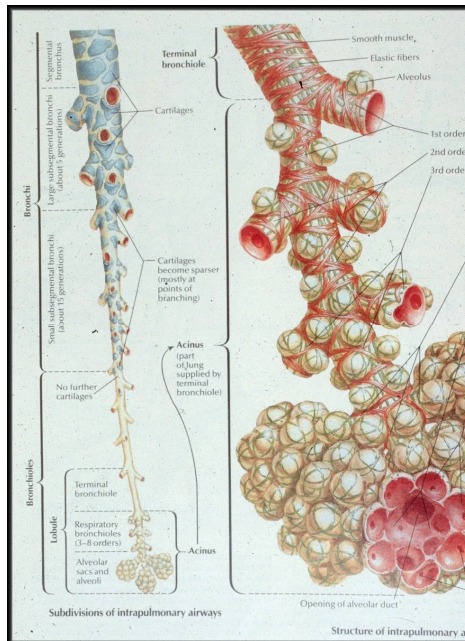




This is a cast of the airways that conduct air to the lungs.

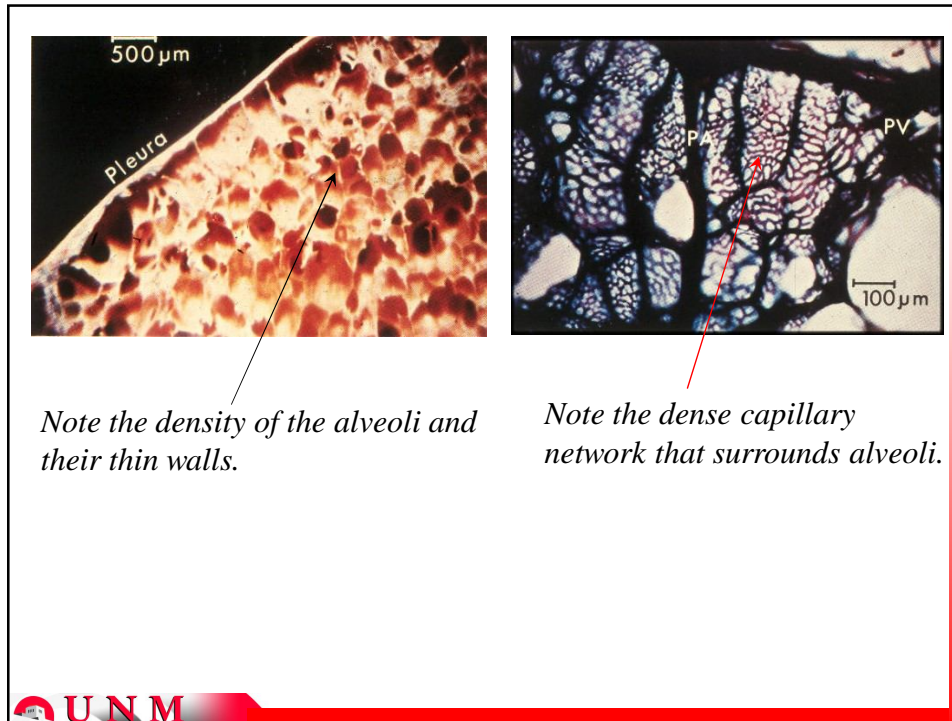
Why is this morphology potentially detrimental to air conductance into and from the lungs?

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Note;
The respiratory zone has the greatest surface area and a dense capillary network.

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Surfactant

A phospholipoprotein molecule, secreted by specialized cells of the lung, that *lines the surface of alveoli and respiratory bronchioles*. Surfactant *lowers the surface tension* of the alveoli membranes, *preventing the collapse* of alveoli during exhalation and *increasing compliance* during inspiration.

