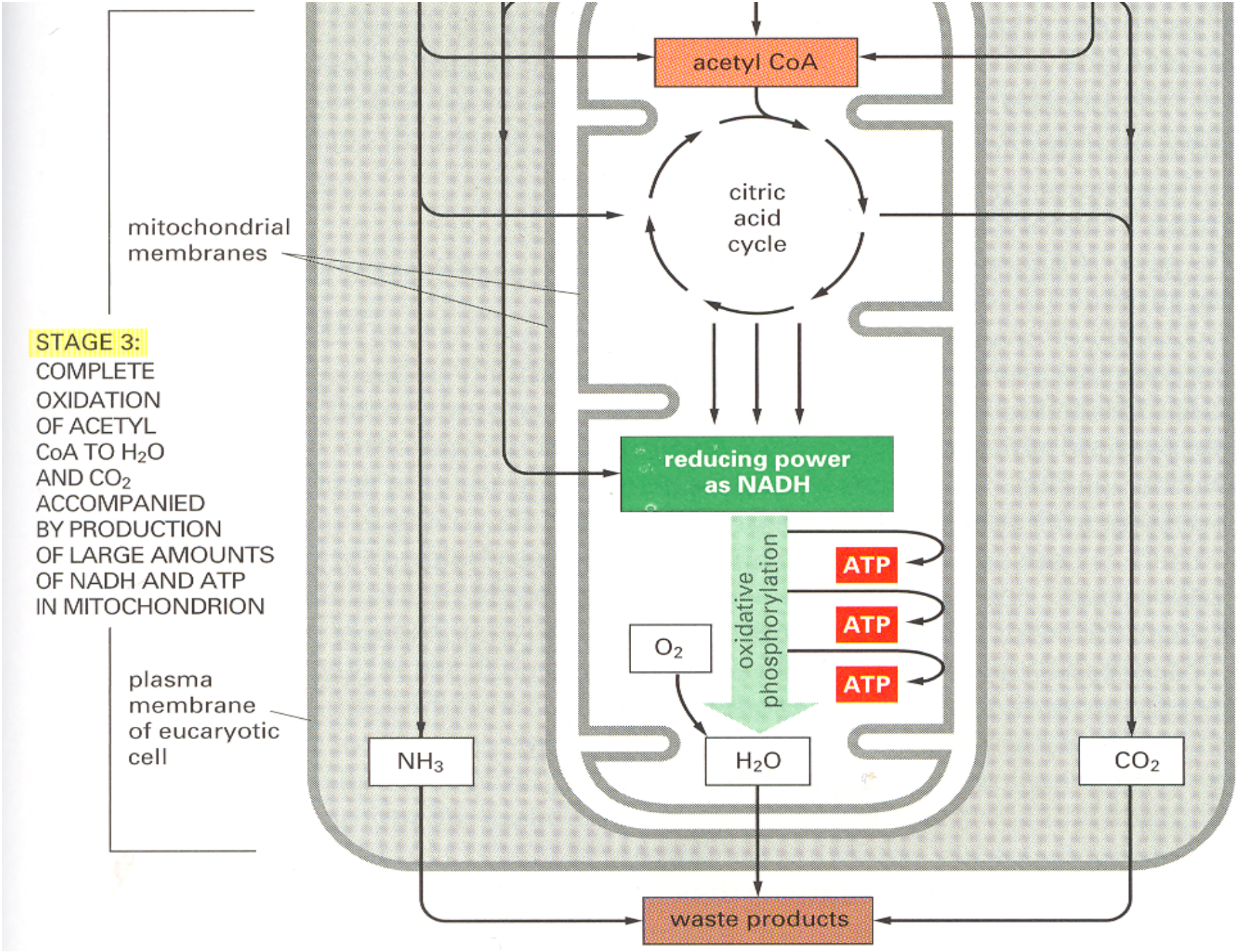
CARDIAC MUSCLE

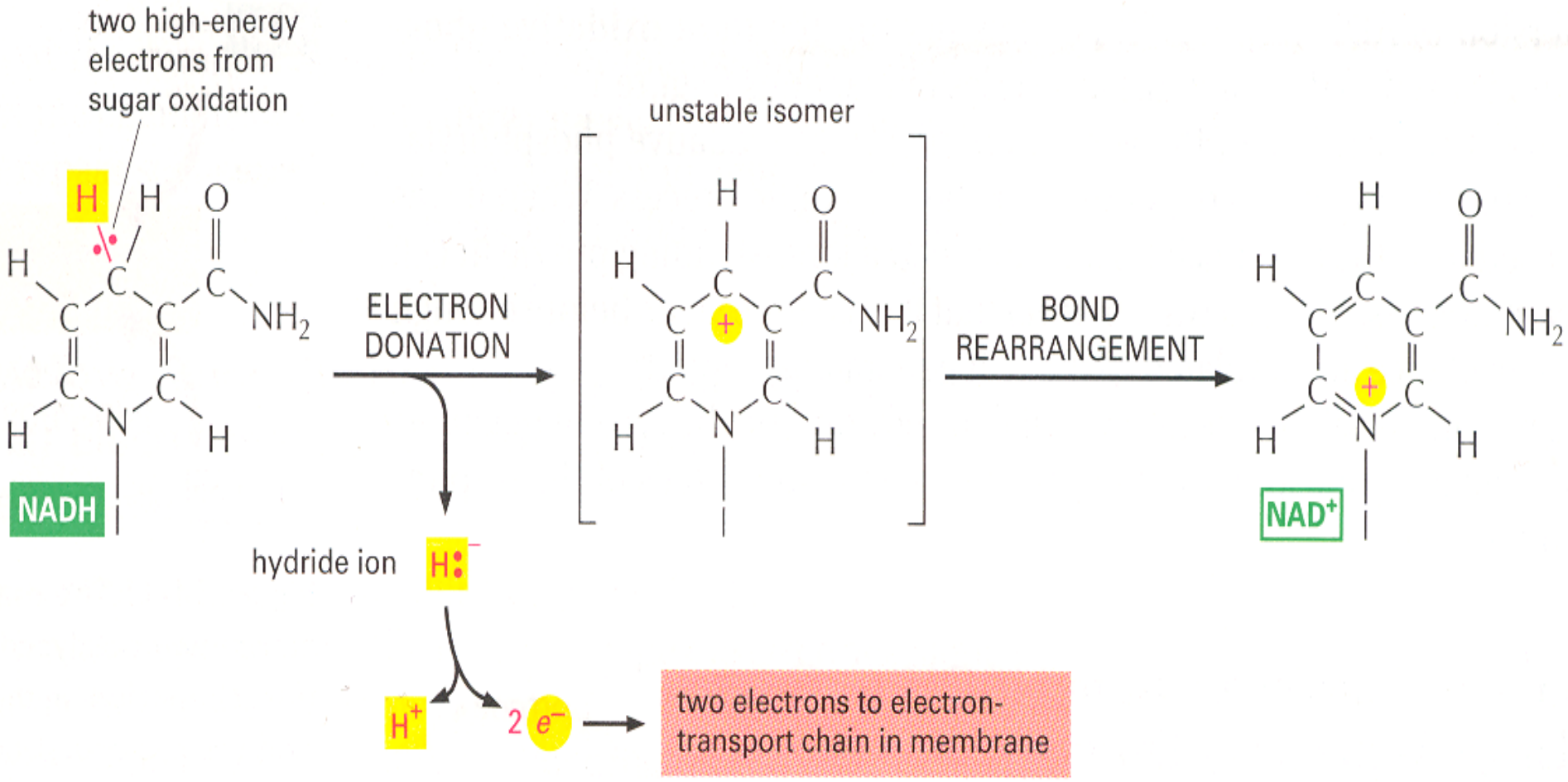


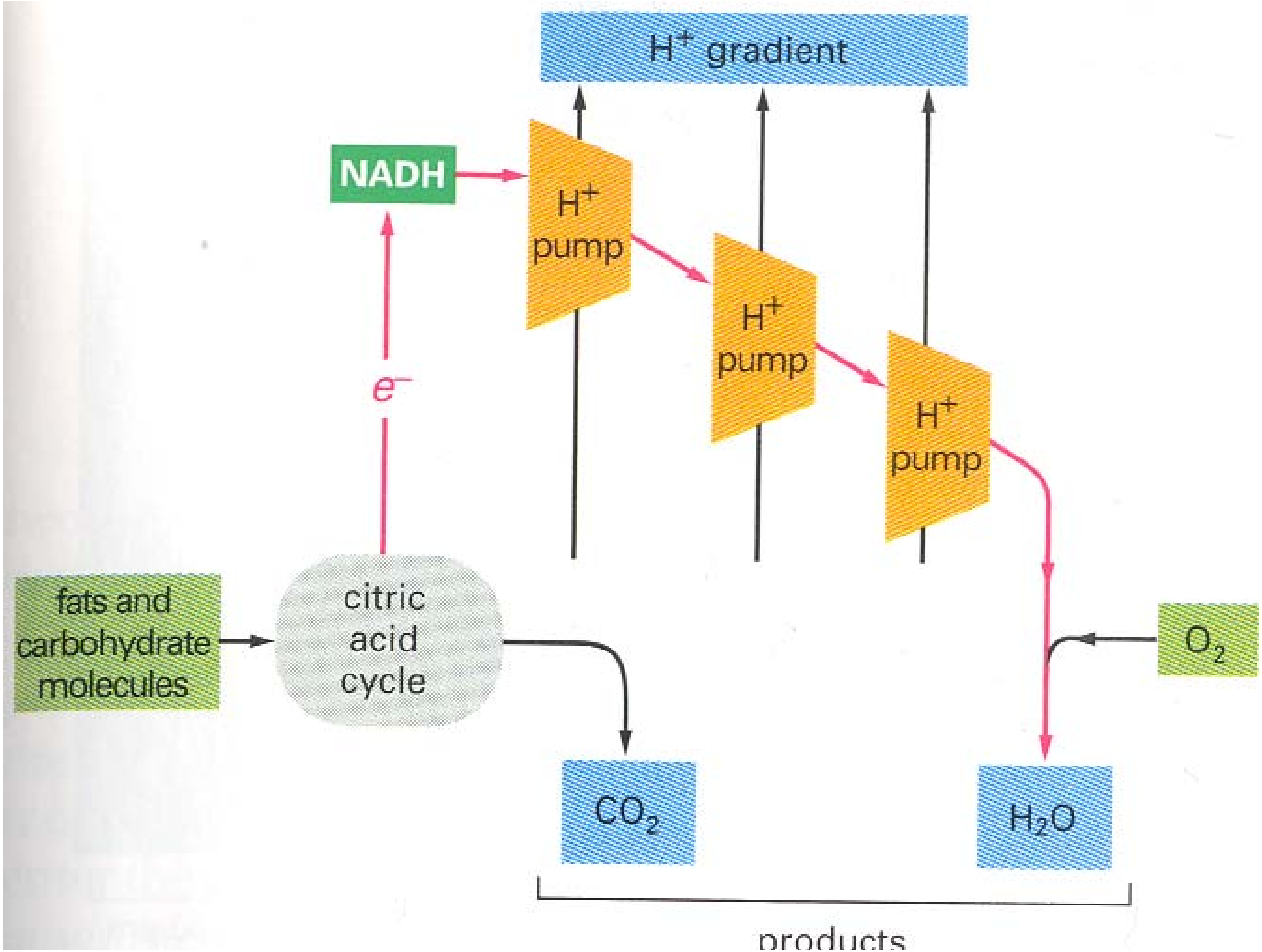
SPERM TAIL

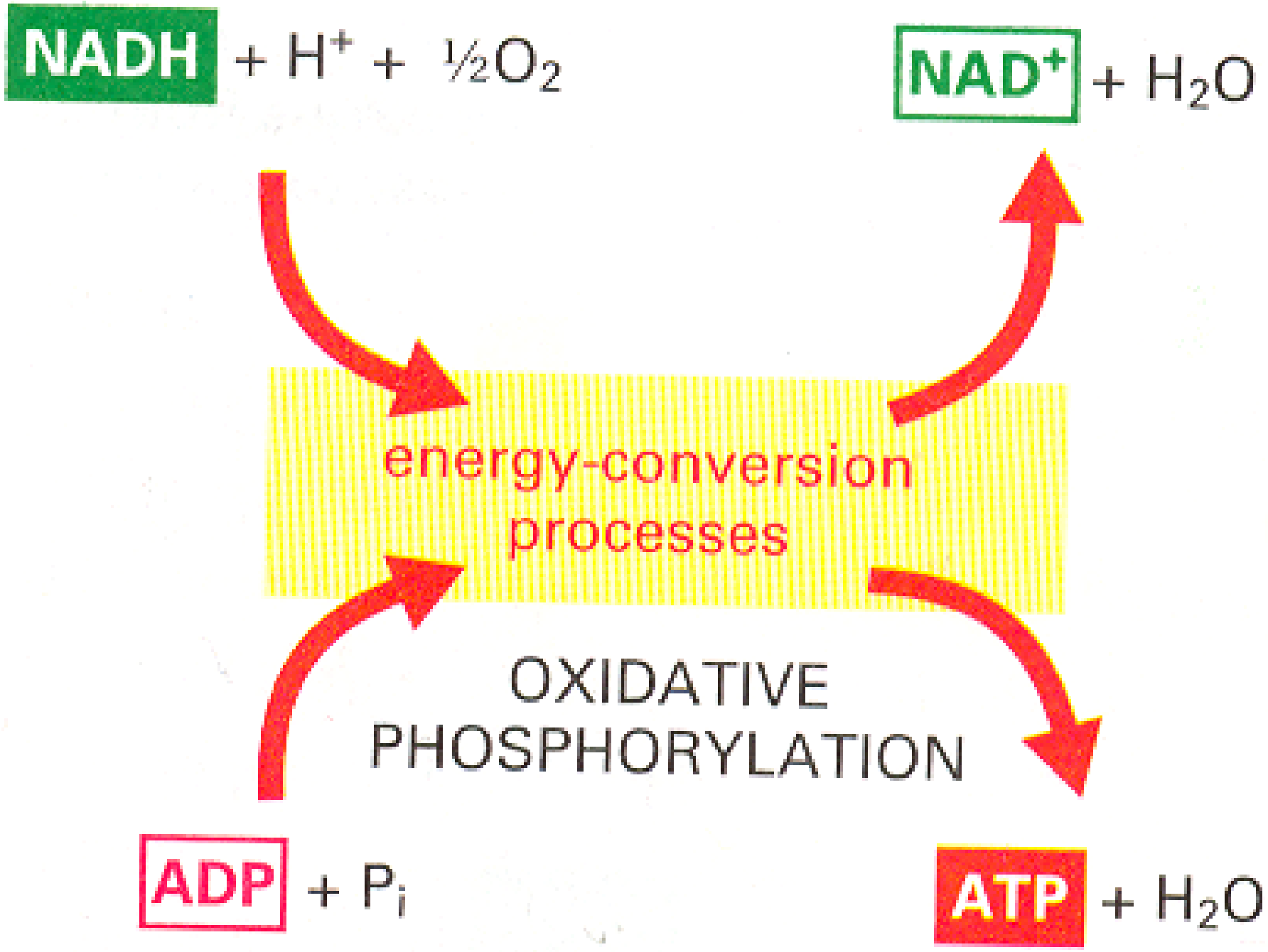


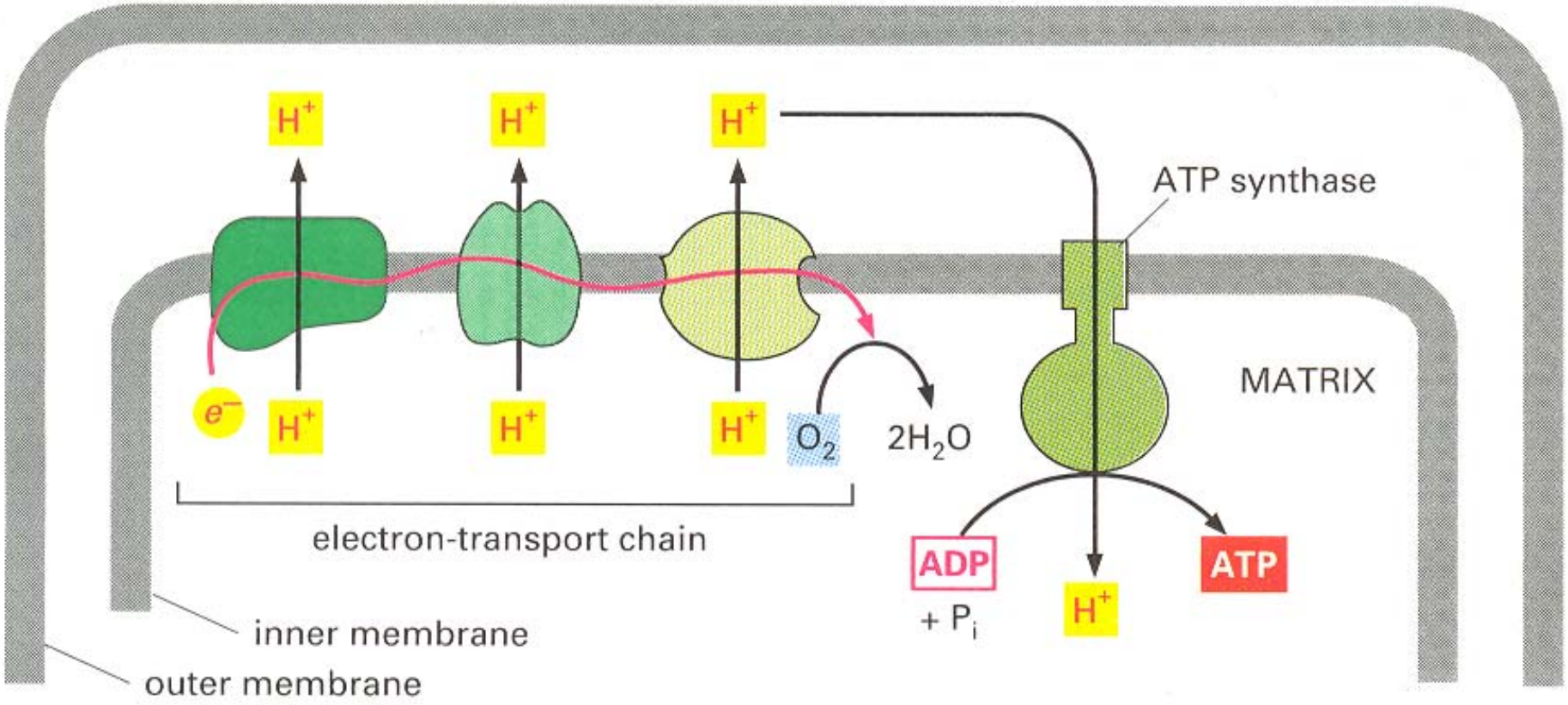

STAGE 1:
BREAKDOWN OF LARGE MACROMOLECULES TO SIMPLE SUBUNITS
STAGE 2:
BREAKDOWN OF SIMPLE SUBUNITS TO ACETYL CoA ACCOMPANIED BY PRODUCTION OF LIMITED AMOUNTS OF ATP AND NADH
proteins
polysaccharides
fats
amino acids
simple sugars
fatty acids and glycerol
glucose
CYTOSOL
glycolysis
ATP
NADH
pyruvate
acetyl CoA

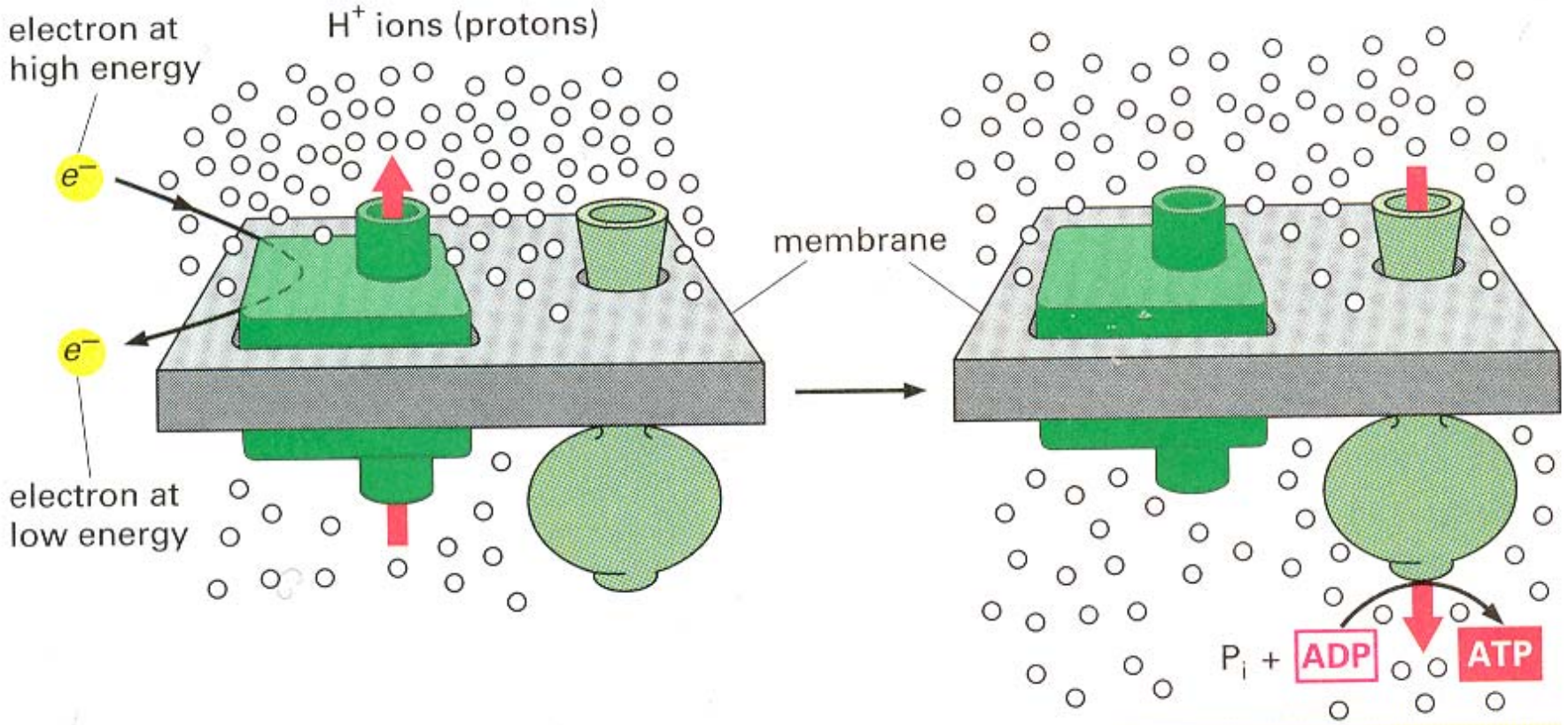

acetyl CoA
citric acid cycle
mitochondrial membranes
STAGE 3: COMPLETE OXIDATION OF ACETYL CoA TO H_2O AND CO_2 ACCOMPANIED BY PRODUCTION OF LARGE AMOUNTS OF NADH AND ATP IN MITOCHONDRION
reducing power as NADH
oxidative phosphorylation
ATP
ATP
ATP
O_2
NH_3
H_2O
CO_2
plasma membrane of eucaryotic cell
waste products


two high-energy electrons from sugar oxidation
NADH
ELECTRON DONATION
unstable isomer
BOND REARRANGEMENT
NAD+
hydride ion
H:−
H+
2 e−
two electrons to electron-transport chain in membrane


H+ gradient
NADH
H+ pump
H+ pump
H+ pump
e−
fats and carbohydrate molecules
citric acid cycle
CO2
H2O
O2
products


NADH + H+ + ½O2
NAD+ + H2O
energy-conversion processes
OXIDATIVE PHOSPHORYLATION
ADP + Pi
ATP + H2O


H+
H+
H+
ATP synthase
MATRIX
e−
H+
H+
H+
O2
2H2O
electron-transport chain
ADP
+ Pi
H+
ATP
inner membrane
outer membrane

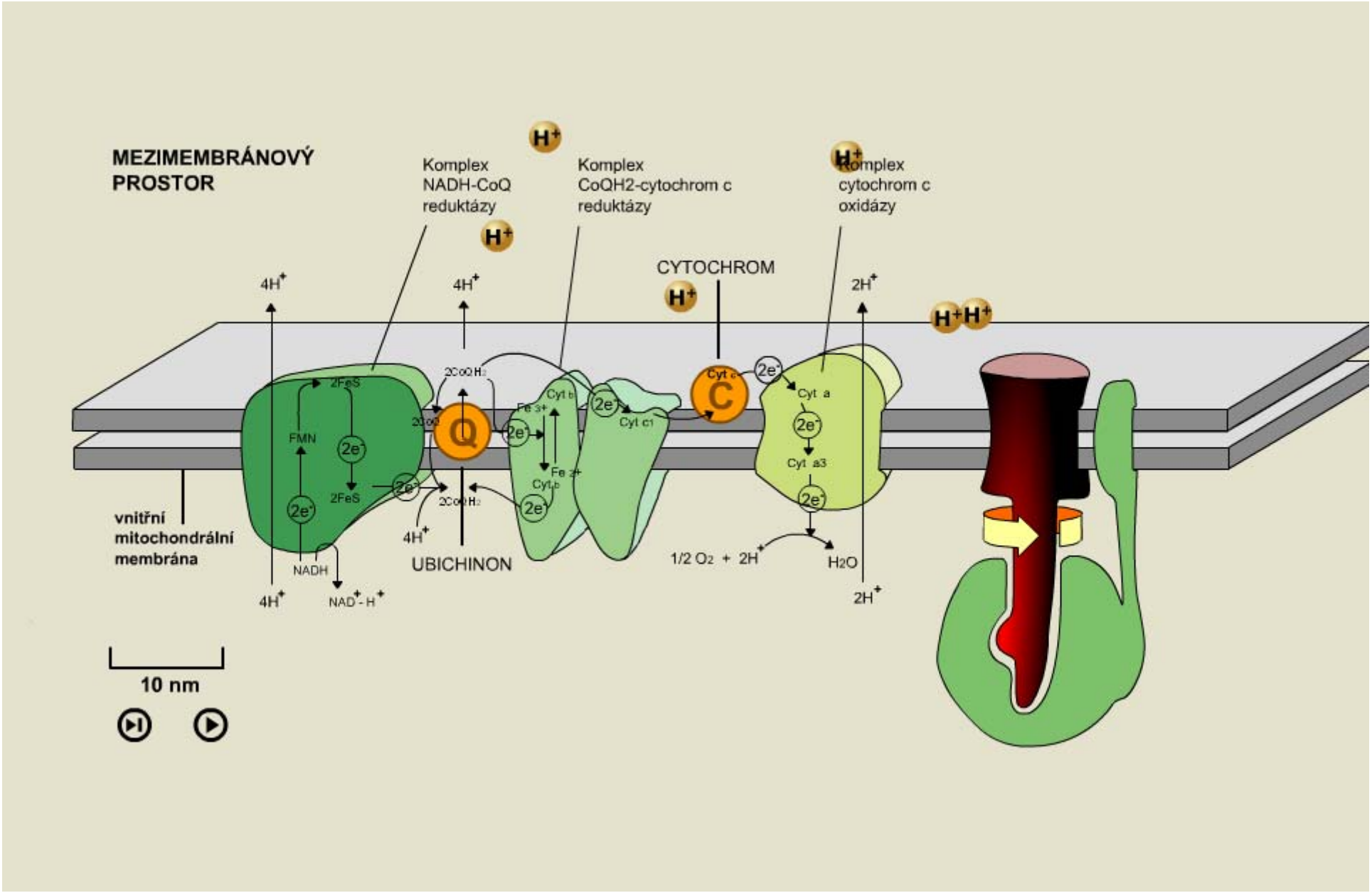


STAGE 1: ELECTRON TRANSPORT DRIVES PUMP THAT PUMPS PROTONS ACROSS MEMBRANE

(A)

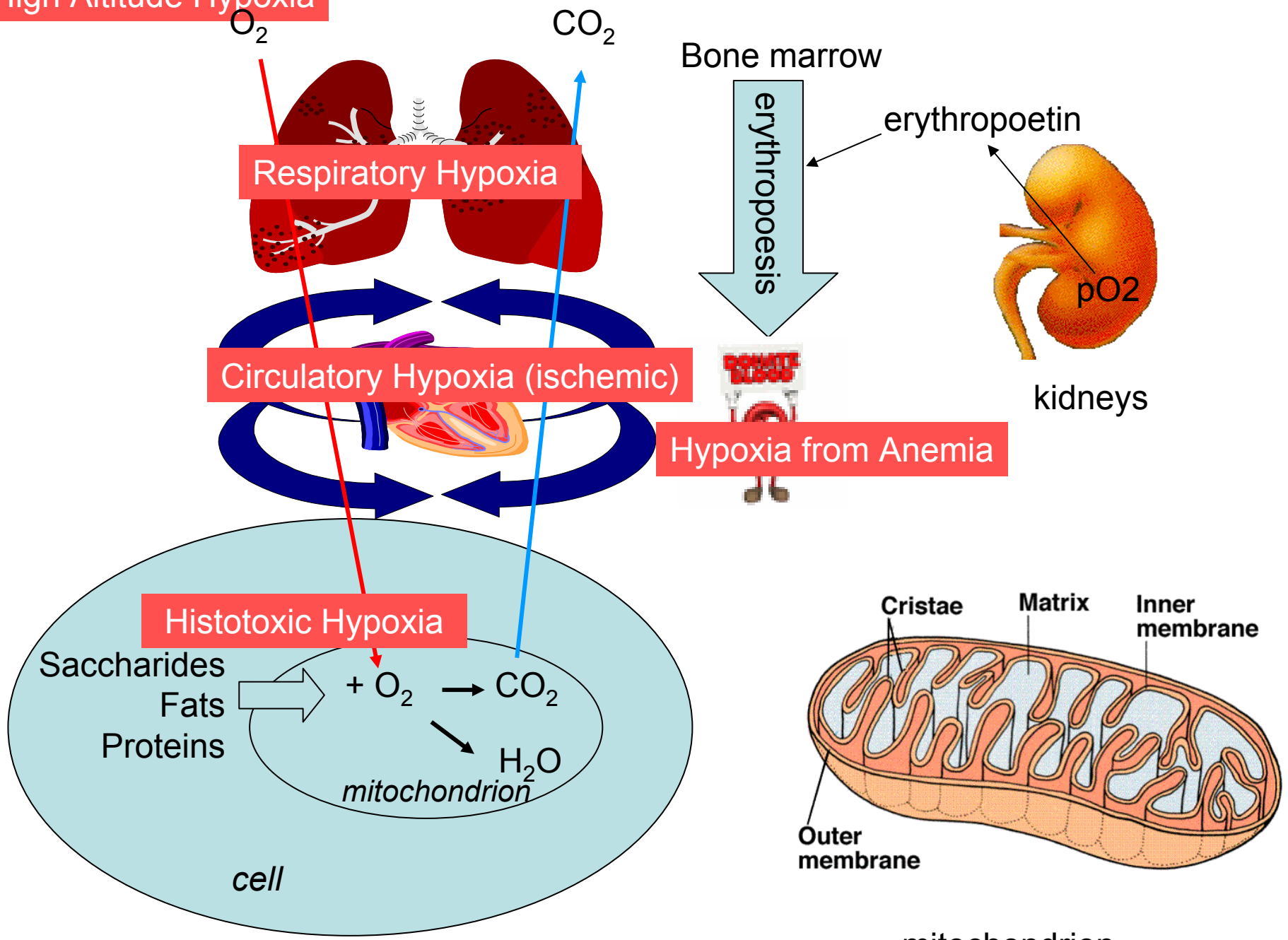
STAGE 2: PROTON GRADIENT IS HARNESSED BY ATP SYNTHASE TO MAKE ATP

(B)


MEZIMEMBRÁNOVÝ PROSTOR
Komplex NADH-CoQ reduktázy
Komplex CoQH2-cytochrom c reduktázy
Komplex cytochrom c oxidázy
CYTOCHROM
4H+
4H+
2H+
H+
H+H+
2FeS
FMN
2e-
2FeS
NADH
NAD+ - H+
4H+
2CoQ
Q
2CoQH2
4H+
UBICHINON
Cyt b
Fe 3+
Cyt c1
Fe 2+
Cyt b
C
Cyt a
Cyt a
Cyt a3
1/2 O2 + 2H+
H2O
2H+
vnitřní mitochondrální membrána
10 nm

