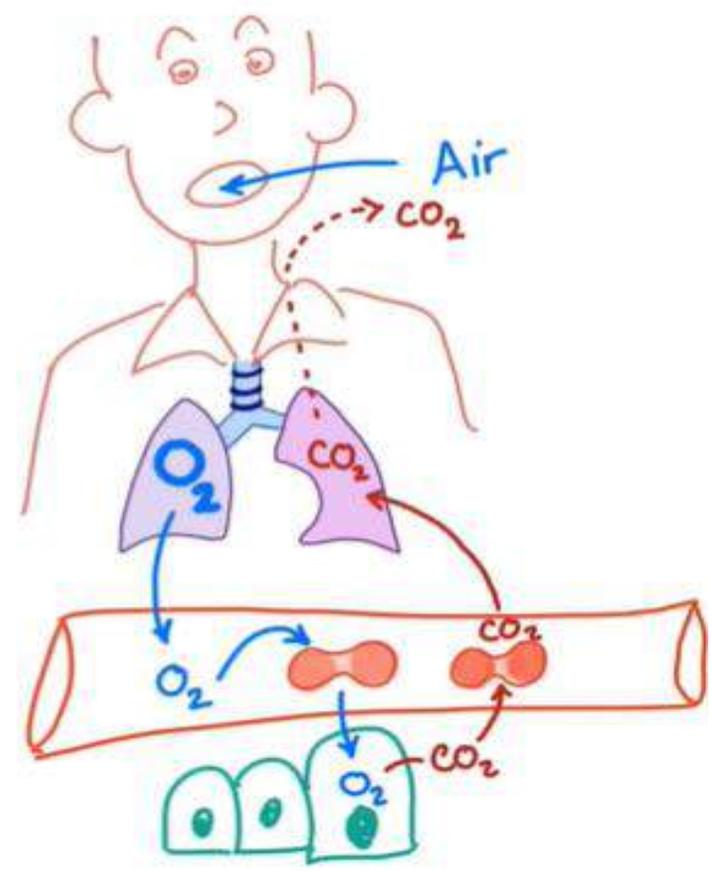
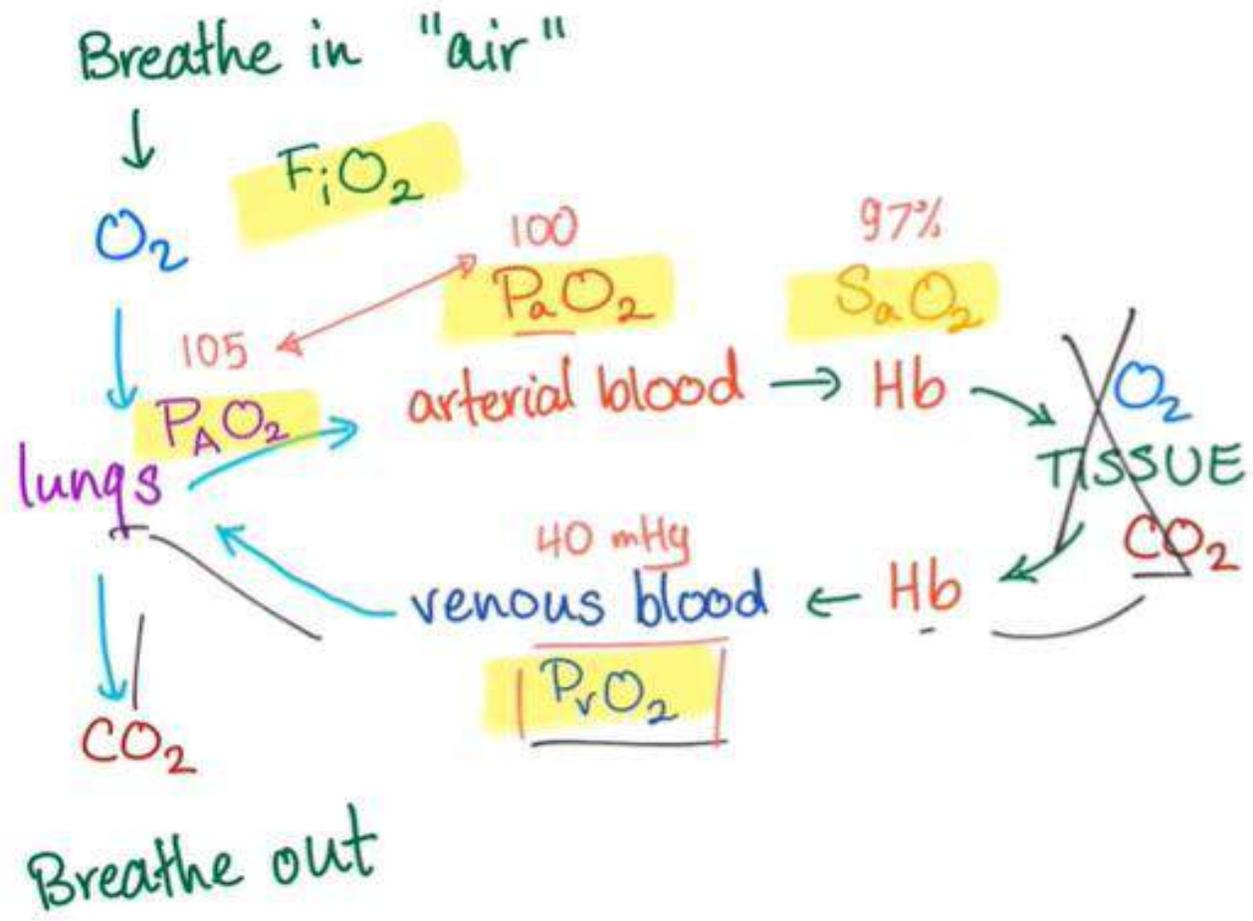