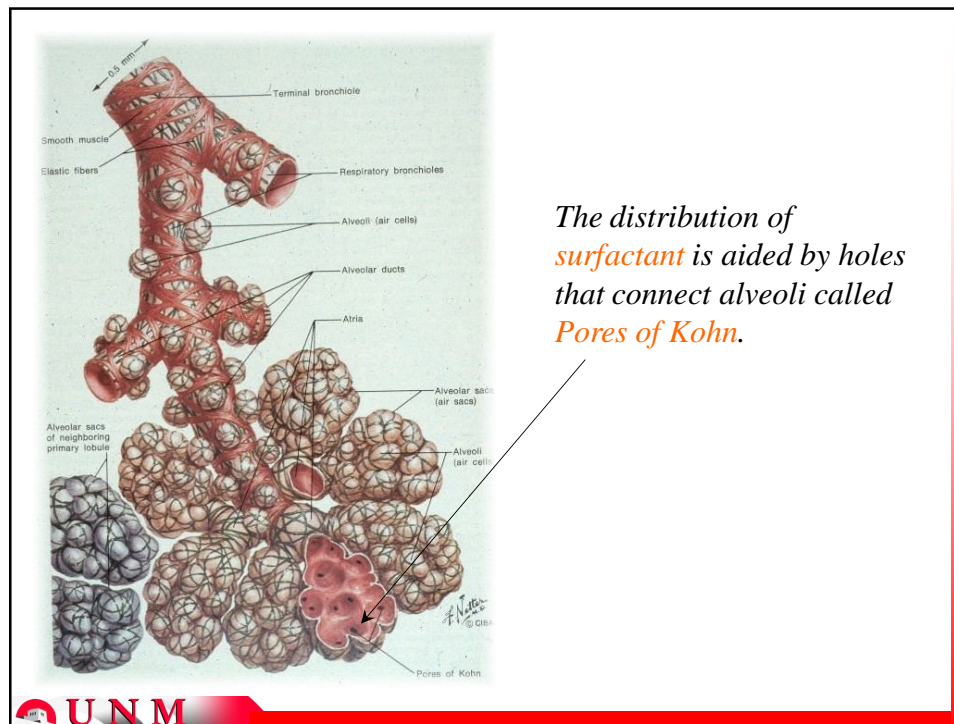
Respiration

The process of gas exchange, which for the human body involves oxygen (O₂) and carbon dioxide (CO₂).

Internal respiration - at the cellular level

External respiration - at the lung

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Ventilation

The movement of air into and from the lung by the process of bulk flow.

$$\text{Ventilation (V}_E\text{) (L/min) = frequency (br/min) x tidal volume (L)}$$

For rest conditions,

$$V_E \text{ (L/min) = } 12 \text{ (br/min) x } 0.5 \text{ (L) = } 6 \text{ L/min}$$

For exercise at VO_2max ,

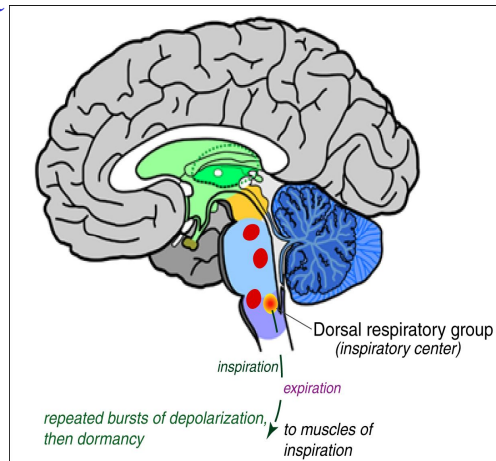
$$V_E \text{ (L/min) = } 60 \text{ (br/min) x } 3.0 \text{ (L) = } 180 \text{ L/min}$$

Compliance - the property of being able to increase size or volume with only small changes in pressure.

Ventilation During Rest

Inspiration is controlled by a repetitive discharge of action potentials from the *inspiratory center*.

Expiration involves the *passive recoil* of the diaphragm.



Minimal regulation from chemoreceptors occurs during normal acid-base conditions.

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Alveolar Ventilation

The volume of “fresh” air that reaches the respiratory zone of the lung.

Alveolar Ventilation (V_A) (L/min)

$$V_A = \text{frequency (br/min)} \times (\text{tidal volume} - 0.15) \text{ (L)}$$

For normal breathing conditions,

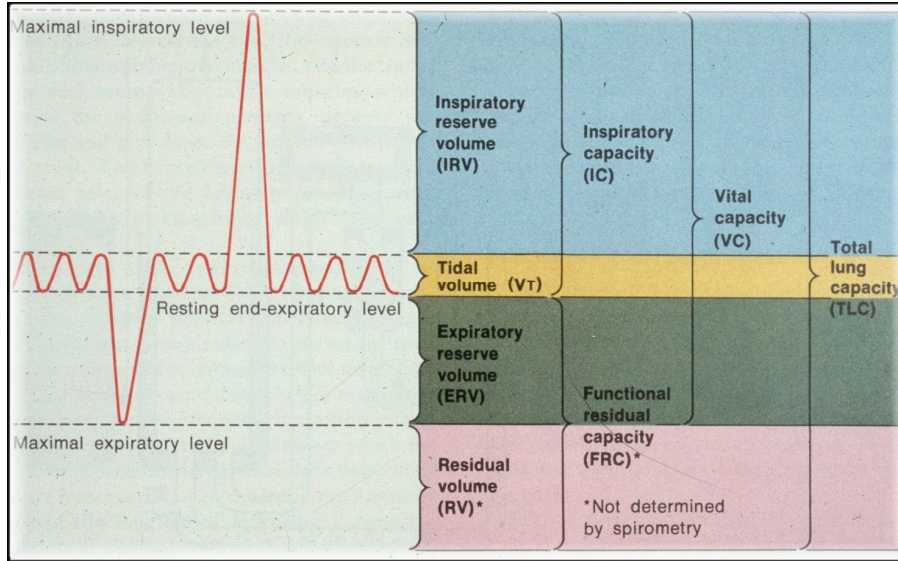
$$\begin{aligned} V_A &= 12 \text{ (br/min)} \times (1.0 - 0.15) \text{ (L)} \\ &= 12 \times 0.85 = 10.2 \text{ L/min} \end{aligned}$$