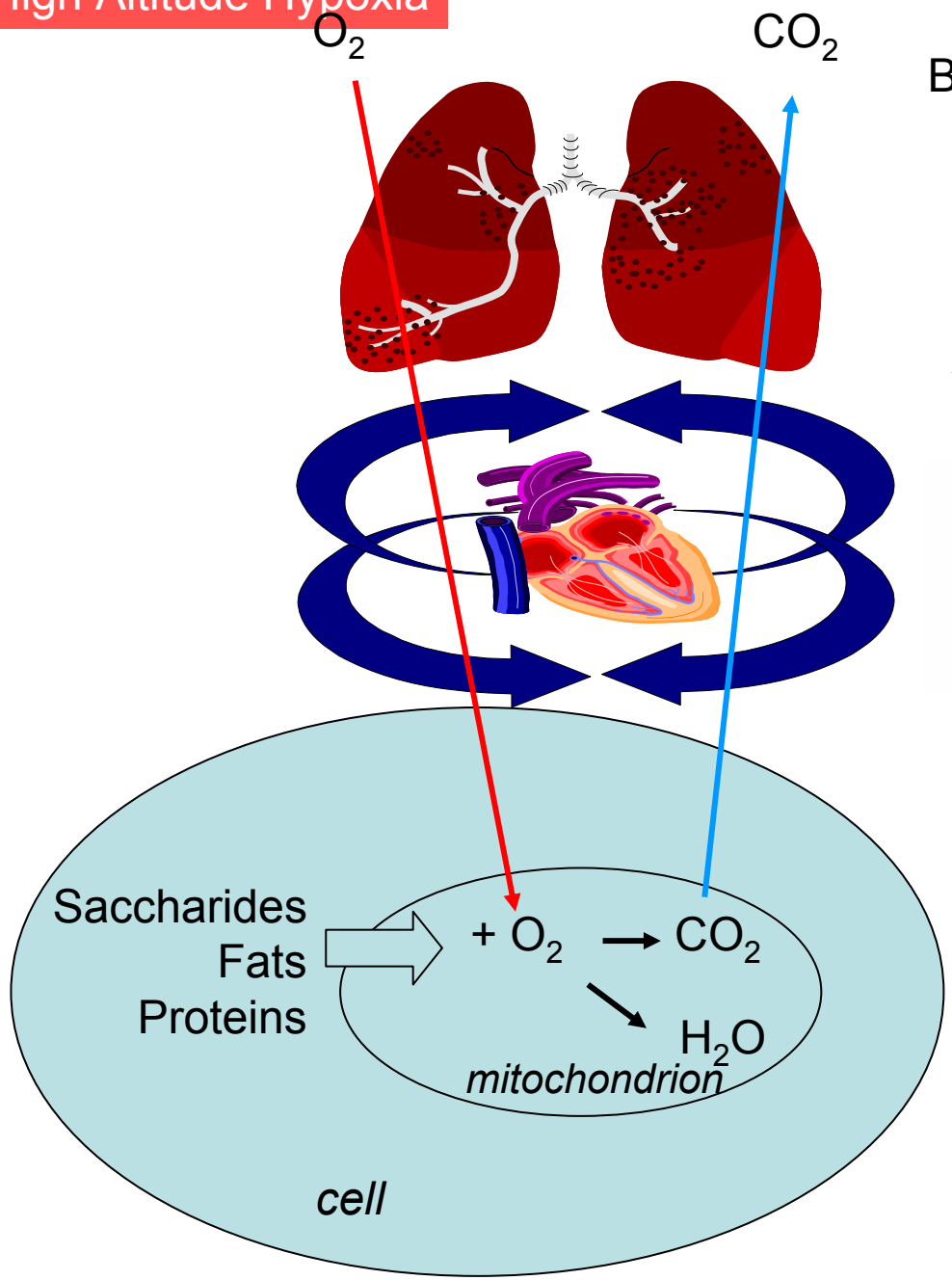

100 nm

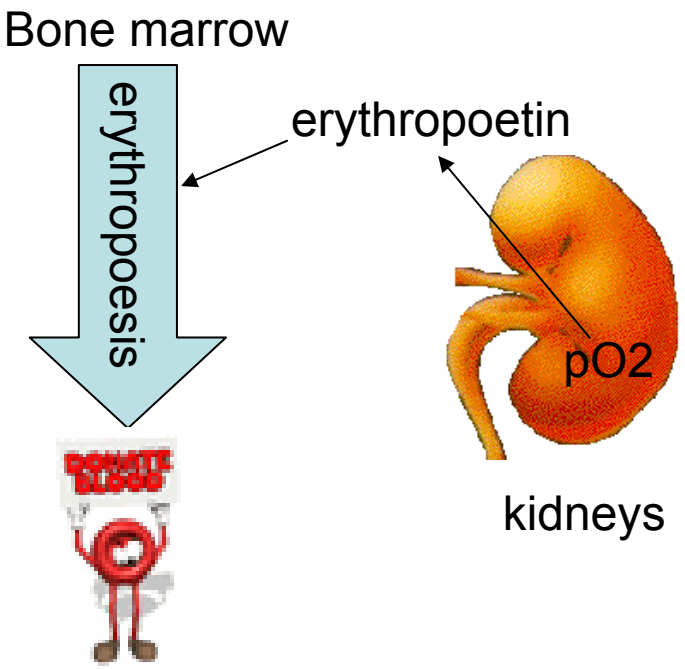
High Altitude Hypoxia

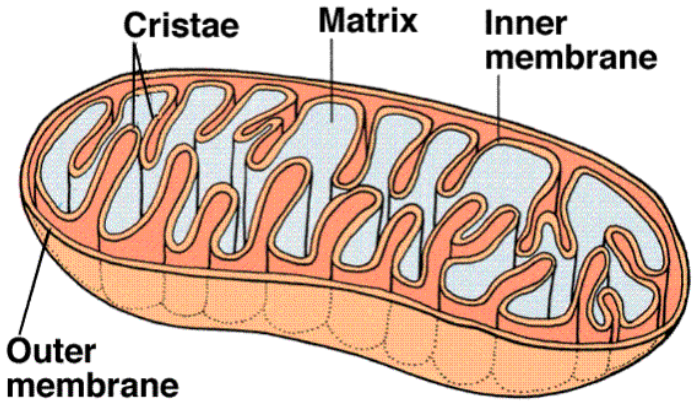


mitochondrion

High Altitude Hypoxia





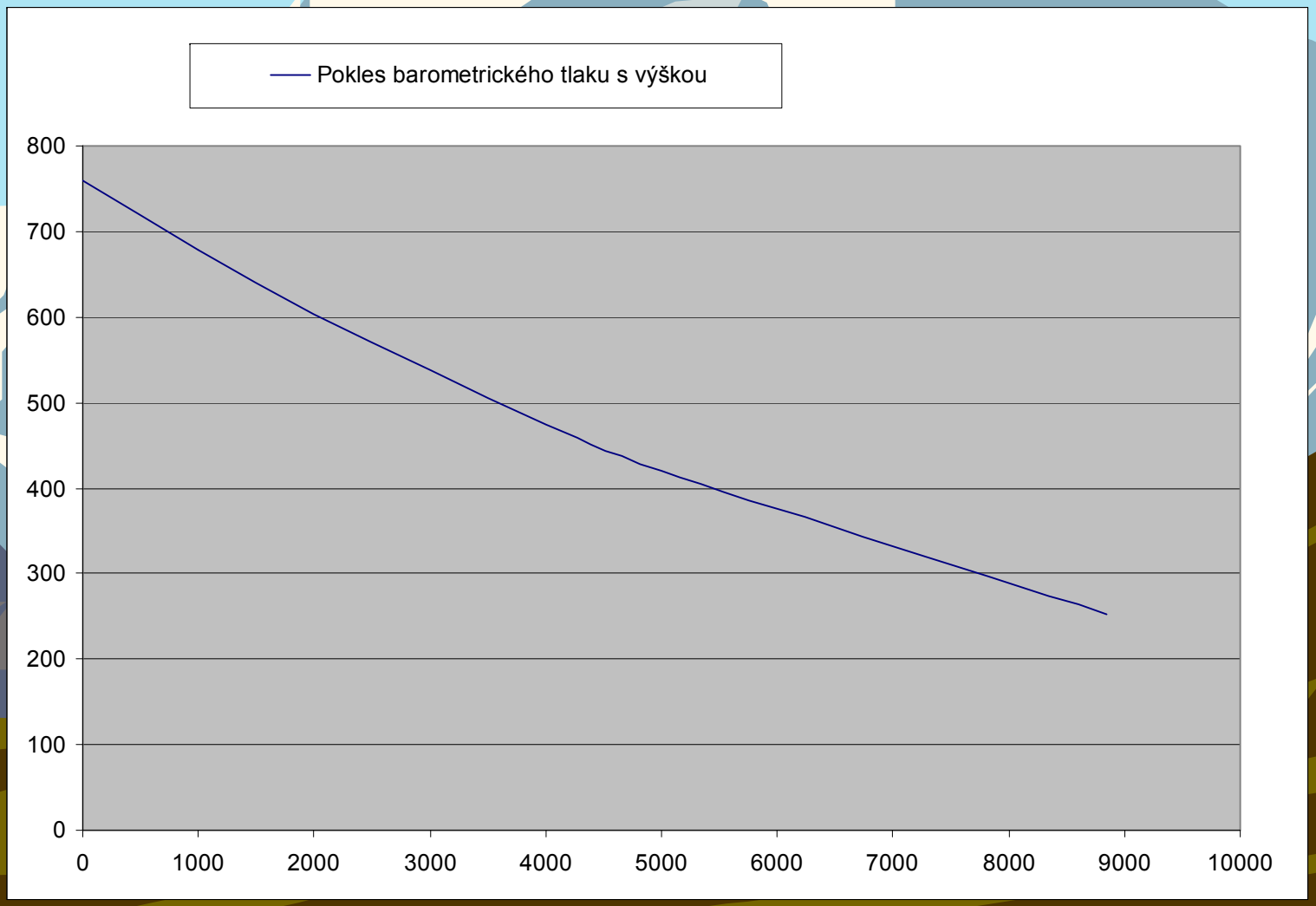


mitochondrion

High Altitude Hypoxia

Climbing

Barometric pressure drop

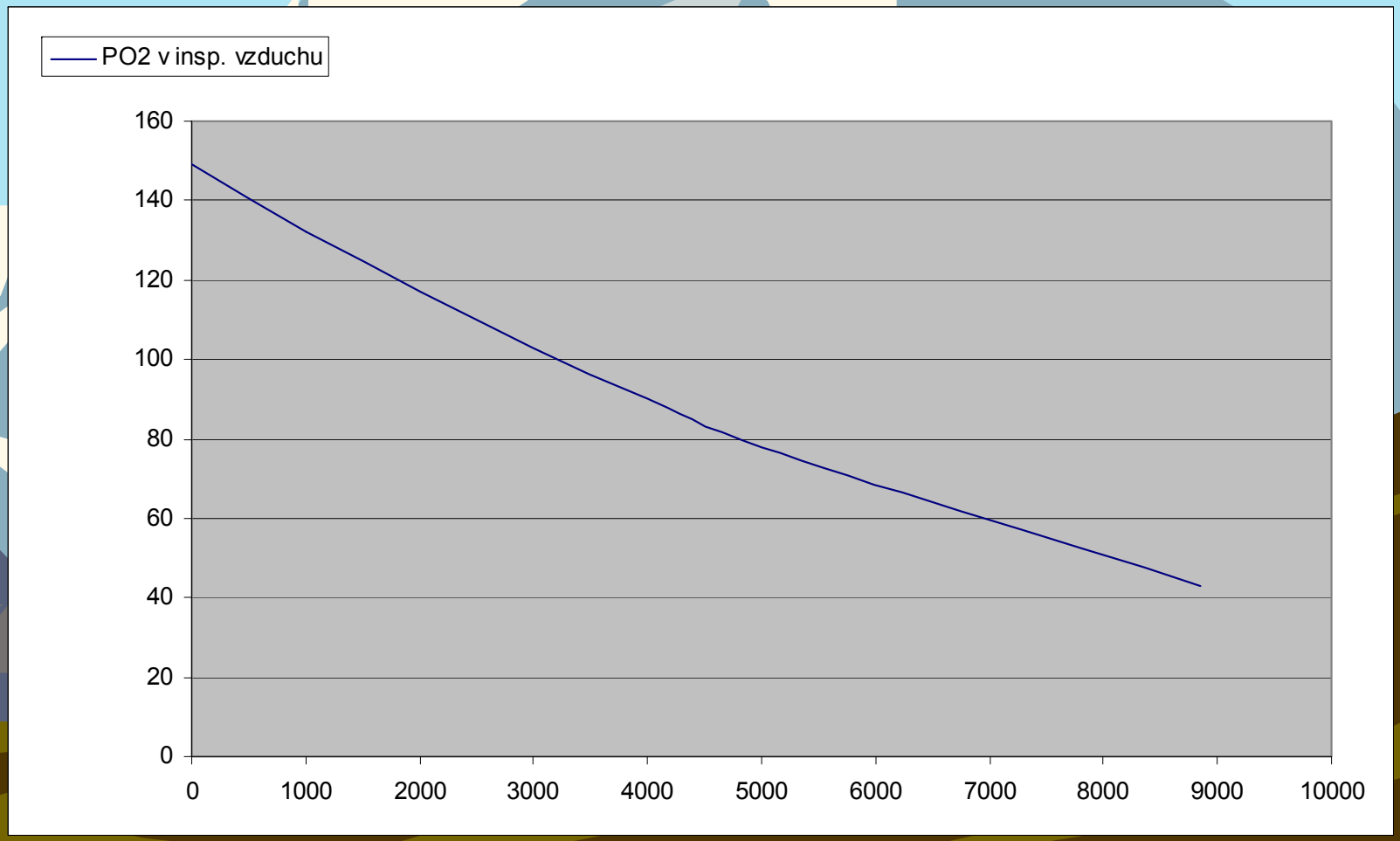


High Altitude Hypoxia

Climbing

Barometric pressure drop

Fall PO2 in inspired air



High Altitude Hypoxia

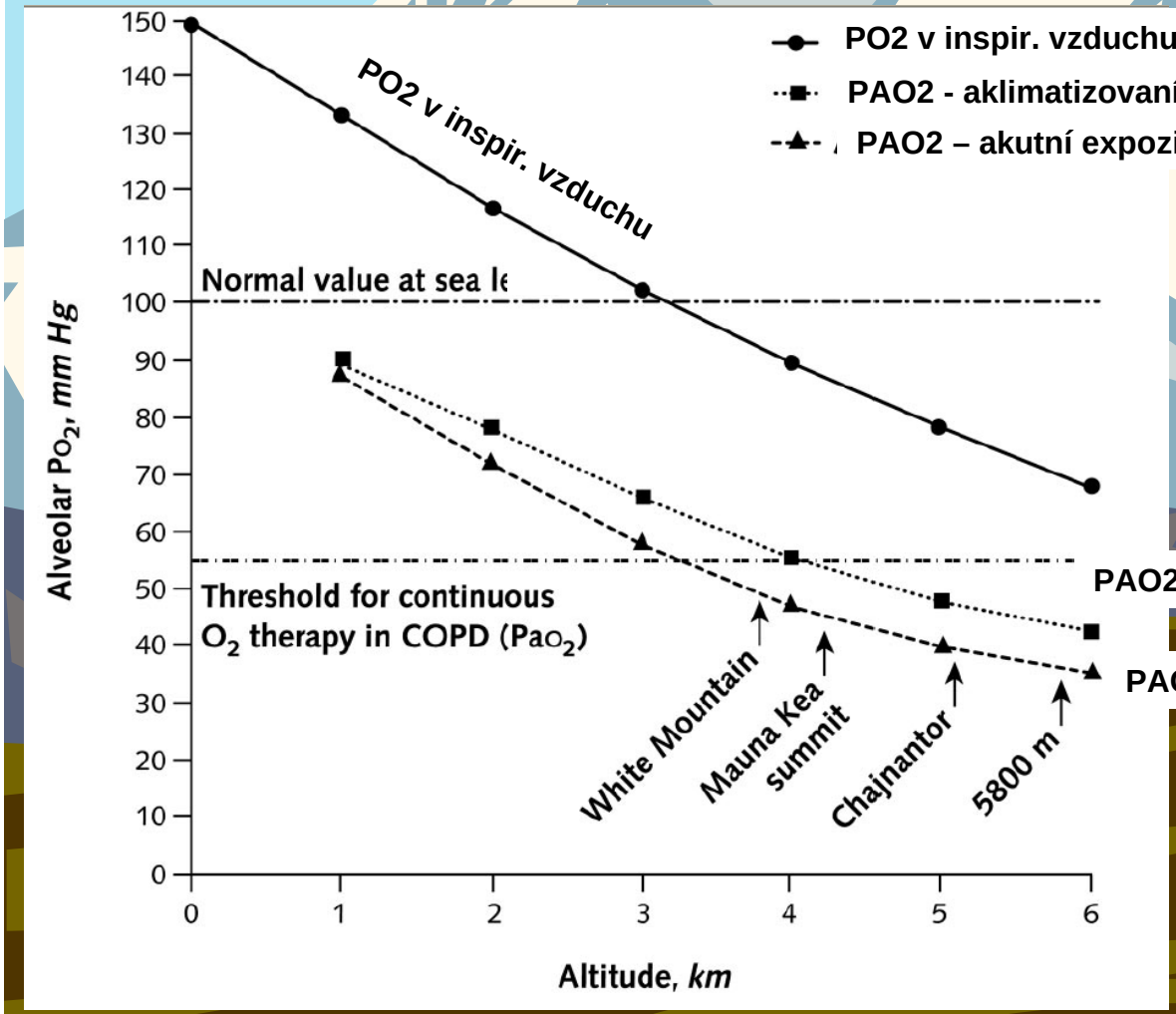
Climbing

Barometric pressure drop

Fall PO2 in inspired air

Fall alveolar PO2

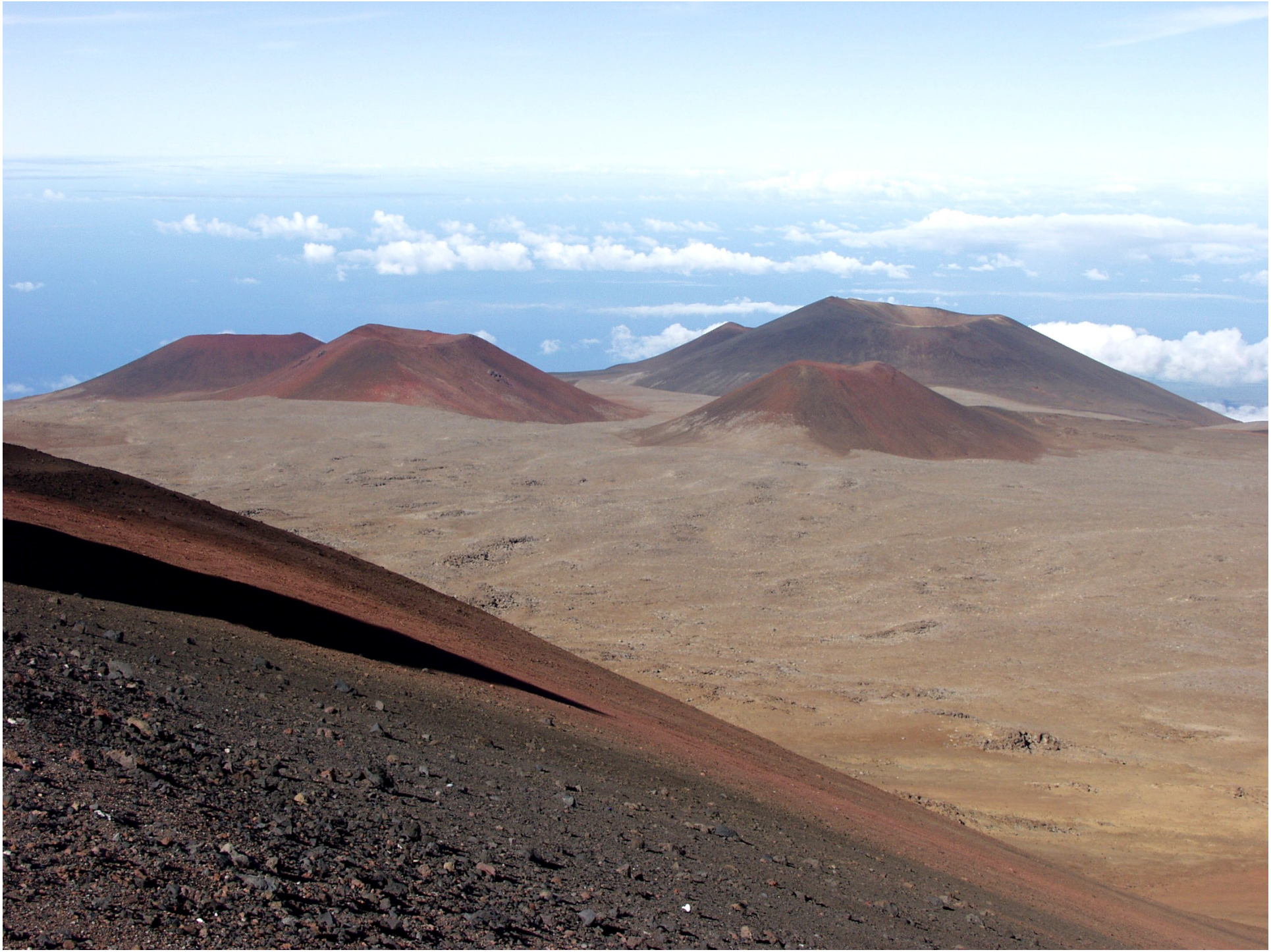
Fall arterial PO2

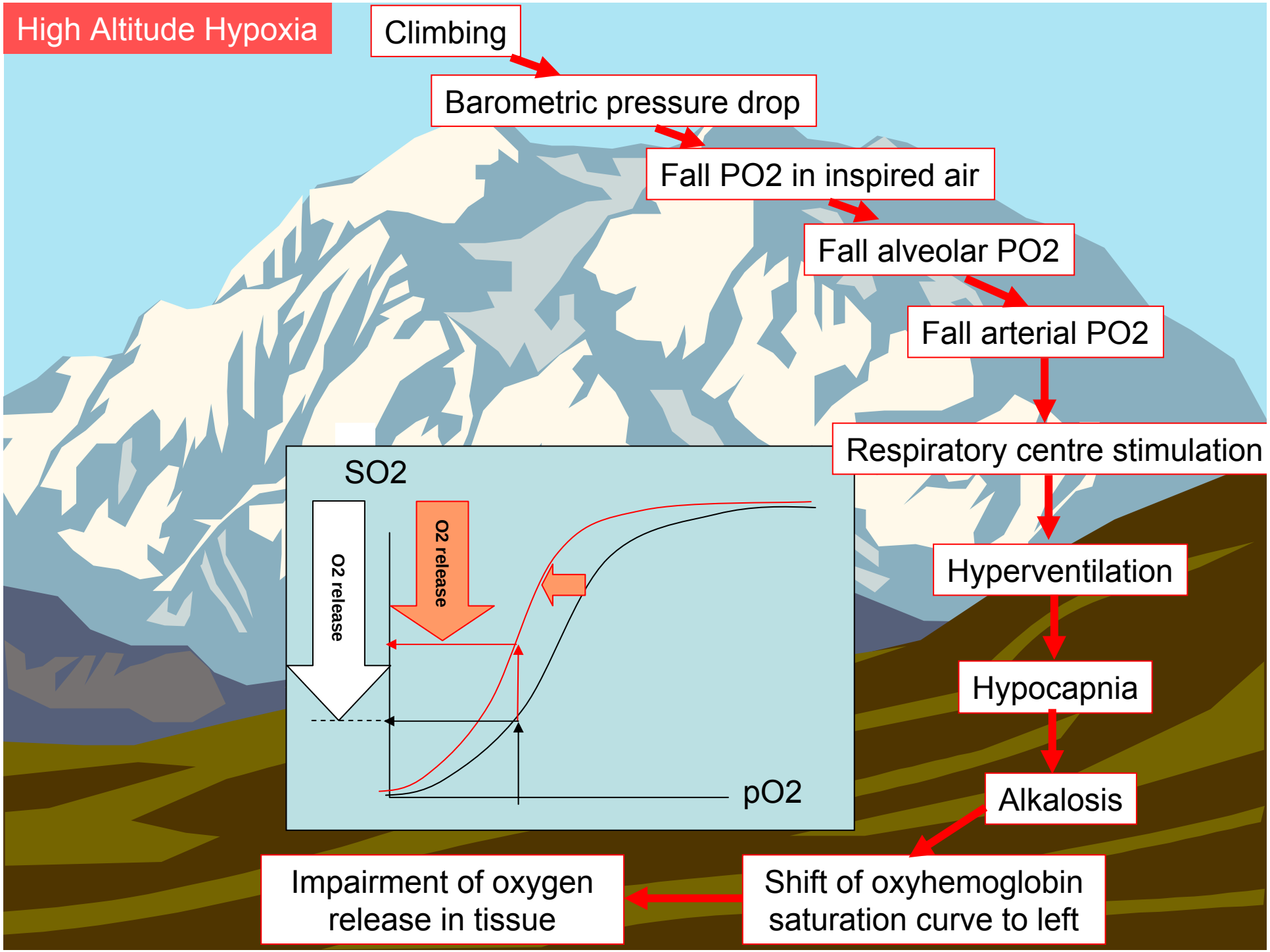


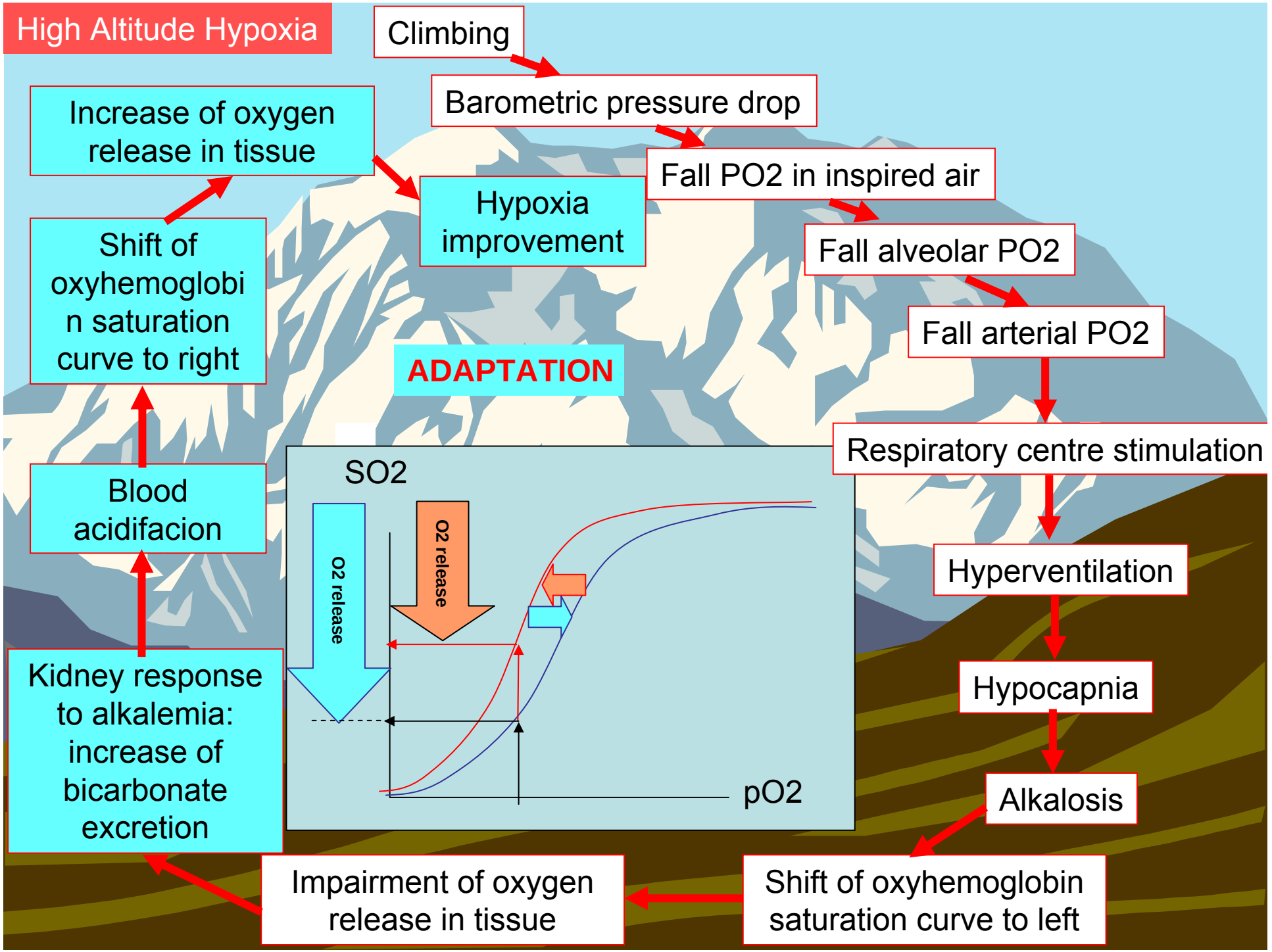
PAO2 – fully acclimated

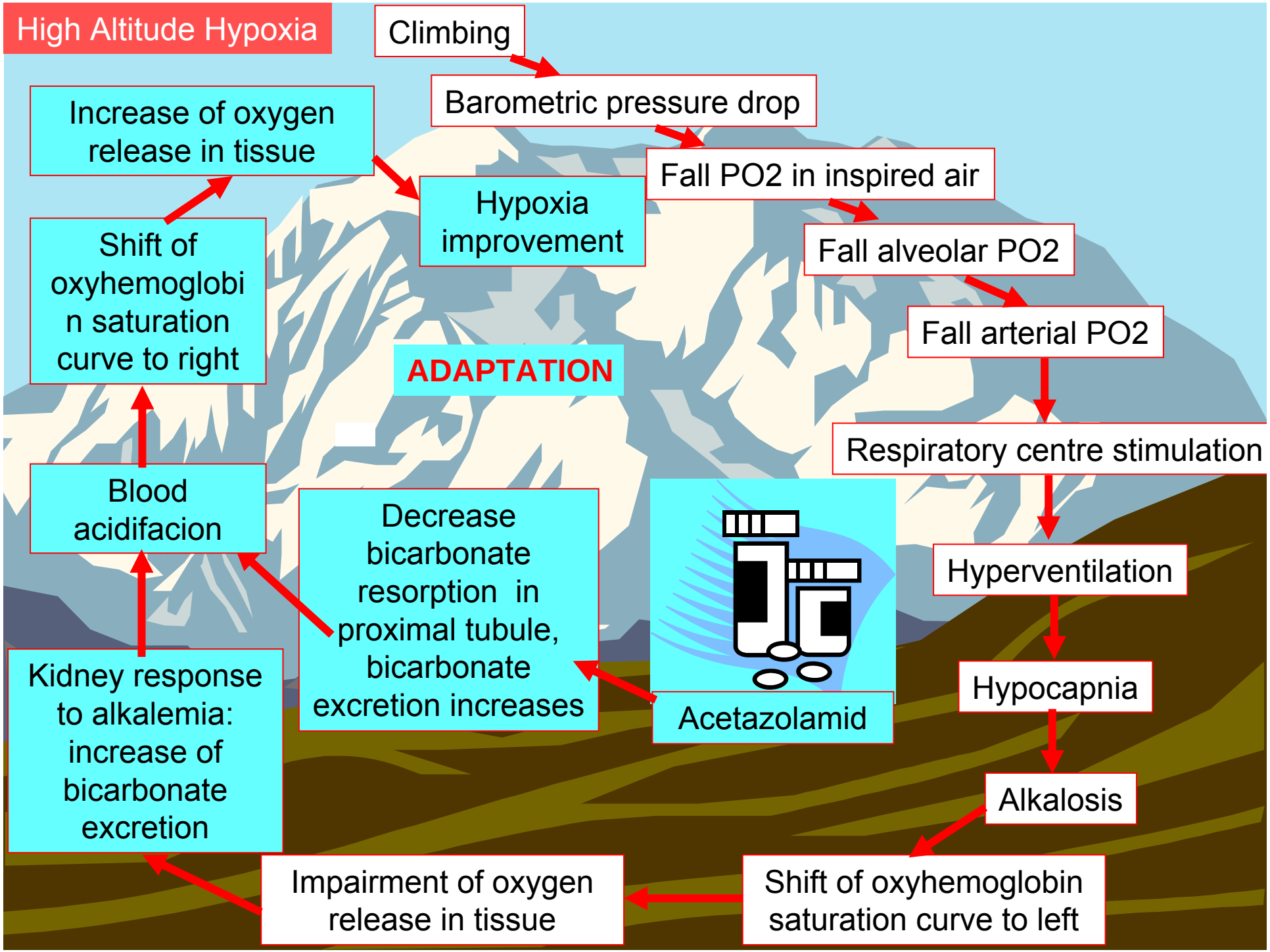
PAO2 – acute exposition

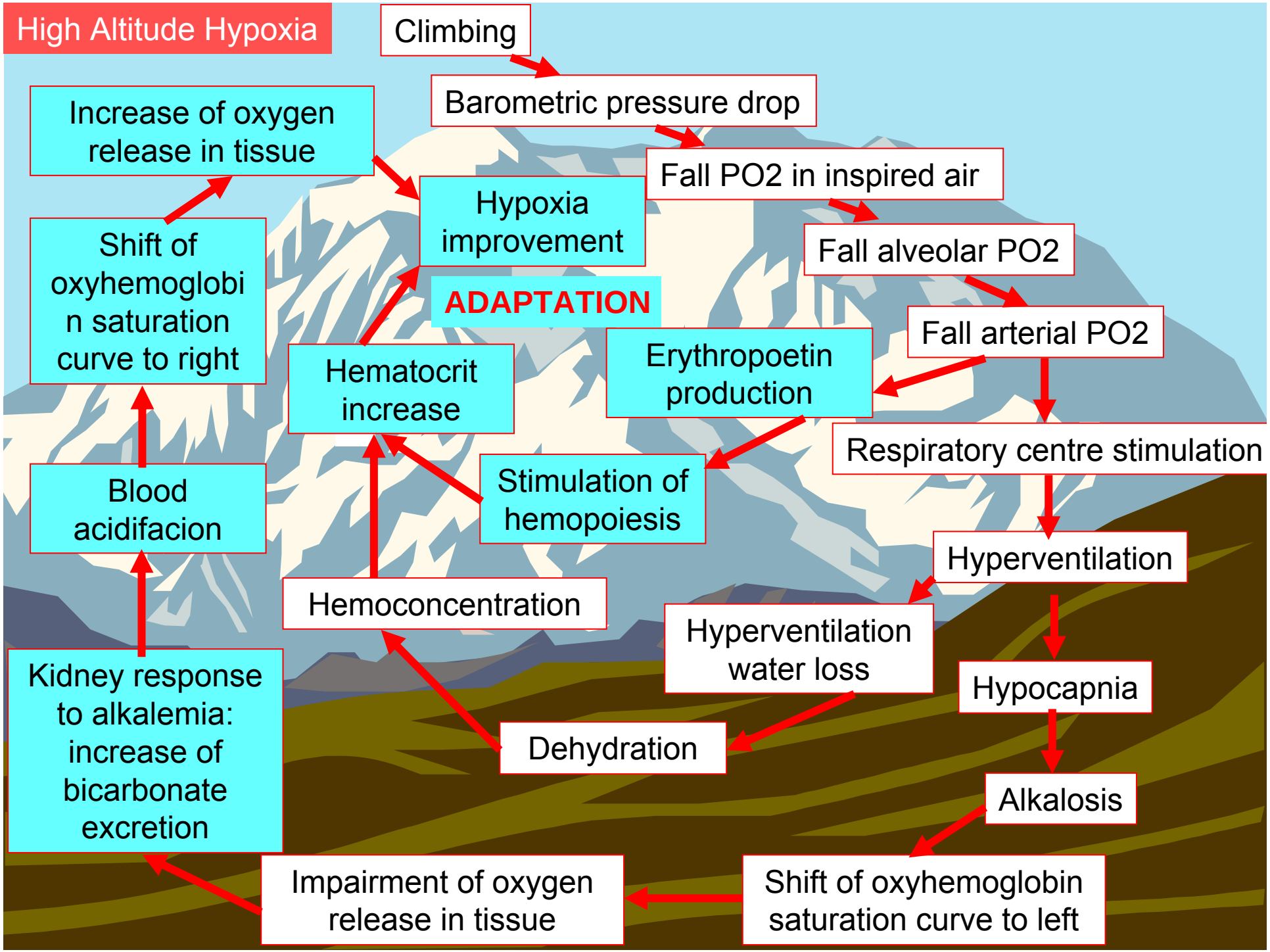

U.S. GEOLOGICAL SURVEY
COOPERATION WRITE THE DIRECTOR WASHINGTON, D.C.
FOR INFORMATION
ELEVATION
ABOVE SEA
FEET
SUMMIT
1955
BENCH MARK

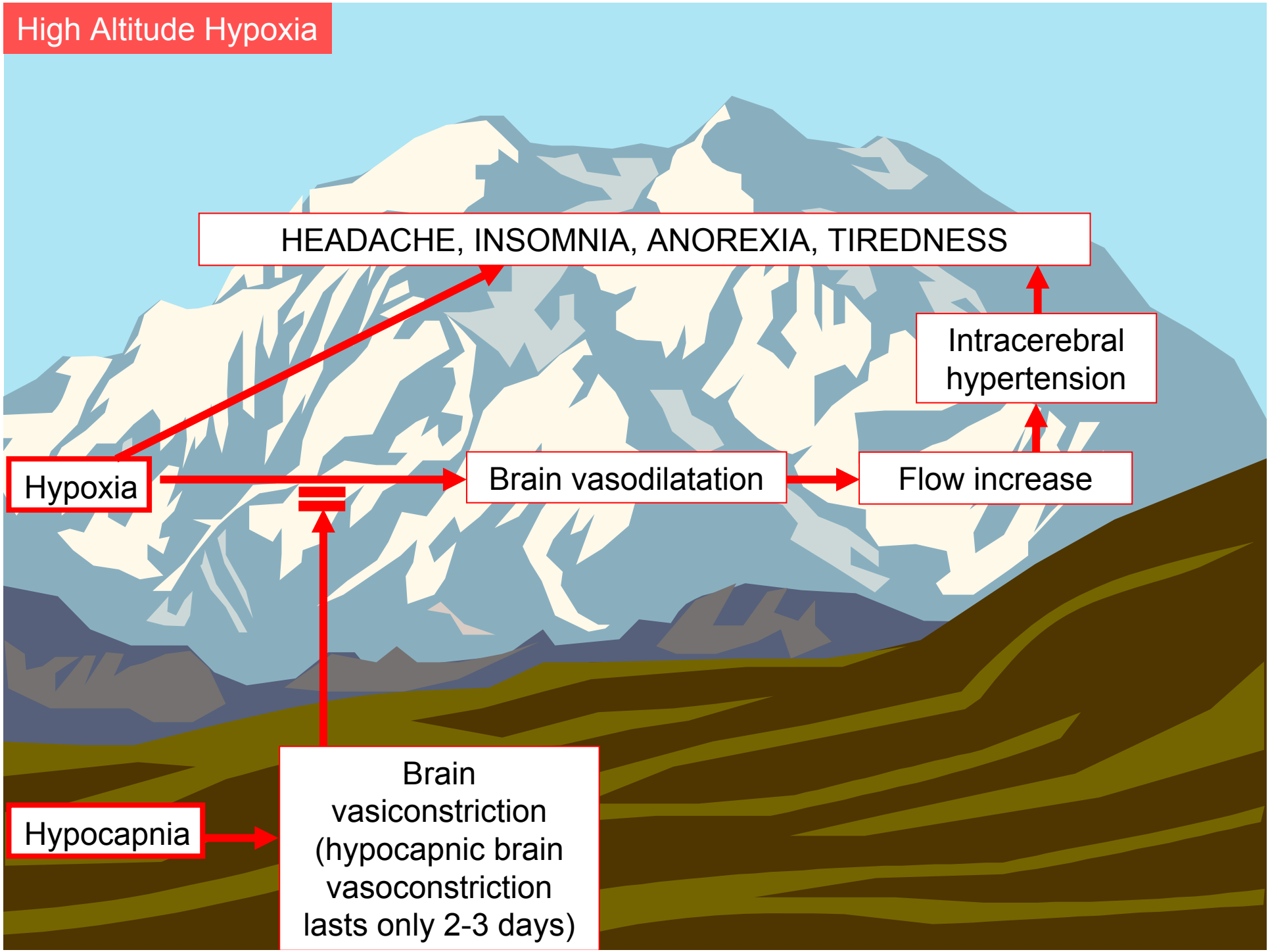



High Altitude Hypoxia
Climbing
Barometric pressure drop
Fall PO2 in inspired air
Fall alveolar PO2
Fall arterial PO2
Respiratory centre stimulation
Hyperventilation
Hypocapnia
Alkalosis
Shift of oxyhemoglobin saturation curve to left
Impairment of oxygen release in tissue
SO2
pO2
O2 release
O2 release

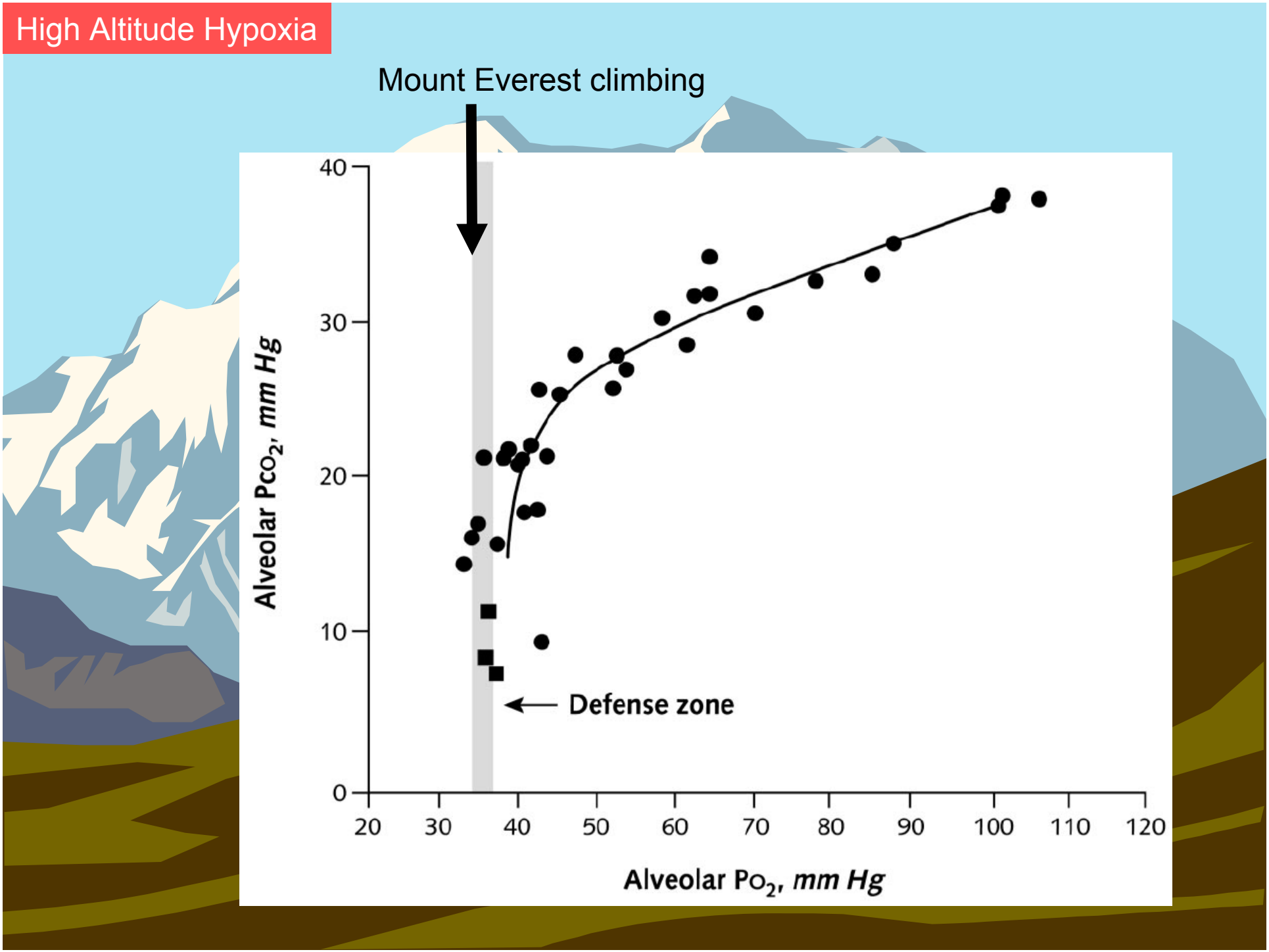

High Altitude Hypoxia
Climbing
Barometric pressure drop
Fall PO2 in inspired air
Fall alveolar PO2
Fall arterial PO2
Respiratory centre stimulation
Hyperventilation
Hypocapnia
Alkalosis
Shift of oxyhemoglobin saturation curve to left
Impairment of oxygen release in tissue
Kidney response to alkalemia: increase of bicarbonate excretion
Blood acidifacion
Shift of oxyhemoglobi n saturation curve to right
Increase of oxygen release in tissue
Hypoxia improvement
ADAPTATION
SO2
O2 release
O2 release
pO2


High Altitude Hypoxia
Climbing
Barometric pressure drop
Fall PO2 in inspired air
Fall alveolar PO2
Fall arterial PO2
Respiratory centre stimulation
Hyperventilation
Hypocapnia
Alkalosis
Shift of oxyhemoglobin saturation curve to left
Impairment of oxygen release in tissue
Kidney response to alkalemia: increase of bicarbonate excretion
Blood acidifacion
Shift of oxyhemoglobi n saturation curve to right
Increase of oxygen release in tissue
Hypoxia improvement
ADAPTATION
Decrease bicarbonate resorption in proximal tubule, bicarbonate excretion increases
Acetazolamid

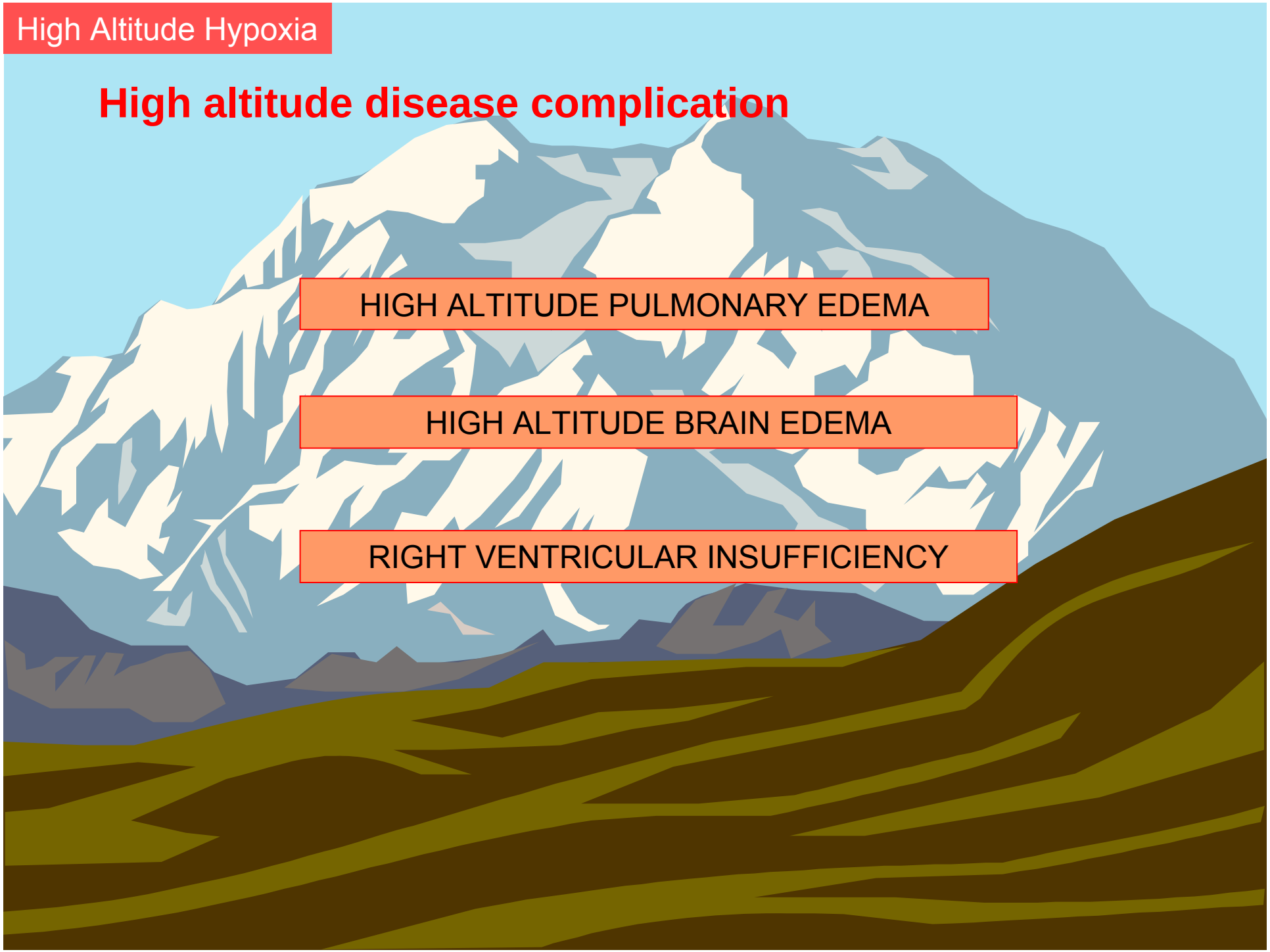

High Altitude Hypoxia
Climbing
Barometric pressure drop
Fall PO2 in inspired air
Fall alveolar PO2
Fall arterial PO2
Respiratory centre stimulation
Hyperventilation
Hypocapnia
Alkalosis
Shift of oxyhemoglobin saturation curve to left
Impairment of oxygen release in tissue
Kidney response to alkalemia: increase of bicarbonate excretion
Blood acidifacion
Shift of oxyhemoglobi n saturation curve to right
Increase of oxygen release in tissue
Hypoxia improvement
ADAPTATION
Hematocrit increase
Erythropoetin production
Stimulation of hemopoiesis
Hemoconcentration
Dehydration
Hyperventilation water loss

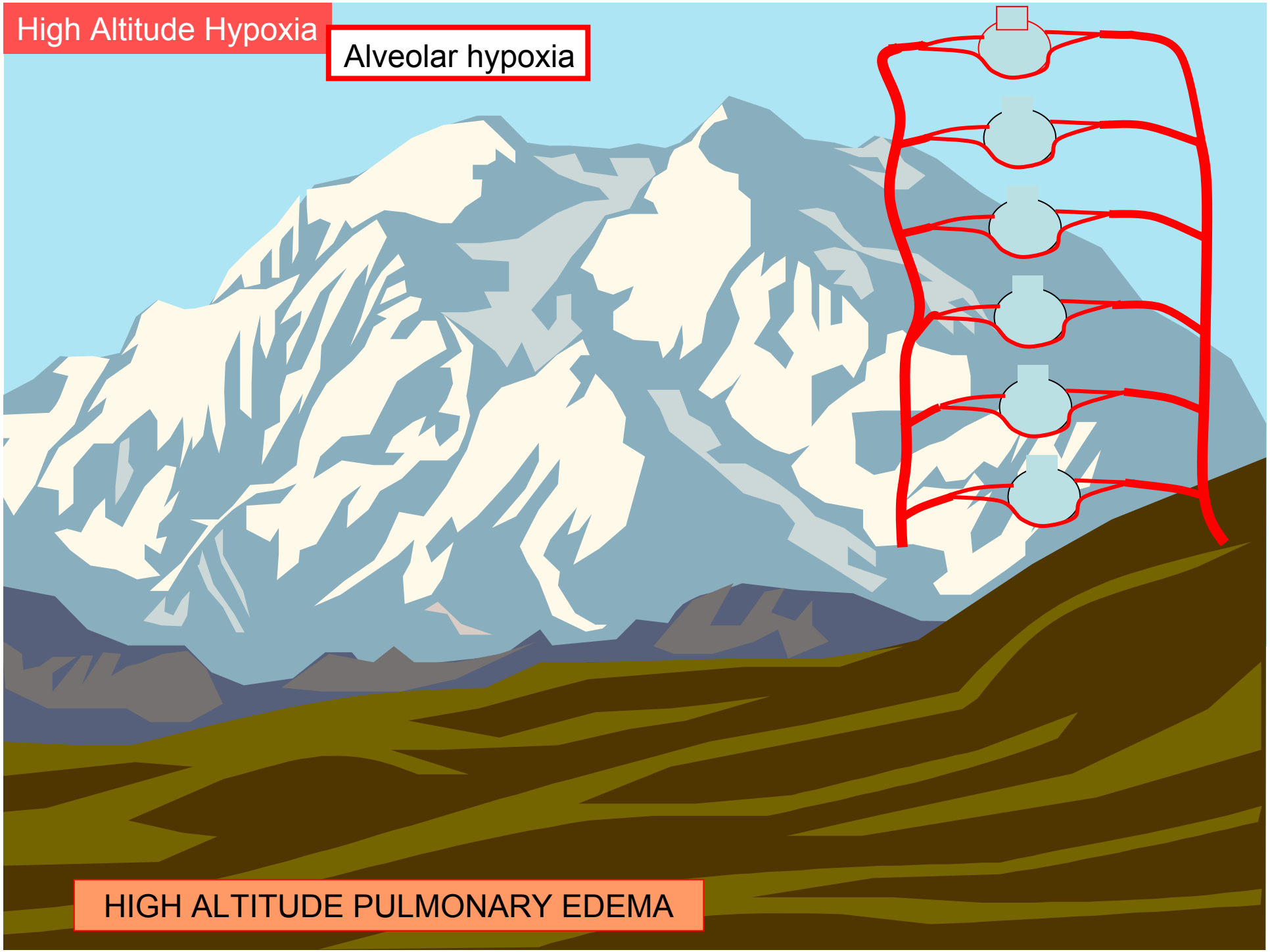

High Altitude Hypoxia
HEADACHE, INSOMNIA, ANOREXIA, TIREDNESS
Intracerebral hypertension
Hypoxia
Brain vasodilatation
Flow increase
Hypocapnia
Brain vasiconstriction (hypocapnic brain vasoconstriction lasts only 2-3 days)