For rapid shallow breathing conditions,

$$\begin{aligned} V_A &= 60 \text{ (br/min)} \times (0.2 - 0.15) \text{ (L)} \quad (8.2b) \\ &= 60 \times 0.05 = 3.0 \text{ L/min} \end{aligned}$$

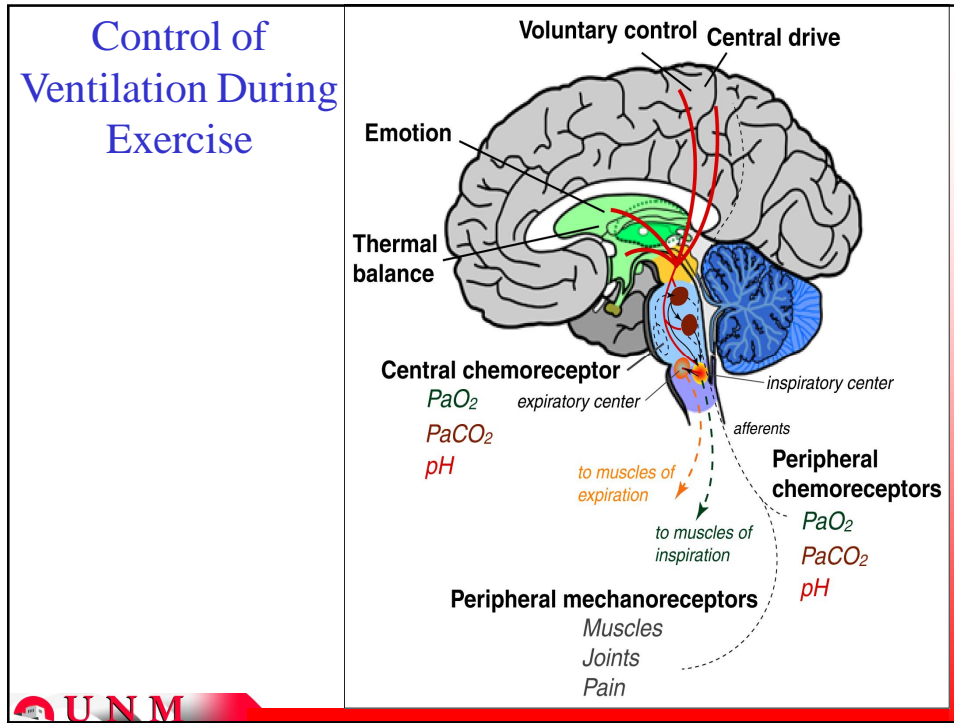
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Lung Volumes and Capacities



Measurement	Abbreviation	Description
Can be measured from spirometry		
Tidal volume	V_T	Volume of air inhaled and exhaled each breath
Inspiratory reserve volume	IRV	Maximum volume of air that can be inhaled after a normal resting end tidal inspiration
Expiratory reserve volume	ERV	Maximum volume of air that can be exhaled after a normal resting end tidal expiration
Inspiratory capacity	IC	Sum of IRV + V_T
Expiratory capacity	EC	Sum of ERV + V_T
Vital capacity	VC	Maximum volume of air exhaled after reaching IC = IC + ERV
Forced vital capacity	FVC	Same as for VC, but with forced rapid exhalation
Forced expiratory volume in 1 s	FEV1	Maximum volume of air that can be expired in 1 s when starting at IC
Maximal voluntary ventilation	MVV	Maximum rate of ventilation that can be attained with voluntary effort
Cannot be measured from spirometry		
Residual volume	RV	Volume of air remaining in the lungs at ERV.
Functional residual capacity	FRC	Sum of RV + ERV
Total lung capacity	TLC	Sum of V_T + IRV + ERV + RV