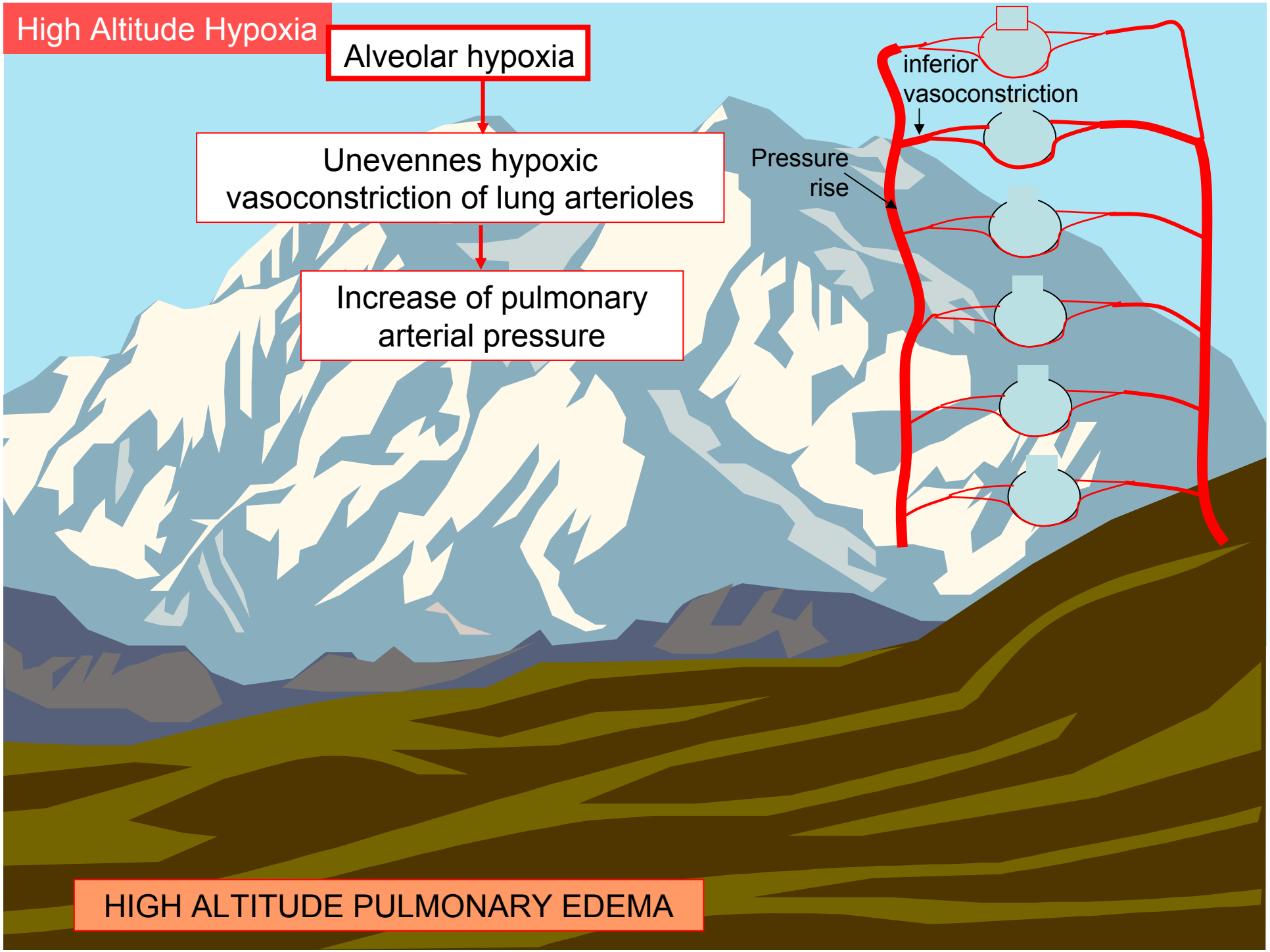
High Altitude Hypoxia

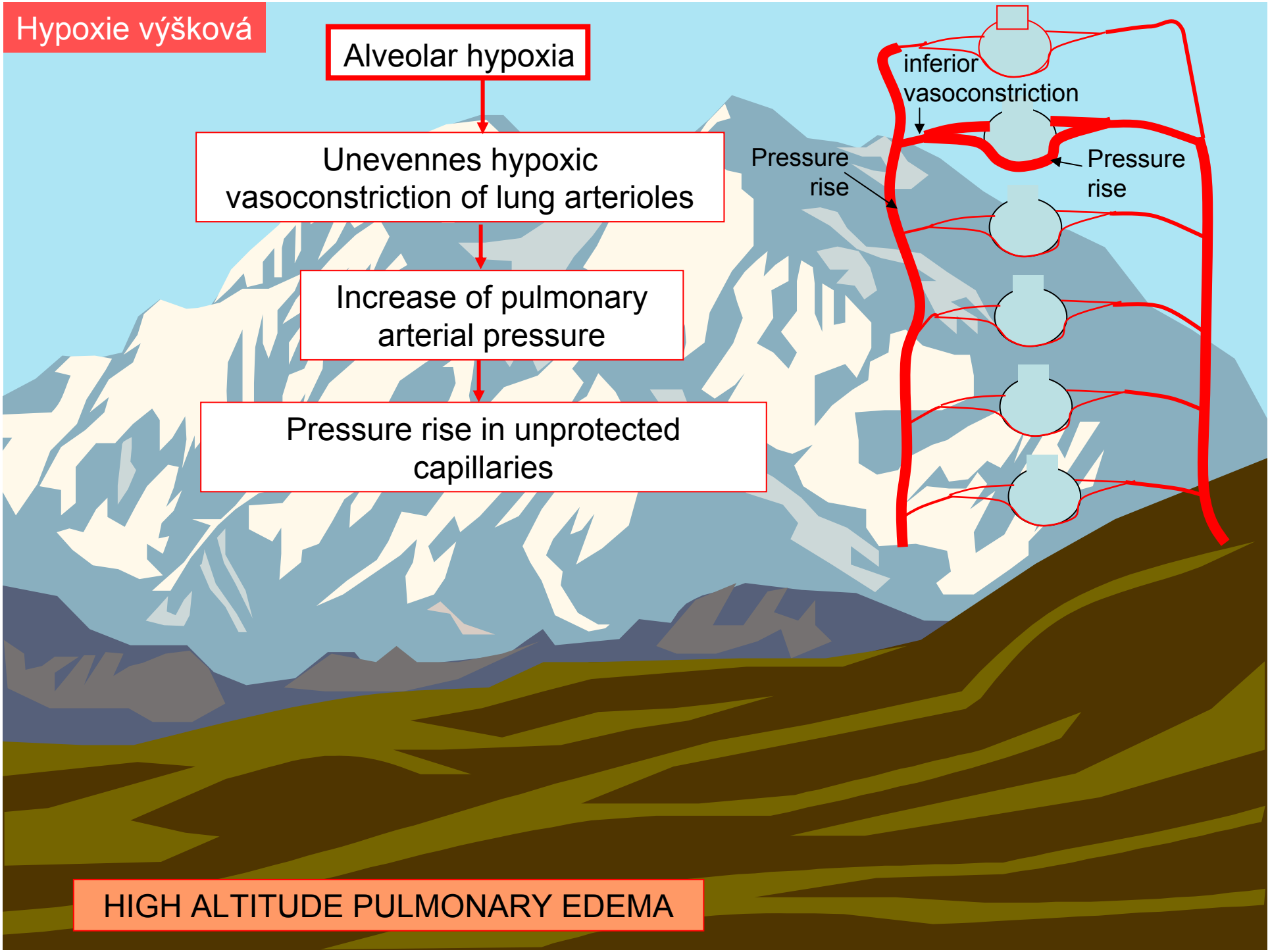


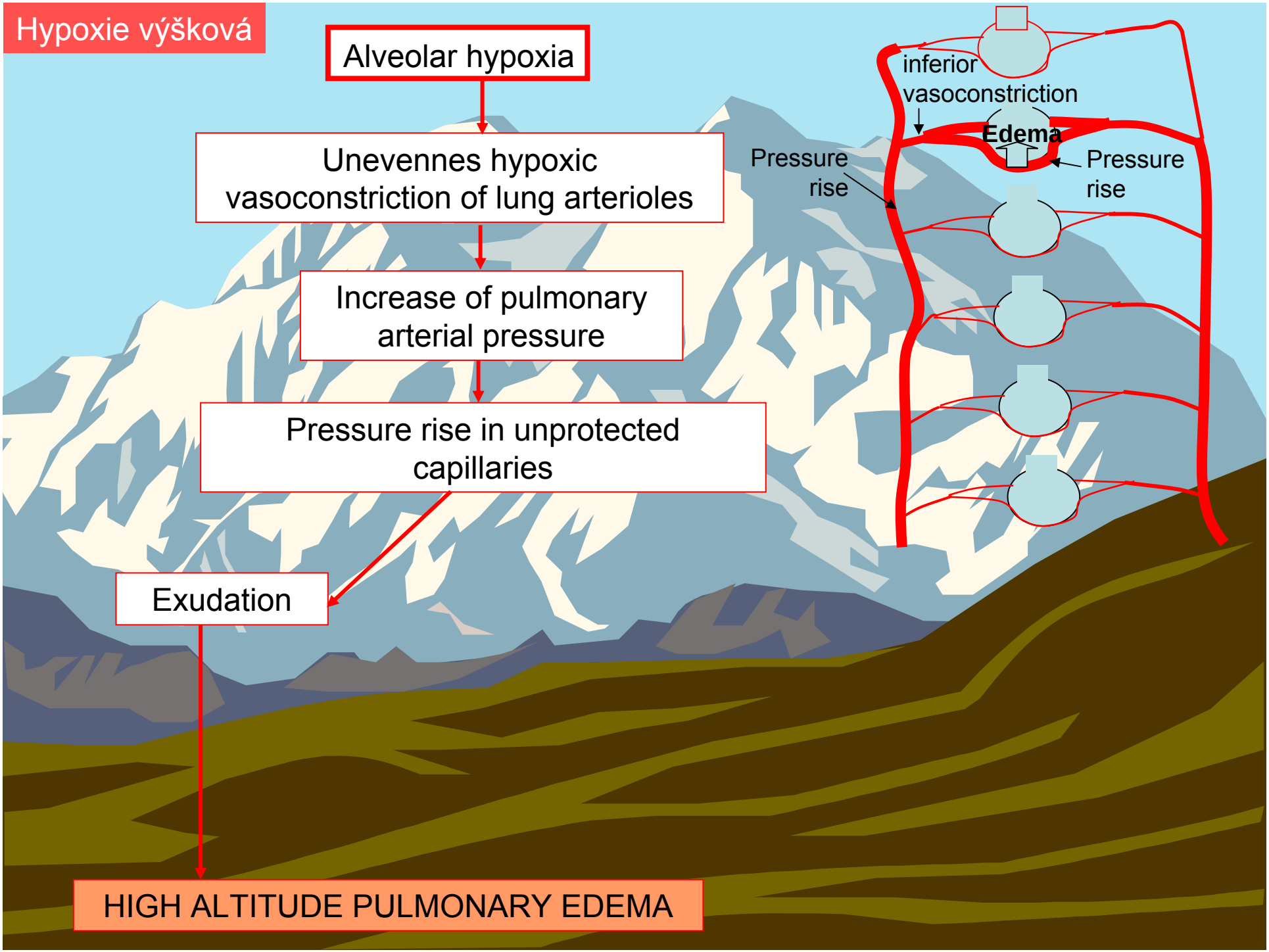
High altitude disease complication

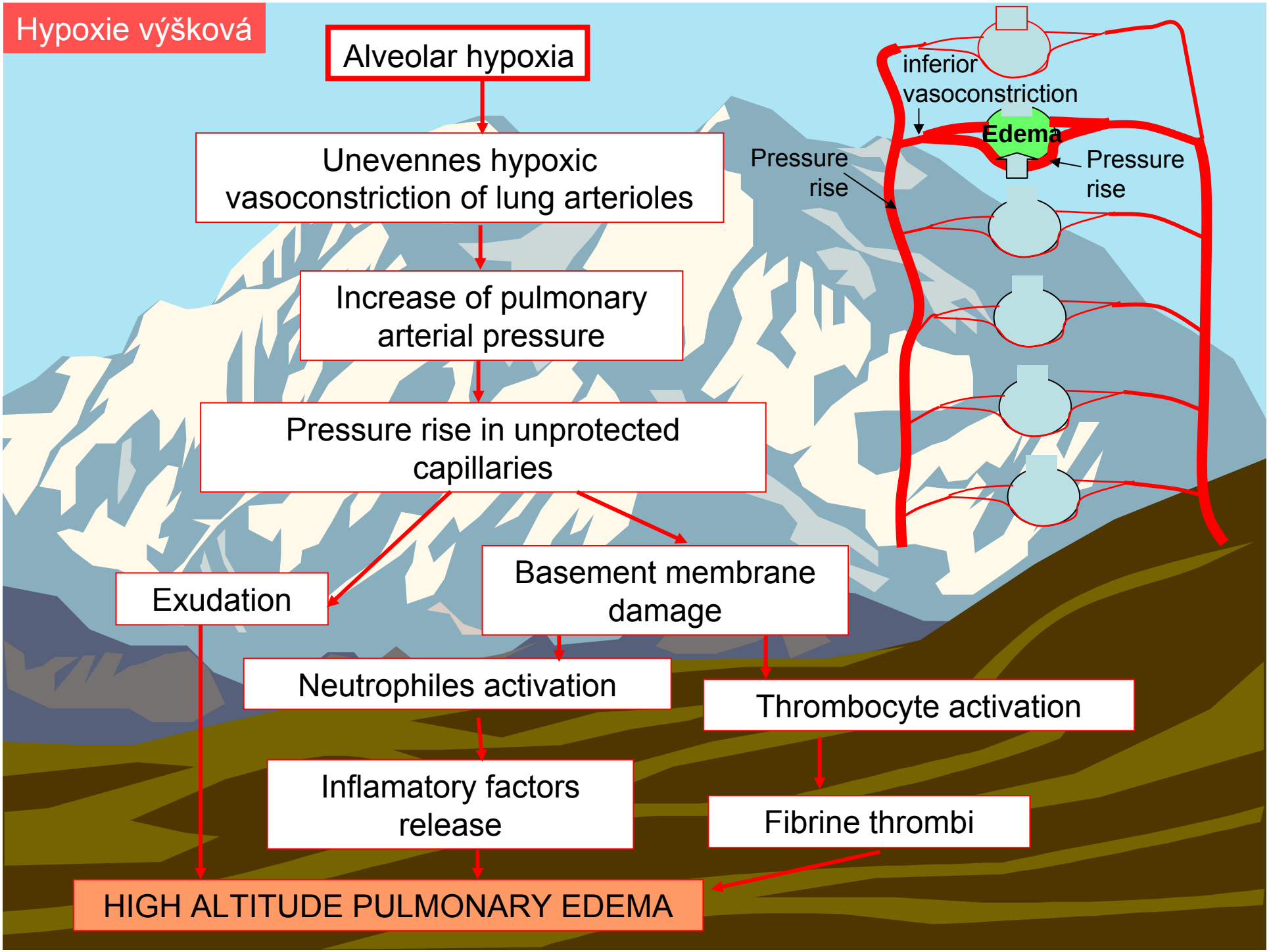


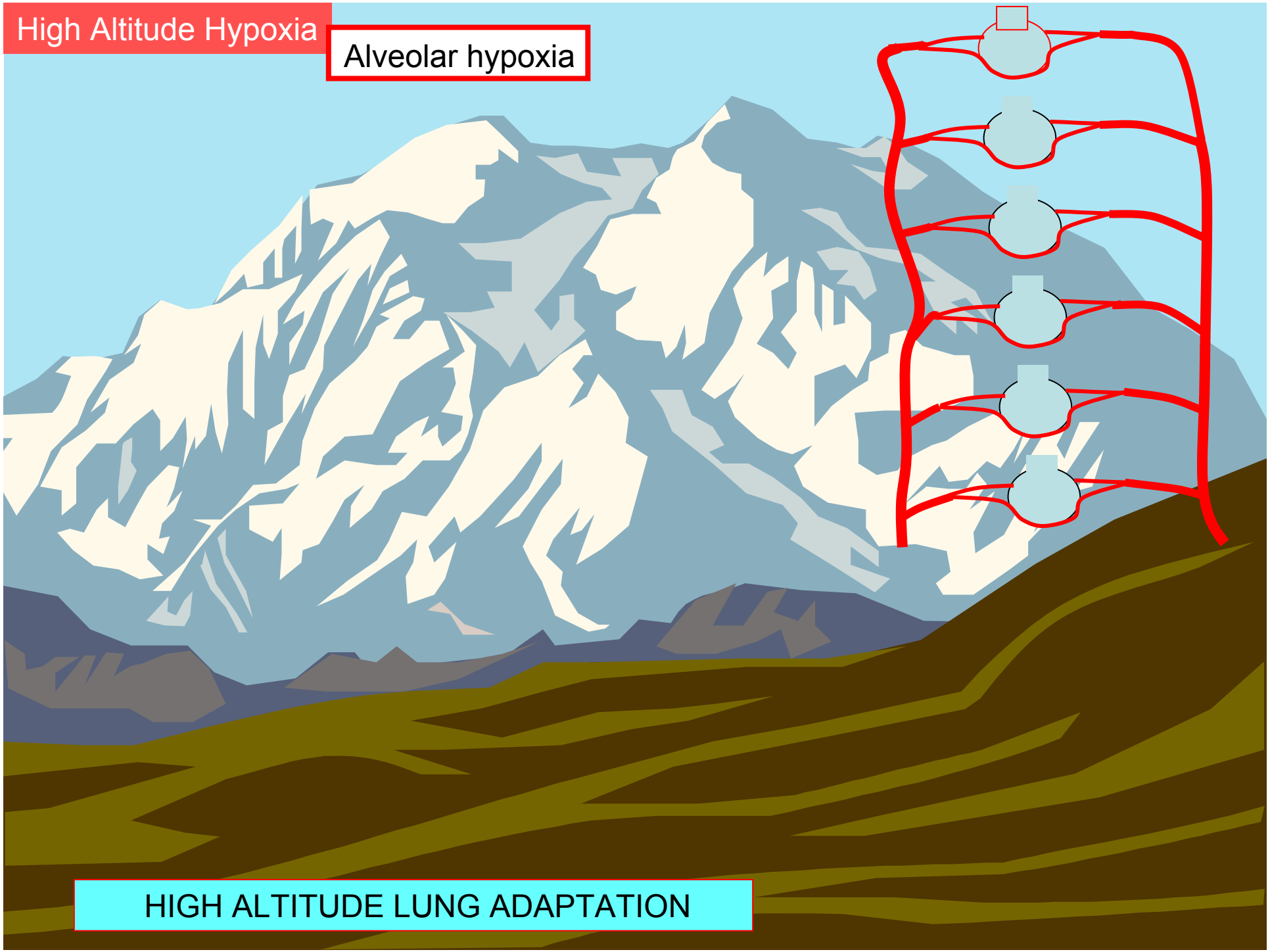

High Altitude Hypoxia
Alveolar hypoxia
HIGH ALTITUDE PULMONARY EDEMA

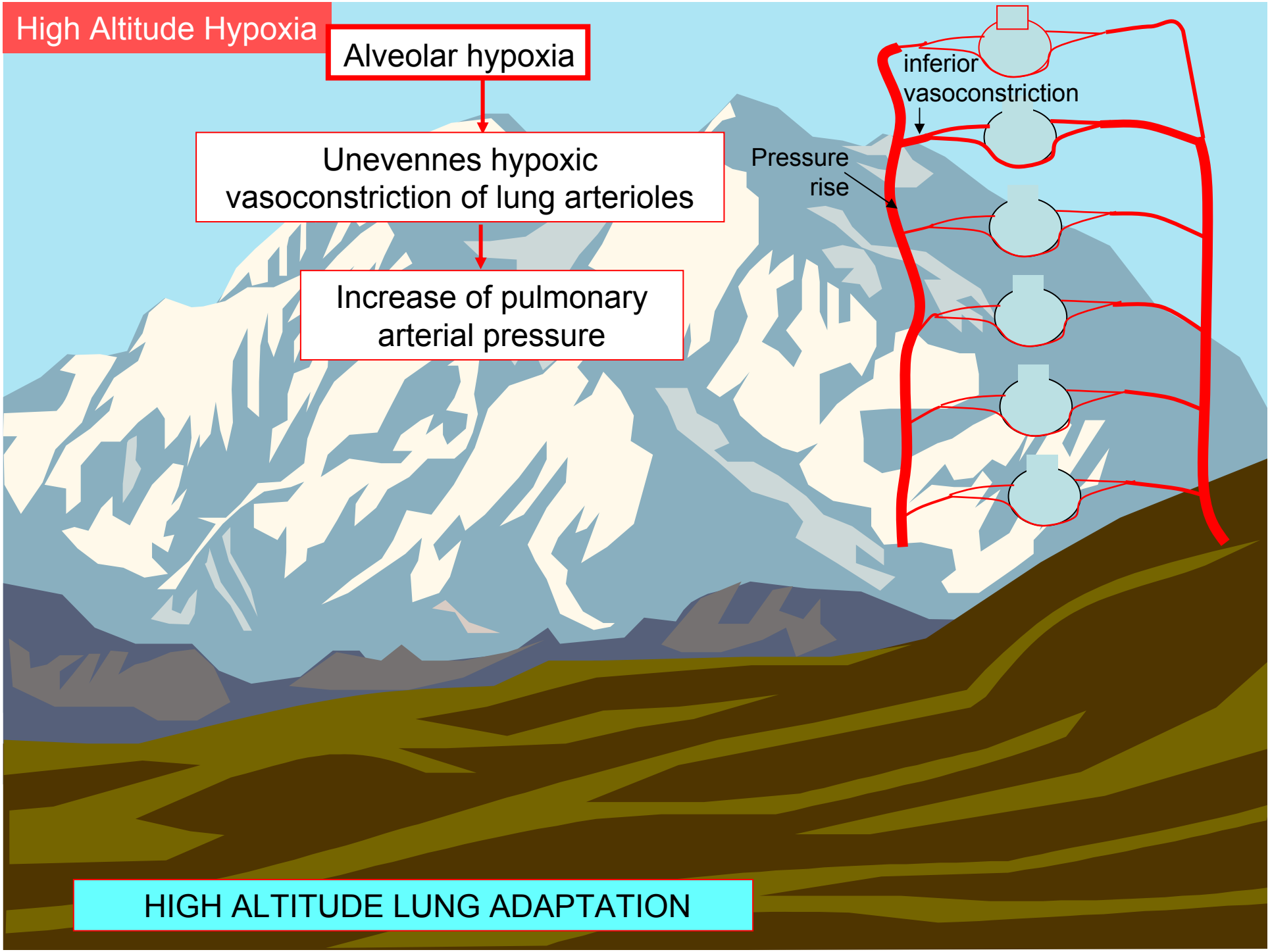

High Altitude Hypoxia
Alveolar hypoxia
Unevennes hypoxic vasoconstriction of lung arterioles
Increase of pulmonary arterial pressure
inferior vasoconstriction
Pressure rise
HIGH ALTITUDE PULMONARY EDEMA

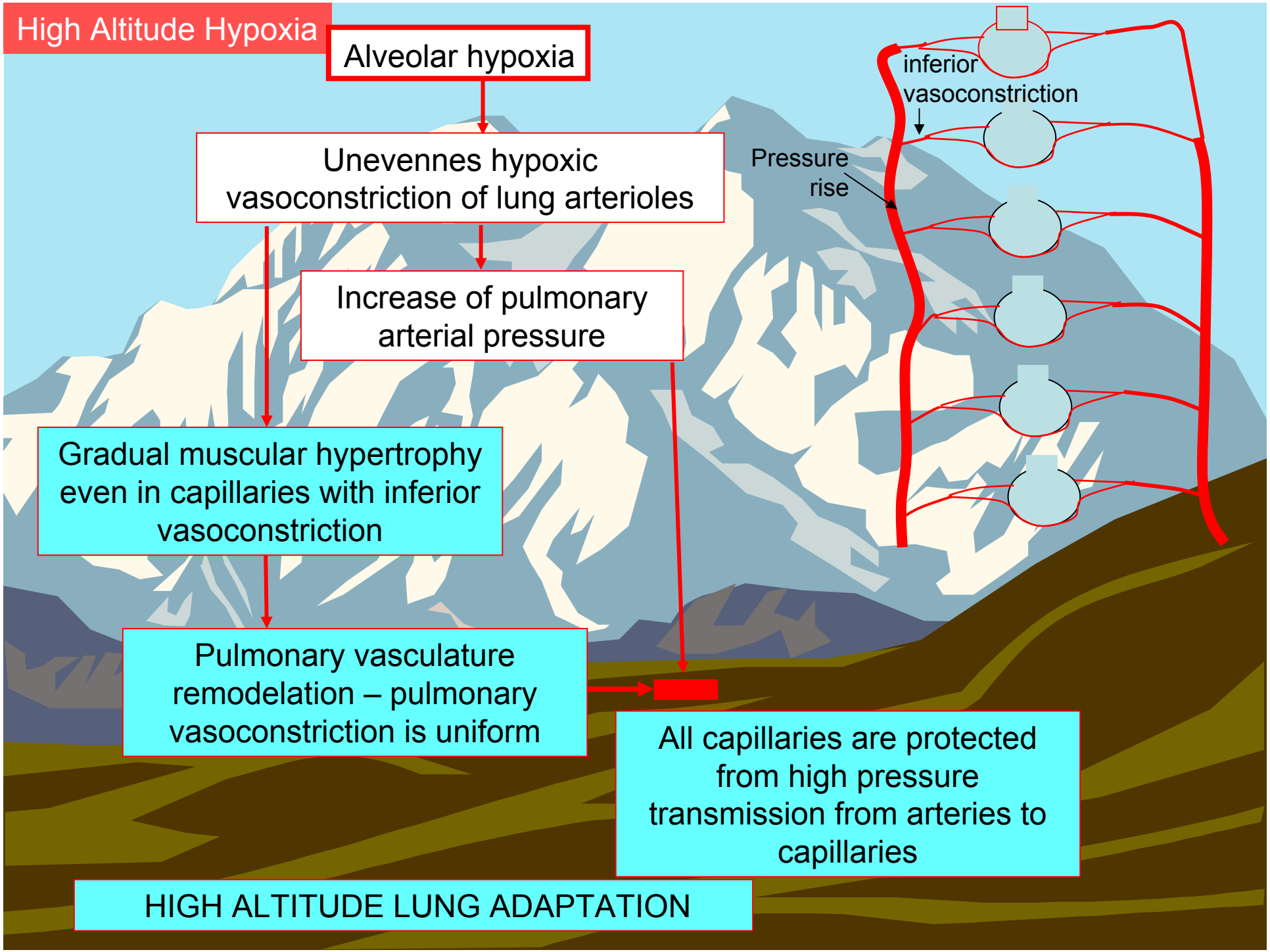

Hypoxie výšková
Alveolar hypoxia
Unevennes hypoxic vasoconstriction of lung arterioles
Increase of pulmonary arterial pressure
Pressure rise in unprotected capillaries
inferior vasoconstriction
Pressure rise
Pressure rise
HIGH ALTITUDE PULMONARY EDEMA

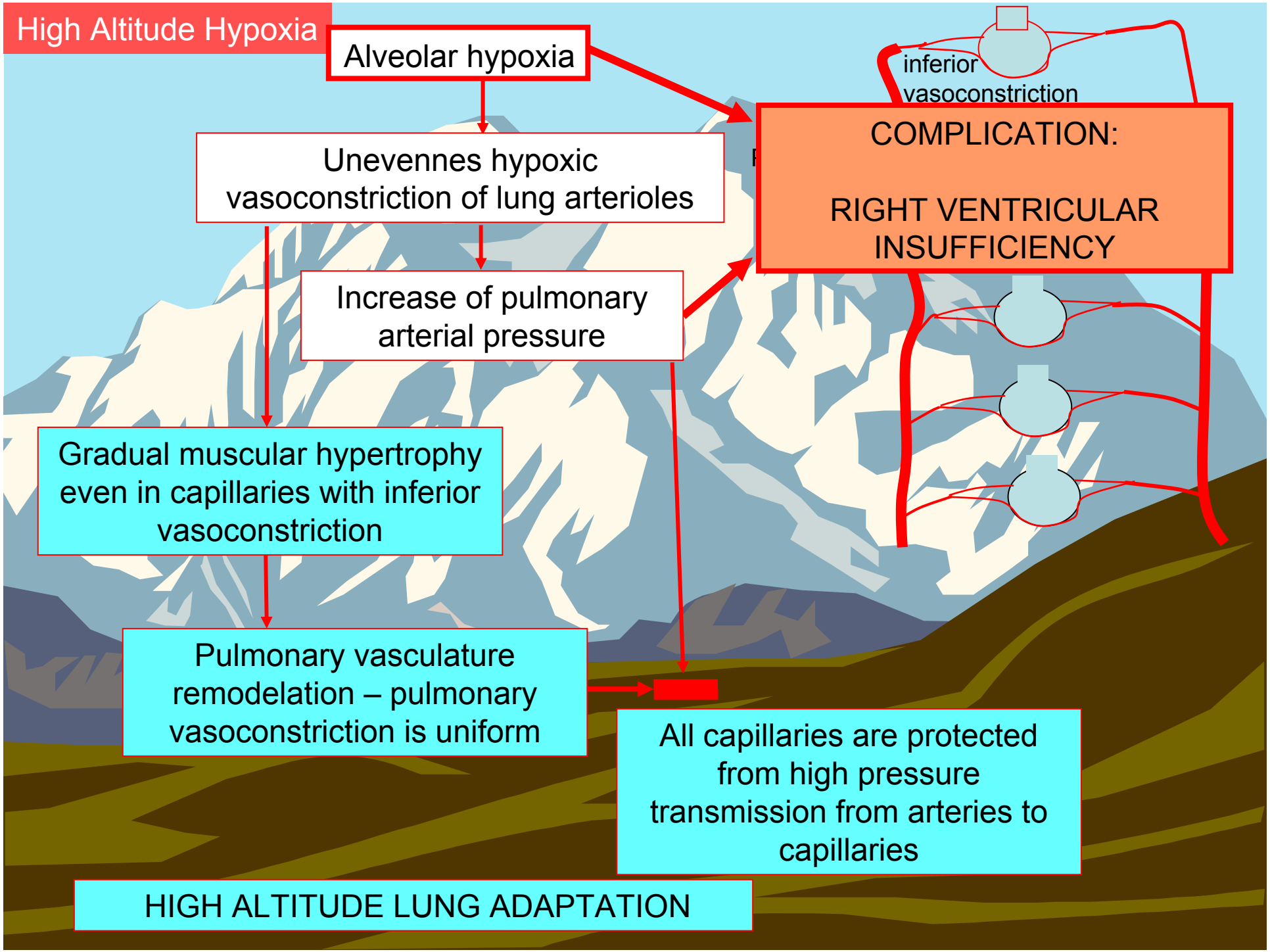

Hypoxie výšková
Alveolar hypoxia
Unevennes hypoxic vasoconstriction of lung arterioles
Increase of pulmonary arterial pressure
Pressure rise in unprotected capillaries
Exudation
HIGH ALTITUDE PULMONARY EDEMA
inferior vasoconstriction
Edema
Pressure rise
Pressure rise

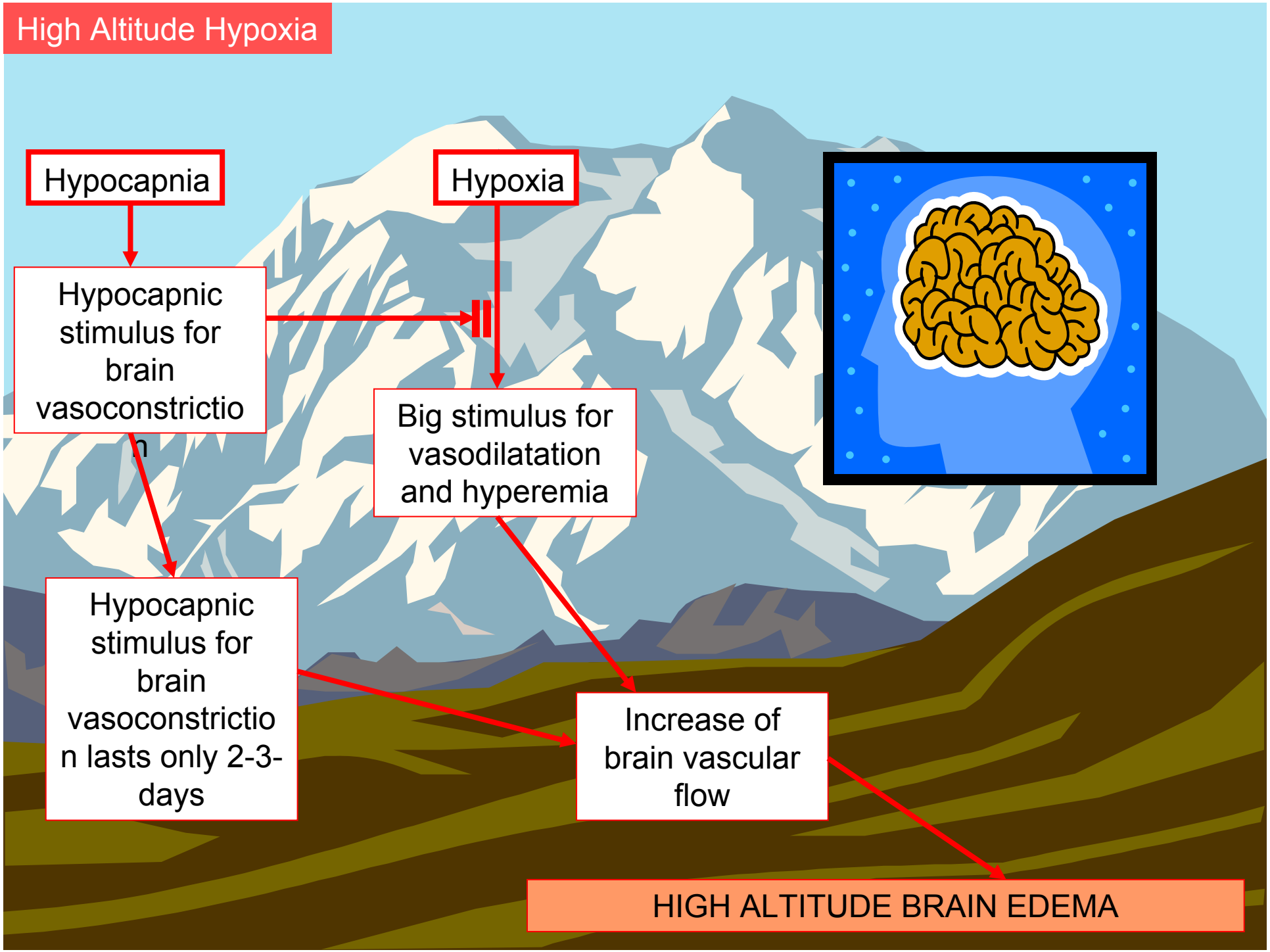

Hypoxie výšková
Alveolar hypoxia
Unevennes hypoxic vasoconstriction of lung arterioles
Increase of pulmonary arterial pressure
Pressure rise in unprotected capillaries
Exudation
Basement membrane damage
Neutrophiles activation
Thrombocyte activation
Inflamatory factors release
Fibrine thrombi
HIGH ALTITUDE PULMONARY EDEMA
inferior vasoconstriction
Edema
Pressure rise
Pressure rise


High Altitude Hypoxia
Alveolar hypoxia
HIGH ALTITUDE LUNG ADAPTATION

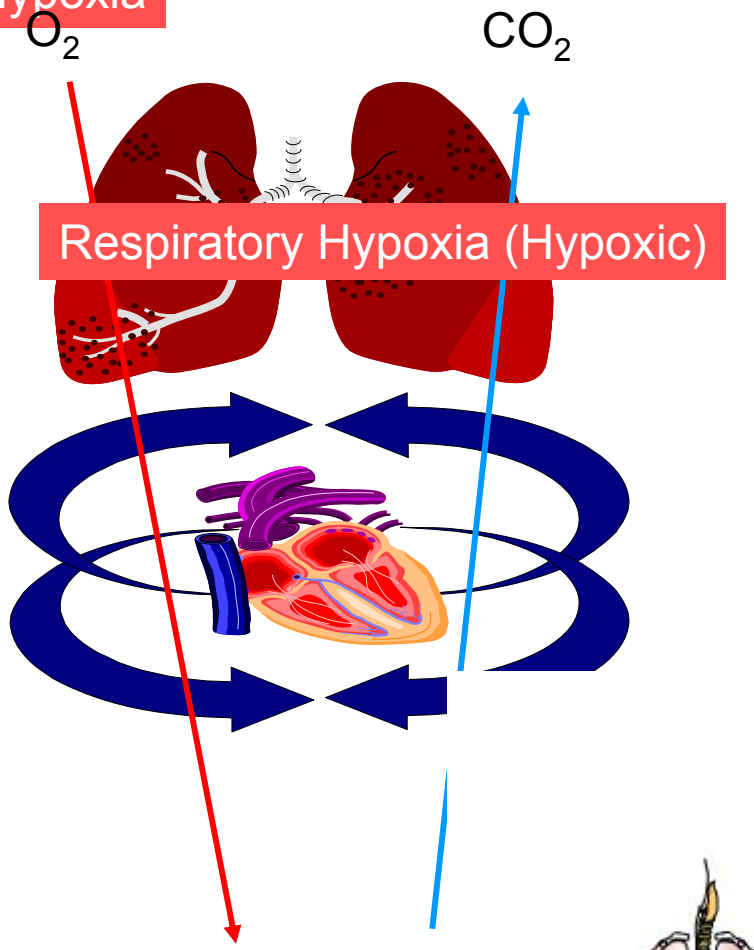

High Altitude Hypoxia
Alveolar hypoxia
Unevennes hypoxic vasoconstriction of lung arterioles
Increase of pulmonary arterial pressure
inferior vasoconstriction
Pressure rise
HIGH ALTITUDE LUNG ADAPTATION

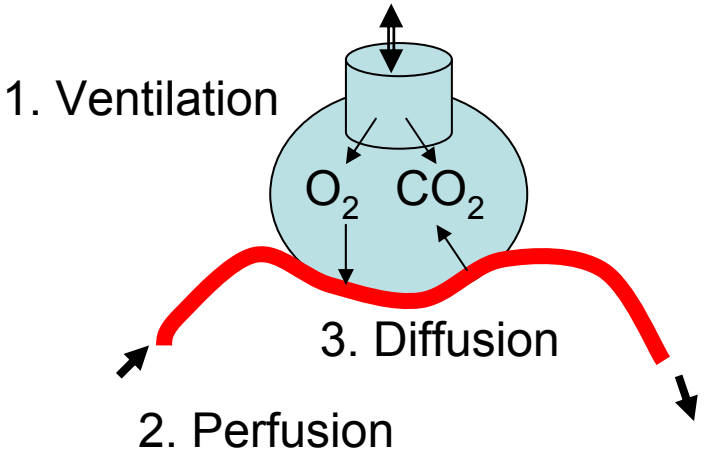

High Altitude Hypoxia
Alveolar hypoxia
Unevennes hypoxic vasoconstriction of lung arterioles
Increase of pulmonary arterial pressure
Gradual muscular hypertrophy even in capillaries with inferior vasoconstriction
Pulmonary vasculature remodelation – pulmonary vasoconstriction is uniform
All capillaries are protected from high pressure transmission from arteries to capillaries
HIGH ALTITUDE LUNG ADAPTATION
inferior vasoconstriction
Pressure rise

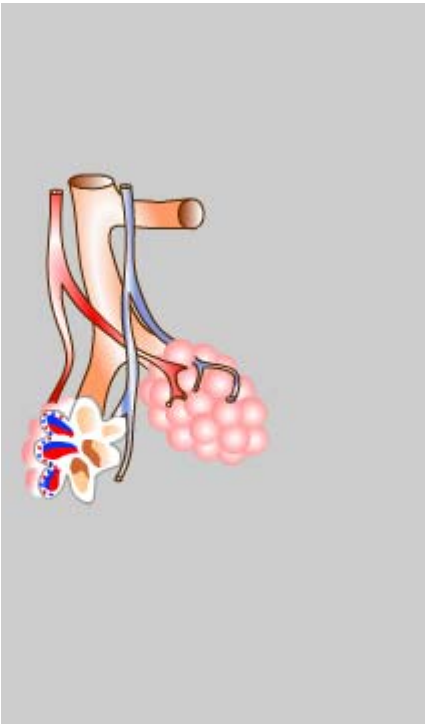

High Altitude Hypoxia
Alveolar hypoxia
Unevennes hypoxic vasoconstriction of lung arterioles
Increase of pulmonary arterial pressure
Gradual muscular hypertrophy even in capillaries with inferior vasoconstriction
Pulmonary vasculature remodelation – pulmonary vasoconstriction is uniform
inferior vasoconstriction
COMPLICATION:
RIGHT VENTRICULAR INSUFFICIENCY
All capillaries are protected from high pressure transmission from arteries to capillaries
HIGH ALTITUDE LUNG ADAPTATION


High Altitude Hypoxia
Hypocapnia
Hypoxia
Hypocapnic stimulus for brain vasoconstrictio n
Big stimulus for vasodilatation and hyperemia
Hypocapnic stimulus for brain vasoconstrictio n lasts only 2-3-days
Increase of brain vascular flow
HIGH ALTITUDE BRAIN EDEMA

High Altitude Hypoxia







What is the function of lungs?

Alveolus

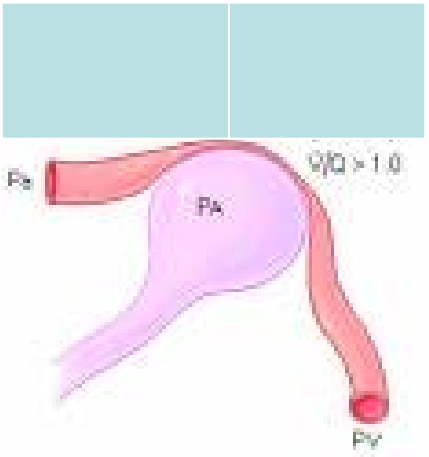
- **Ventilation** – mechanical function of the lung – get air in and out
- **Perfusion** with blood – get blood in and out
- **Diffusion** – get gas molecules from air to blood and back
- **Matching** of ventilation and perfusion

Possible respiratory system disturbances

- // **ventilation**
- // **perfusion**
- // **distribution** of ventilation and per-fusion

= ventilation perfusion mismatch

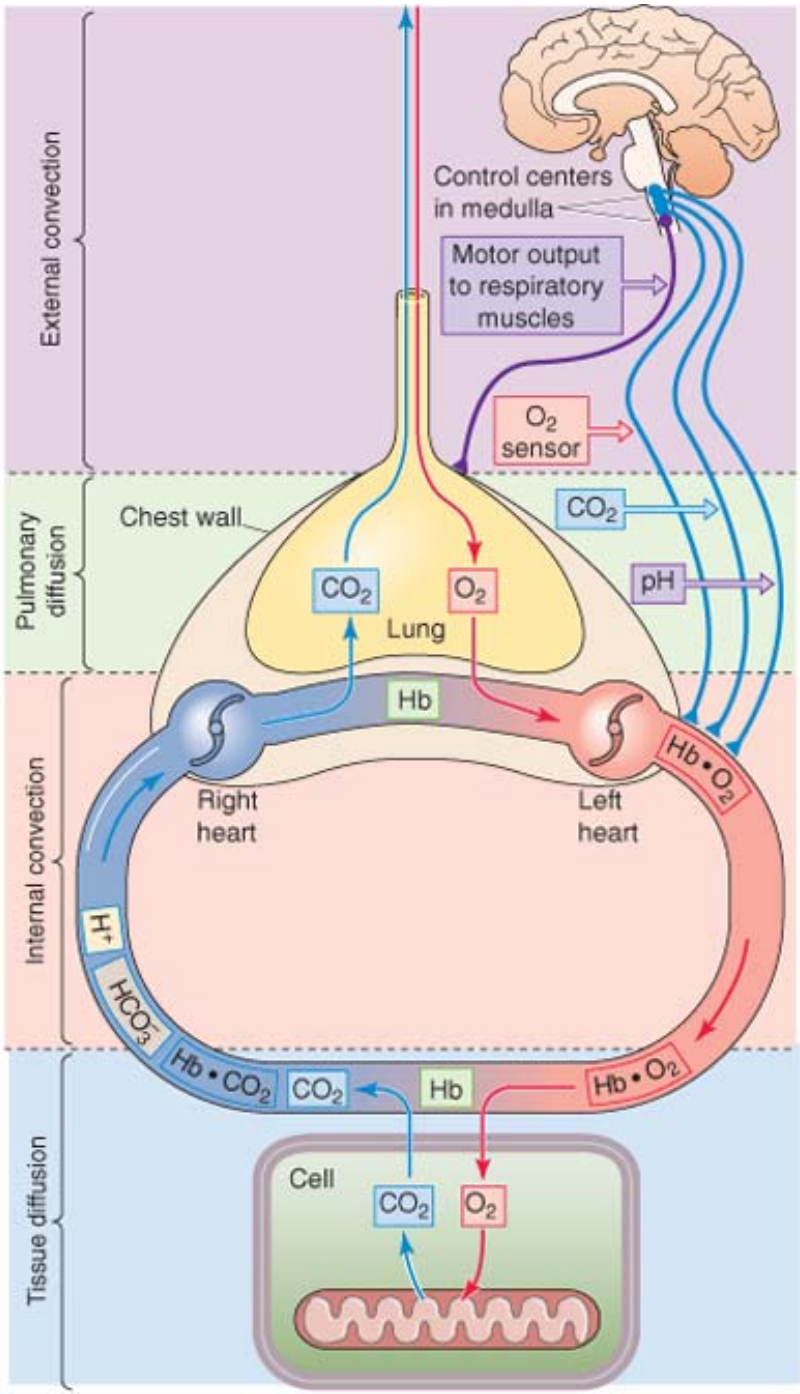
- // **diffusion**
- ***Important: Ventilation, perfusion and their distribution are feedback regulated processes.***
- ***Disturbance:***
  - ***1. In the effector part (lungs, resp. muscles for ventilation, heart for perfusion)***
  - ***2. In the regulator part (sensors, CNS eg. in uremia, liver in hepatopulmonary syndrome)***



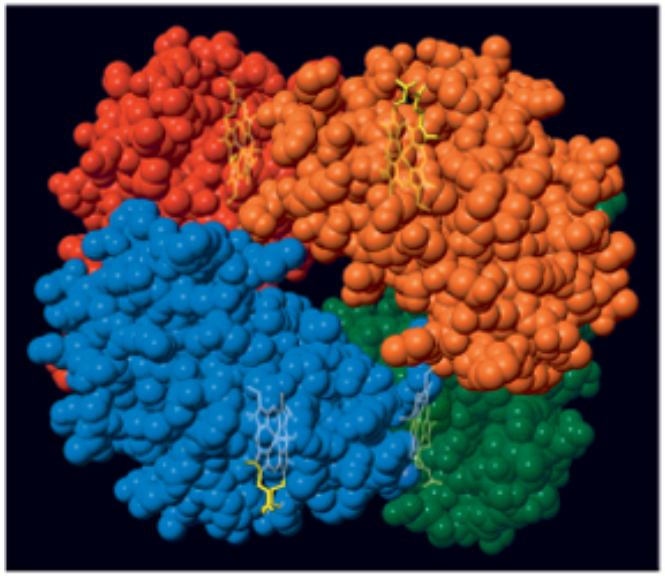
The overall measure of respiratory system function

- **pO2** & **pCO2 in arterial blood -** („**Astrup**“)
- O2 solubility in water is low => need of Hemoglobin
- **pO2** = 13,3 kPa = **100 Torr**
- **pCO2** = 5,3 kPa = **40 Torr**

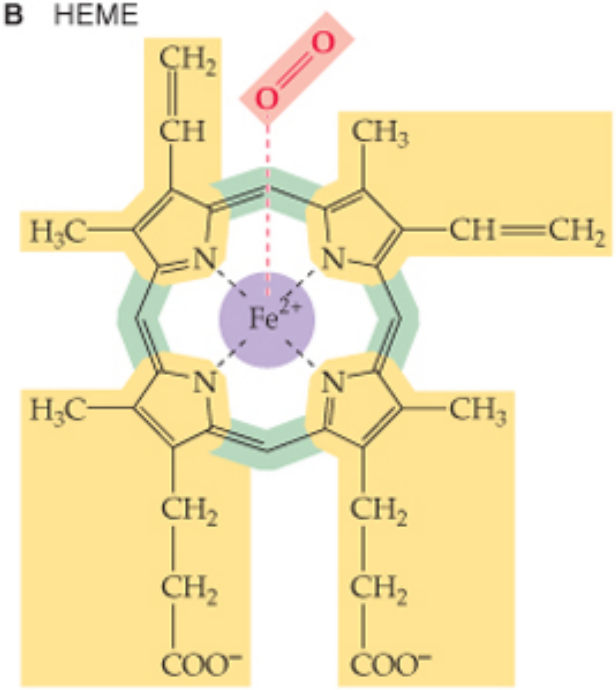
(1 kPa = 10 cm H2O = 7,6 mmHg or Torr)


External convection
Pulmonary diffusion
Internal convection
Tissue diffusion
Control centers in medulla
Motor output to respiratory muscles
O_2 sensor
CO_2
pH
Chest wall
CO_2
O_2
Lung
Hb
Right heart
Left heart
$Hb \cdot O_2$
H^+
HCO_3^-
$Hb \cdot CO_2$
CO_2
Hb
$Hb \cdot O_2$
Cell
CO_2
O_2