Diffusion of Gases

The gases of respiration (O_2 and CO_2) diffuse down pressure gradients that exist between,

- a. pulmonary blood and the alveoli
- b. systemic capillary blood and cells

Gas	Air Fraction	Sea Level $P_B=760$ mmHg $P_{B-47}=713 * 0.9906 = 706.3$		5,280 ft (1,610 m) $P_B=620$ mmHg $P_{B-47}=573 * 0.9906 = 567.6$	
		Alveolar Fraction	$P_{A gas}$ (mmHg)	$P_{A gas}$ (mmHg)	$P_{A gas}$ (mmHg)
Nitrogen	0.78084	0.7868	561	447	
Oxygen	0.209476	0.1472	104	84	
Carbon Dioxide	0.000314	0.0566	40	32	

The data of alveolar partial pressures at any barometric pressure (altitude) can be calculated from memorizing the bold values.

The factors that govern the directionality and magnitude of gas diffusion are?

What Determines Gas Exchange?

The factors that govern the directionality and magnitude of gas diffusion are,

- the gas diffusion capacity
- **the gas partial pressure gradient**
- characteristics of the medium through which diffusion occurs (hydration, thickness, cross sectional area)

The fact that alveolar and blood gas partial pressures approximately reach equilibrium within the lung enables researchers to estimate arterial blood gas partial pressures from alveolar partial pressures!



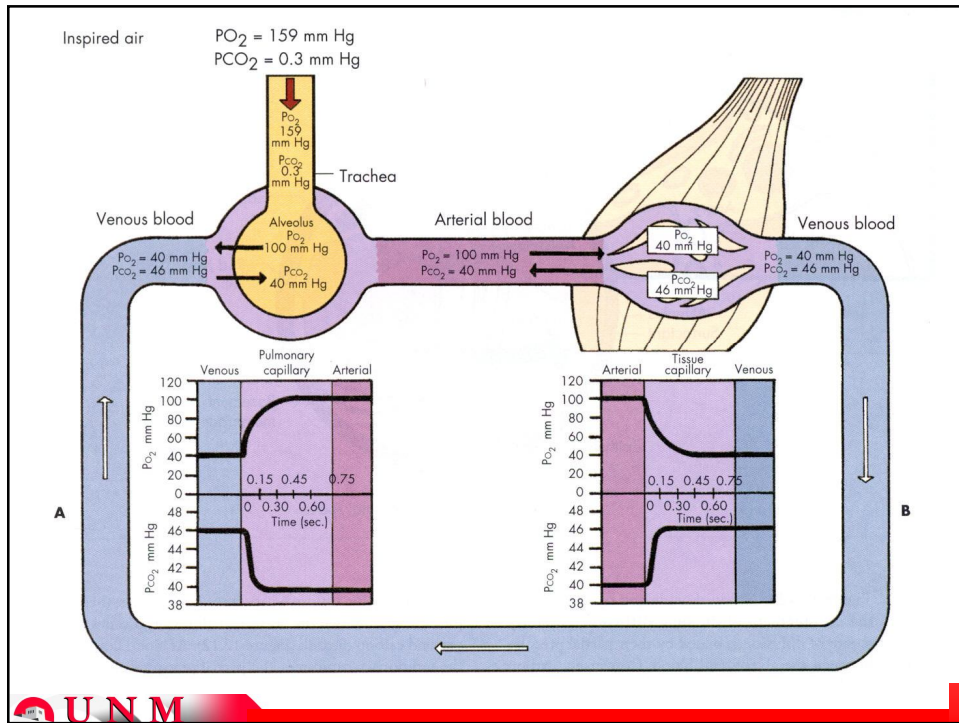
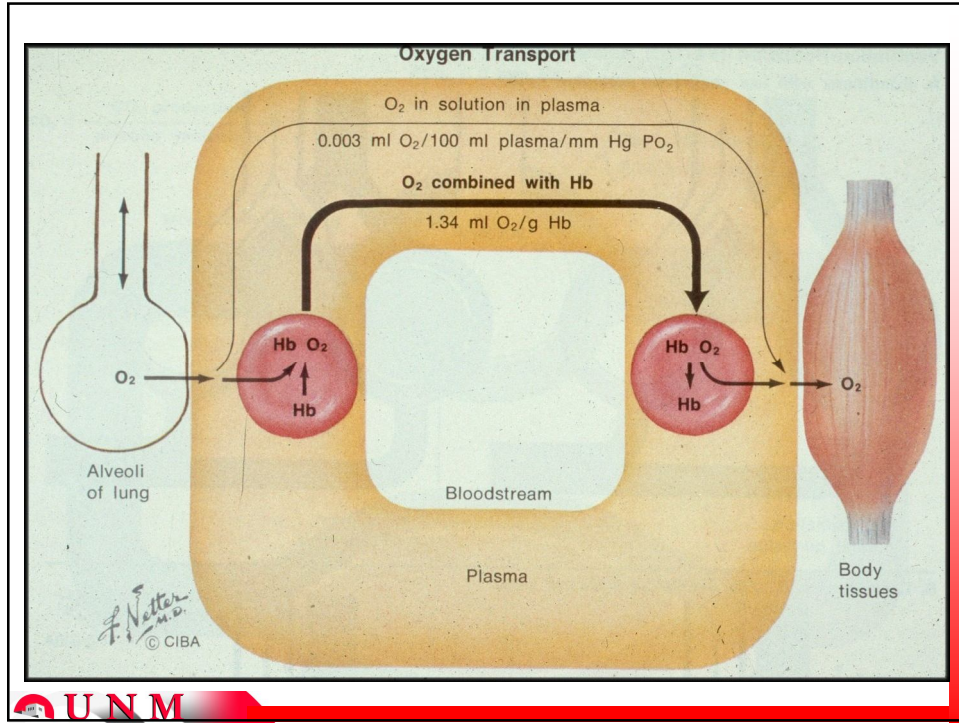
Gas Partial Pressures in Atmospheric and Alveolar Air