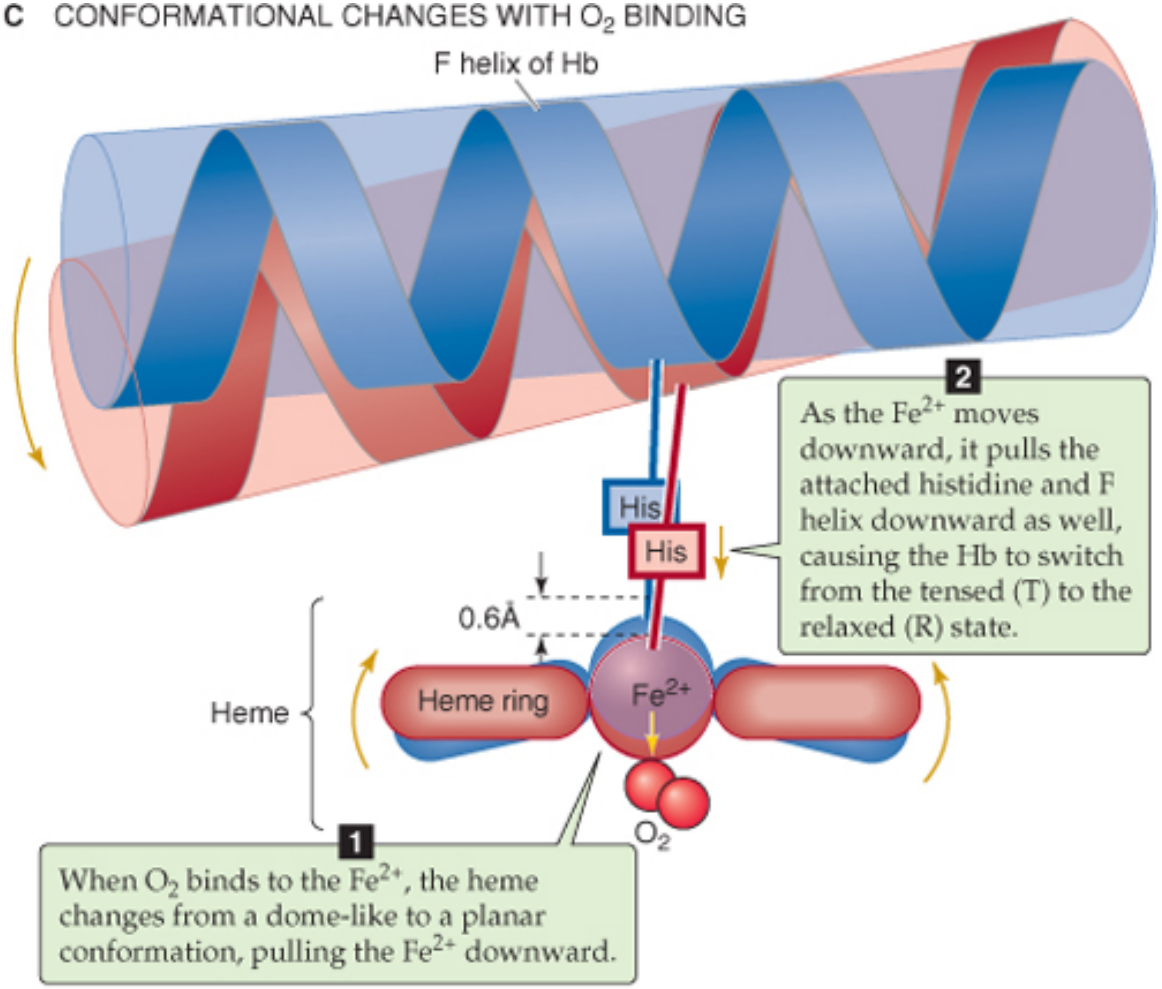
A HEMOGLOBIN TETRAMER

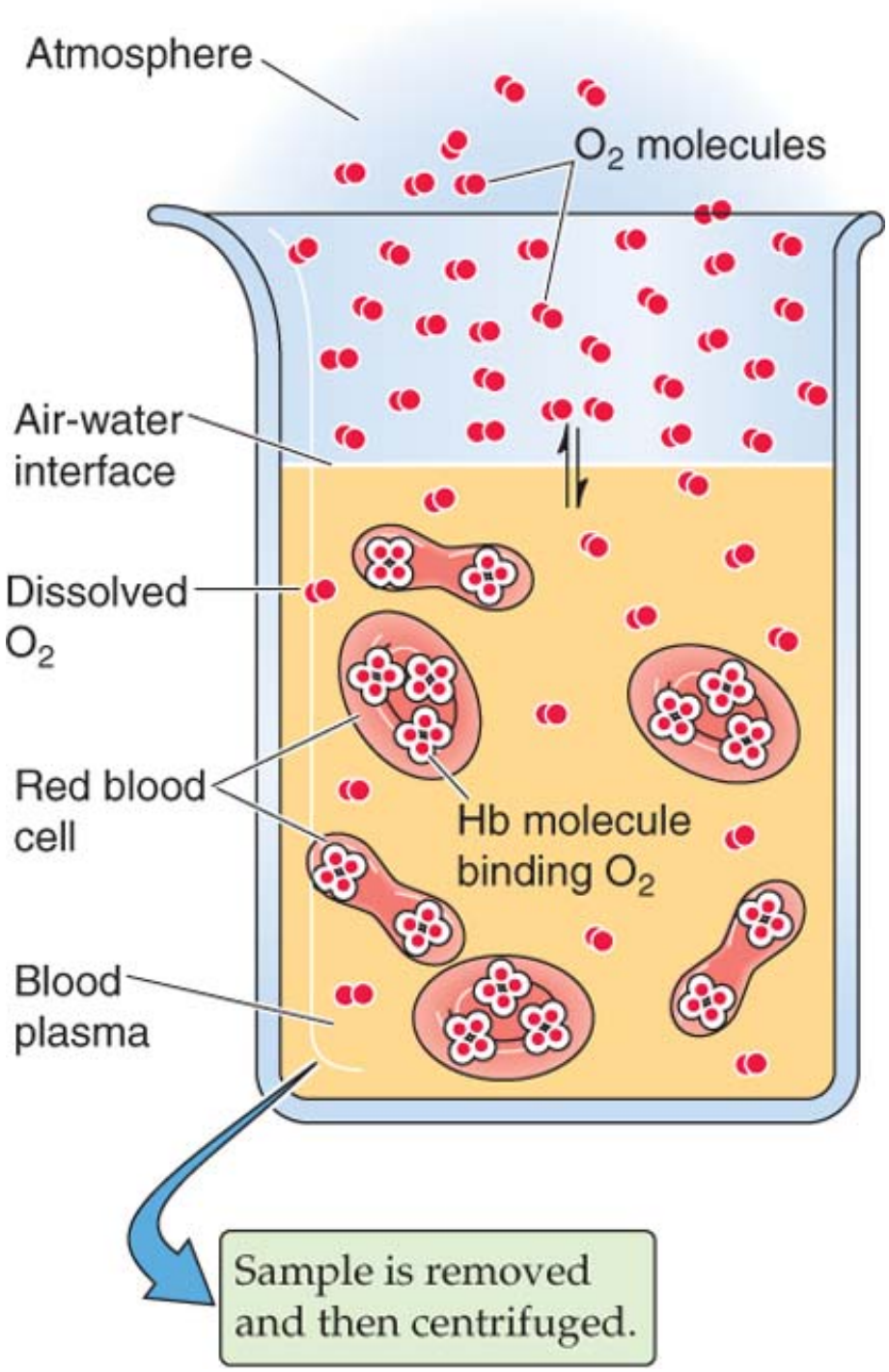


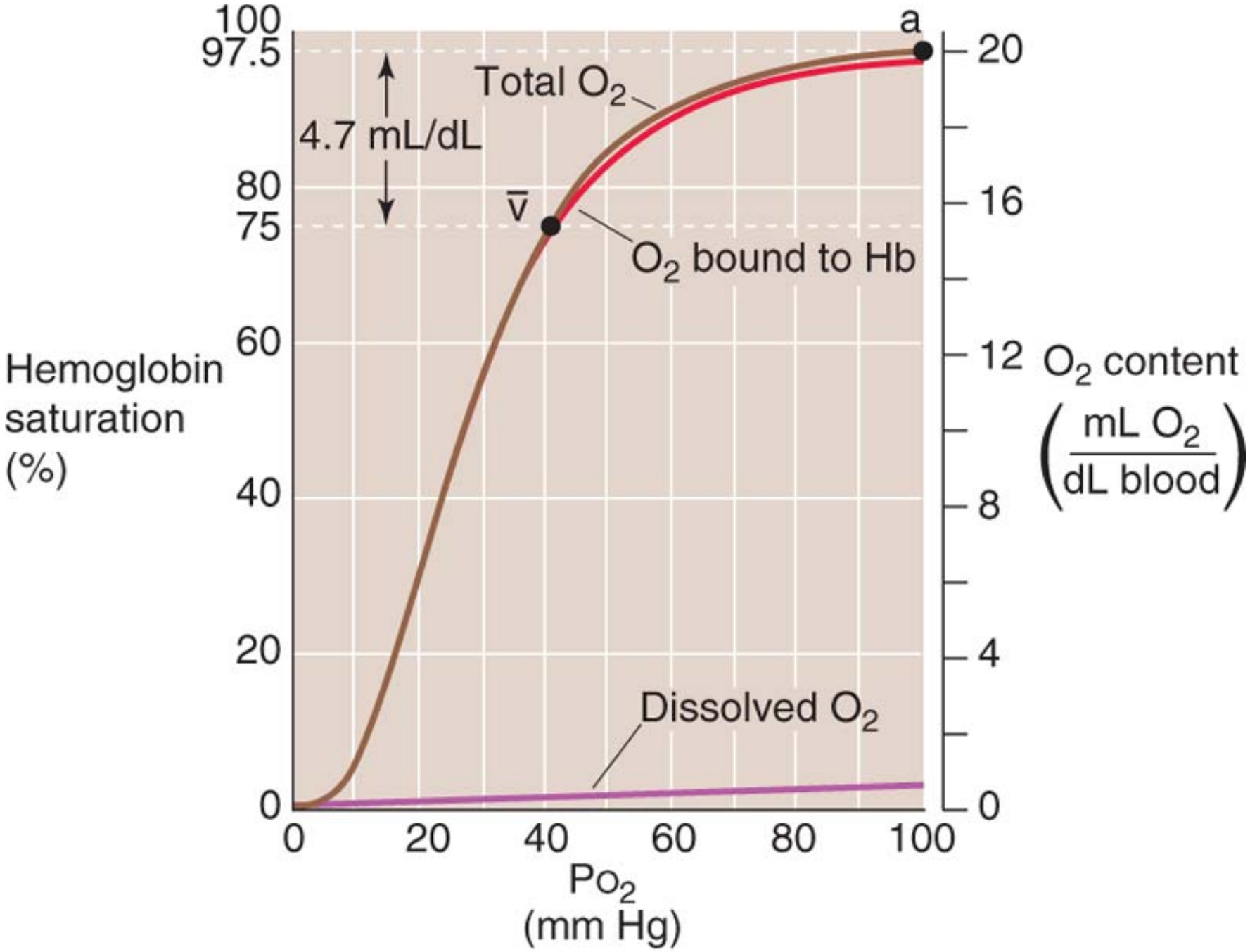
B HEME

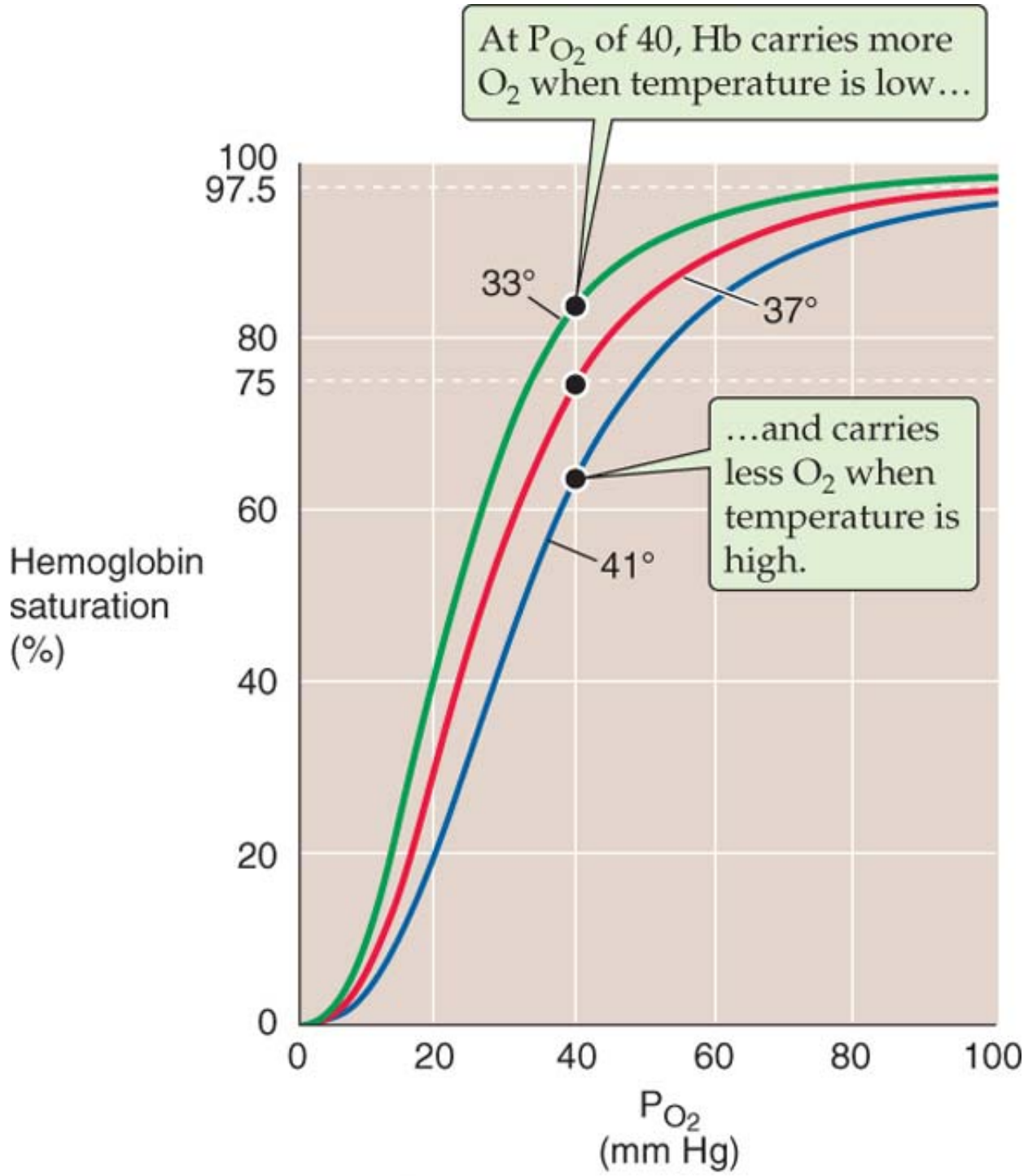


C CONFORMATIONAL CHANGES WITH O_2 BINDING

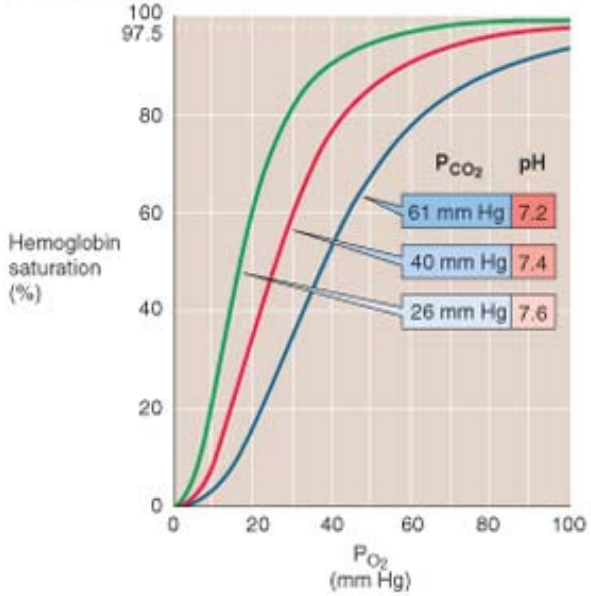



Atmosphere
O_2 molecules
Air-water interface
Dissolved O_2
Red blood cell
Hb molecule binding O_2
Blood plasma
Sample is removed and then centrifuged.

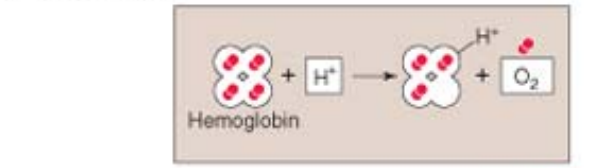

100
97.5
80
75
60
40
20
0
a
Total O_2
4.7 mL/dL
$\bar{v}$
O_2 bound to Hb
Dissolved O_2
Hemoglobin saturation (%)
0
20
40
60
80
100
P_{O_2} (mm Hg)
20
16
12
8
4
0
O_2 content $\left(\frac{\text{mL } O_2}{\text{dL blood}}\right)$

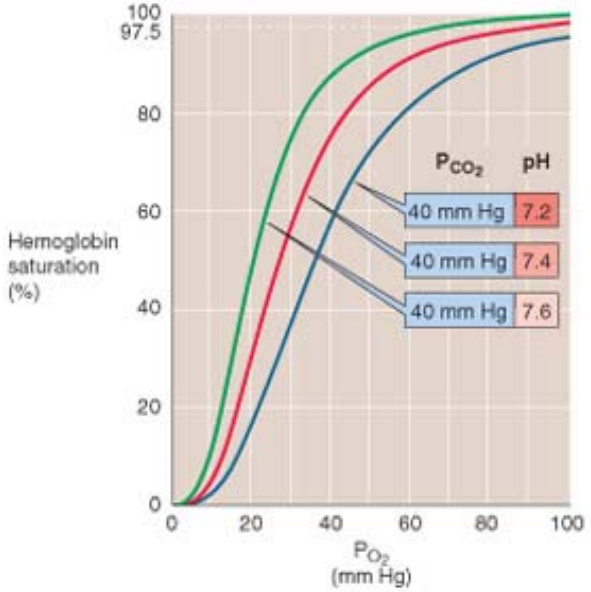

At P_{O_2} of 40, Hb carries more O_2 when temperature is low…
100
97.5
80
75
60
40
20
0
33°
37°
41°
…and carries less O_2 when temperature is high.
Hemoglobin saturation (%)
0
20
40
60
80
100
P_{O_2} (mm Hg)

A RESPIRATORY ACID-BASE DISTURBANCES

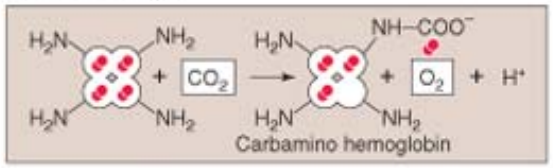


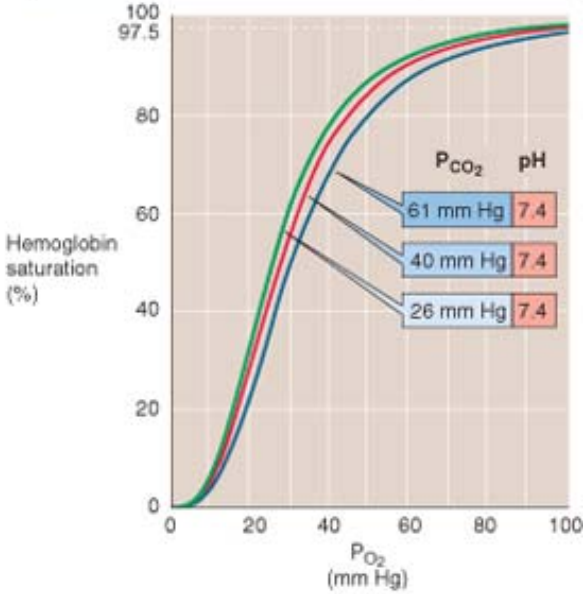
B EFFECT OF pH

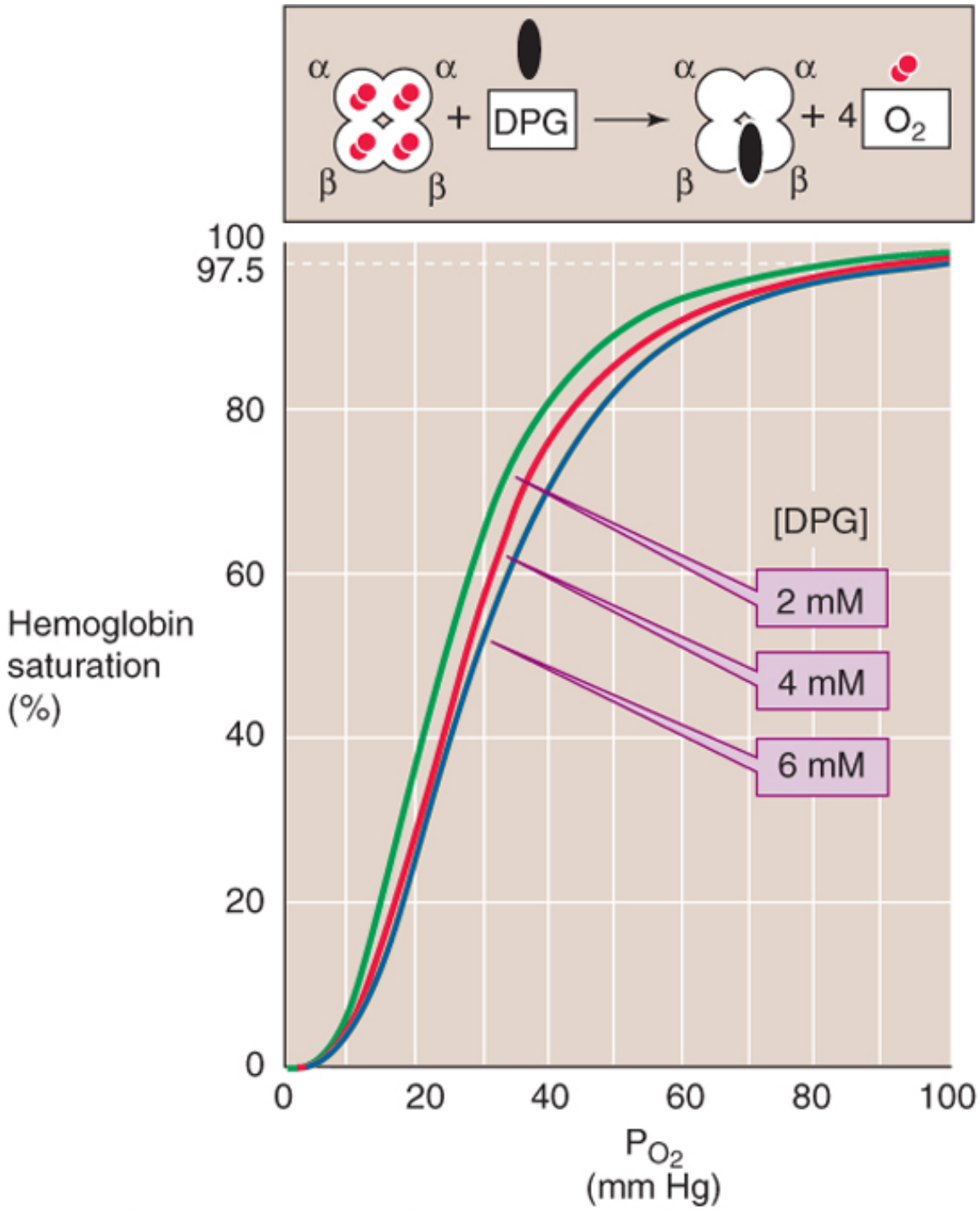


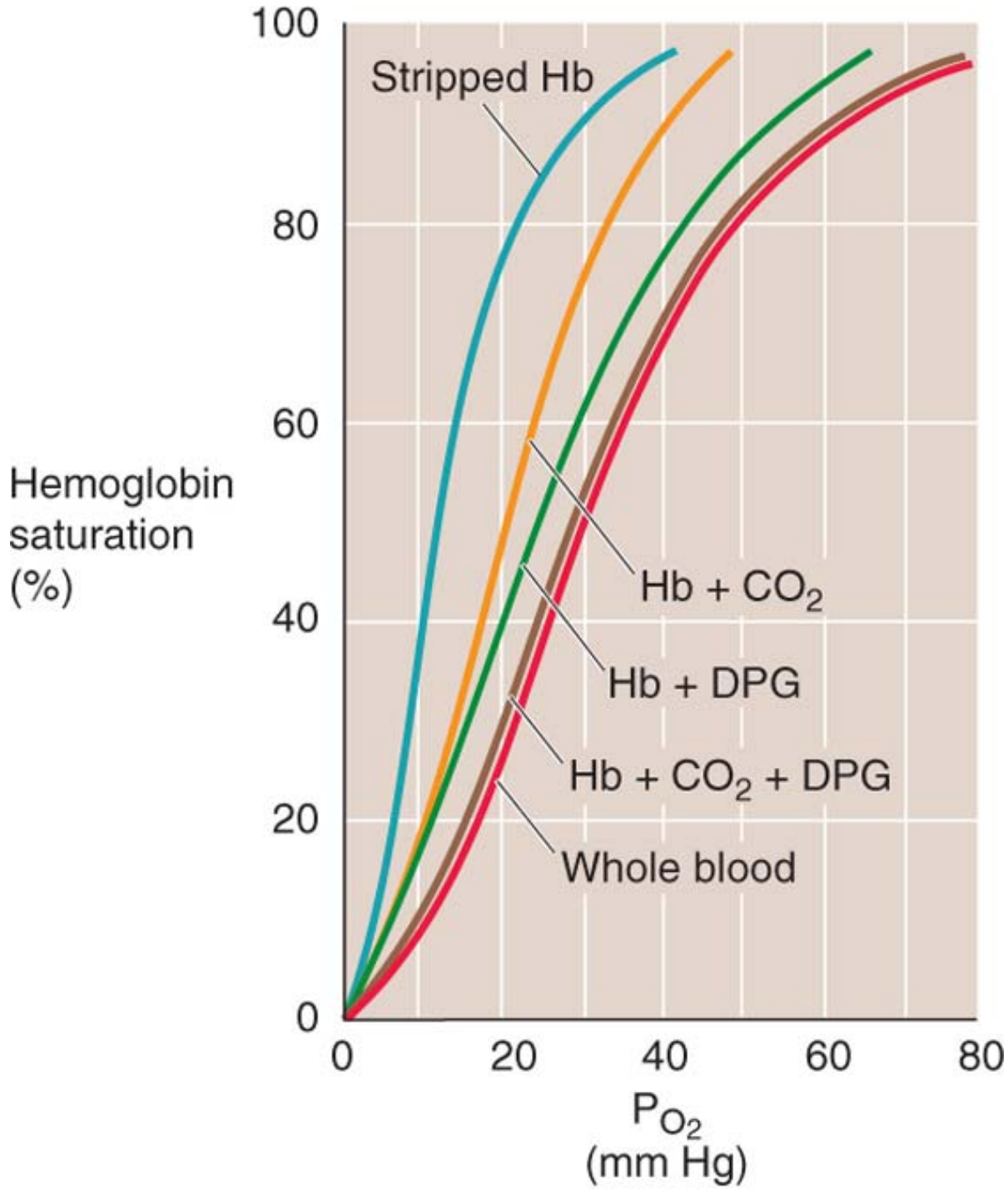


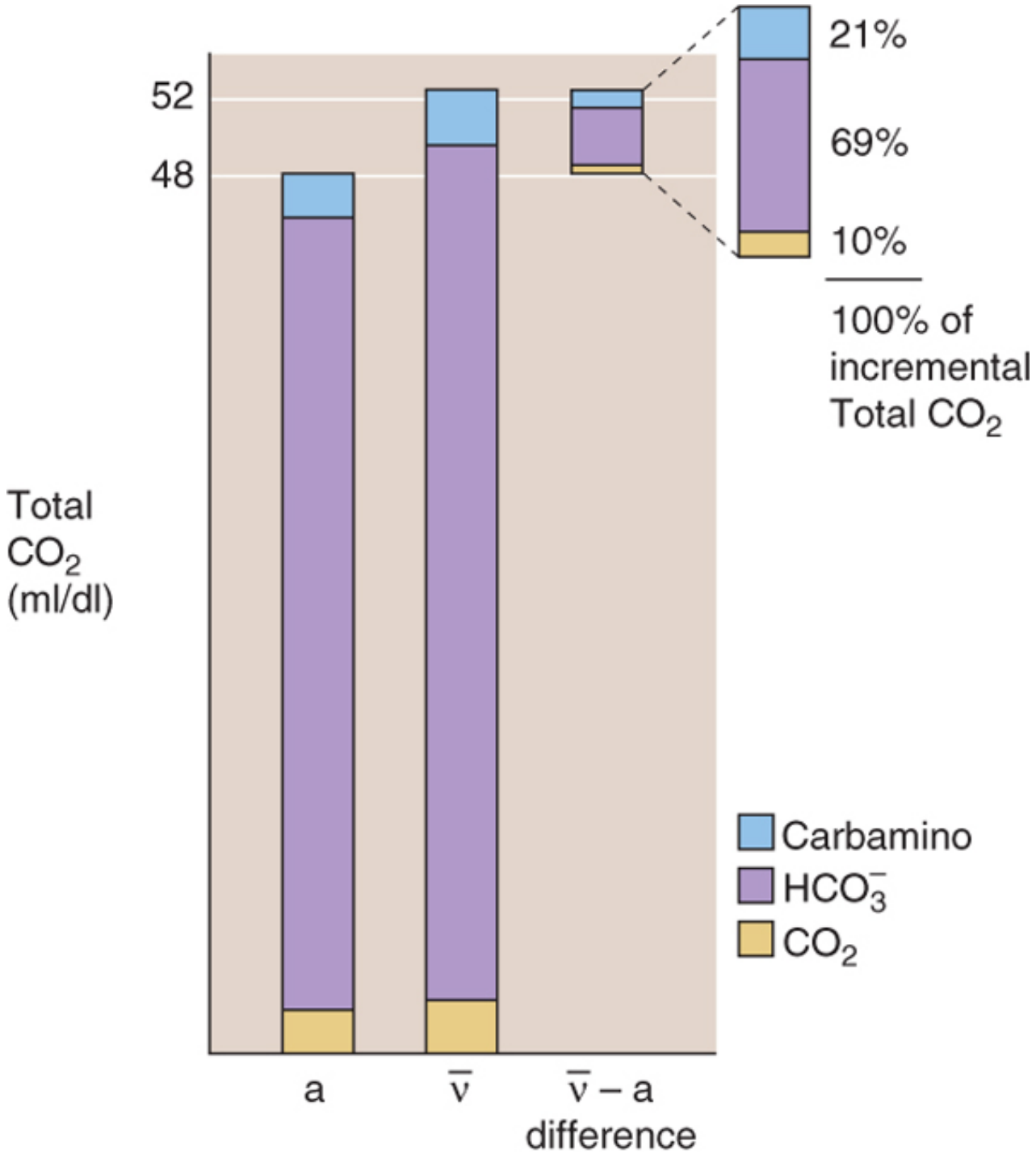
C EFFECT OF CO_2

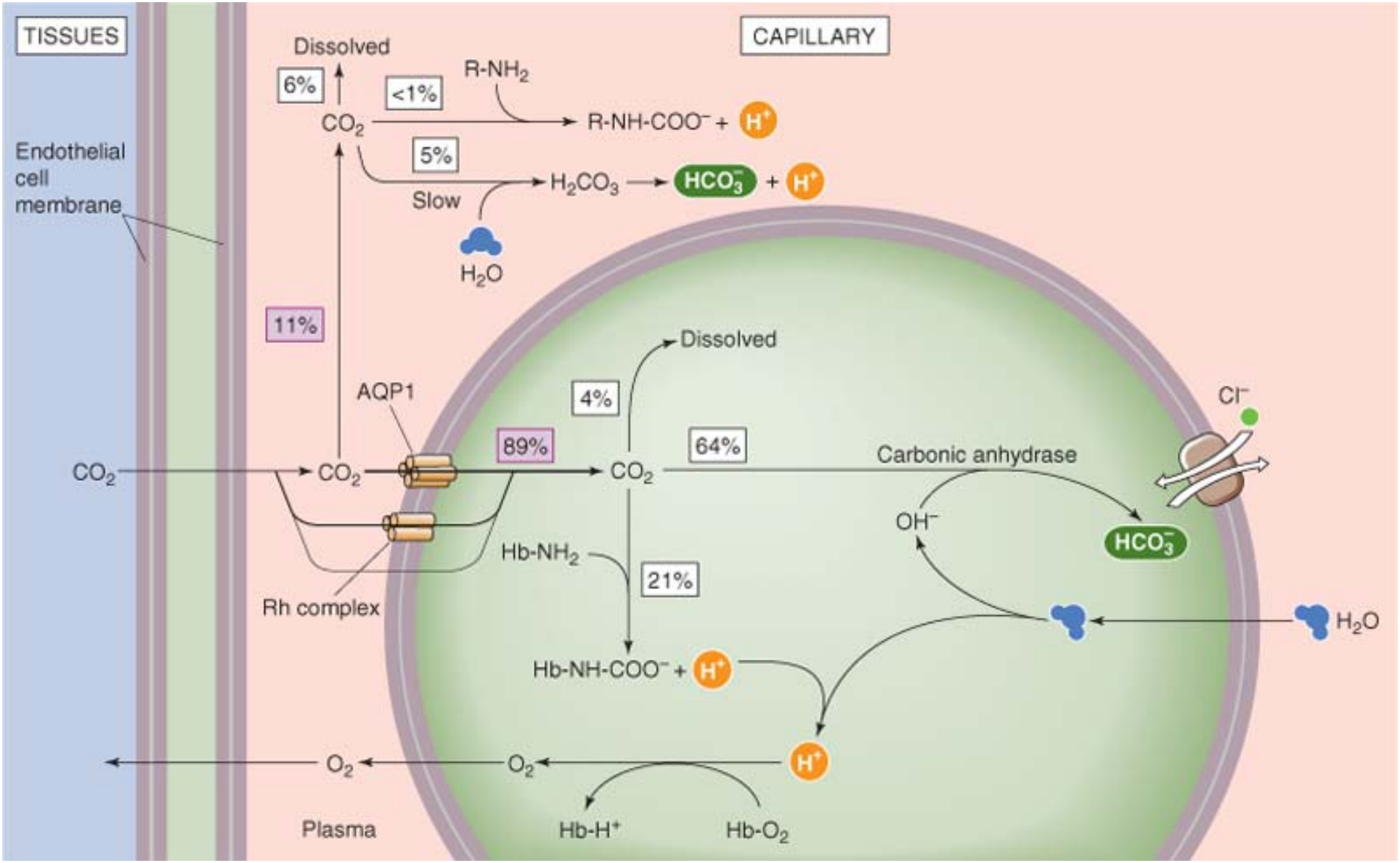


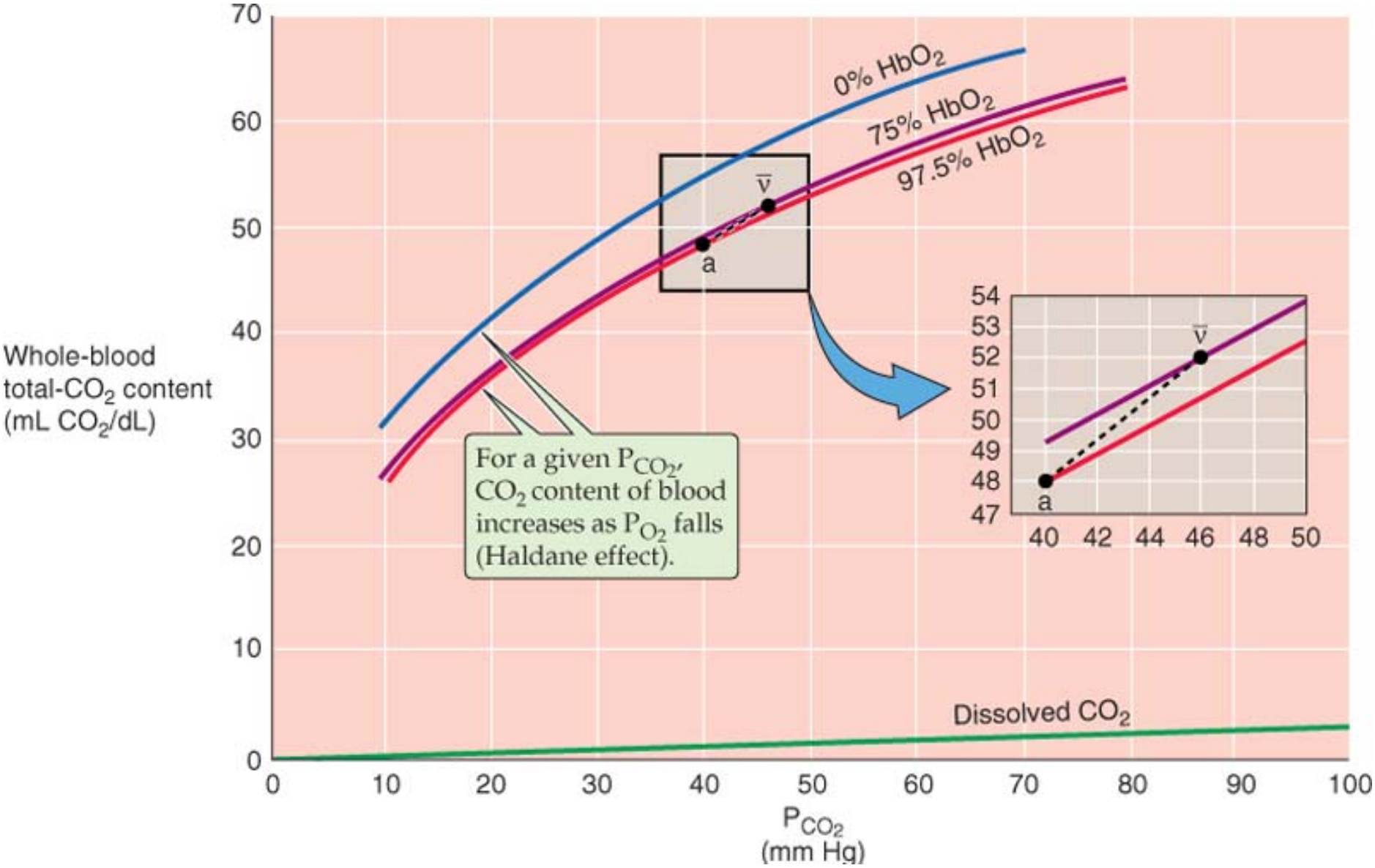


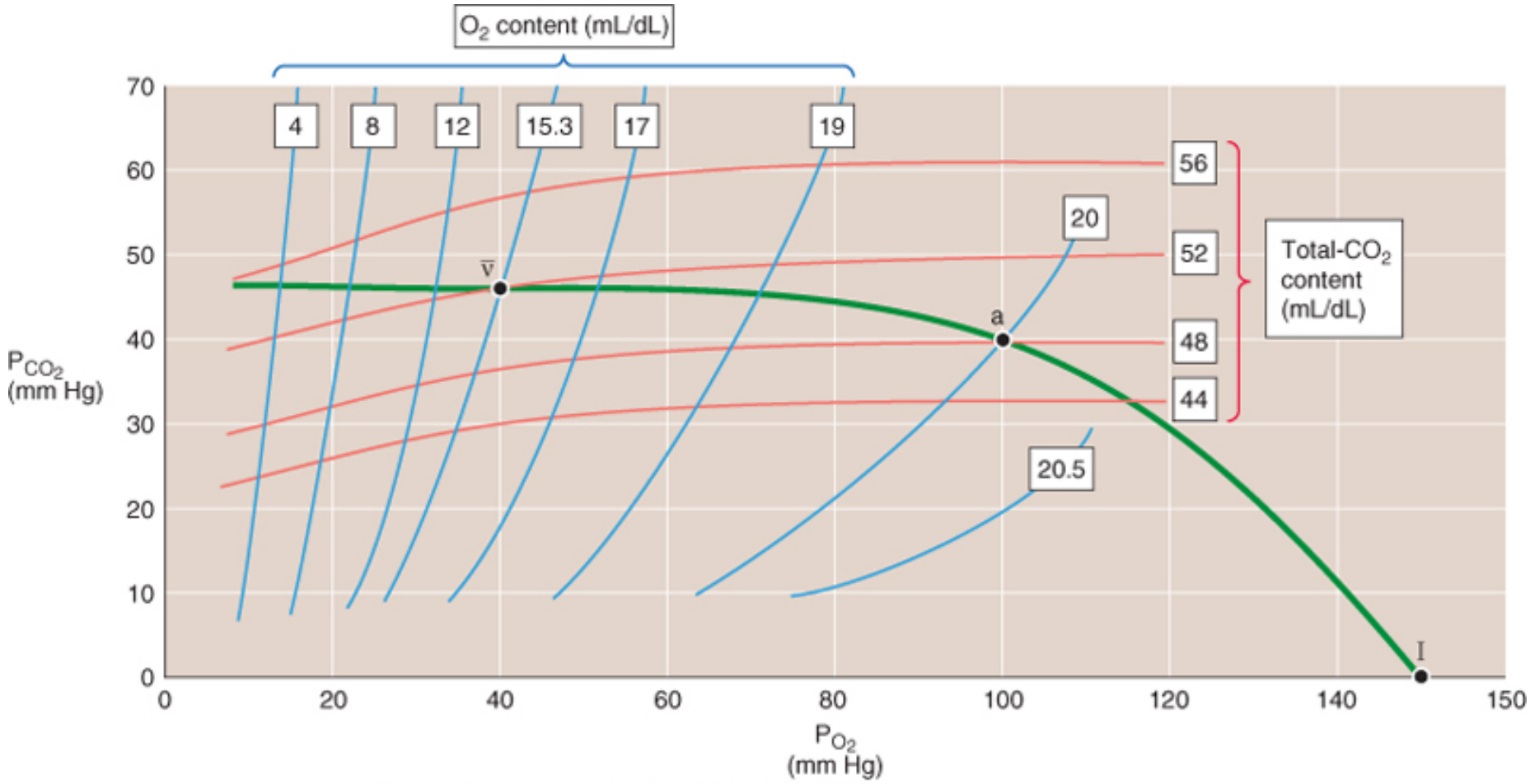

α
α
β
β
+
DPG
α
α
β
β
+ 4
O_2
100
97.5
80
60
40
20
0
Hemoglobin saturation (%)
[DPG]
2 mM
4 mM
6 mM
0
20
40
60
80
100
P_{O_2} (mm Hg)

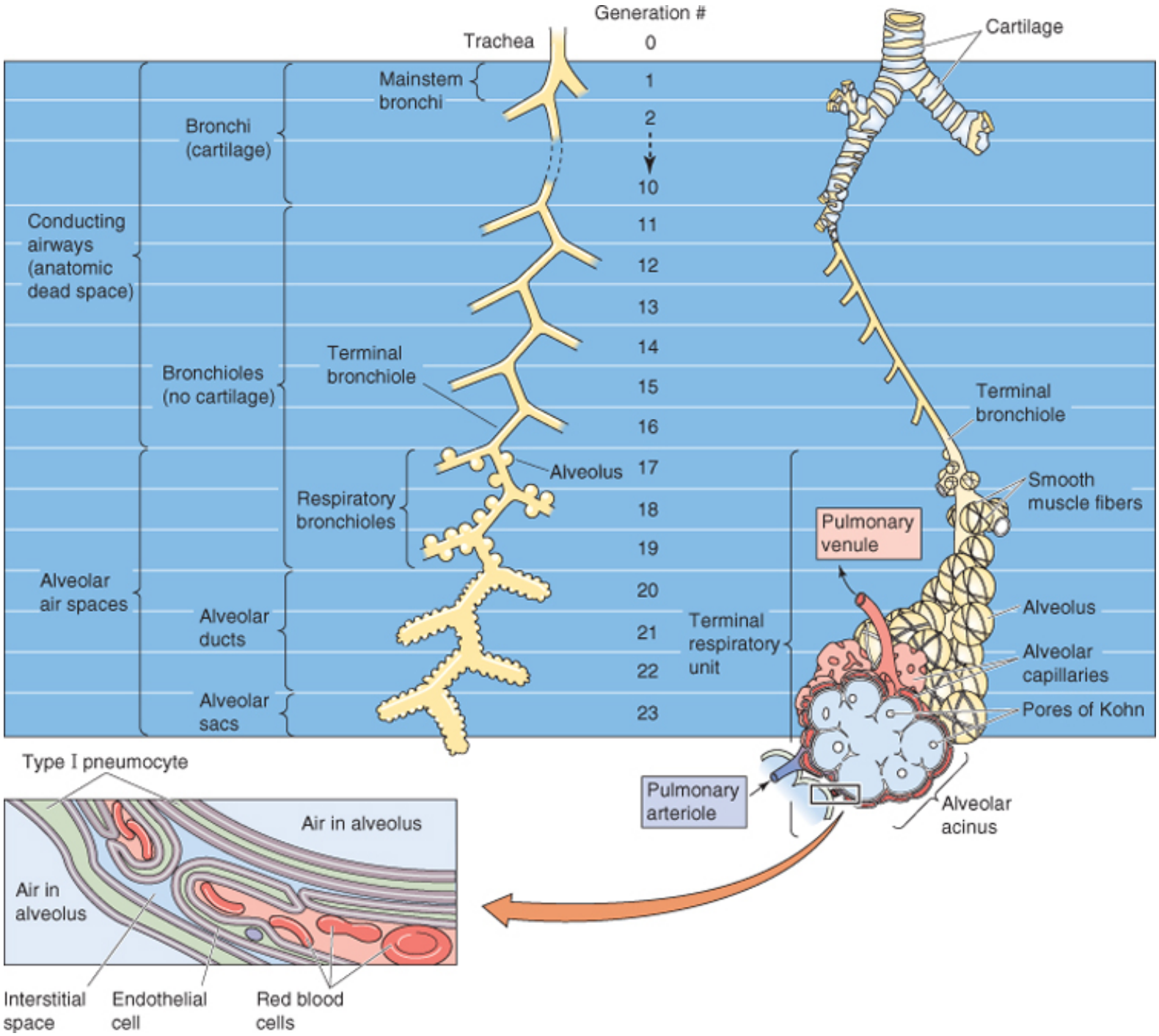

100
80
60
40
20
0
Stripped Hb
Hb + CO_2
Hb + DPG
Hb + CO_2 + DPG
Whole blood
Hemoglobin saturation (%)
0
20
40
60
80
P_{O_2} (mm Hg)

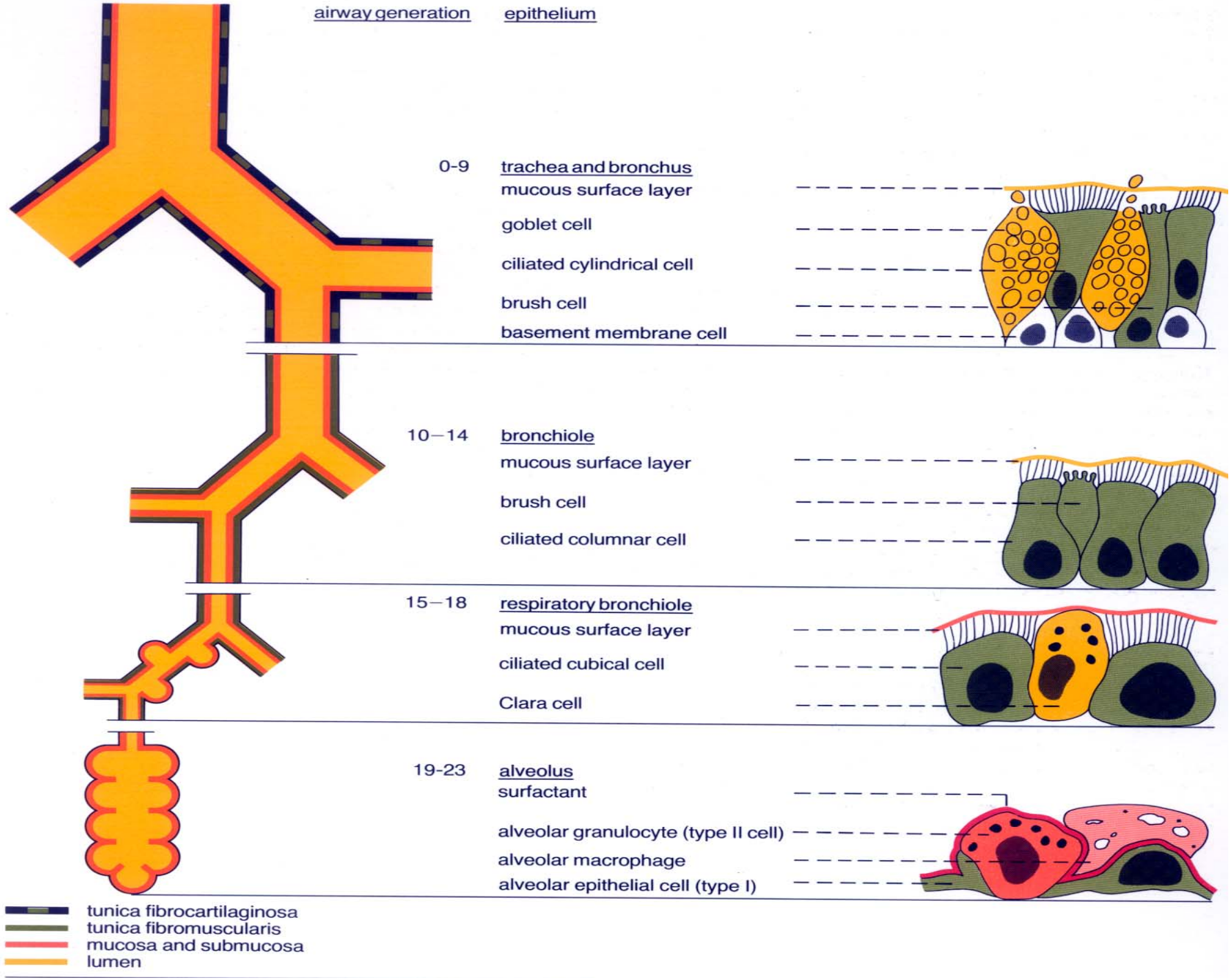

52
48
21%
69%
10%
100% of incremental Total CO_2
Total CO_2 (ml/dl)
Carbamino
HCO_3^-
CO_2
a
$\bar{v}$
$\bar{v}$ – a difference

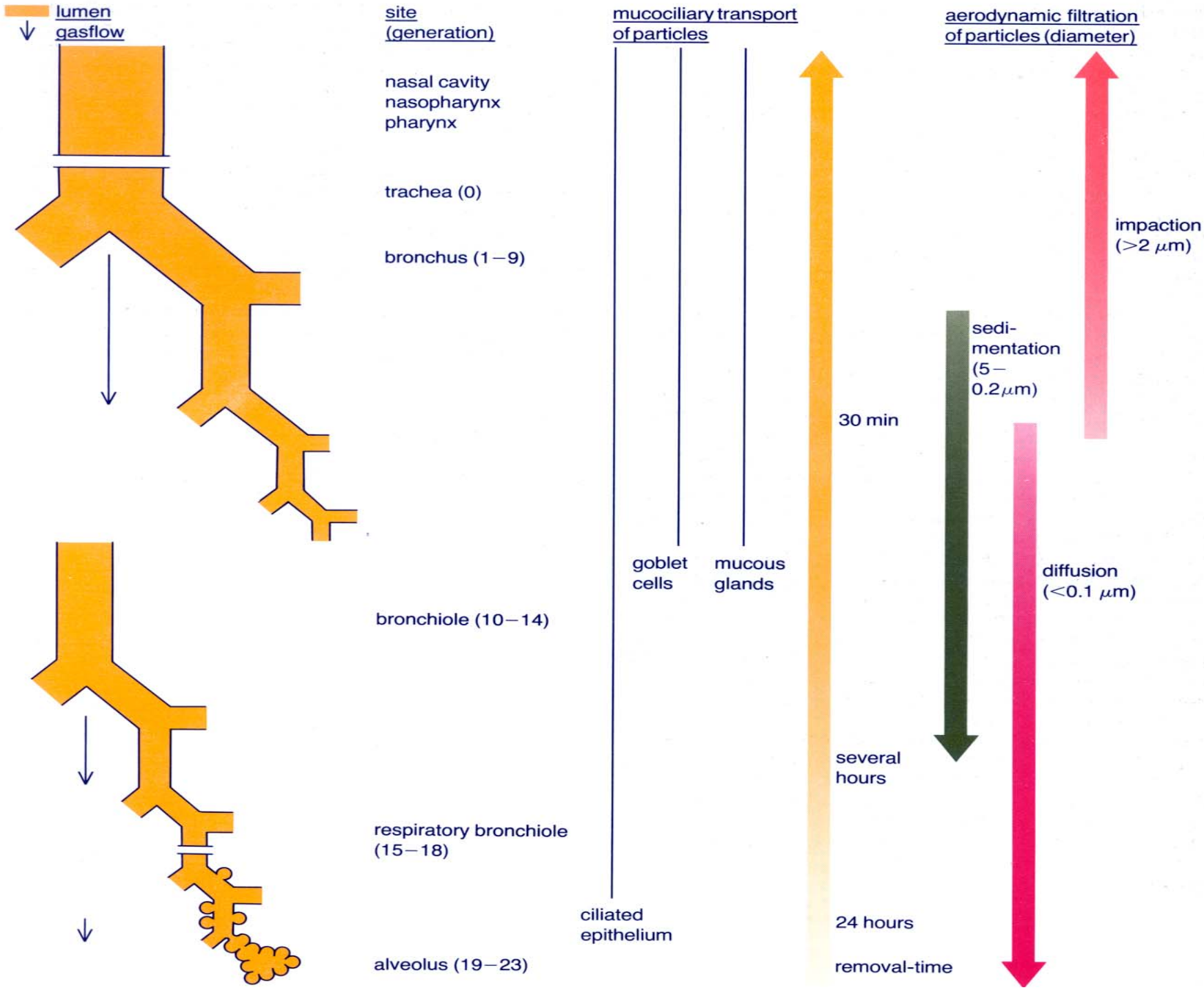

TISSUES
CAPILLARY
Endothelial cell membrane
Dissolved
6%
<1%
R-NH2
CO2
R-NH-COO⁻ + H⁺
5%
H2CO3
HCO3⁻ + H⁺
Slow
H2O
11%
Dissolved
4%
AQP1
89%
64%
Carbonic anhydrase
Cl⁻
CO2
CO2
CO2
OH⁻
HCO3⁻
Hb-NH2
21%
Rh complex
H2O
Hb-NH-COO⁻ + H⁺
H⁺
O2
O2
Plasma
Hb-H⁺
Hb-O2