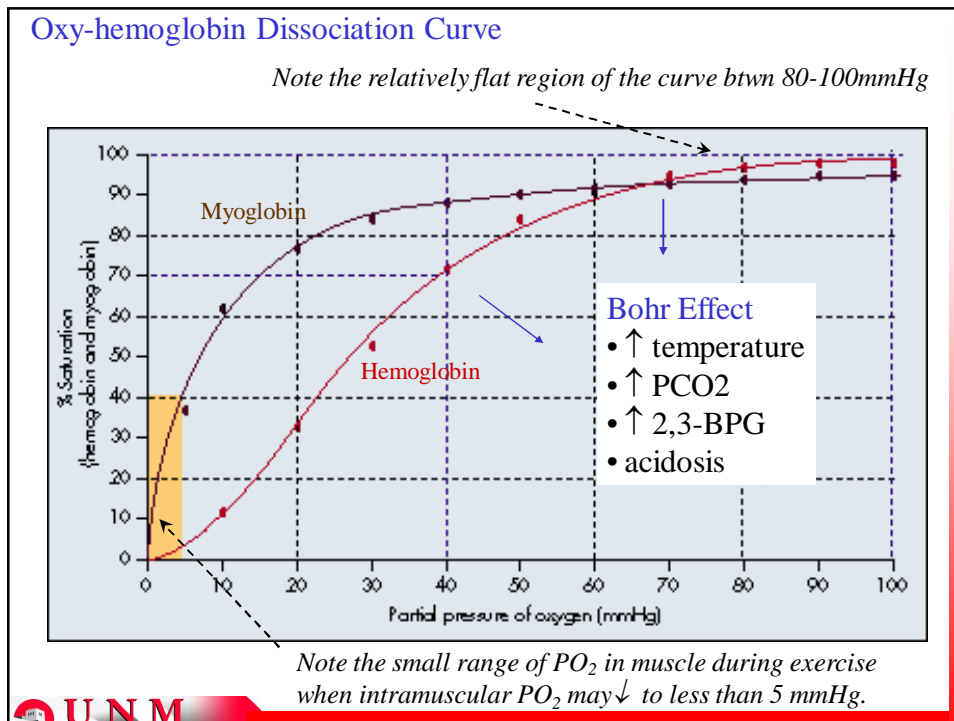
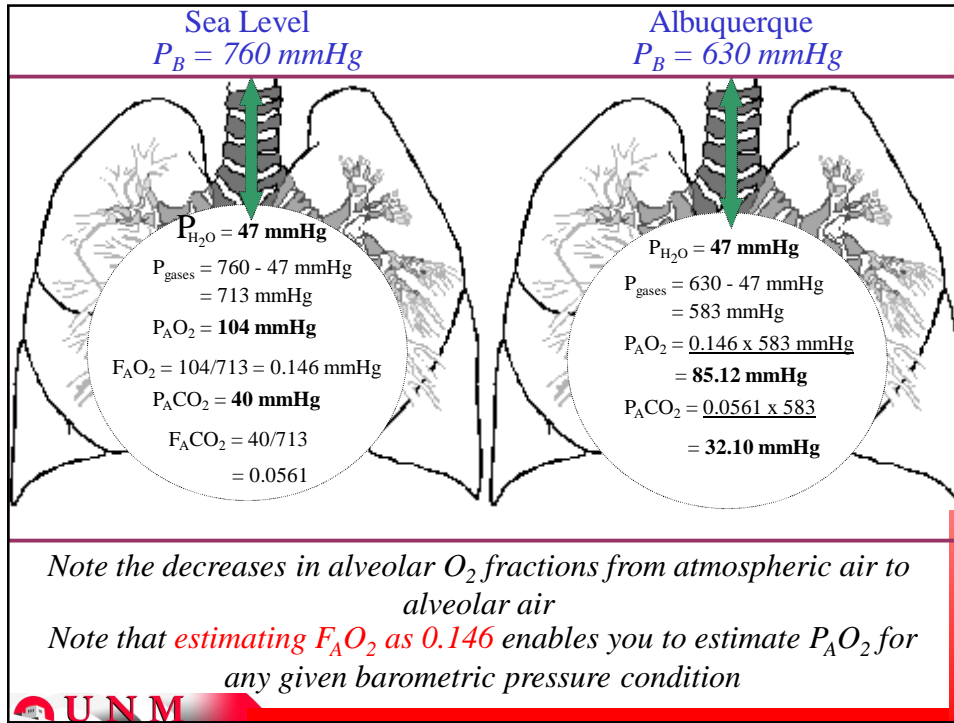
Gas	Air* Fraction	Air* Partial Pressure	Alveolar Fraction [^]	Alveolar Partial Pressure
H₂O	0	0	----	47
O₂	0.2095	159.0	0.1459	104
CO₂	0.0003	0.3	0.0561	40
N₂	0.7808	600.6	0.7980	569

* assumes dry air at sea level, $P_B=760$ mmHg

[^] note that the water vapor pressure is removed to calculate alveolar gas fractions







Exchange of gas at the cellular level

(i.e., systemic capillary blood and muscle cells)

In addition to the Bohr and Haldane effects, unloading of oxygen is also aided by the molecule **myoglobin**.

Myoglobin is found within skeletal muscle fibers and is similar to hemoglobin in that it contains a *heme prosthetic group* that can *bind oxygen*.

When PaO₂ drops **below 60 mmHg**, myoglobin has a higher affinity for oxygen than does hemoglobin.

This allows for a unidirectional transfer of O₂ from **hemoglobin (blood) to the myoglobin (muscle fiber)**.

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Transport of Oxygen in the Blood

Oxygen is transported in blood bound to **hemoglobin** (Hb). 1 gram of Hb can maximally bind 1.34 mL of oxygen (1.34 mL O₂/g Hb @ 100% saturation).

Table 8.1: Examples of hemoglobin (Hb) and oxygen carrying capacity conditions (98% saturation and pH = 7.4)

<i>Population/Condition</i>	<i>[Hb]</i>	<i>mL O₂/L</i>
<i>Males</i>	14.0	183.8
<i>Females</i>	12.0	157.6
<i>Blood Doping</i>	18.0	236.4
<i>Anemia</i>	< 10.0	< 131.3

[Hb] = g/100 mL

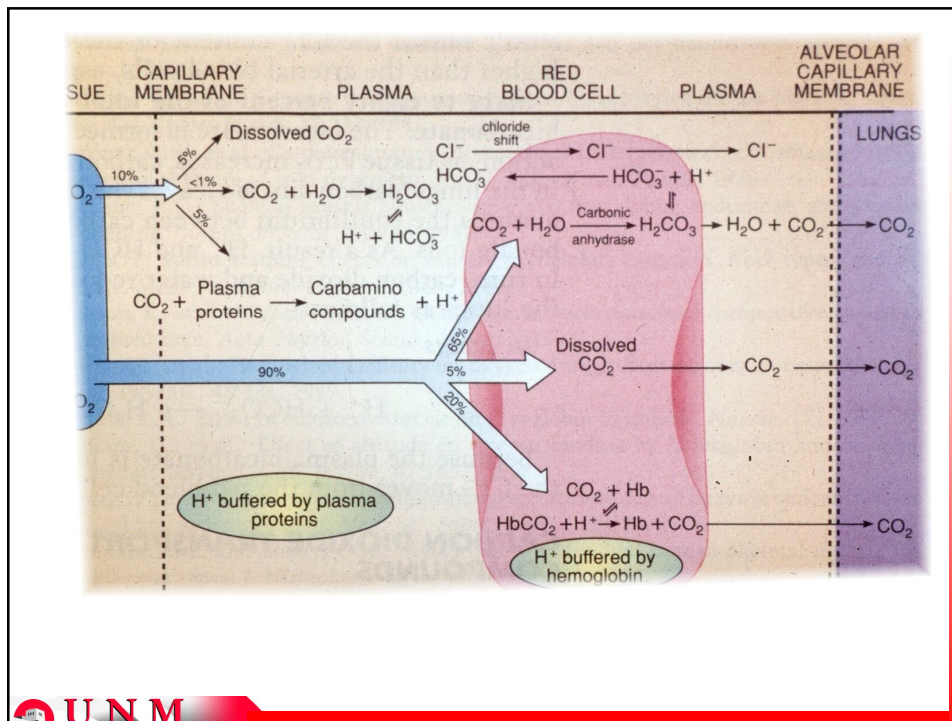
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The oxygen content (CaO₂) of blood can be calculated;