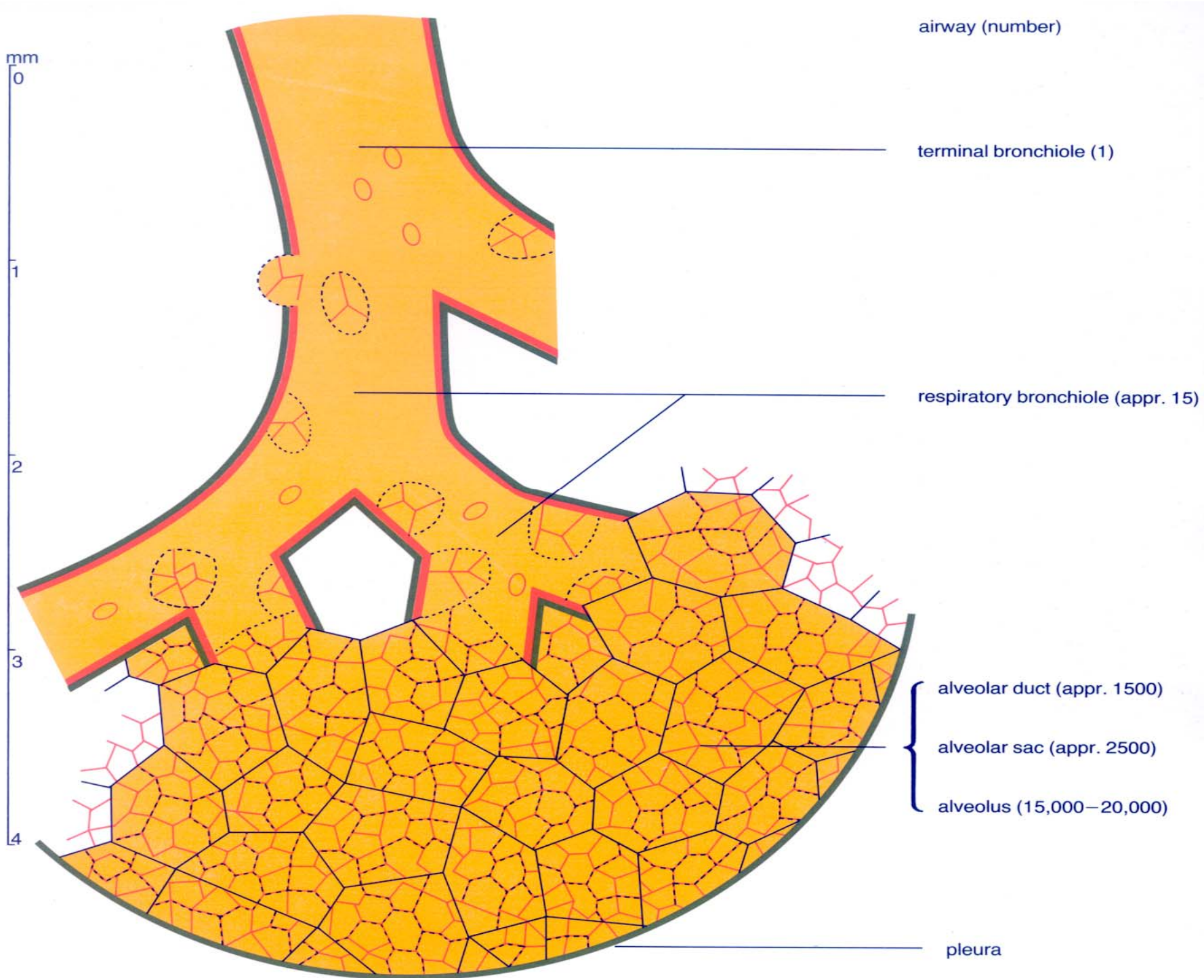

Whole-blood total-CO_2 content (mL CO_2/dL)
70
60
50
40
30
20
10
0
0% HbO_2
75% HbO_2
97.5% HbO_2
$\bar{v}$
a
For a given P_{CO_2}, CO_2 content of blood increases as P_{O_2} falls (Haldane effect).
54
53
52
51
50
49
48
47
40 42 44 46 48 50
Dissolved CO_2
0 10 20 30 40 50 60 70 80 90 100
P_{CO_2} (mm Hg)

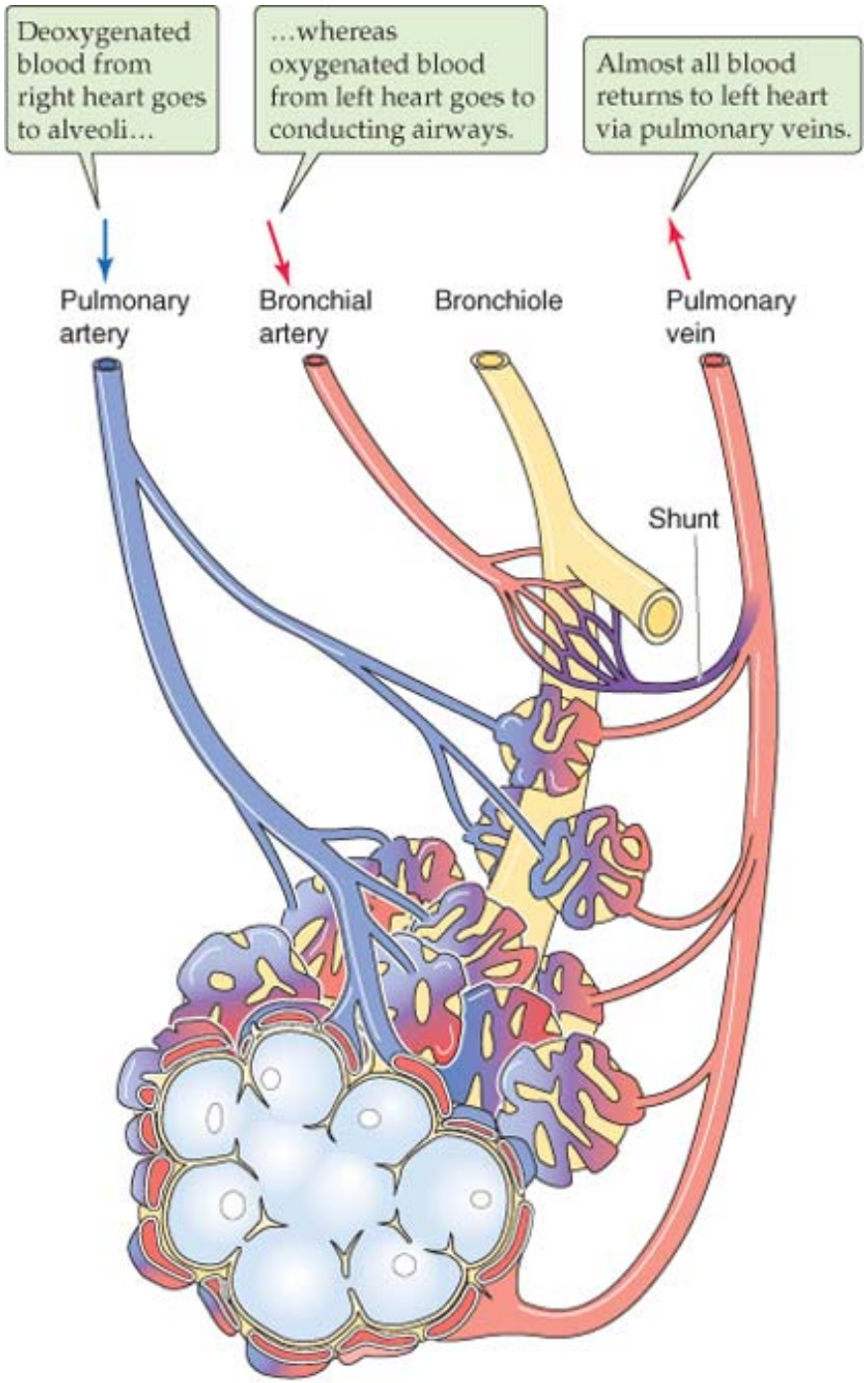

O_2 content (mL/dL)
4
8
12
15.3
17
19
20
20.5
56
52
48
44
Total-CO_2 content (mL/dL)
$\bar{v}$
a
I
P_{CO_2} (mm Hg)
P_{O_2} (mm Hg)
0
10
20
30
40
50
60
70
0
20
40
60
80
100
120
140
150

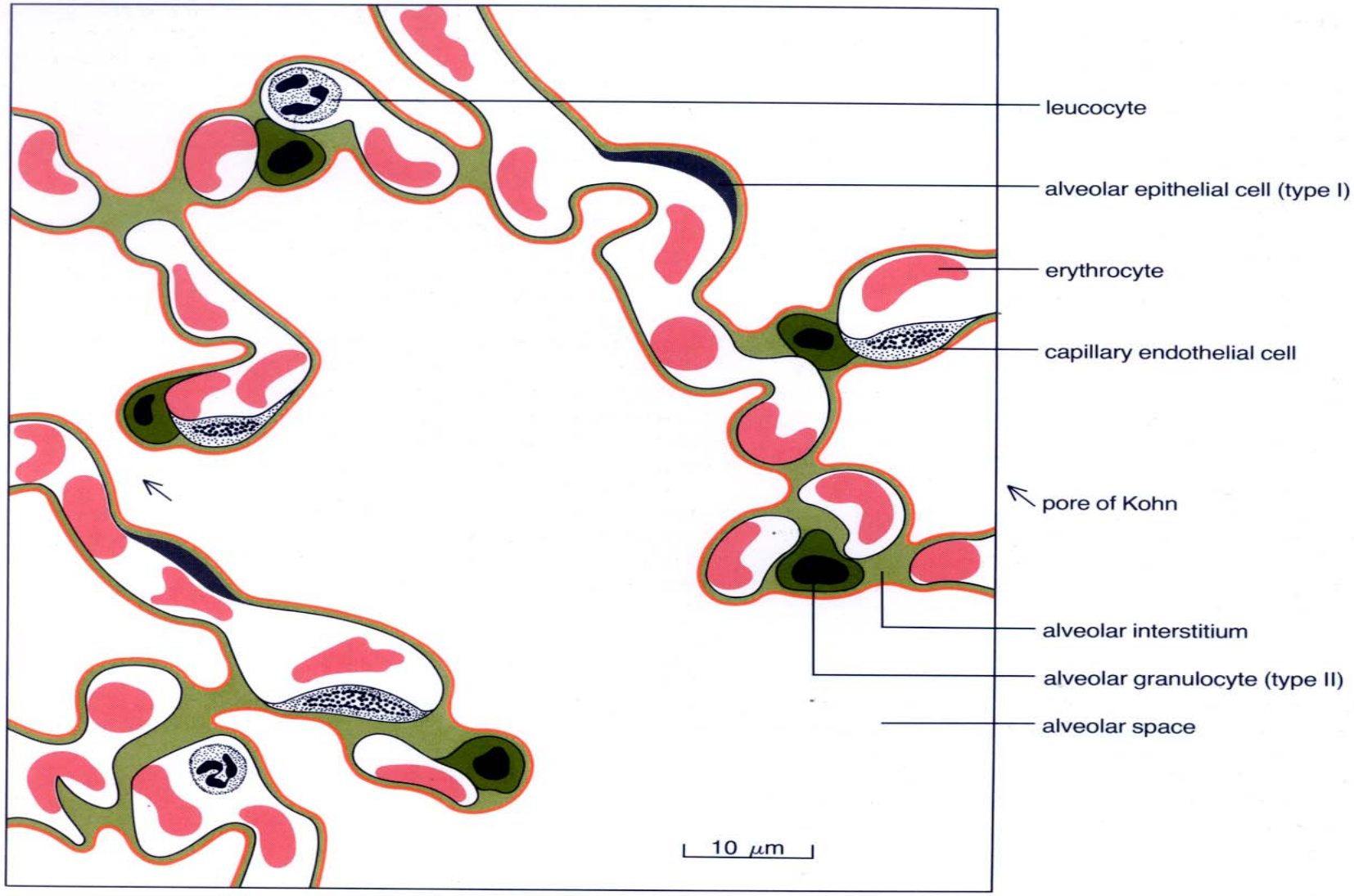

Generation #
Trachea
0
Mainstem bronchi
1
Bronchi (cartilage)
2
10
Conducting airways (anatomic dead space)
11
12
13
Terminal bronchiole
14
Bronchioles (no cartilage)
15
16
Alveolus
17
Respiratory bronchioles
18
19
Alveolar air spaces
20
Alveolar ducts
21
22
Alveolar sacs
23
Cartilage
Terminal bronchiole
Smooth muscle fibers
Pulmonary venule
Alveolus
Terminal respiratory unit
Alveolar capillaries
Pores of Kohn
Pulmonary arteriole
Alveolar acinus
Type I pneumocyte
Air in alveolus
Air in alveolus
Interstitial space
Endothelial cell
Red blood cells


airway generation
epithelium
0-9
trachea and bronchus
mucous surface layer
goblet cell
ciliated cylindrical cell
brush cell
basement membrane cell
10–14
bronchiole
mucous surface layer
brush cell
ciliated columnar cell
15–18
respiratory bronchiole
mucous surface layer
ciliated cubical cell
Clara cell
19-23
alveolus
surfactant
alveolar granulocyte (type II cell)
alveolar macrophage
alveolar epithelial cell (type I)
tunica fibrocartilaginosa
tunica fibromuscularis
mucosa and submucosa
lumen


lumen
gasflow
site
(generation)
nasal cavity
nasopharynx
pharynx
trachea (0)
bronchus (1–9)
bronchiole (10–14)
respiratory bronchiole
(15–18)
alveolus (19–23)
mucociliary transport
of particles
goblet
cells
mucous
glands
ciliated
epithelium
30 min
several
hours
24 hours
removal-time
aerodynamic filtration
of particles (diameter)
impaction
(>2 μm)
sedi-
mentation
(5–
0.2μm)
diffusion
(<0.1 μm)

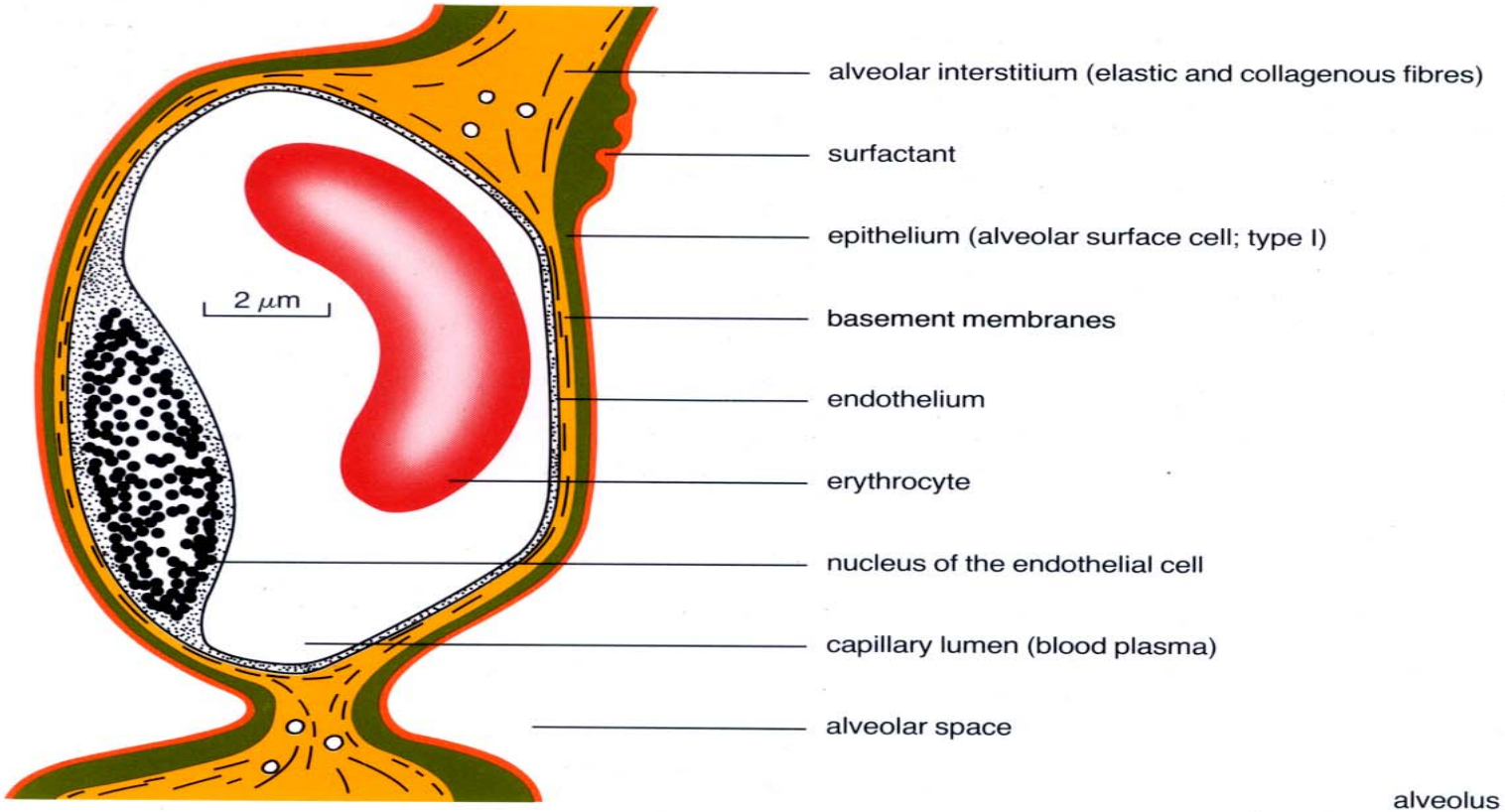

airway (number)
mm
0
1
2
3
4
terminal bronchiole (1)
respiratory bronchiole (appr. 15)
alveolar duct (appr. 1500)
alveolar sac (appr. 2500)
alveolus (15,000–20,000)
pleura

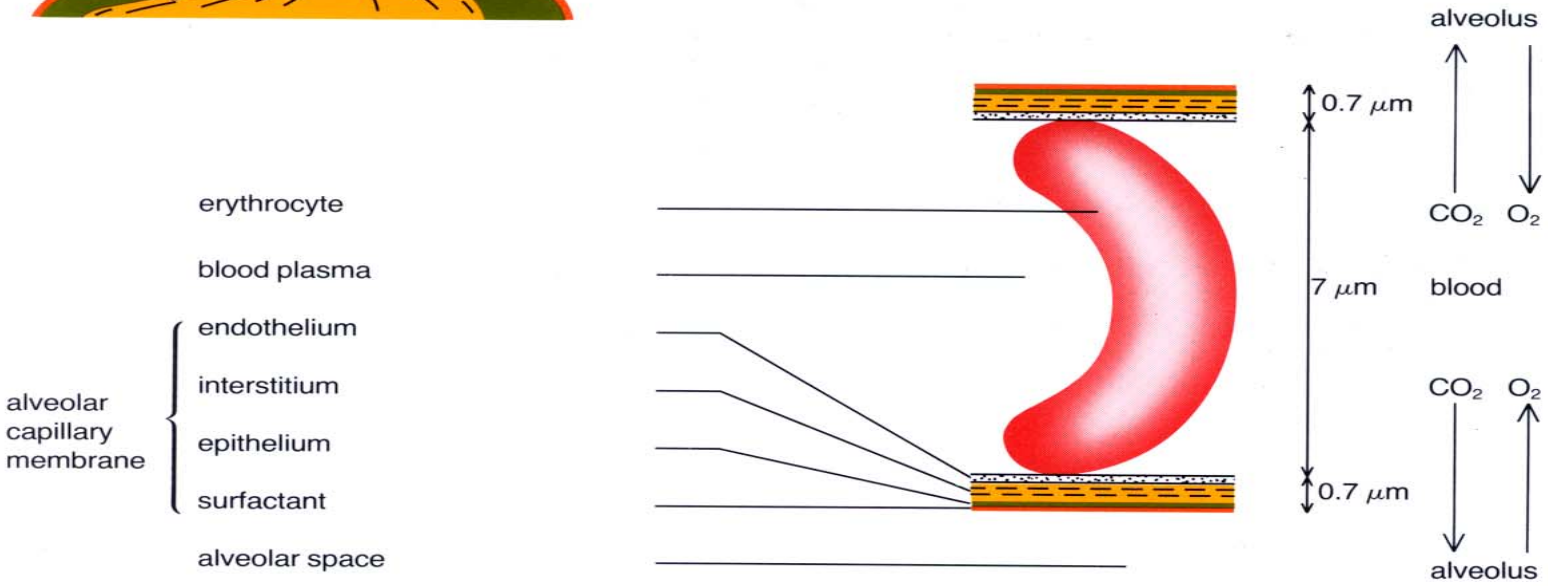

Deoxygenated blood from right heart goes to alveoli…
…whereas oxygenated blood from left heart goes to conducting airways.
Almost all blood returns to left heart via pulmonary veins.
Pulmonary artery
Bronchial artery
Bronchiole
Pulmonary vein
Shunt

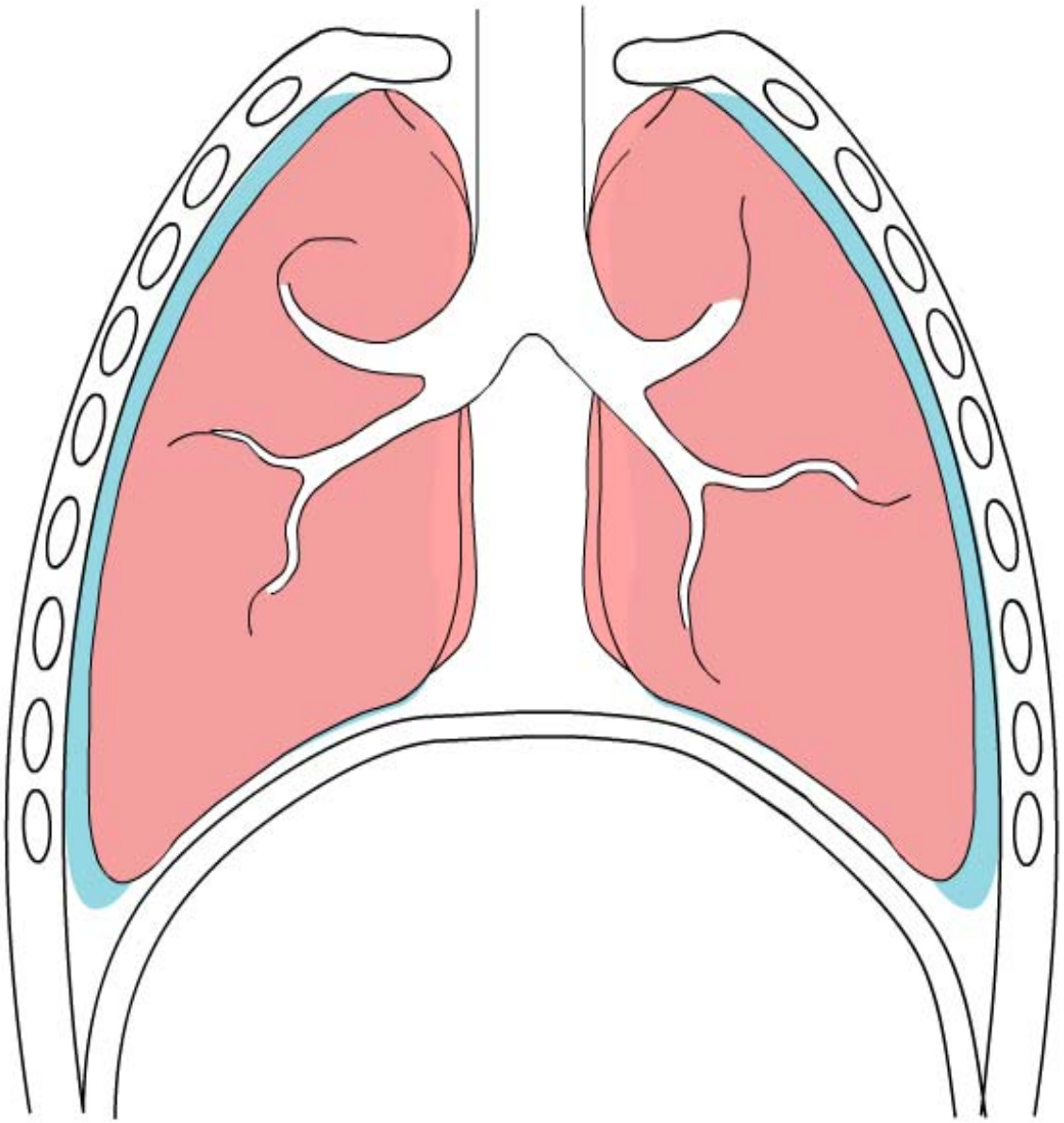


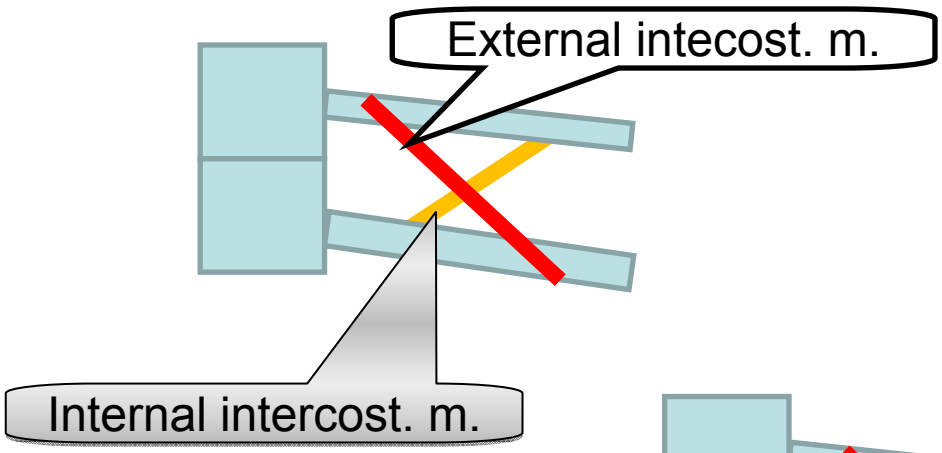
dimensiones of the alveolar-capillary membrane

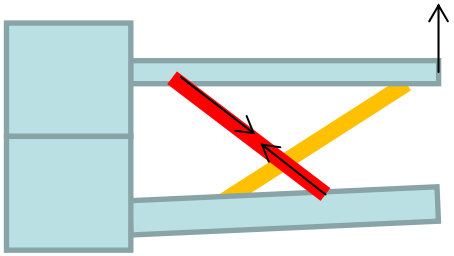
overall thickness:	0.30–1.00 µm
alveolar epithelium:	0.15–0.35 µm
epithelial basement membrane:	0.05–0.20 µm
endothelial basement membrane:	0.05–0.40 µm
capillary endothelium:	0.05–0.25 µm


alveolar interstitium (elastic and collagenous fibres)
surfactant
epithelium (alveolar surface cell; type I)
2 μm
basement membranes
endothelium
erythrocyte
nucleus of the endothelial cell
capillary lumen (blood plasma)
alveolar space

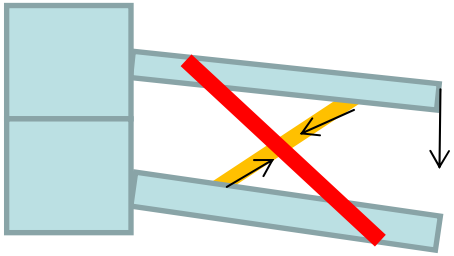

erythrocyte
blood plasma
alveolar capillary membrane
endothelium
interstitium
epithelium
surfactant
alveolar space
0.7 μm
7 μm
0.7 μm
alveolus
CO2
O2
blood
CO2
O2
alveolus



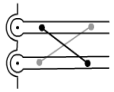


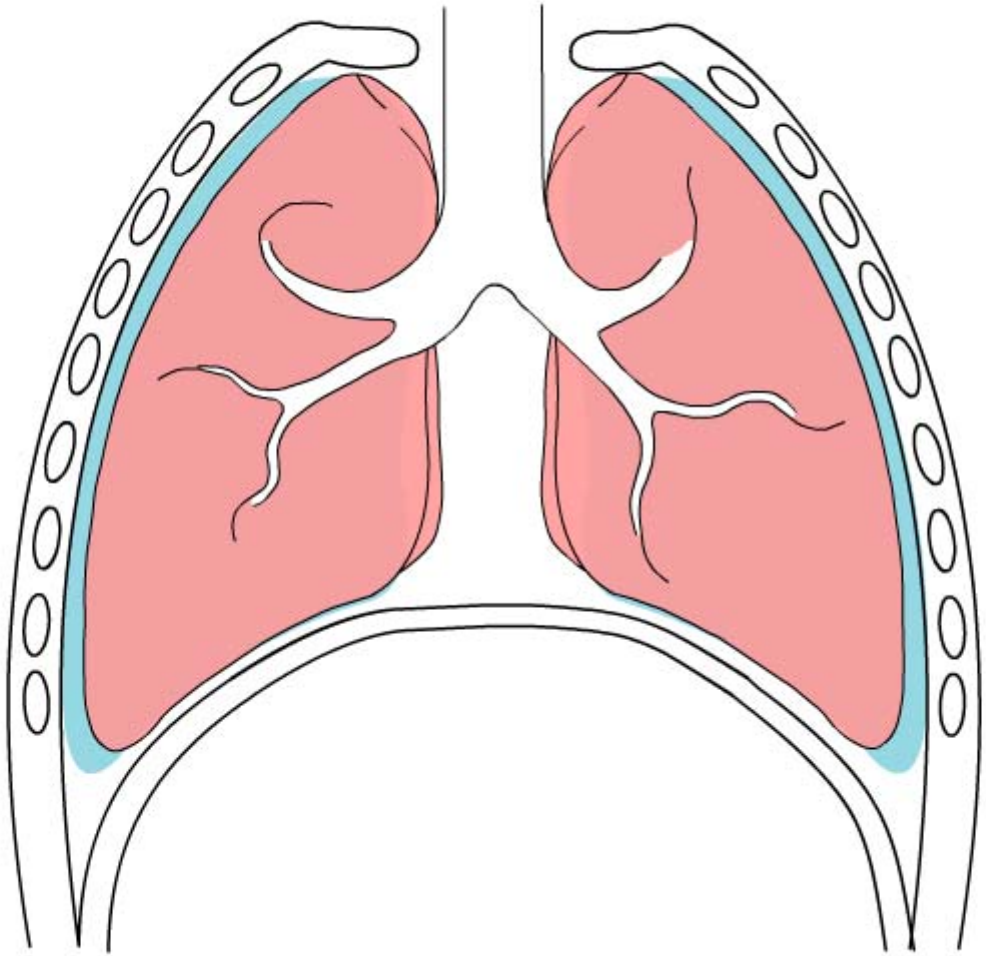


inspiration



expiration

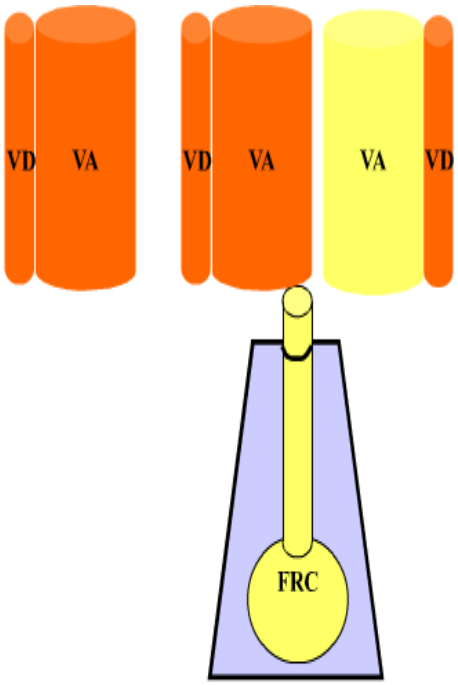




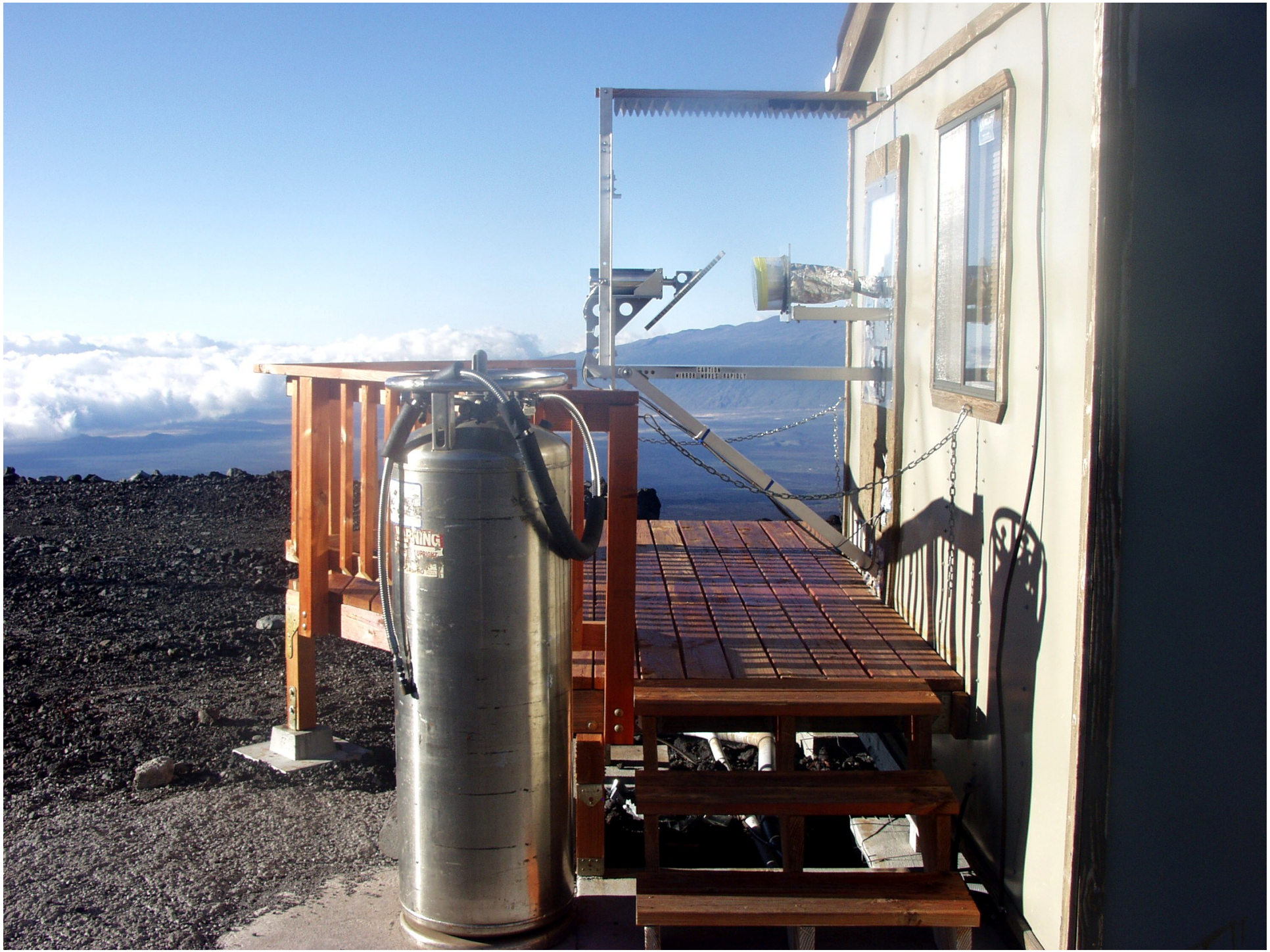
Pneumothorax

Alveolar ventilation

VE=VD+VA



FRC = Function residual capacity



COMMERCE

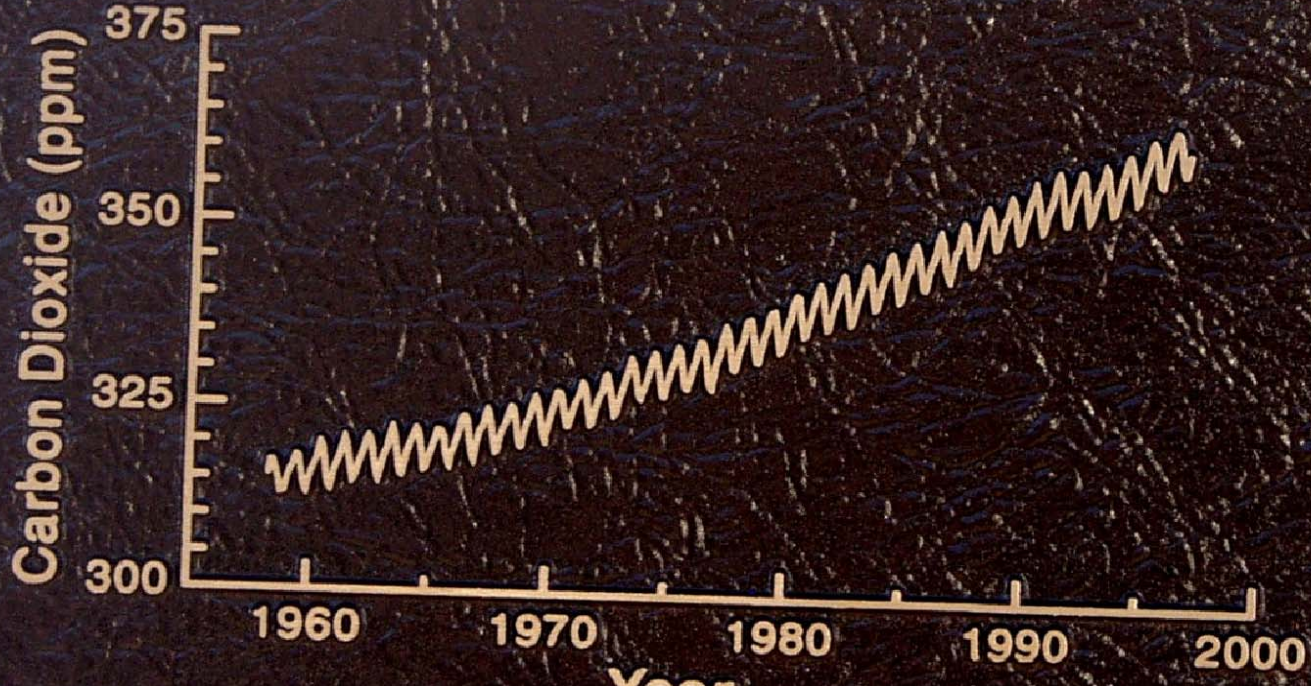
Keeling Building

NOAA

Named in honor of

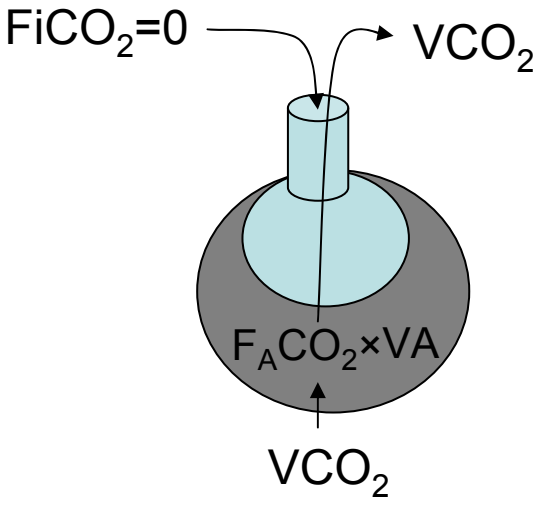
Professor Charles David Keeling,
Scripps Institution of Oceanography,

who initiated continuous CO_2 measurements at this site in 1958



November 1997

Alveolar ventilation



$$VCO2 = F_ACO2 * VA$$

$$VA = VCO2/F_ACO2$$

$$P_ACO2 = F_ACO2 \times \text{Barometric pressure}$$

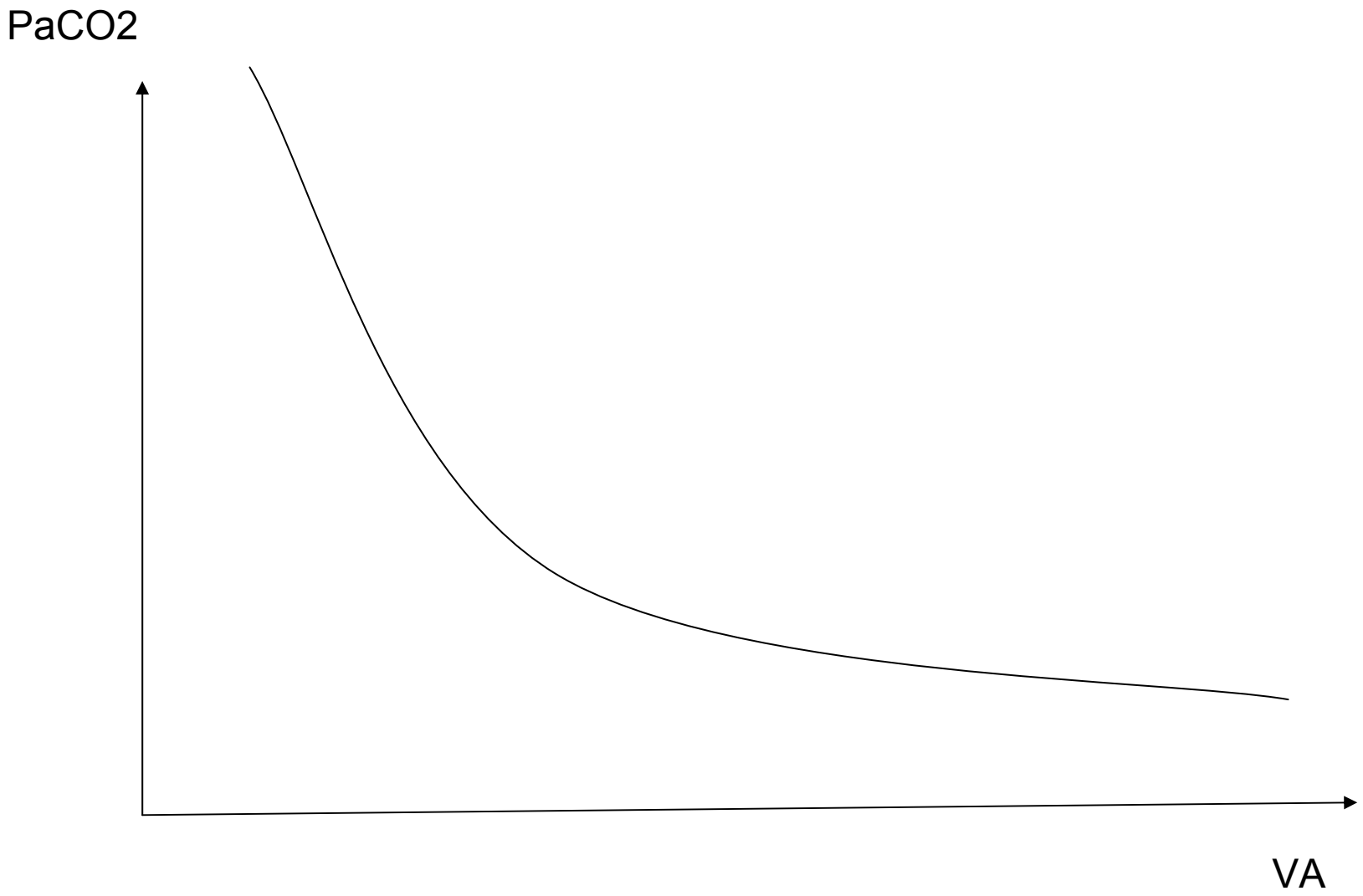
$$VA = k_1 \times VCO2/P_ACO2$$

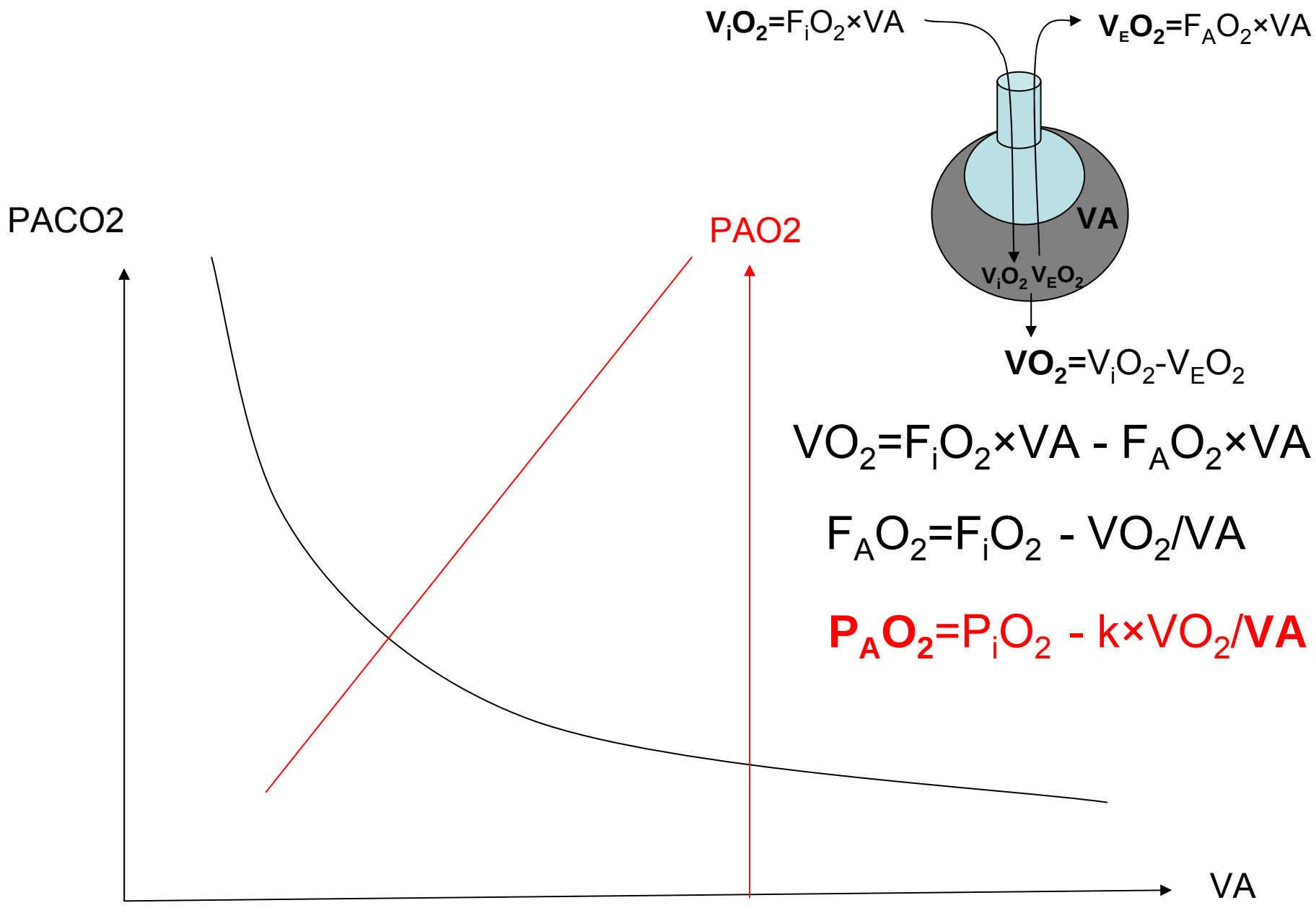
$$P_ACO2 = k_2 \times VCO2/VA$$

STPD BTPS

$$\frac{P \times V}{T} = R \qquad \frac{(P - P_{H_2O}) \times V_{BTPS}}{273 + t°_{patient}} = \frac{760 \times V_{BTPS}}{273}$$