$$\begin{aligned} \text{CaO}_2 &= [\text{Hb}] \times \text{O}_2/\text{g Hb} \times \text{Hb-O}_2 \text{ saturation} \\ &= 150 \text{ g/L} \times 1.34 \text{ mL O}_2/\text{g} \times 0.98 \\ &= 197 \text{ mL O}_2/\text{L} \end{aligned}$$

Another small source of oxygen in blood is the volume of *oxygen dissolved in plasma*. However, due to the low solubility of oxygen, this value is small and approximates,

$$\begin{aligned} \text{dissolved O}_2 &= 0.003 \text{ mL} / 100 \text{ mL blood} / \text{mmHg PO}_2 \\ &\sim 0.3 \text{ mL} / 100 \text{ mL at sea level (PaO}_2 \sim 100 \text{ mmHg)} \end{aligned}$$



Carbonic anhydrase

$$\text{CO}_2 + \text{H}_2\text{O} \rightarrow \text{H}_2\text{CO}_3 \rightarrow \text{H}^+ + \text{HCO}_3^-$$

Carbonic acid Bicarbonate

Buffering


When acid is produced from metabolism, the liberated proton can bind with bicarbonate, eventually forming CO₂, which is then expired by the lung.

Carbonic anhydrase

$$\text{CO}_2 + \text{H}_2\text{O} \leftarrow \text{H}_2\text{CO}_3 \leftarrow \text{H}^+ + \text{HCO}_3^-$$

Carbonic acid Bicarbonate

The bicarbonate-carbon dioxide system relies on ventilation for proper function as a buffer system.




Carbonic anhydrase

$$\uparrow\text{CO}_2 + \text{H}_2\text{O} \leftarrow \text{H}_2\text{CO}_3 \leftarrow \text{H}^+ + \text{HCO}_3^-$$

Carbonic acid Bicarbonate

It is this increased production of carbon dioxide from the bicarbonate buffering of acid that accounts for the increase in **RER above 1.0** during intense exercise.

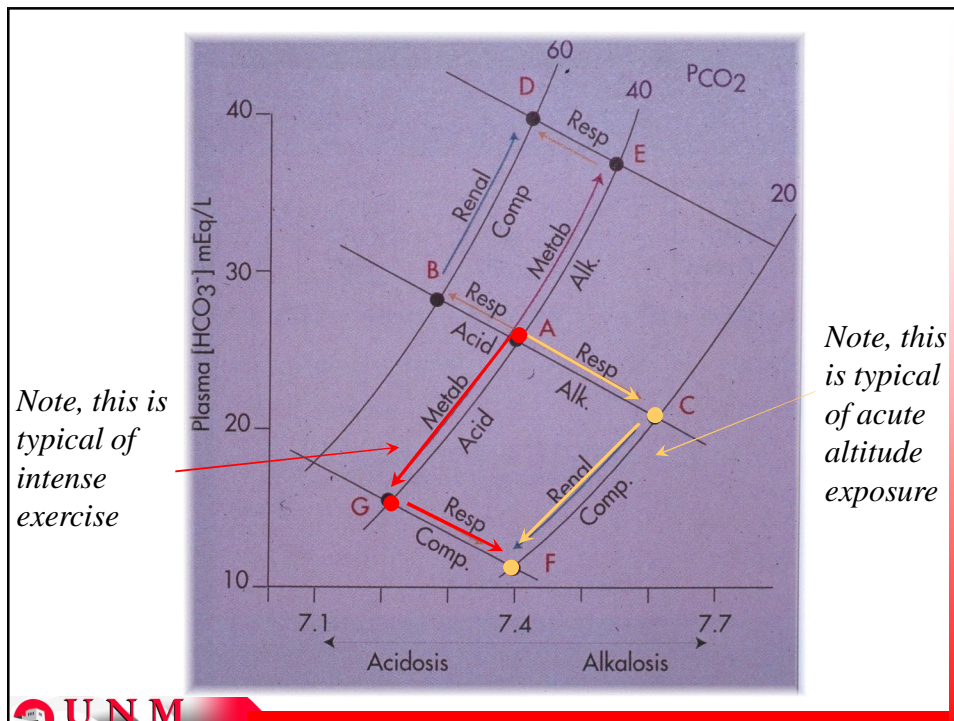
Remember, the respiratory exchange ratio (RER) is calculated by;

$$\text{VCO}_2 / \text{VO}_2$$


Transport of Carbon Dioxide in the Blood

The volume of CO₂ in the blood is approximately 10-fold greater than O₂.

Transport Location	Form	Percentage
Plasma (<i><10%</i>)	Dissolved	5
	$\text{CO}_2 + \text{H}_2\text{O} \rightarrow \text{H}_2\text{CO}_3 \rightarrow \text{H}^+ + \text{HCO}_3^-$	<1
	Bound to proteins	5
Red Blood Cell (<i>90%</i>)	$\text{CO}_2 + \text{H}_2\text{O} \rightarrow \text{H}_2\text{CO}_3 \rightarrow \text{H}^+ + \text{HCO}_3^-$	65
	Dissolved	5
	Bound to hemoglobin	20



Acidosis

Quantified by the pH scale, where pH equals the negative logarithm of the hydrogen ion concentration ($[H^+]$)

$$pH = -\log [H^+] \quad \text{or} \quad [H^+] = 10^{-pH}$$

Normal blood pH is $\sim 7.4 = [H^+] = \underline{0.00000004 \text{ M}}$

The main determinants of blood pH are;

- Balance of proton release/consumption
- Buffer capacity
- $PaCO_2$
- Renal excretion of electrolytes, protons and bases
- Ventilation
- Concentration of HCO_3^- and other bases or acids

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Acute Adaptations of Pulmonary Function During Exercise

After the onset of exercise there is;

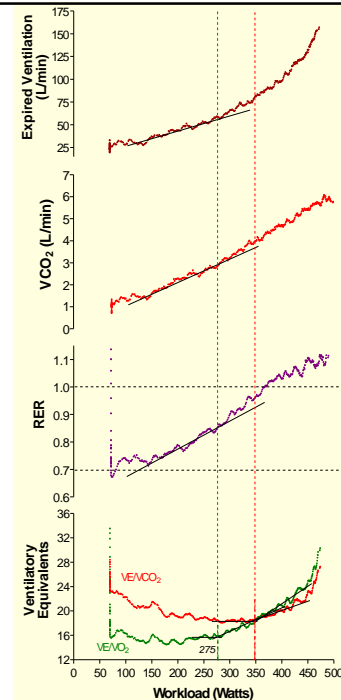
- ▶ a rapid \uparrow in ventilation
(proportional to intensity)
- ▶ a similar rapid \uparrow in pulmonary blood flow
- ▶ an improved V_E vs Q relationship in the lung
(both ventilation & perfusion become more evenly distributed)
- ▶ \uparrow lung compliance
- ▶ airway dilation and \downarrow resistance to air flow
(enlargement of trachea, dilation of bronchi & bronchioles)

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Ventilatory Threshold

Exercise intensity at which there is a deviation from linearity in *ventilation* and an increase in VE/VO_2 .

The abrupt increase in acidosis and subsequent increase in $PaCO_2$, disproportionately increases ventilation above that of oxygen use.



Mechanics of Ventilation

As ventilation increases, **both tidal volume and breathing frequency increase**, with an eventual plateau seen in tidal volume.

As ventilation and intensity increase;

- ↑ air remaining in lung after expiration
- ↑ inspiratory pleural pressure
- ↑ work of breathing

Exercise-Induced Hypoxemia

A lowering of partial pressure of oxygen (PaO_2) (**hypoxia**) resulting in a reduced CaO_2 (**hypoxemia**) during exhausting exercise in *highly endurance-trained individuals*, even at sea level.

- decreased pulmonary transit time
- decreased / uneven diffusion capacities in lung
- venoarterial shunts
- ventilation-perfusion inequalities
- inadequate hyperventilation

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