$$P_ACO2_{[torr]} = 0{,}863 * VCO2_{[ml/min\ STPD]}/VA_{[l/min\ BTPS]}$$


PaCO2
VA

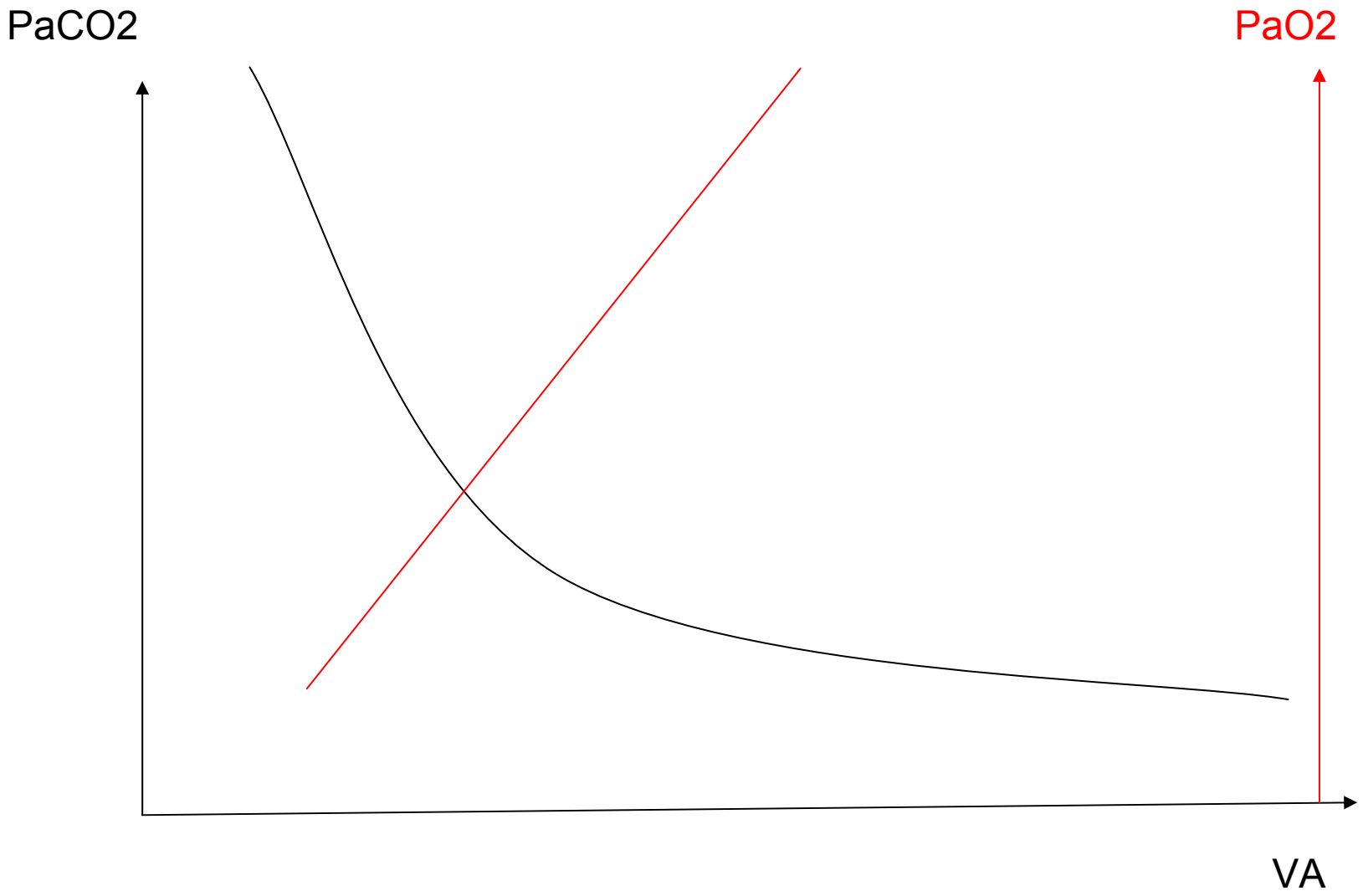


$$VO_2=F_iO_2\times VA - F_AO_2\times VA$$

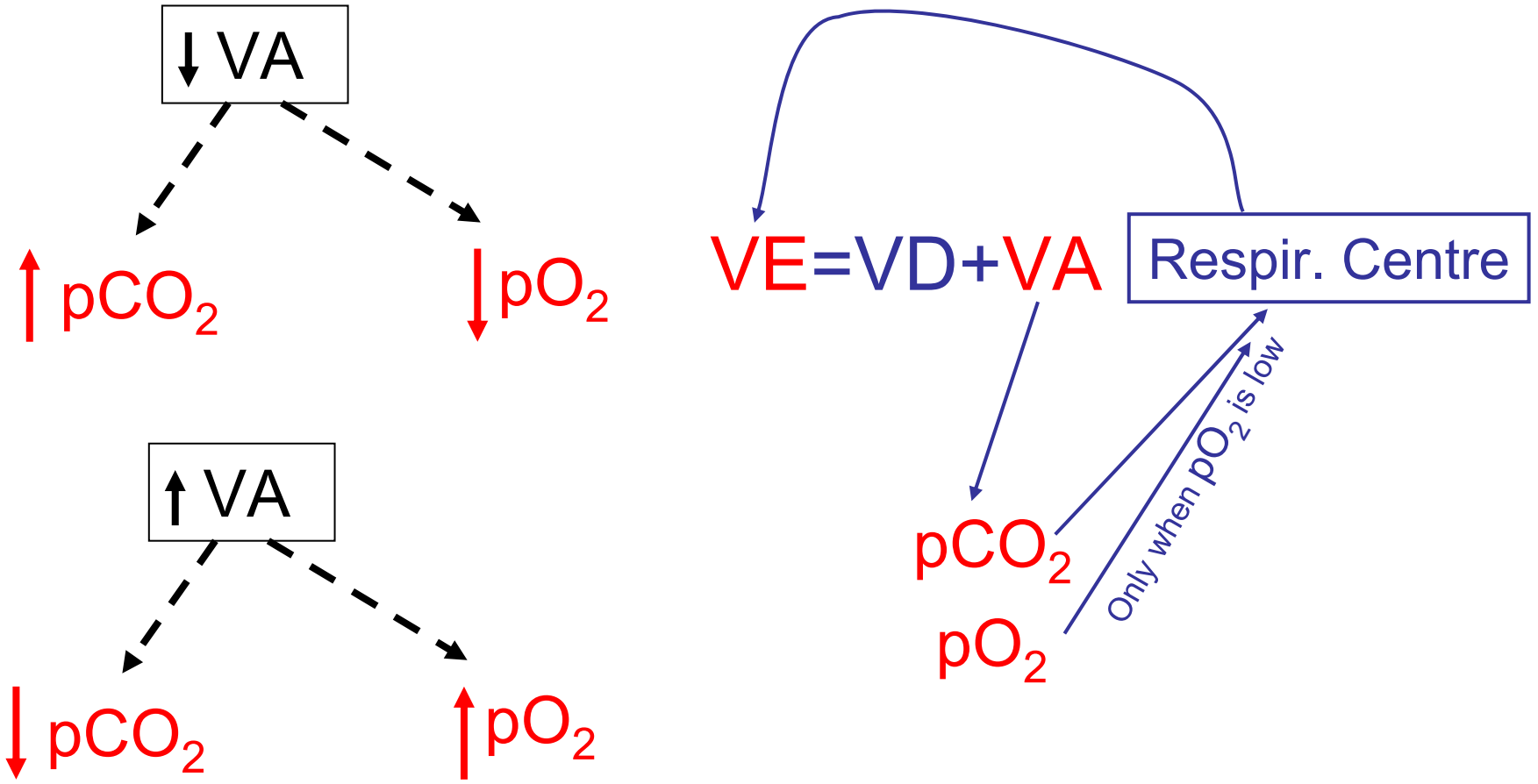
$$F_AO_2=F_iO_2 - VO_2/VA$$

$$\mathbf{P_AO_2}=P_iO_2 - k\times VO_2/\mathbf{VA}$$

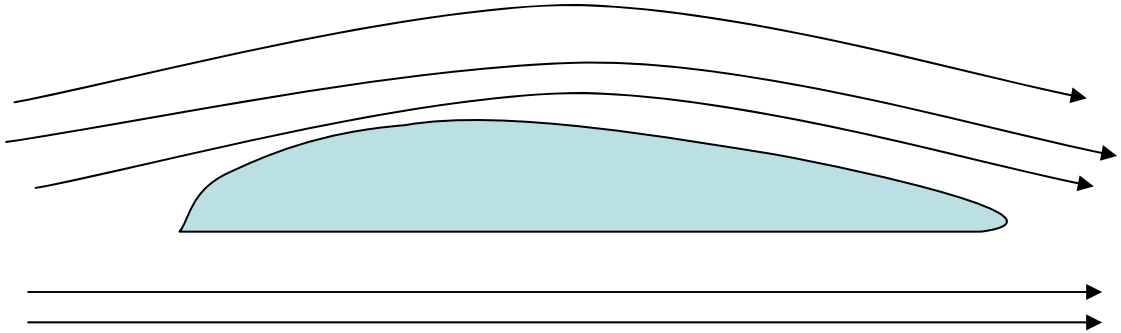
$$P_ACO2_{[torr]} = 0{,}863*VCO2_{[ml/min\ STPD]}/VA_{[l/min\ BTPS]}$$


PaCO2
PaO2
VA

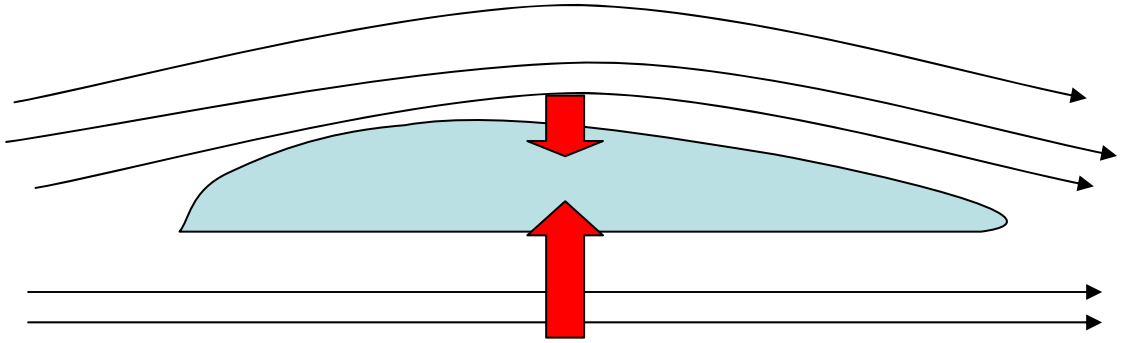
Alveolar Ventilation Controls Rate of Breathing by Influencing pCO2 and pO2



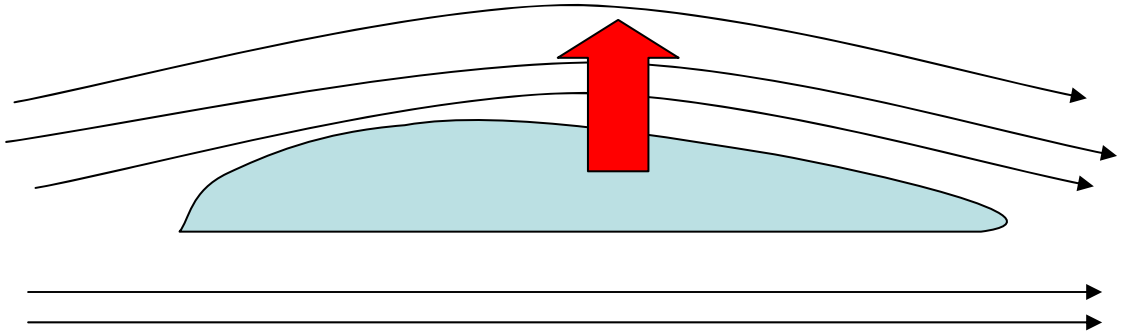
Why the airplane flies?



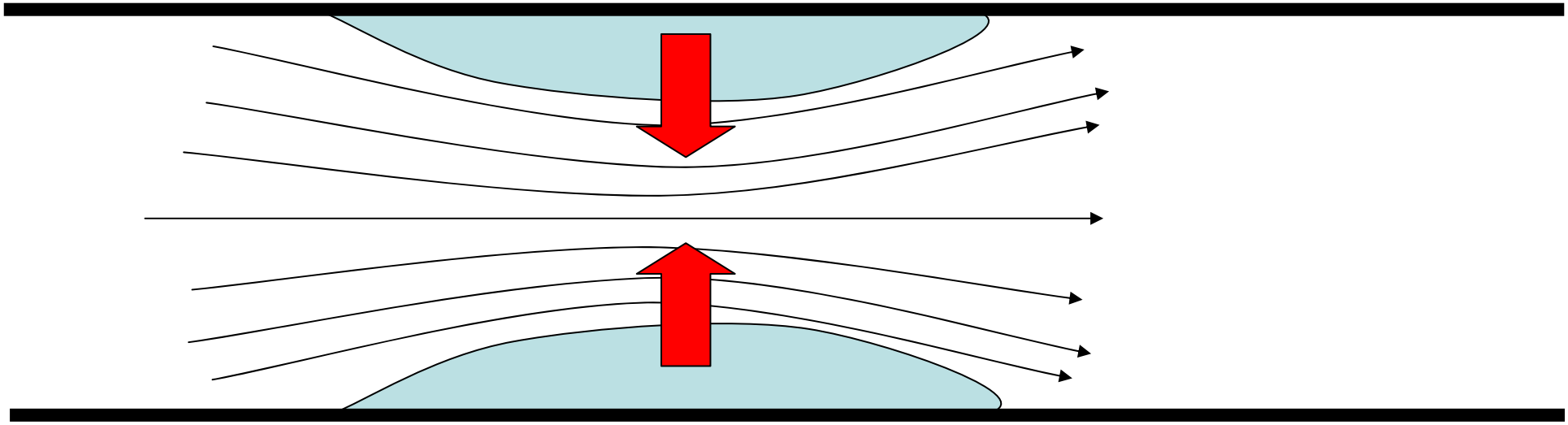
Why the airplane flies?



Why the airplane flies?

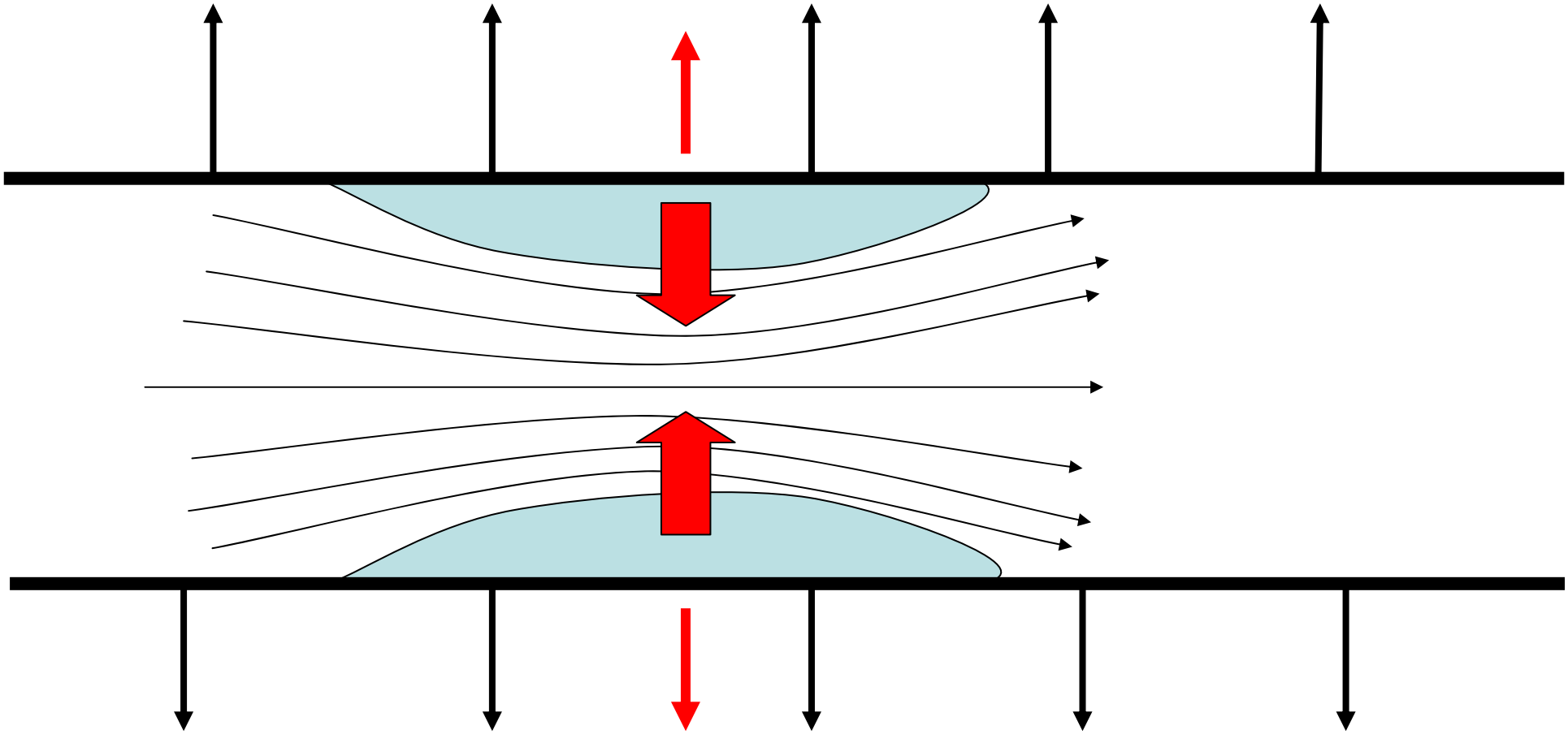


Narrowing of bronchiole (bronchoconstriction, mucus…)



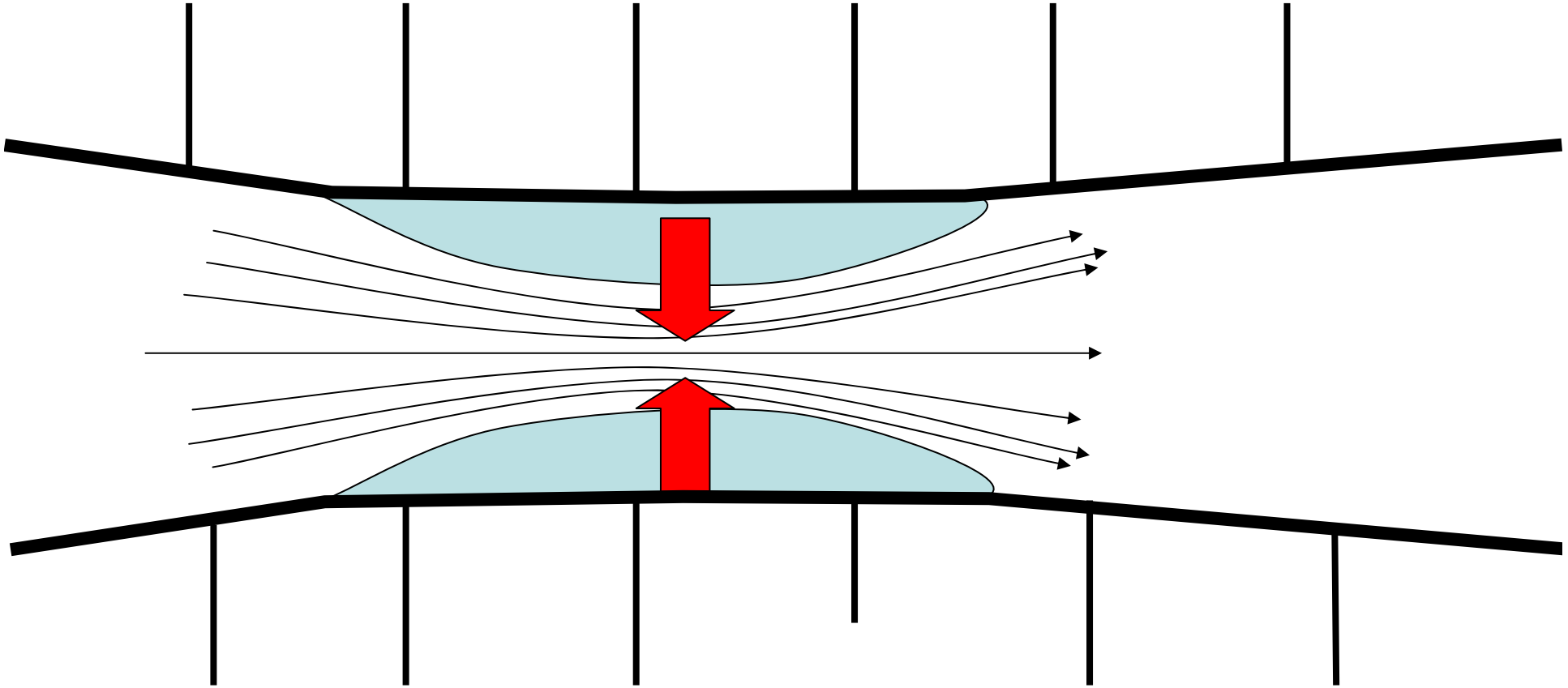
Narrowing of bronchiole (bronchoconstriction, mucus..)

Inspiration – the narrowing is opposed by neg. introthoratic pressure



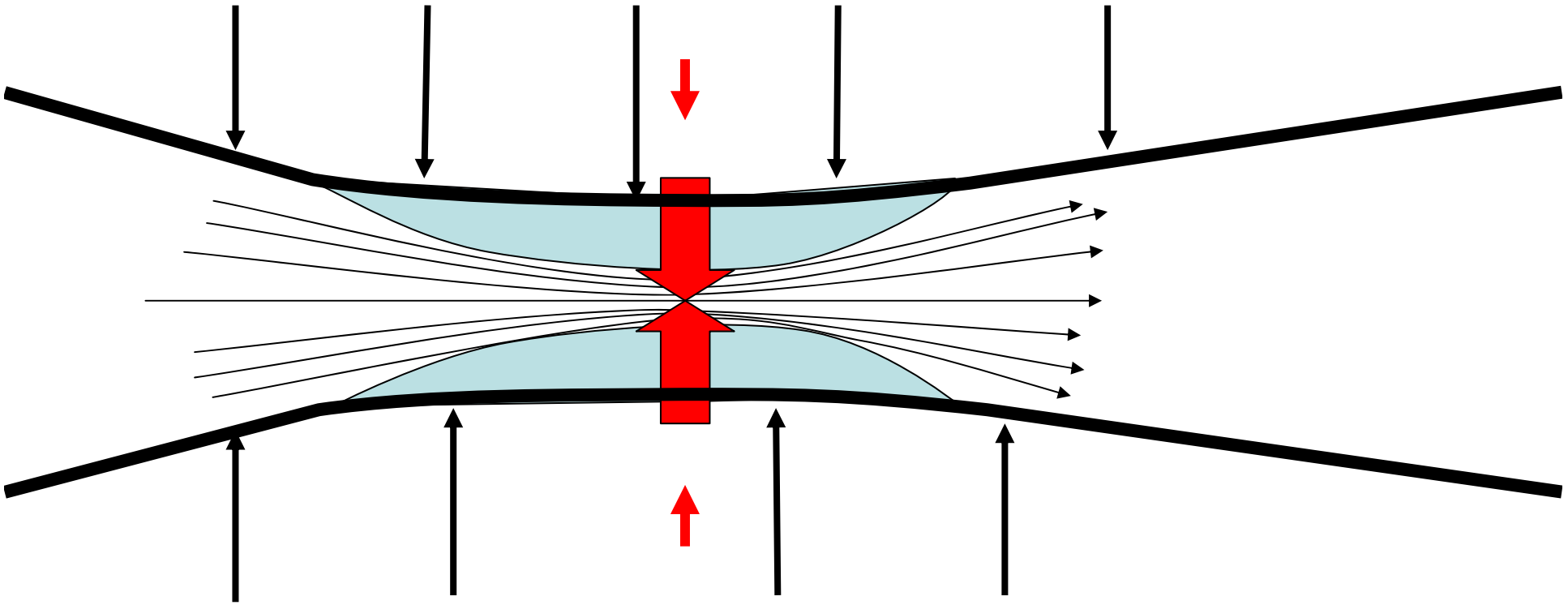
Narrowing of a bronchiole (bronchoconstriction, mucus..)

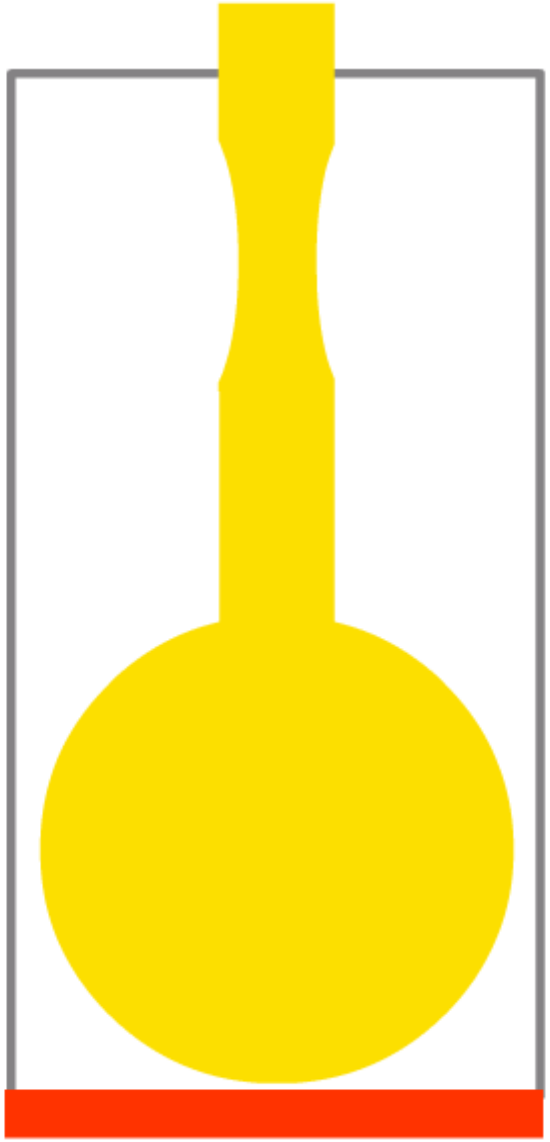
Expiration – nothing opposes the narrowing of a bronchiole



Narrowing of a bronchiole (bronchoconstriction, mucus..)

Forceful expiration - leads to worsening of the obstruction



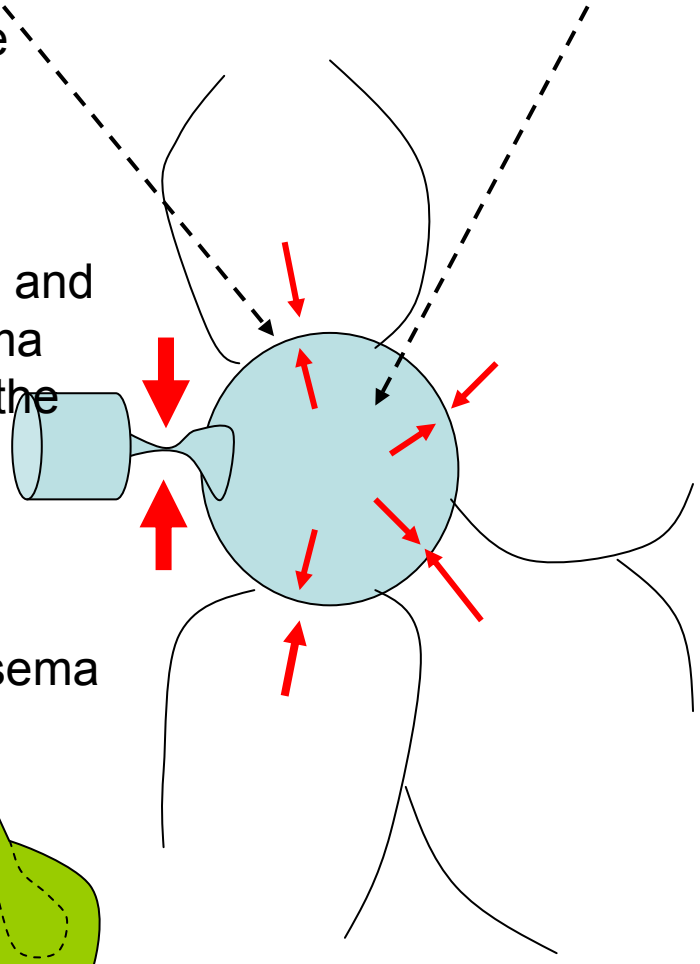


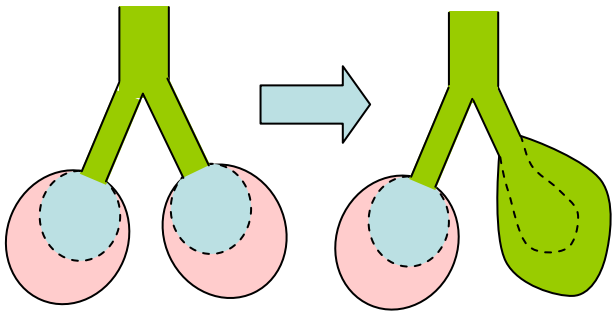
It is expiration that is difficult with intrathoratic obstruction

Air Captioning – premature closure of bronchioli

The air trapped in alveoli during expiration exerts pressure on the alveolar membrane

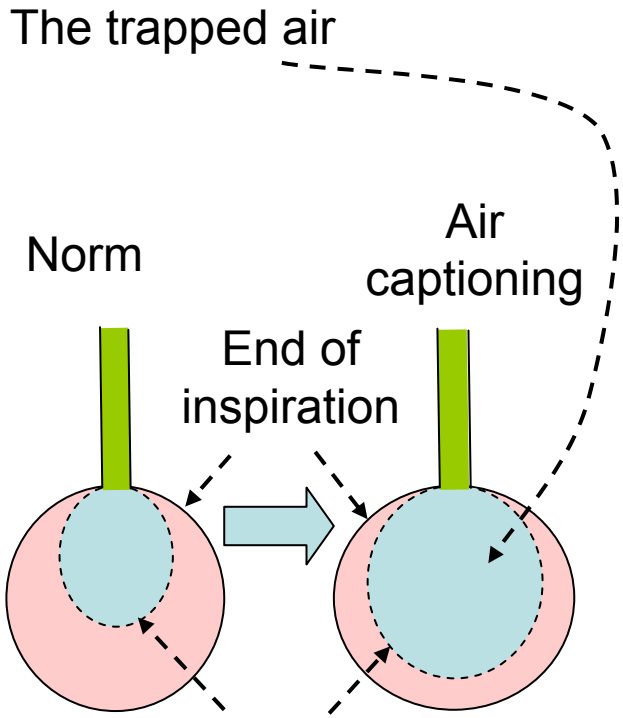
Tendency to alveolar membrane destruction and evolution of emphysema bullae, thus enlarging the dead space





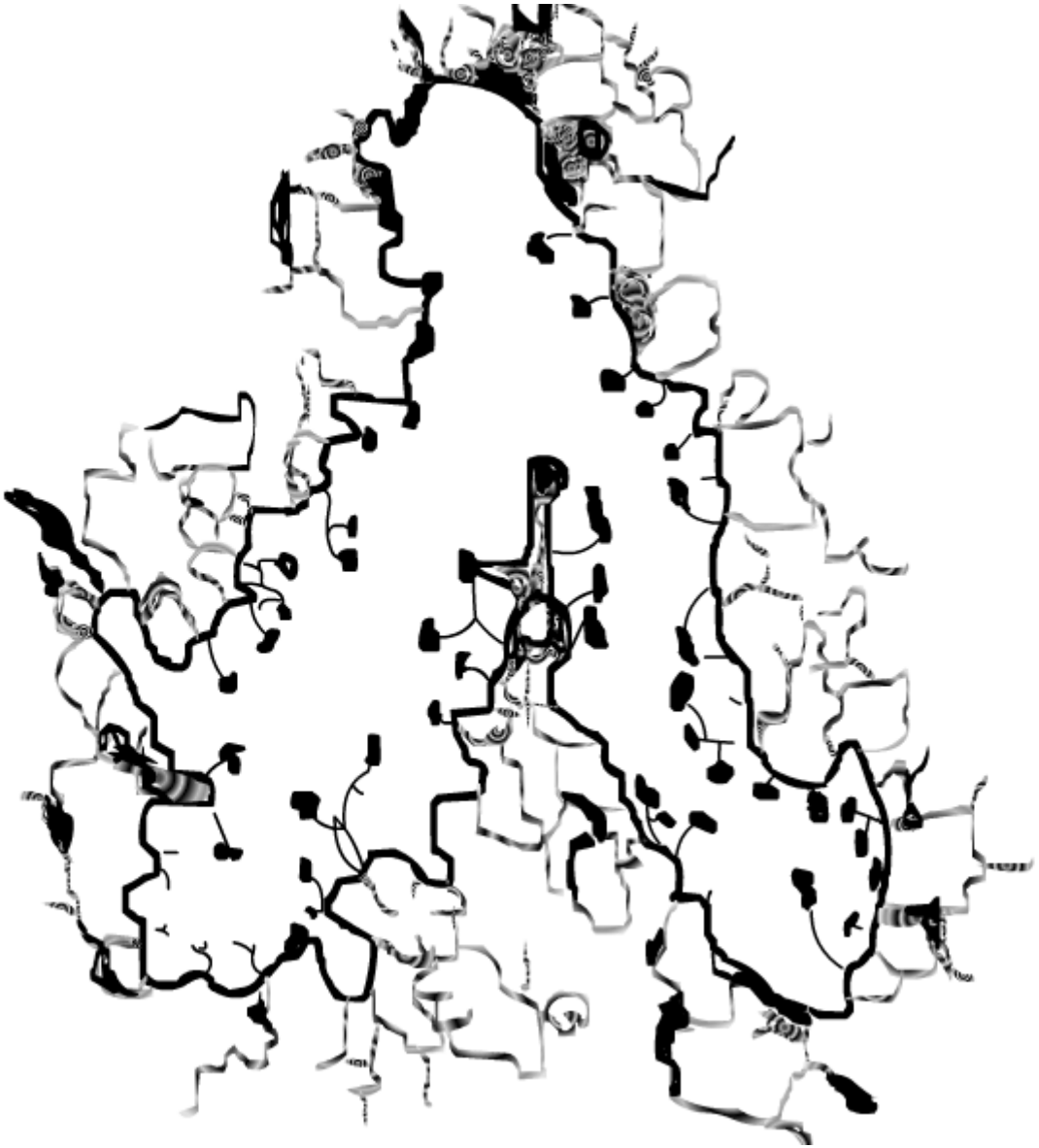
VA VD

The trapped air



VD VA

The alveolus doesn't manage to empty itself, thus, alveolar ventilation decreases

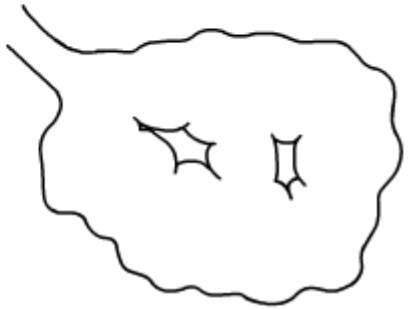


Non-emphysematous lung

VE = VD + VA

Centrilobular emphysema

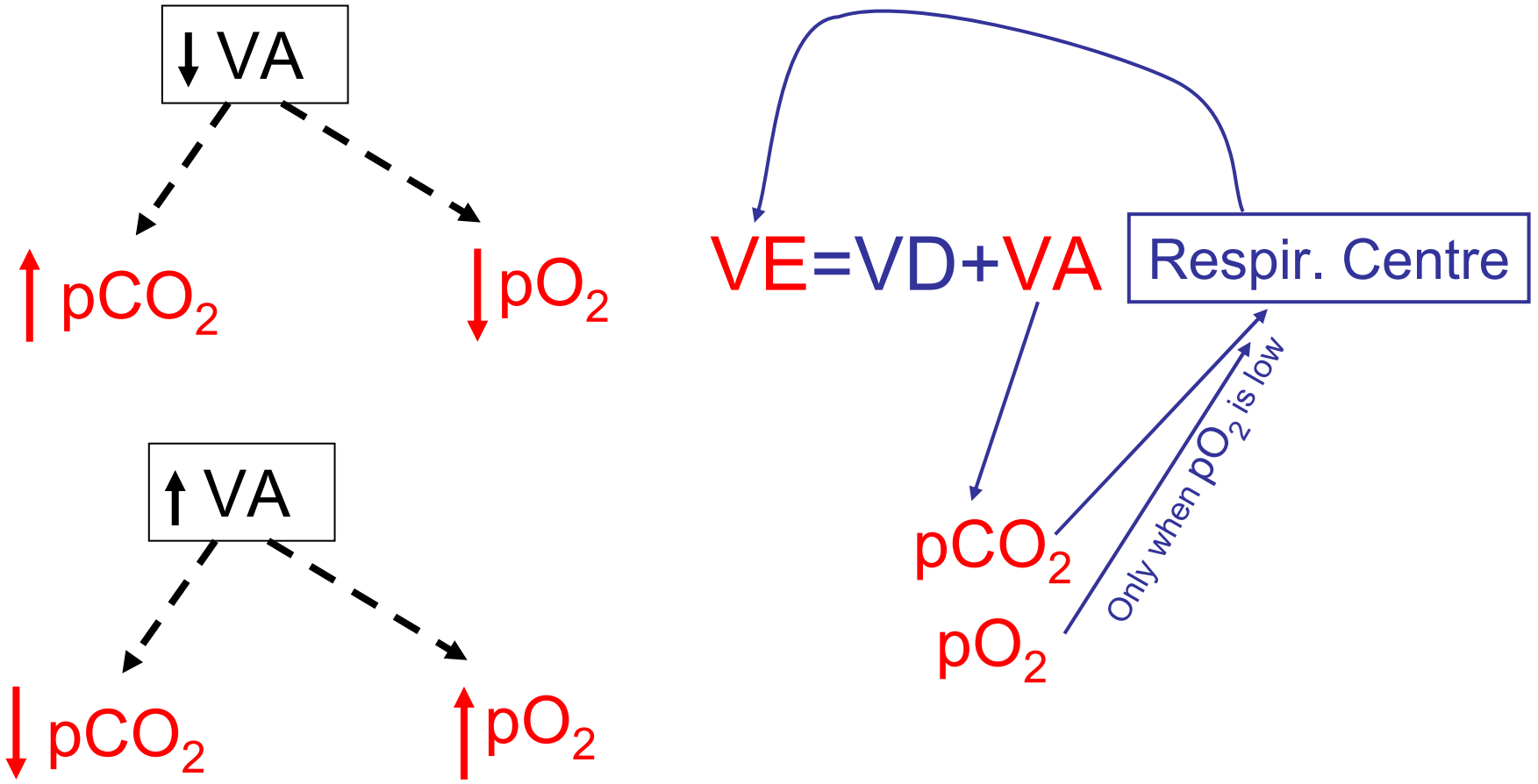
$$VE = VD + VA \longrightarrow VE = \mathbf{VD} + VA$$



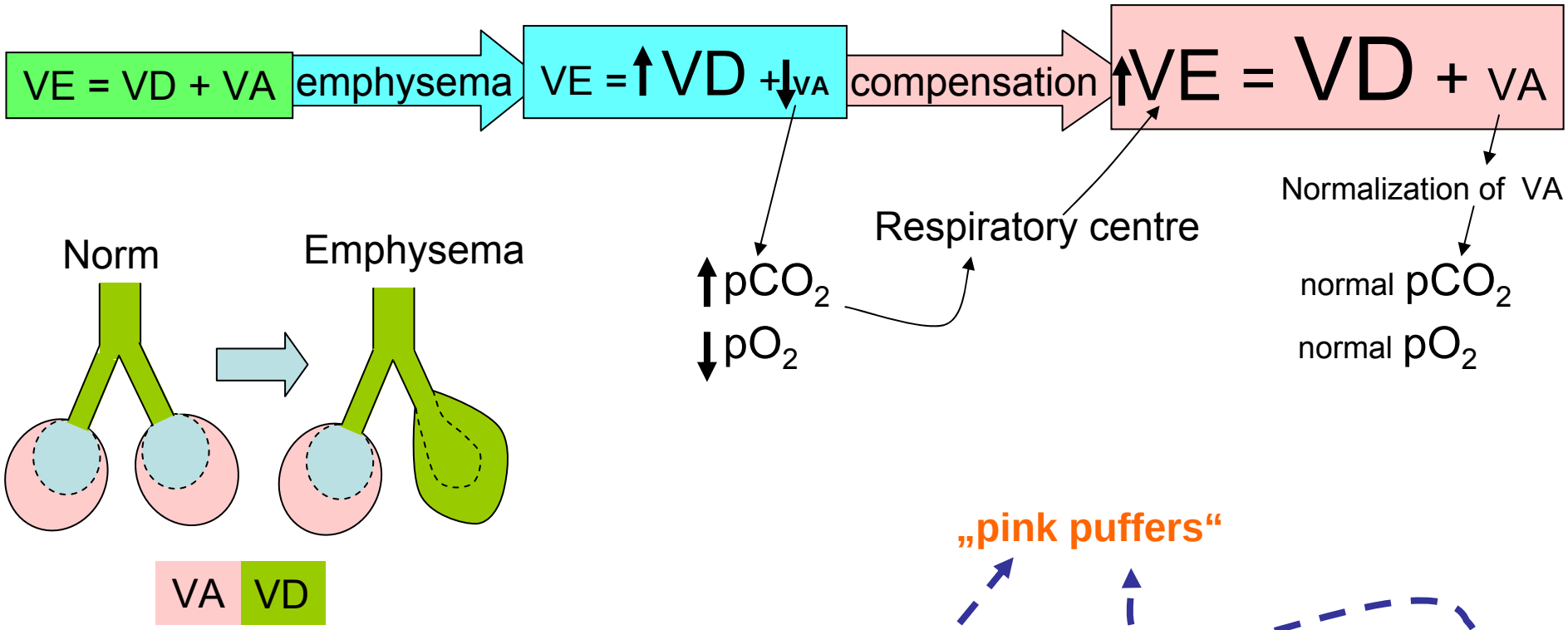
Panlobular emphysema

$$VE = VD + VA \longrightarrow VE = \mathbf{VD} + VA$$

Alveolar Ventilation Controls Rate of Breathing by Influencing pCO2 and pO2



Emphysematous form of the chronic obstructive lung disease



„pink puffers“

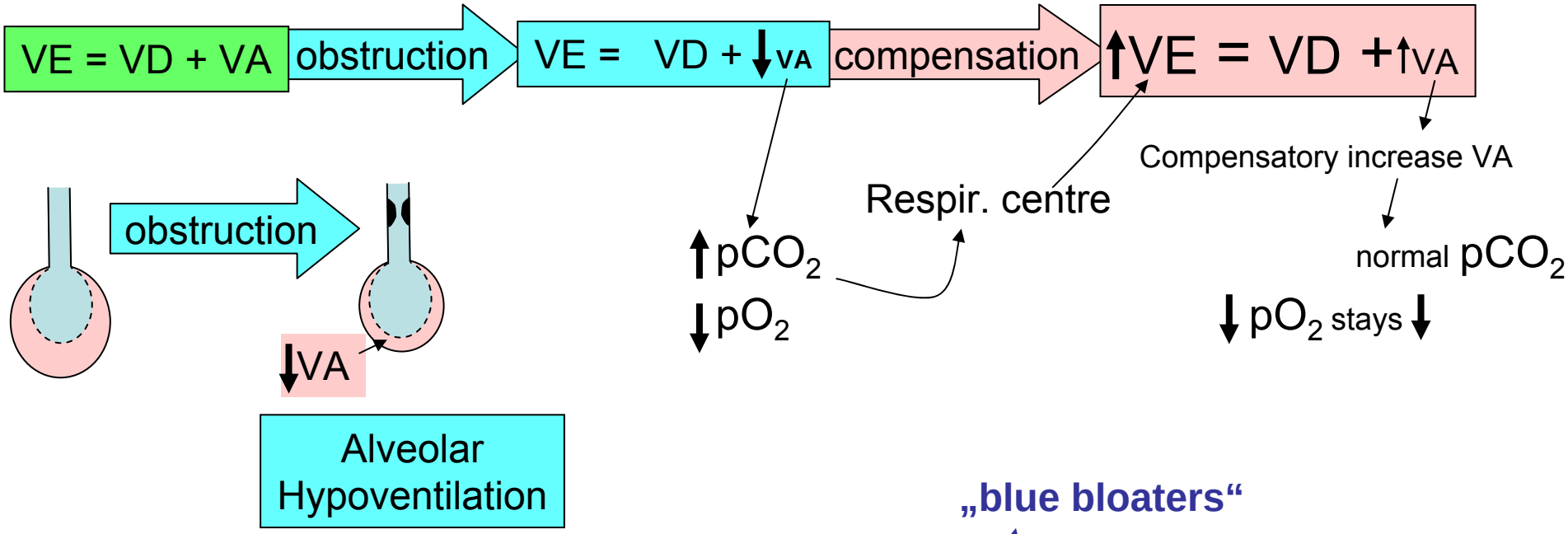
Normal P_aO_2 – no hypoxia (until total resp. failure)

Greater VE to bring to normal VA

Greater volume must be ventilated per minute
Increased respiratory work

Feeling of dyspnoea

Obstructive form of the chronic obstructive lung disease

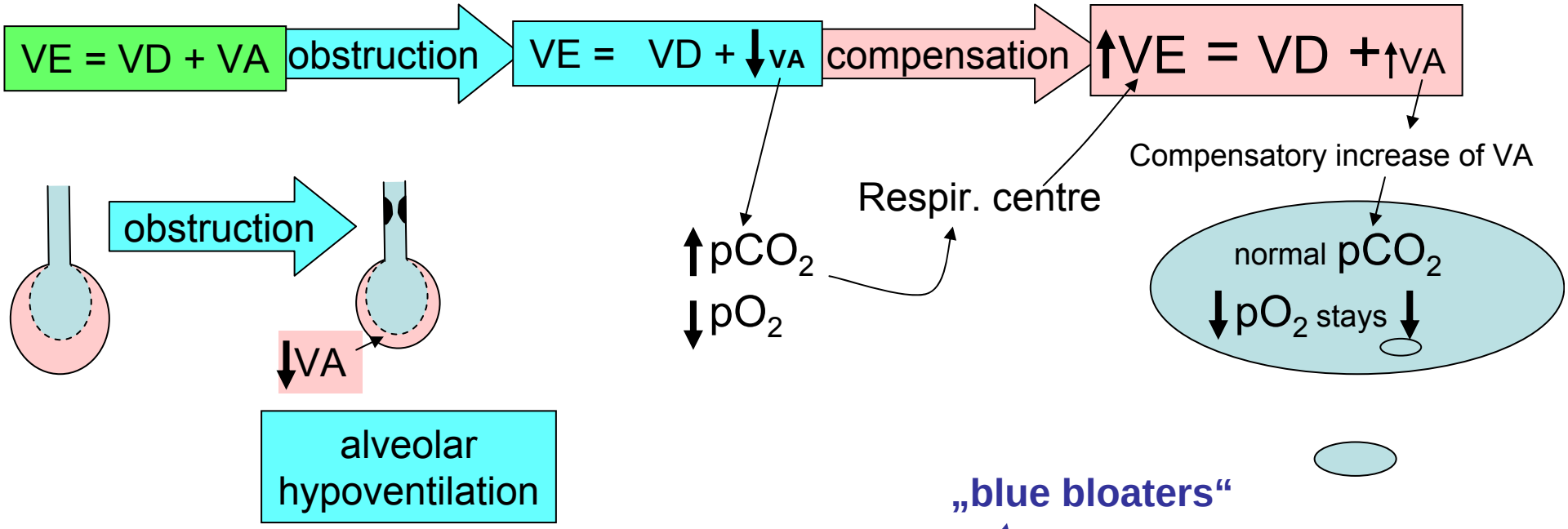


„blue bloaters“

Normal P_aCO_2, lower PaO_2 – hypoxia

Increased VE in order to increase the VA

Obstructive form of the chronic obstructive lung disease



„blue bloaters“

Normal P_aCO_2, decreased PaO_2 – hypoxia

Increased VE in order to increase VA

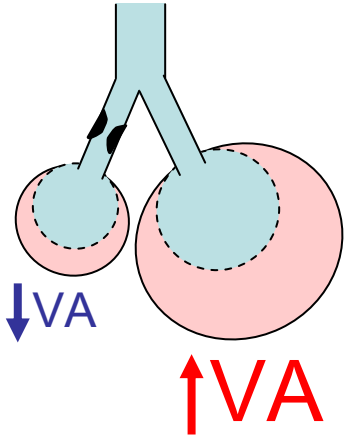
Why pCO_2 “manages” to normalize and pO_2 not?

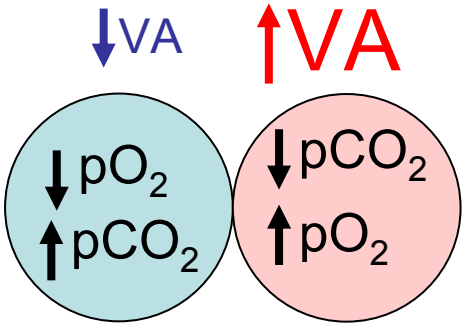
Obstructive form of the chronic obstructive lung disease

VE = VD + VA → obstruction → VE = VD + ↓VA

VA = (VA1 + VA2)

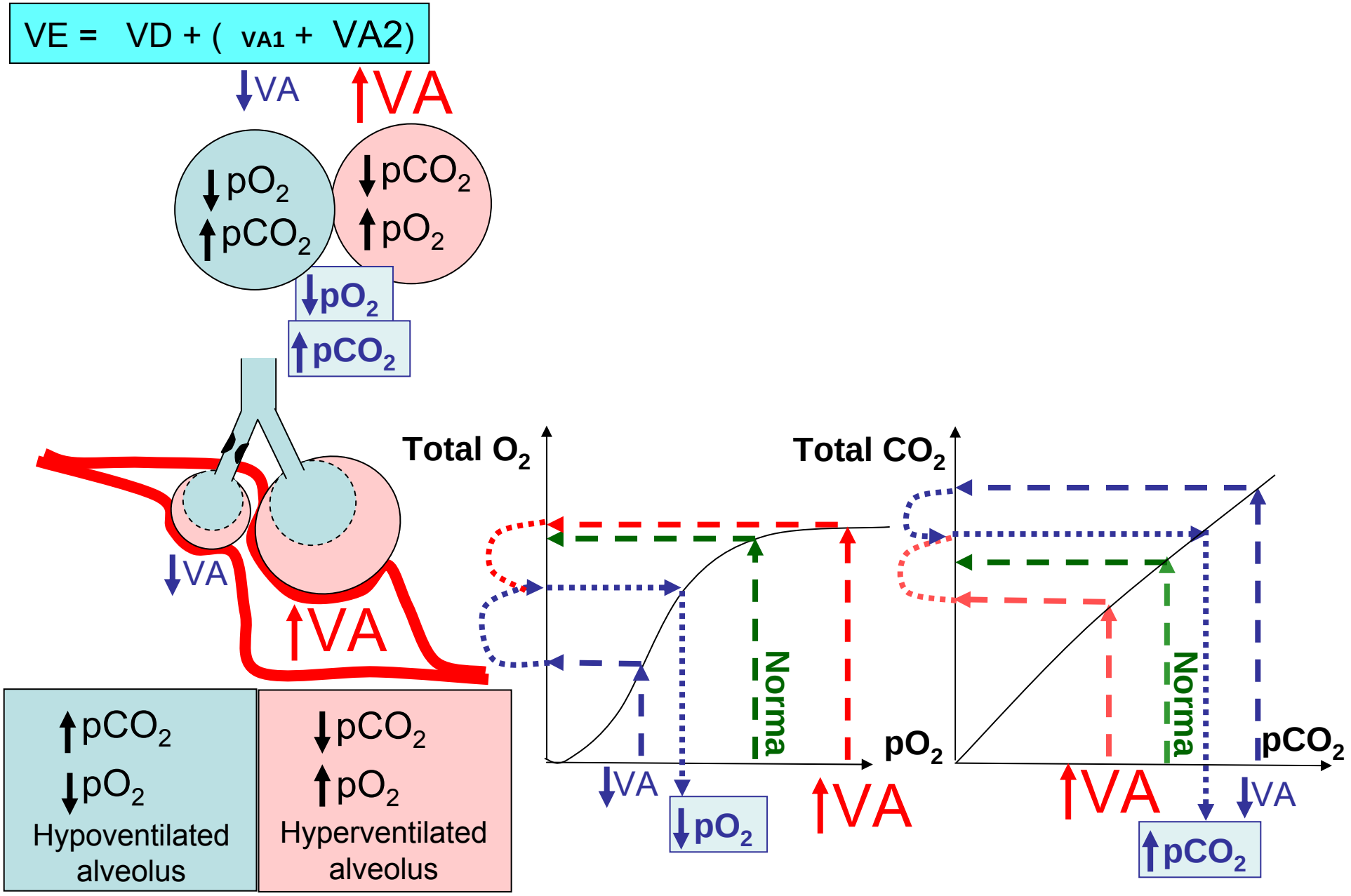
VE = VD + (VA1+VA2) → obstruction → VE = VD + (↓VA1 + VA2)



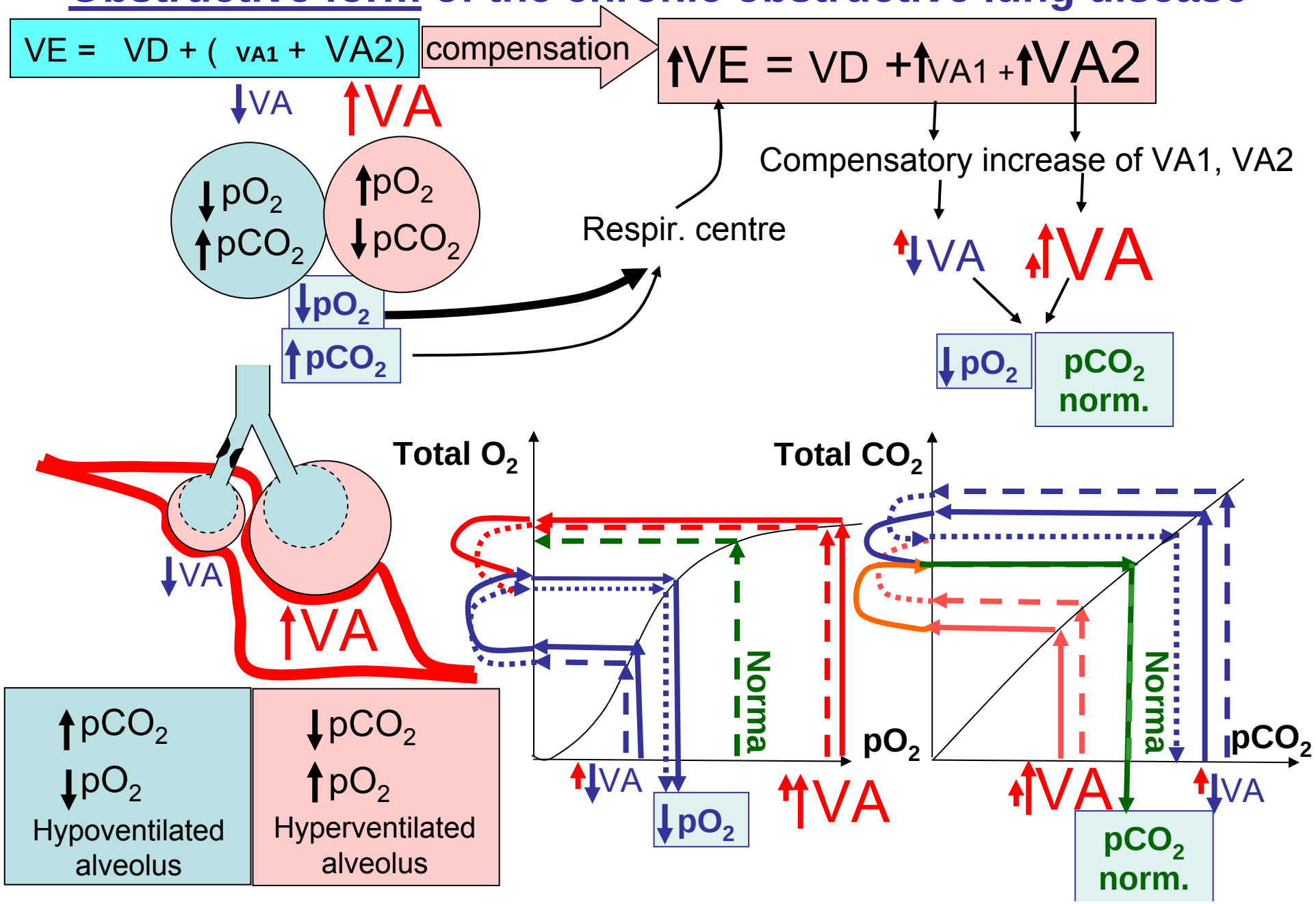


↑pCO_2 ↓pO_2 Hypoventilated alveolus	↓pCO_2 ↑pO_2 Hyperventilated alveolus

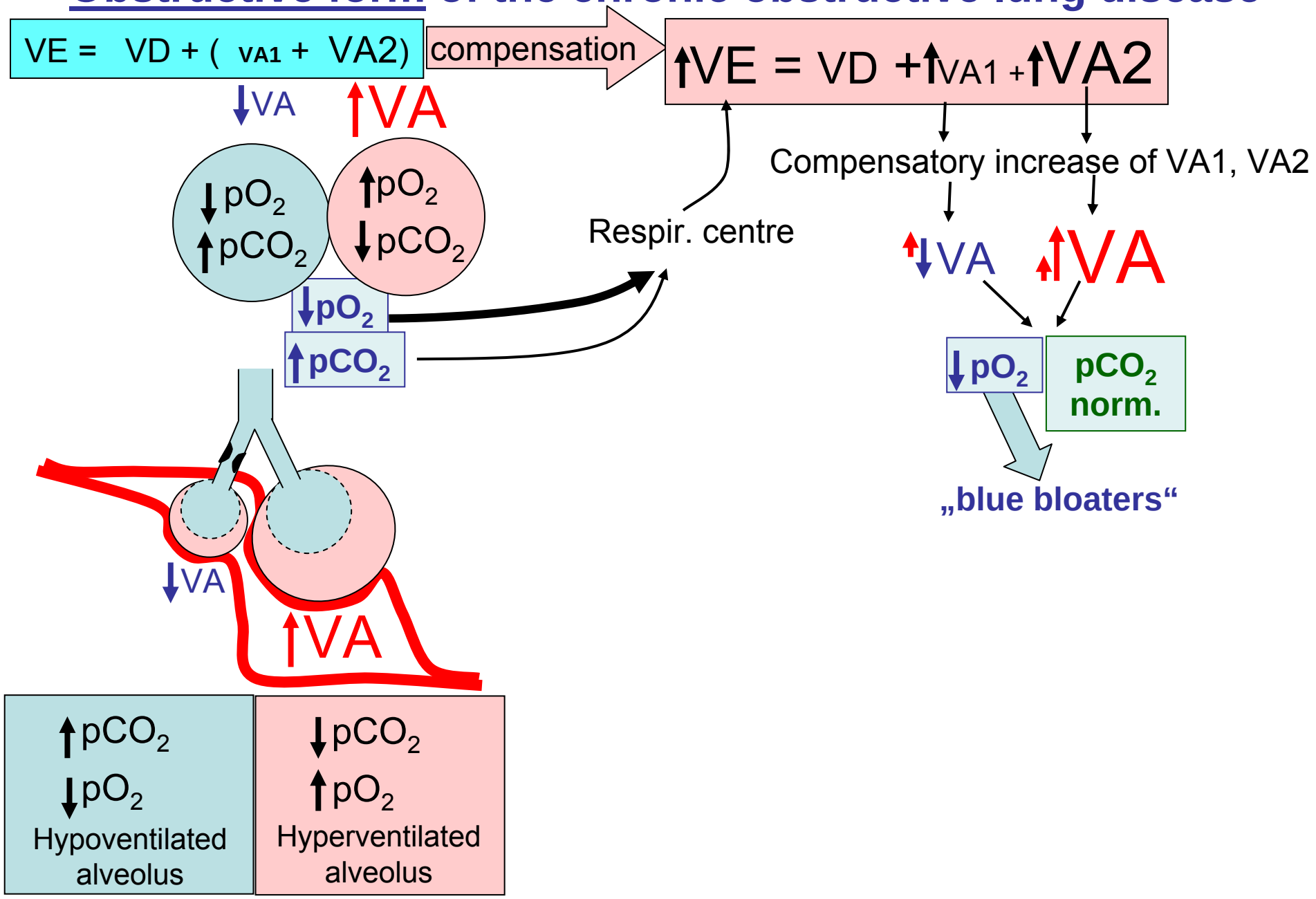
Obstructive form of the chronic obstructive lung disease



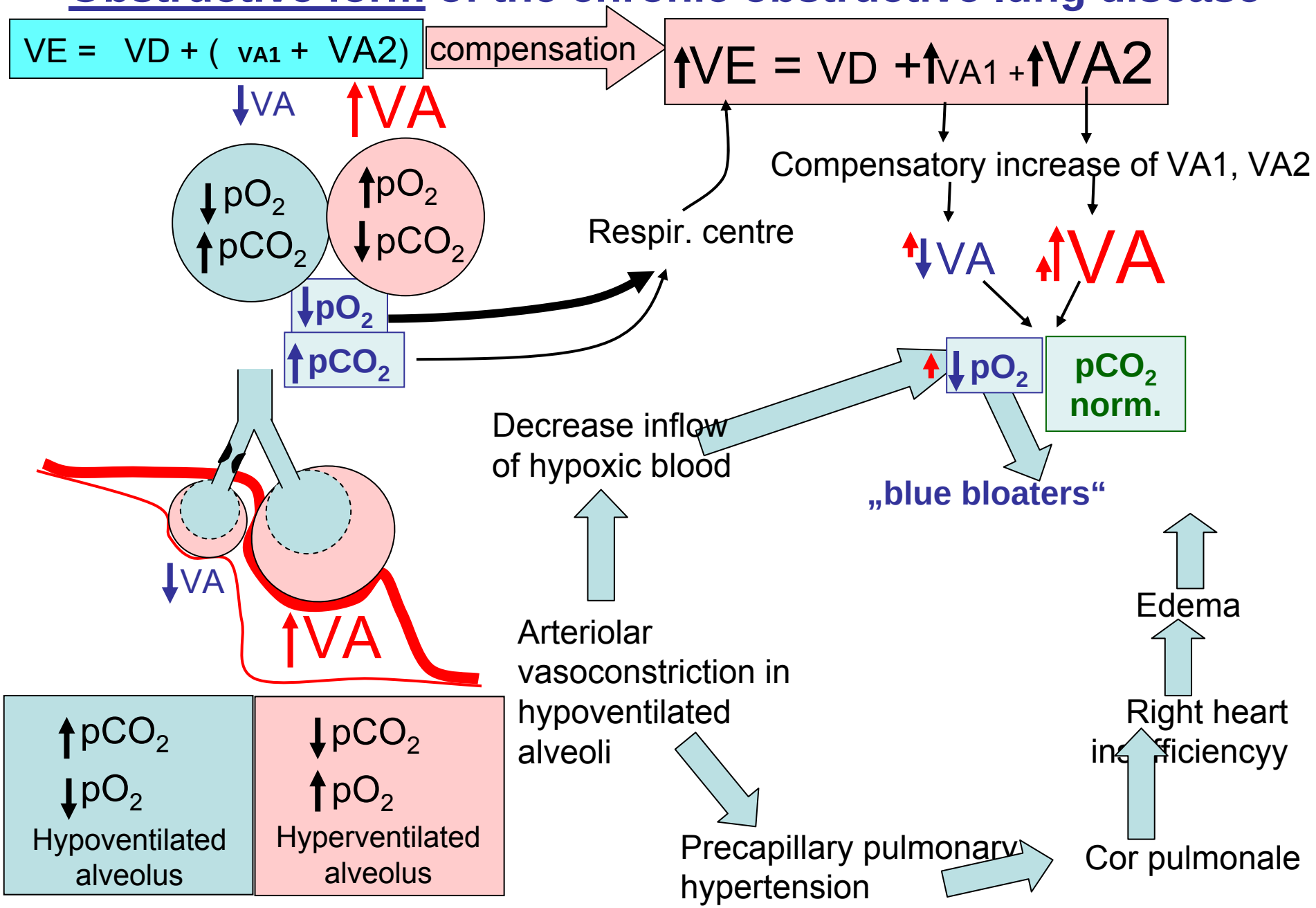
Obstructive form of the chronic obstructive lung disease



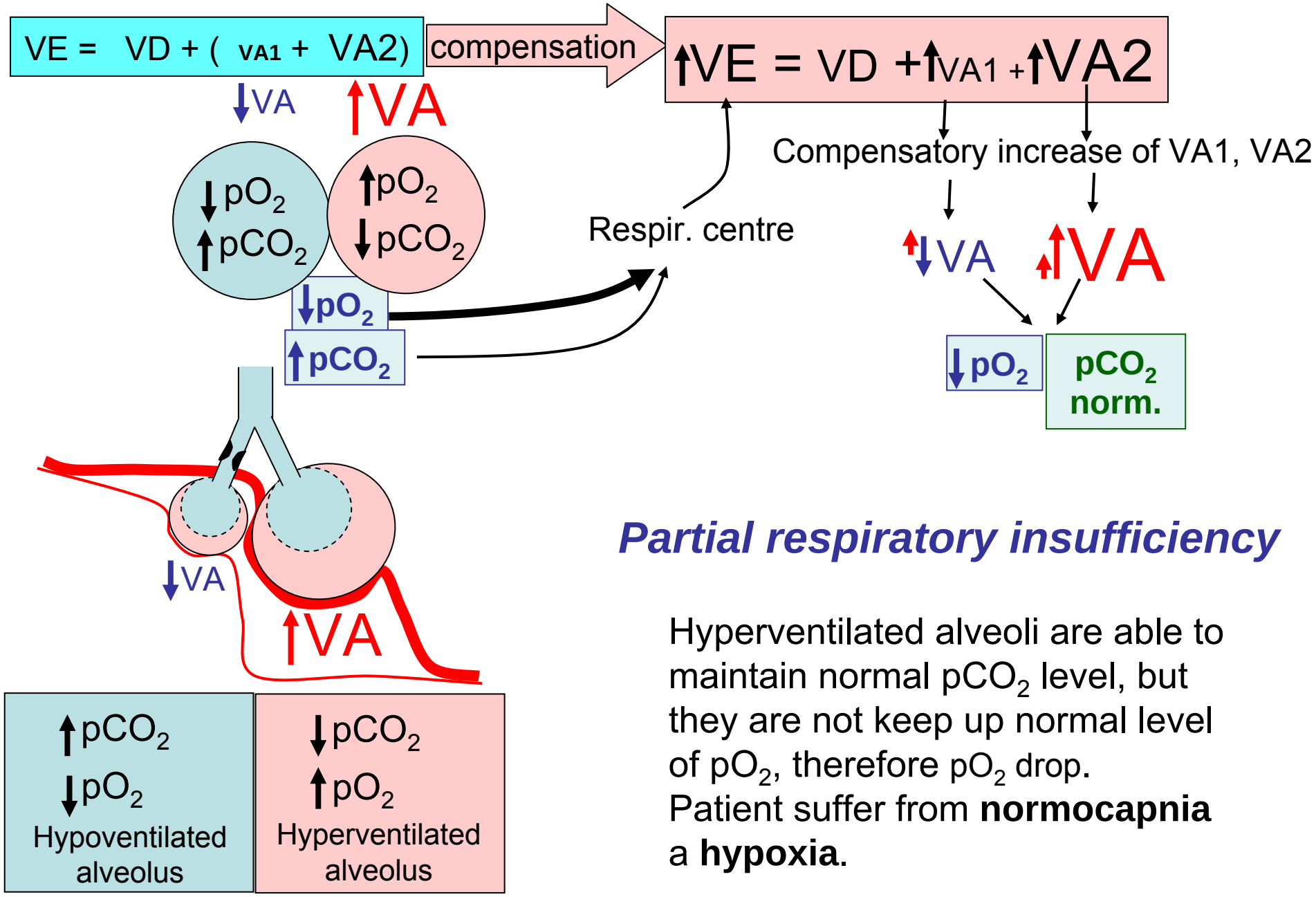
Obstructive form of the chronic obstructive lung disease



Obstructive form of the chronic obstructive lung disease



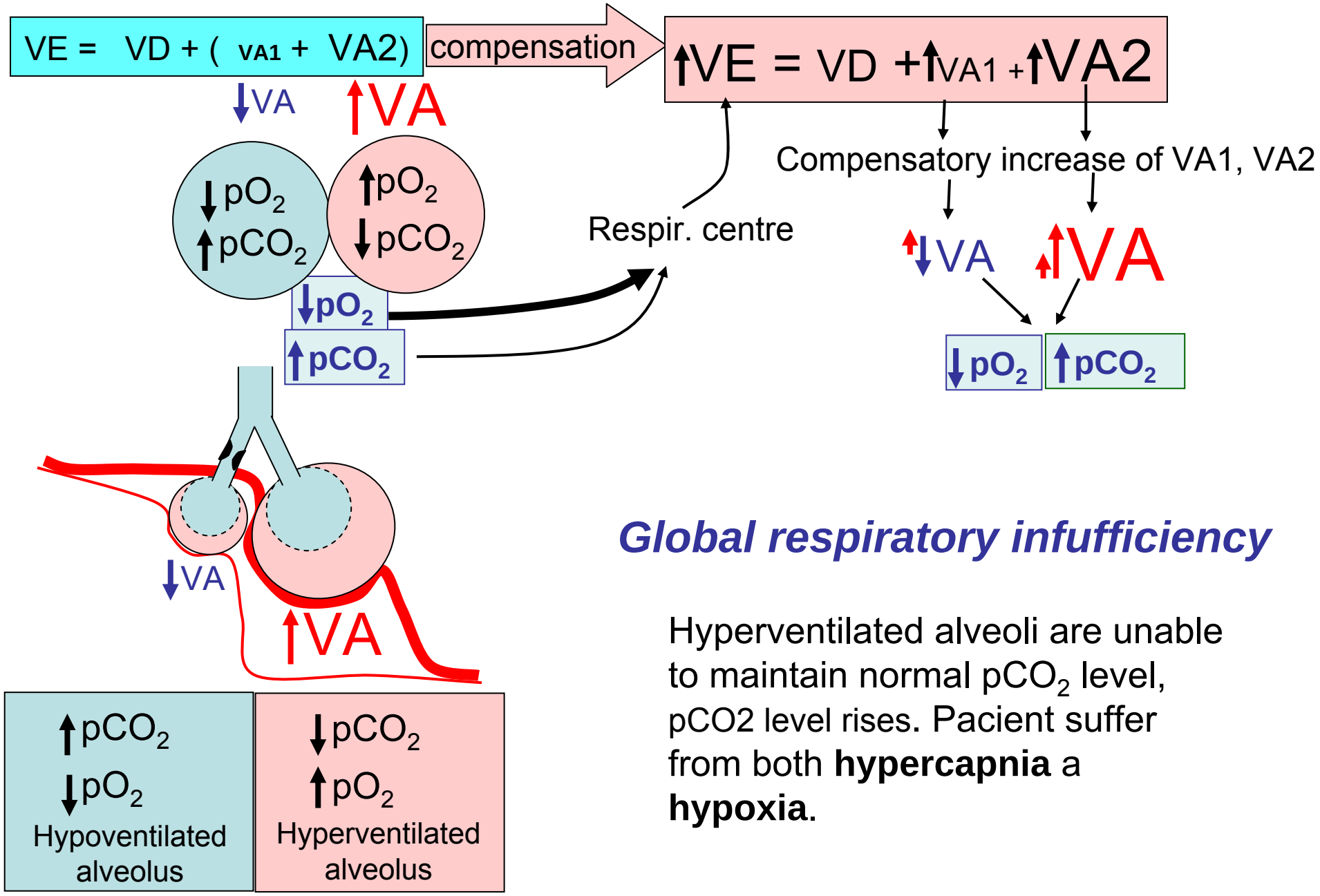
Partial respiratory insufficiency



Partial respiratory insufficiency

Hyperventilated alveoli are able to maintain normal pCO_2 level, but they are not keep up normal level of pO_2, therefore pO_2 drop. Patient suffer from **normocapnia** a **hypoxia**.

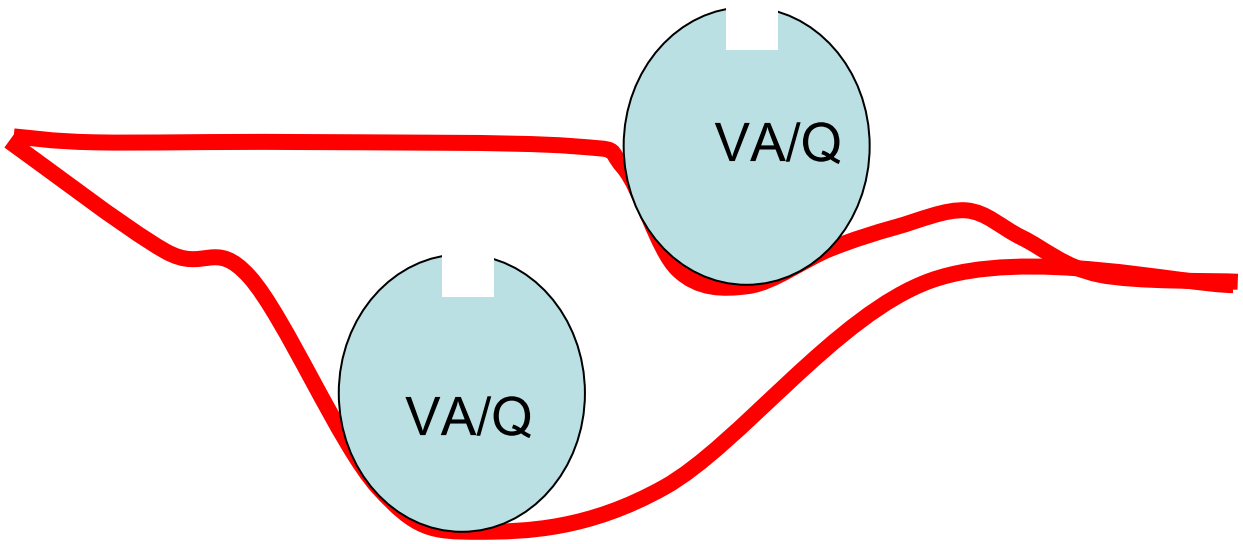
Global respiratory insufficiency



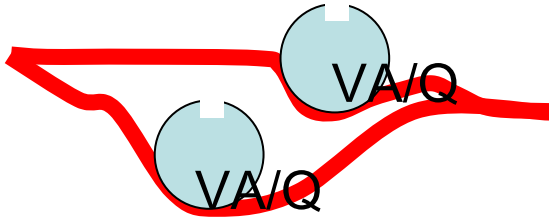
Global respiratory infufficiency

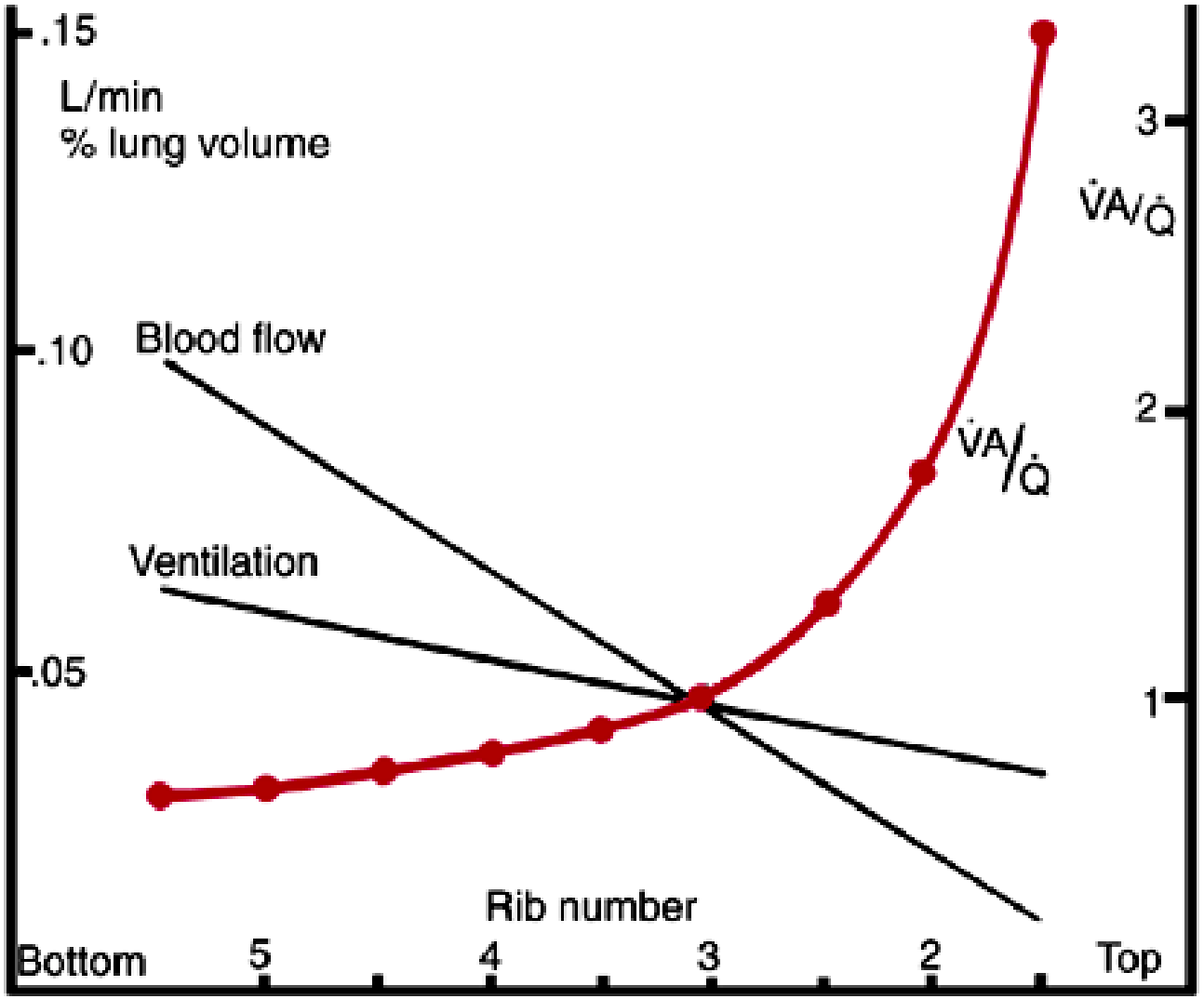
Hyperventilated alveoli are unable to maintain normal pCO_2 level, pCO2 level rises. Pacient suffer from both **hypercapnia** a **hypoxia**.

Ventilation-perfusion

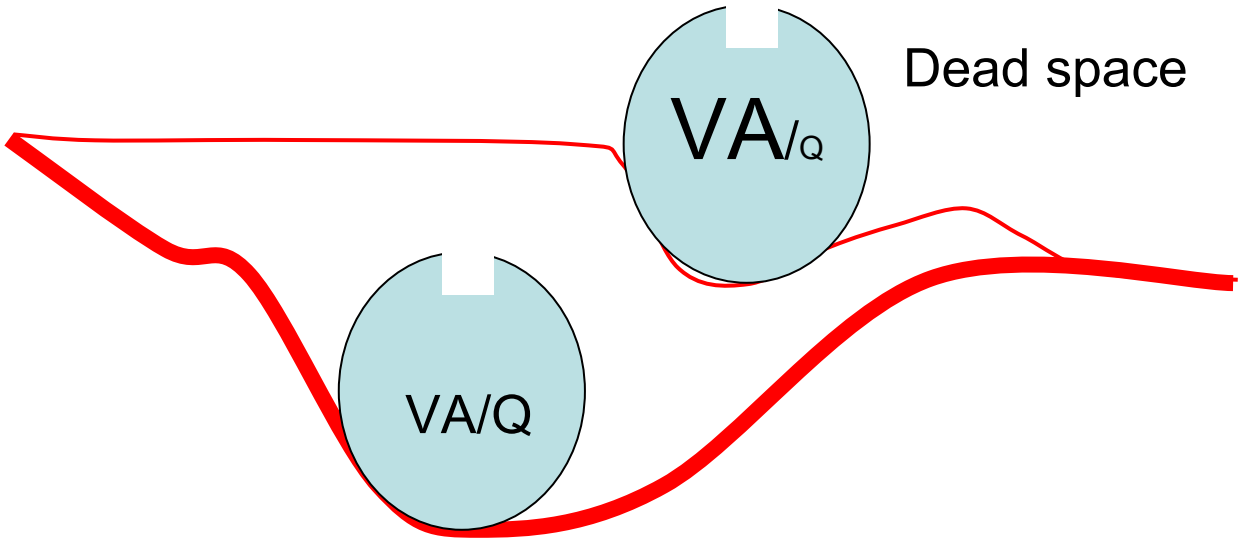


Ventilation-perfusion

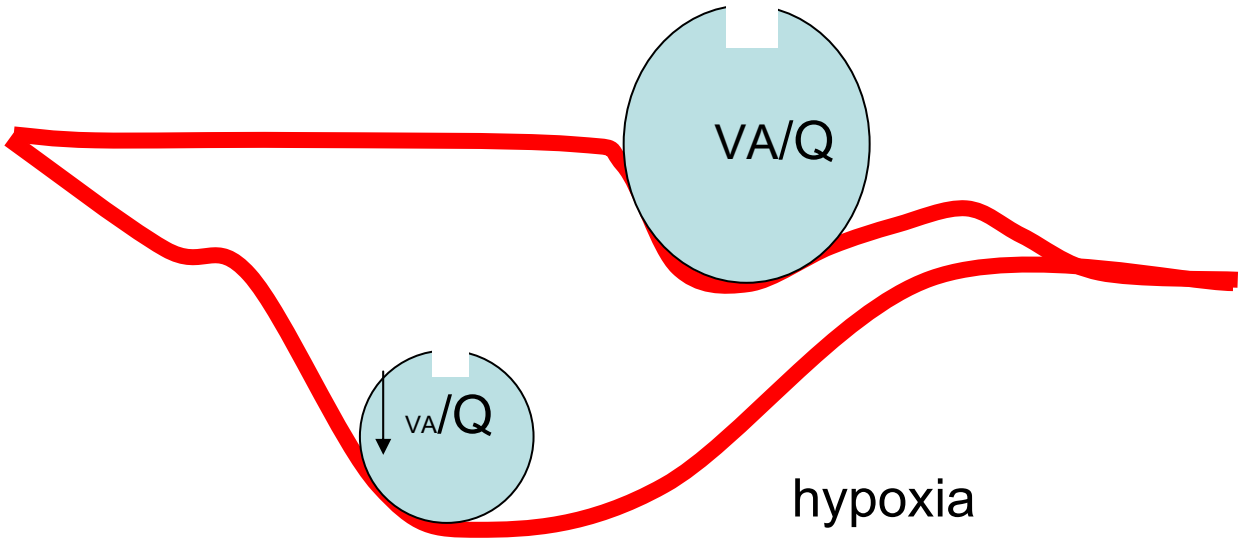




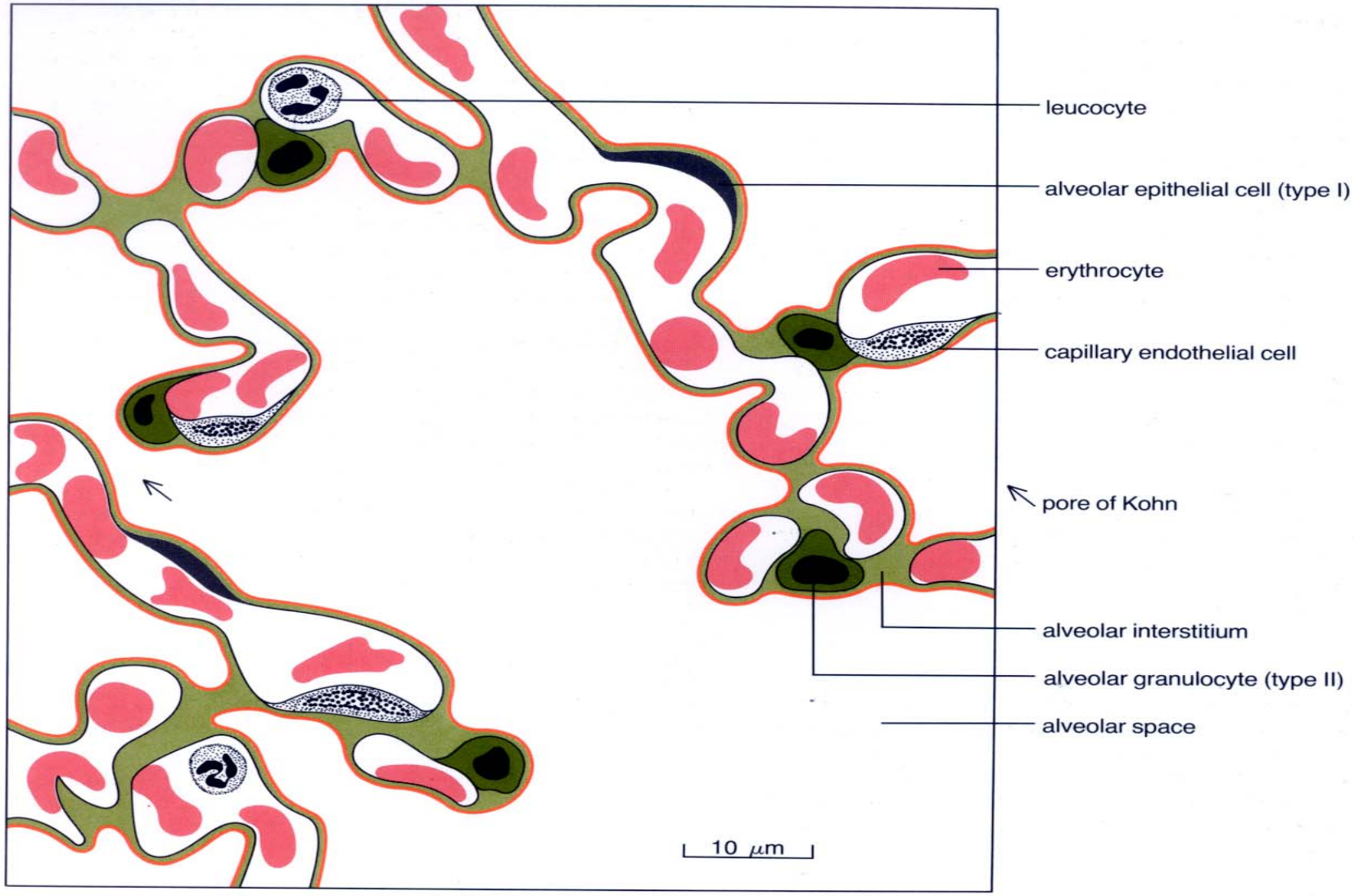
Ventilation-perfusion



Ventilation-perfusion

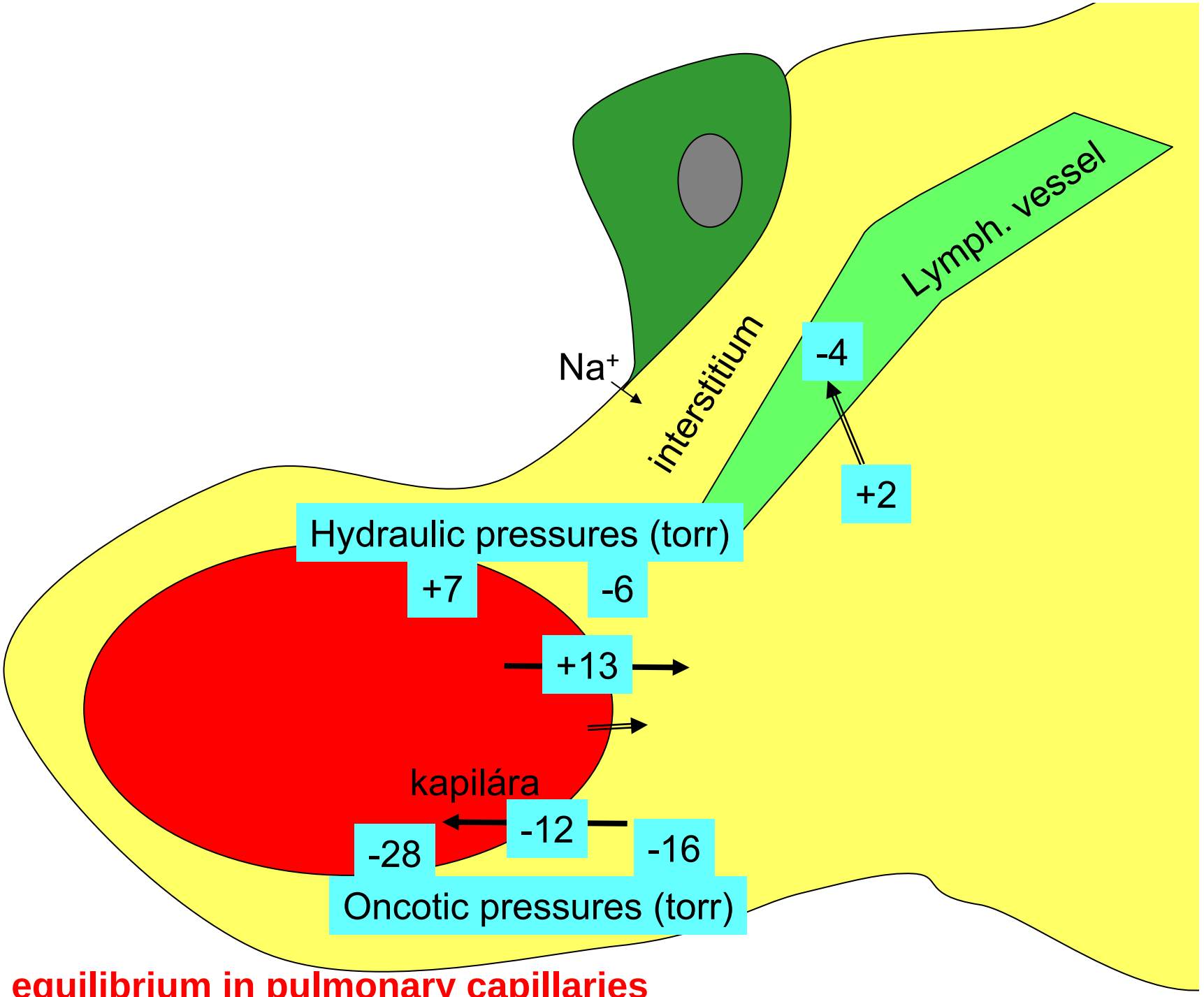


Right-left shunt

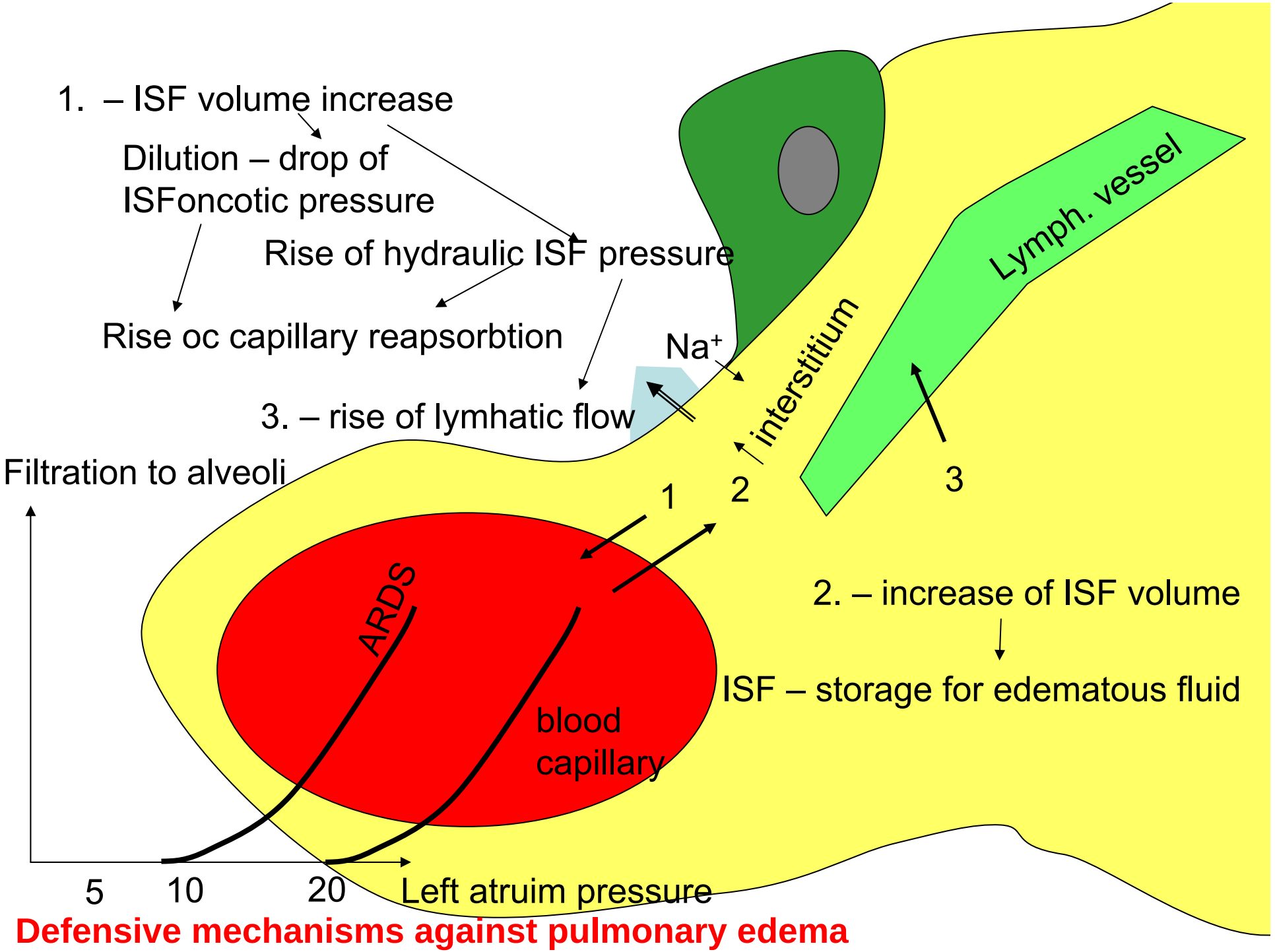


dimensiones of the alveolar-capillary membrane

overall thickness:	0.30–1.00 µm
alveolar epithelium:	0.15–0.35 µm
epithelial basement membrane:	0.05–0.20 µm
endothelial basement membrane:	0.05–0.40 µm
capillary endothelium:	0.05–0.25 µm



Starling equilibrium in pulmonary capillaries



Defensive mechanisms against pulmonary edema

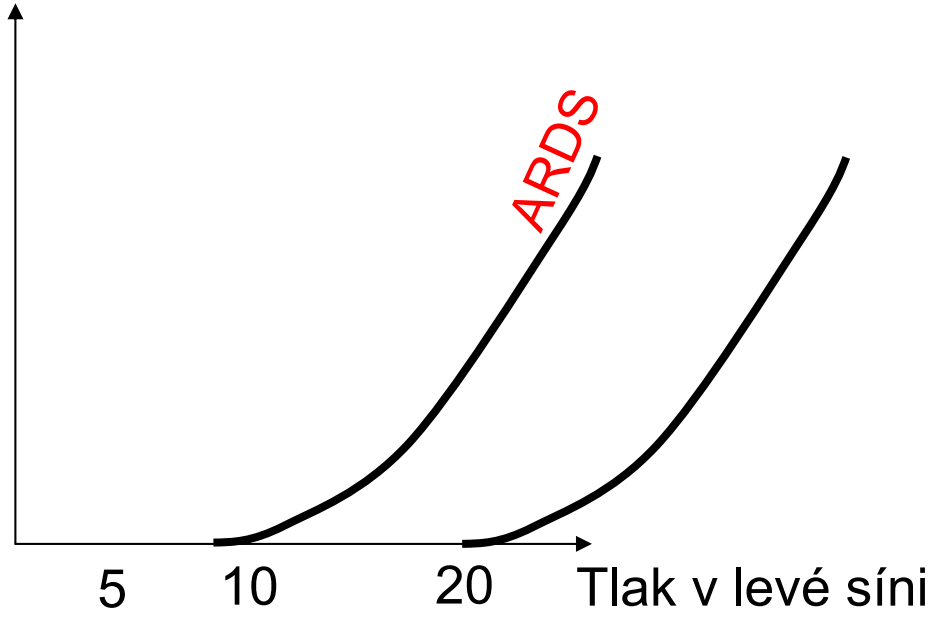
Acute Respiratory Distress Syndrome ARDS

Acute respiratory distress + risk factor (infection, aspiration, pankreatitis, trauma)

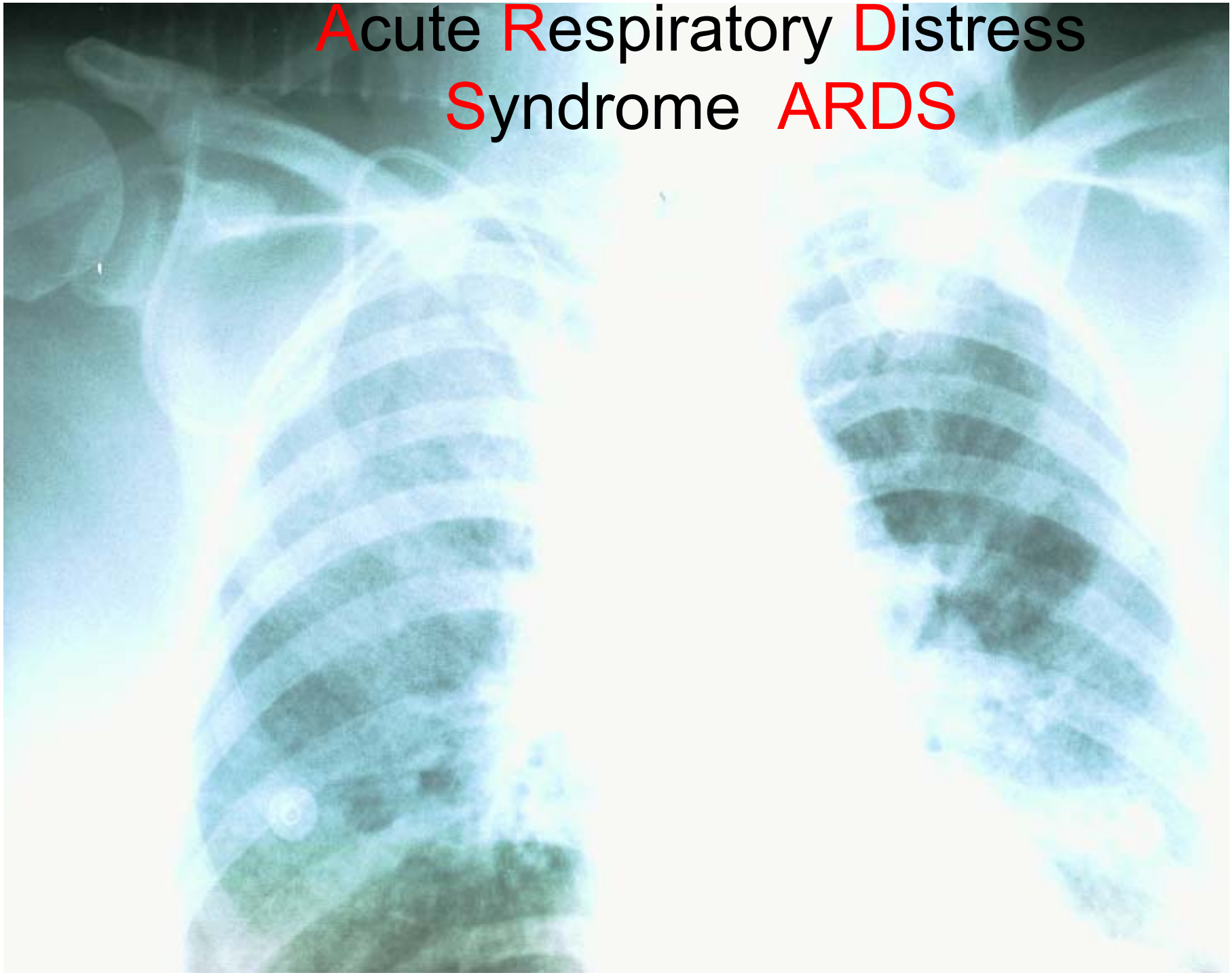
Hypoxemia

BIlateral pulmonary inflitration on RTG

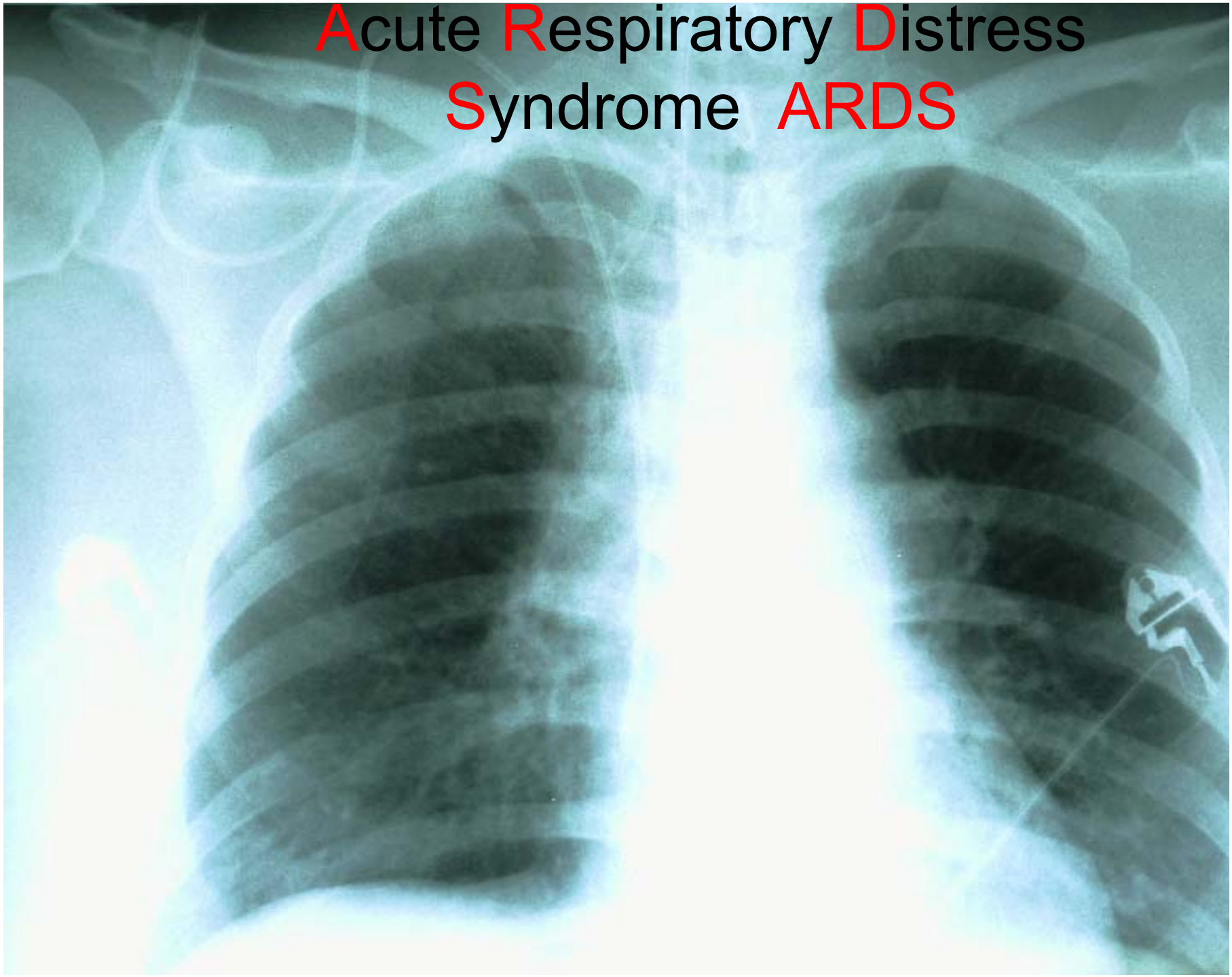
Normal right atrial pressure (pulmonary wedge pressure< 18 torr)



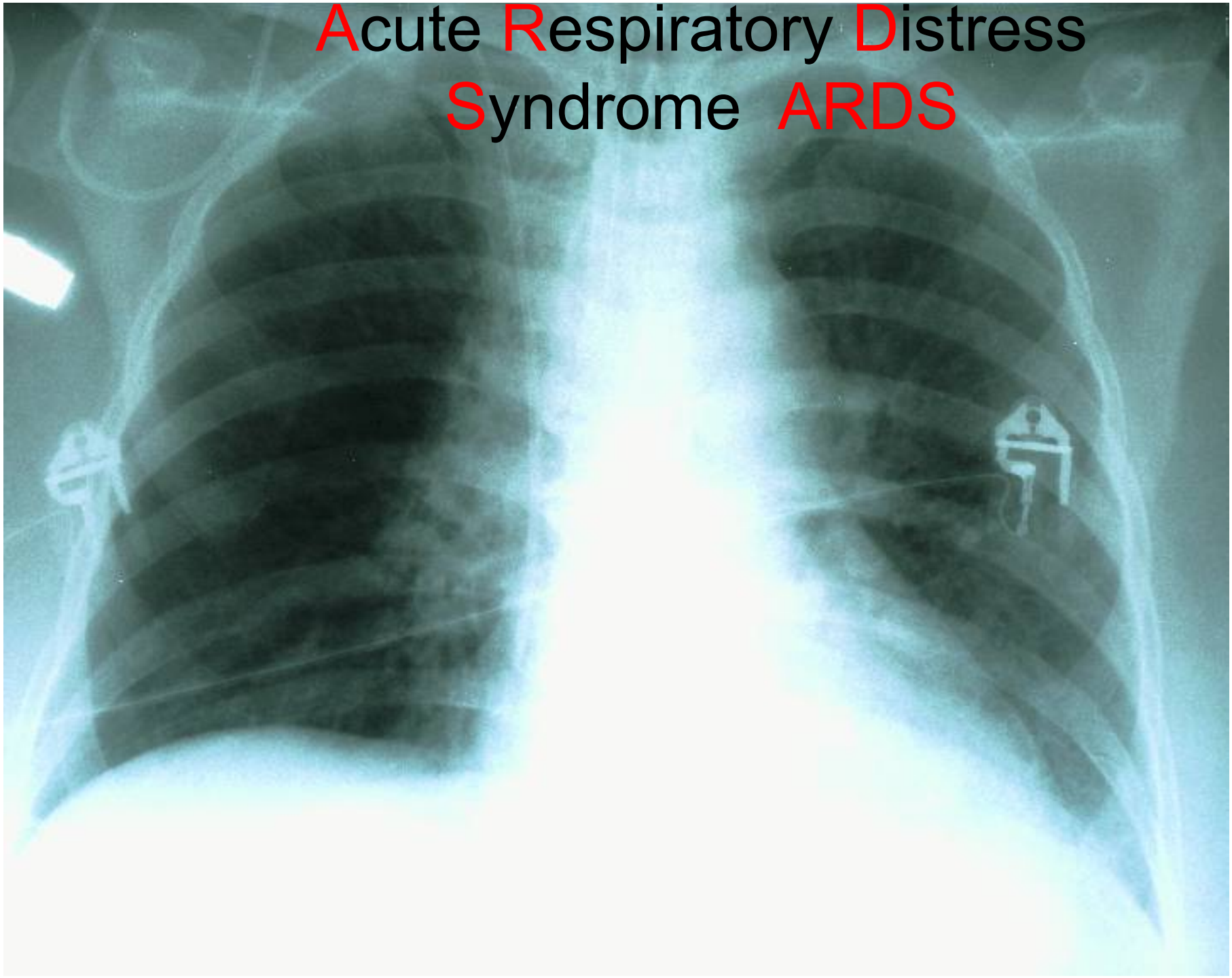
Acute Respiratory Distress Syndrome ARDS



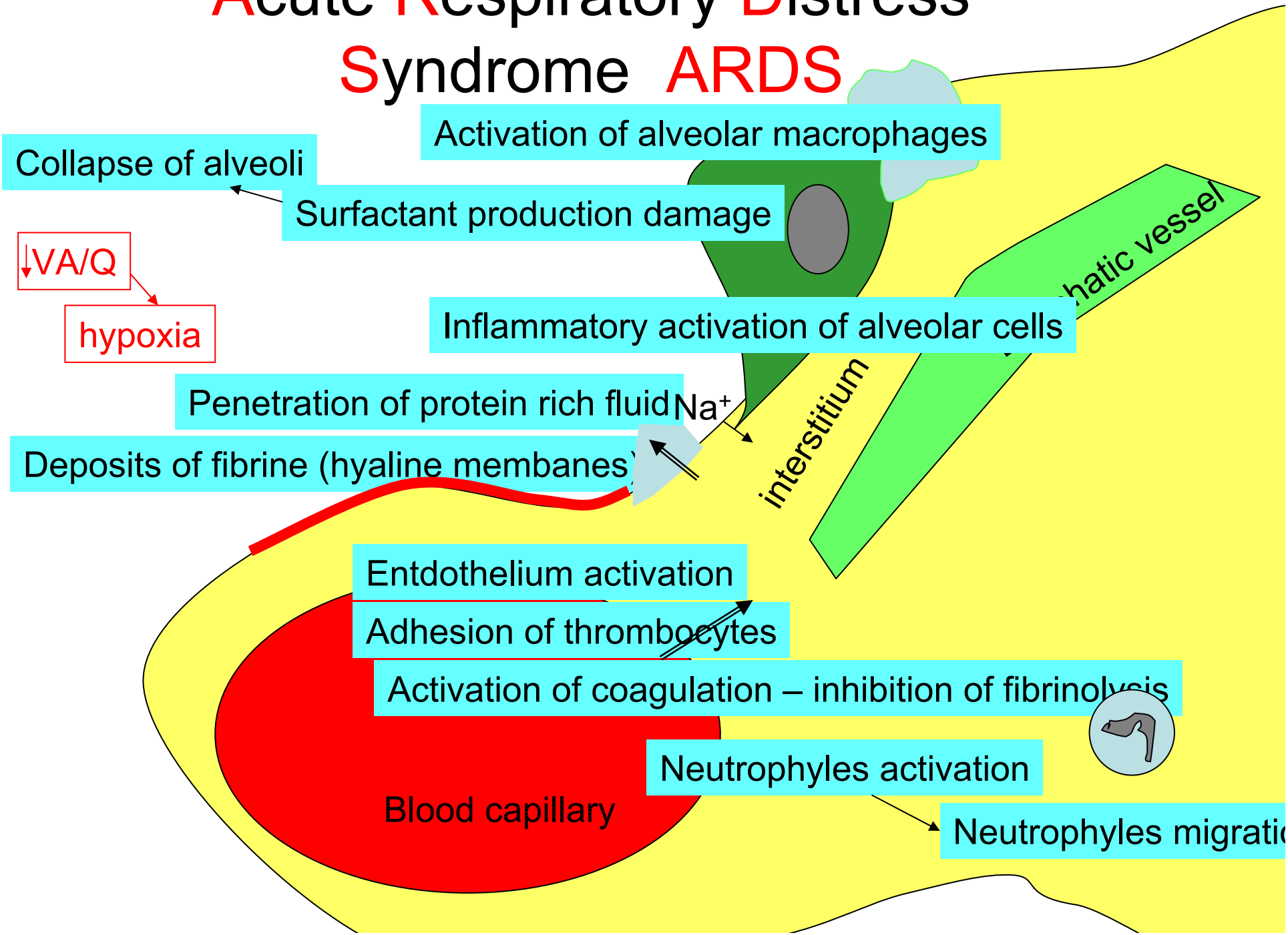
Acute Respiratory Distress Syndrome ARDS

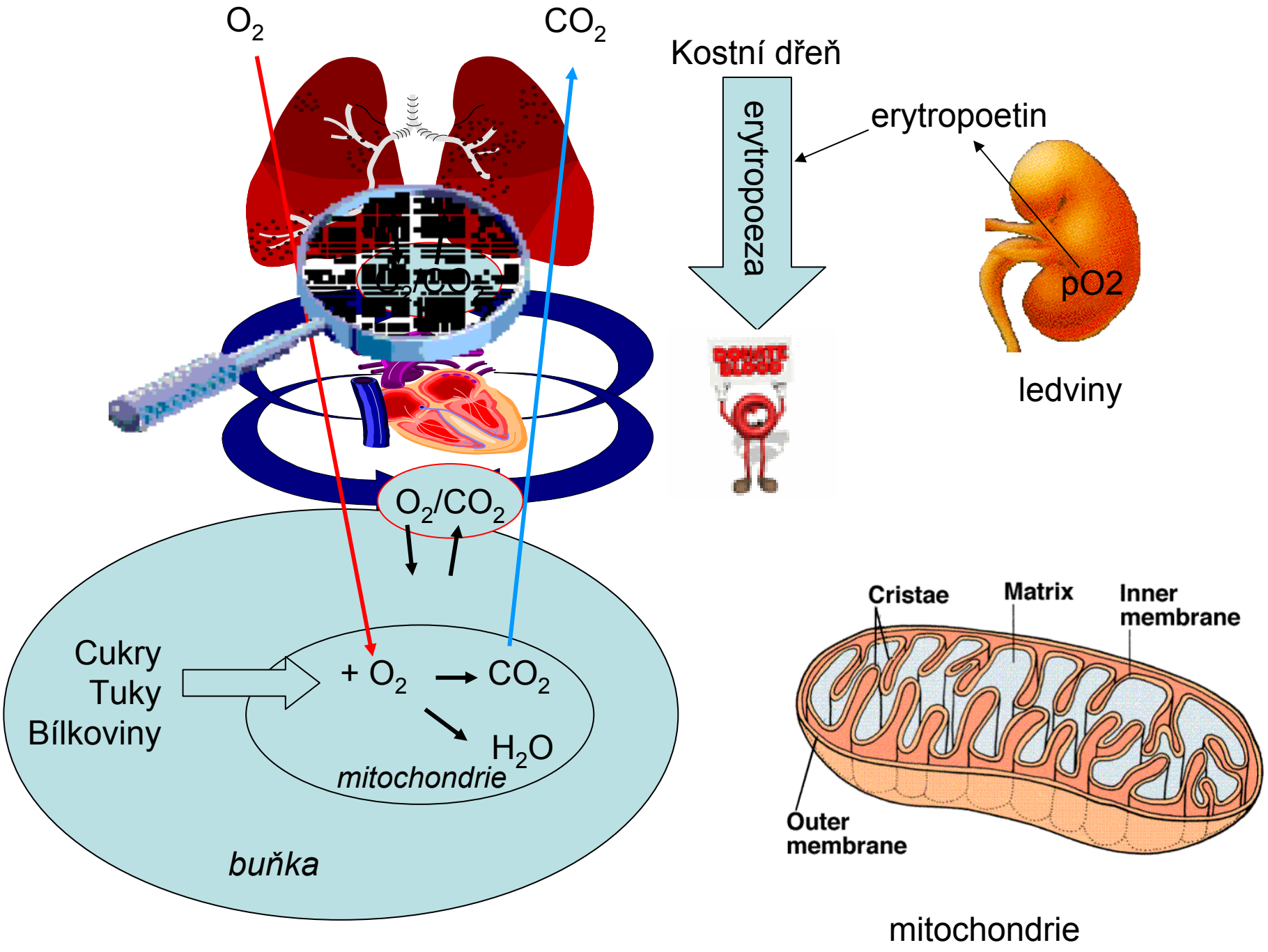


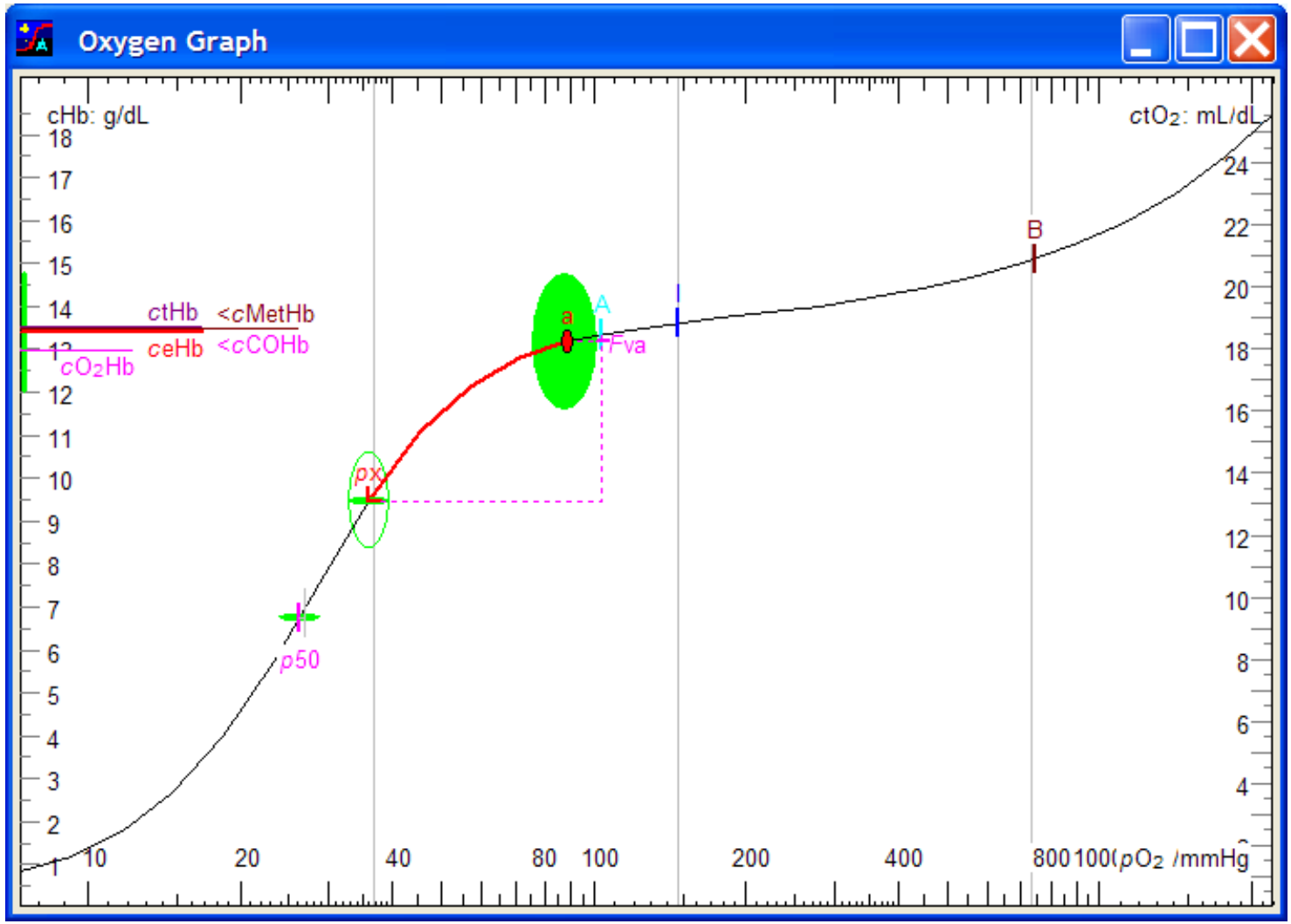
Acute Respiratory Distress Syndrome ARDS

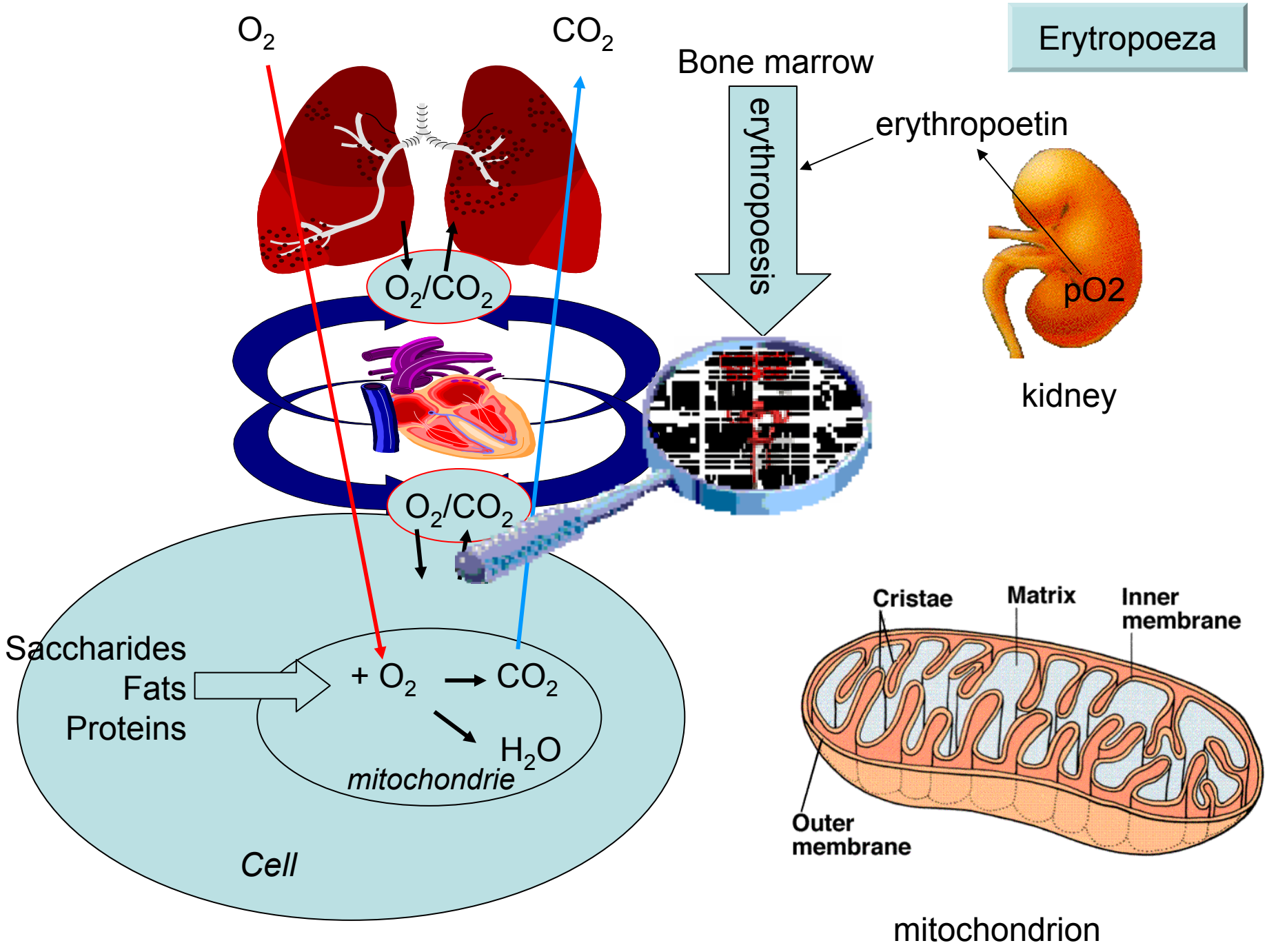


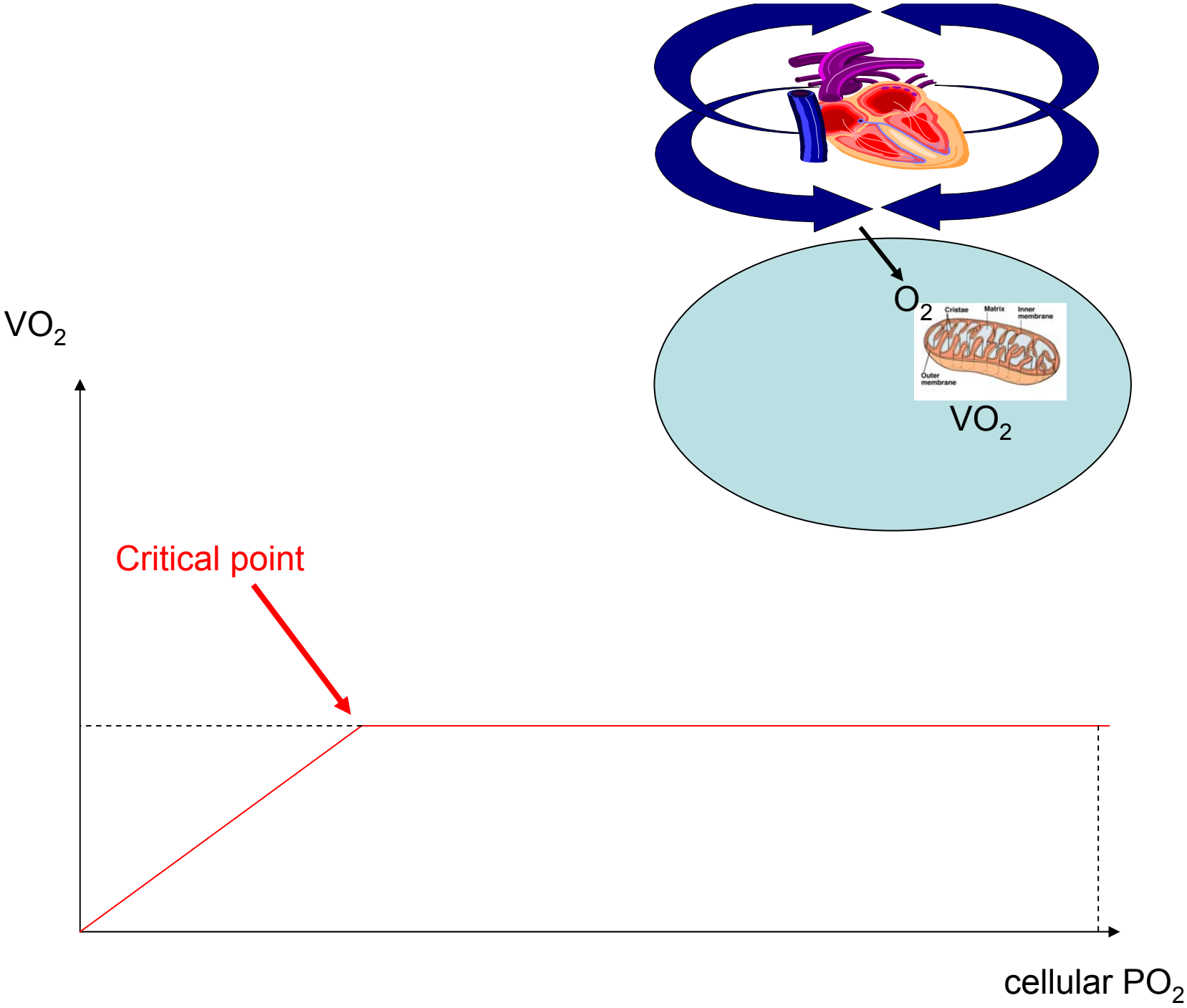
Acute Respiratory Distress Syndrome ARDS

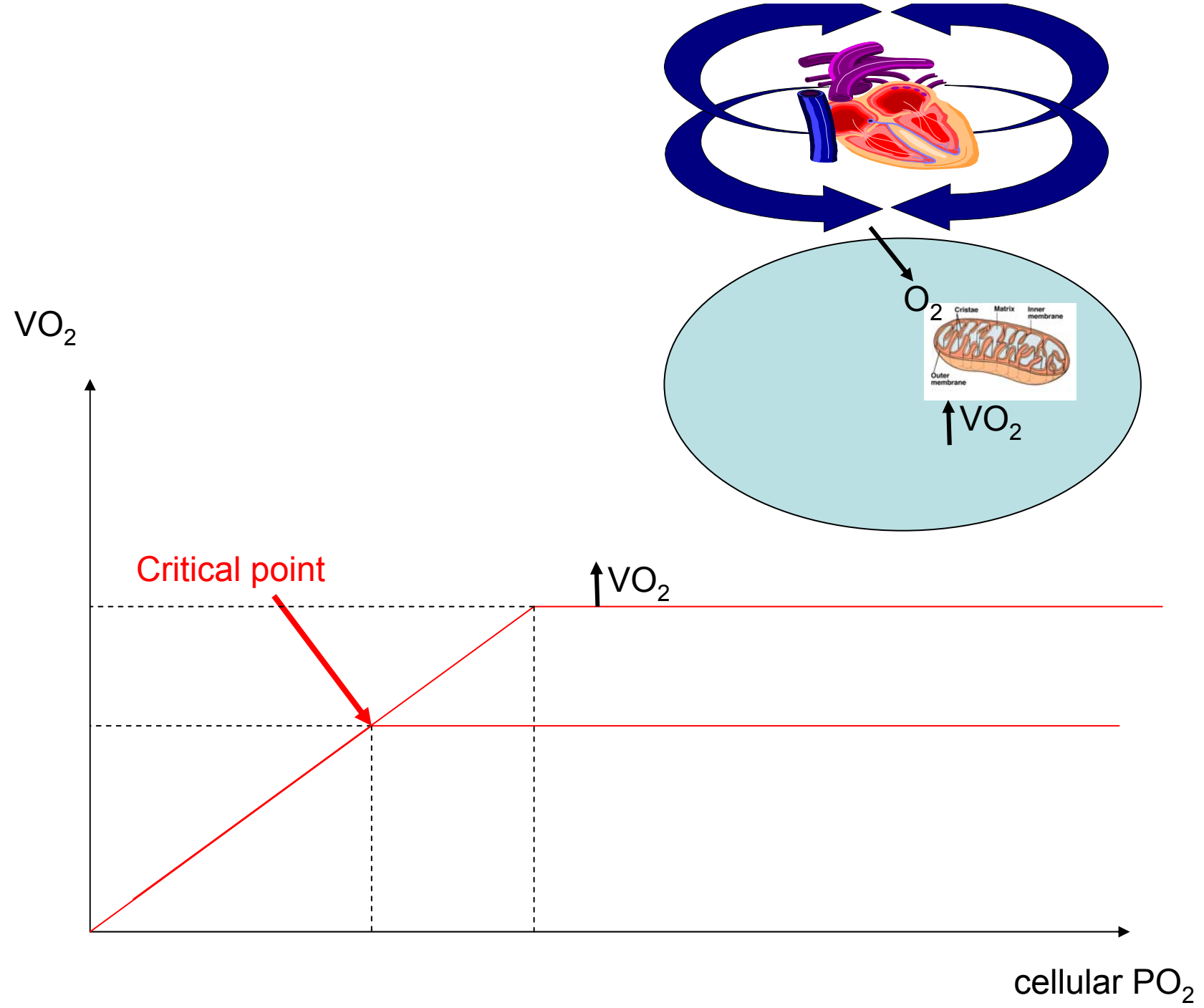


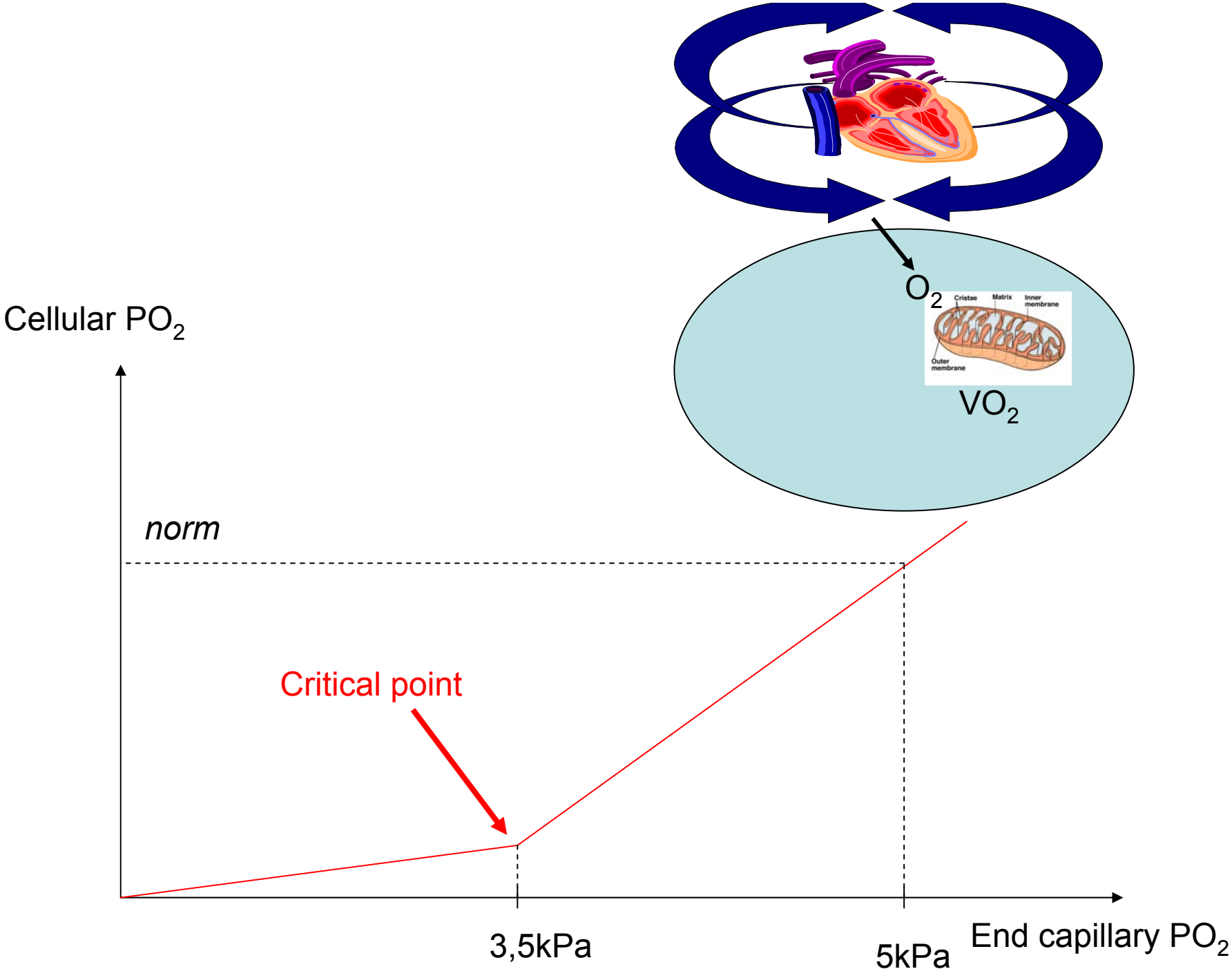

O_2
CO_2
O_2/CO_2
O_2/CO_2
Cukry
Tuky
Bílkoviny
+ O_2
CO_2
H_2O
mitochondrie
buňka
Kostní dřeň
erytropoeza
erytropoetin
pO2
ledviny
Cristae
Matrix
Inner membrane
Outer membrane
mitochondrie

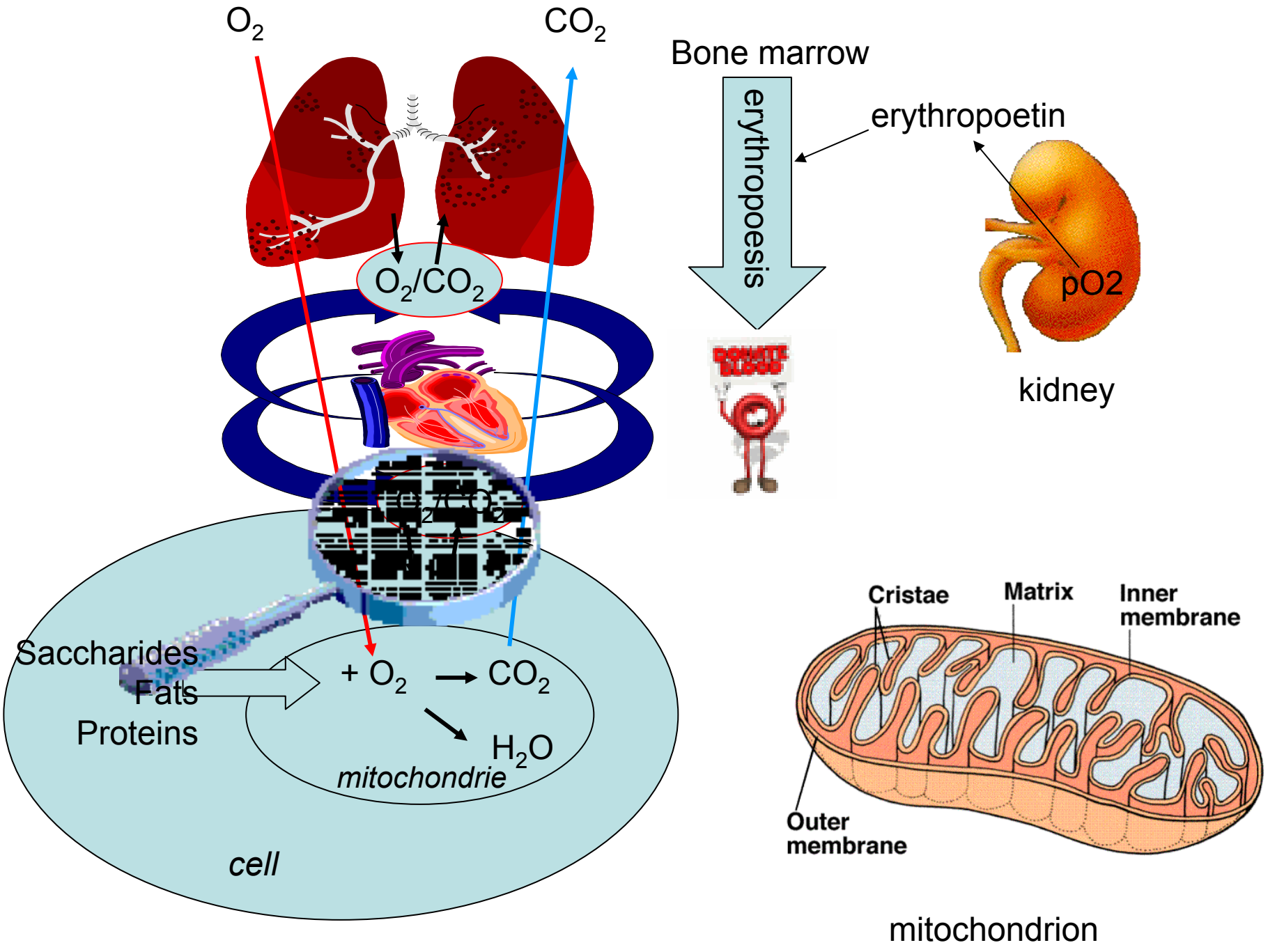

Oxygen Graph
cHb: g/dL
ctO2: mL/dL
ctHb
<cMetHb
ceHb
<cCOHb
cO2Hb
a
A
Fva
B
px
p50
(pO2 /mmHg


Erytropoeza
O_2
CO_2
Bone marrow
erythropoesis
erythropoetin
pO2
kidney
O_2/CO_2
O_2/CO_2
Saccharides
Fats
Proteins
+ O_2
CO_2
H_2O
mitochondrie
Cell
Cristae
Matrix
Inner membrane
Outer membrane
mitochondrion


VO_2
Critical point
O_2
Cristae
Matrix
Inner membrane
Outer membrane
VO_2
cellular PO_2


VO_2
O_2
Cristae
Matrix
Inner membrane
Outer membrane
VO_2
Critical point
VO_2
cellular PO_2


O2
Cristae
Matrix
Inner membrane
Outer membrane
VO2
Cellular PO2
norm
Critical point
3,5kPa
5kPa
End capillary PO2


O_2
CO_2
O_2/CO_2
Saccharides
Fats
Proteins
+ O_2
CO_2
H_2O
mitochondrie
cell
Bone marrow
erythropoesis
erythropoetin
pO2
kidney
Cristae
Matrix
Inner membrane
Outer membrane
mitochondrion