# Hypoxia

Dr. Arwa Rawashdeh



- Ventilation (V): getting air into the alveoli.
- Perfusion (Q): blood flow to the lung.

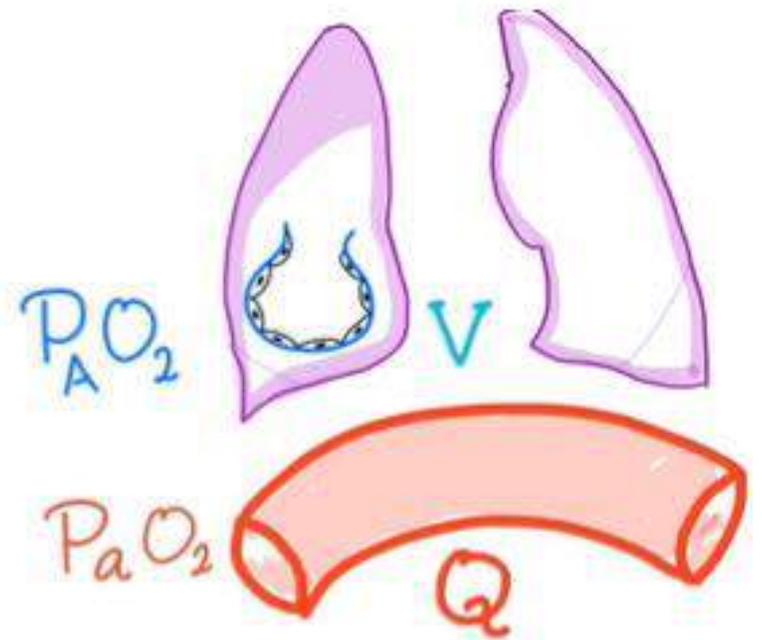
## OXYGEN

In the air:  $F_i O_2 = 21\%$

partial pressure or  $P_i O_2 = 150 \text{ mmHg}$

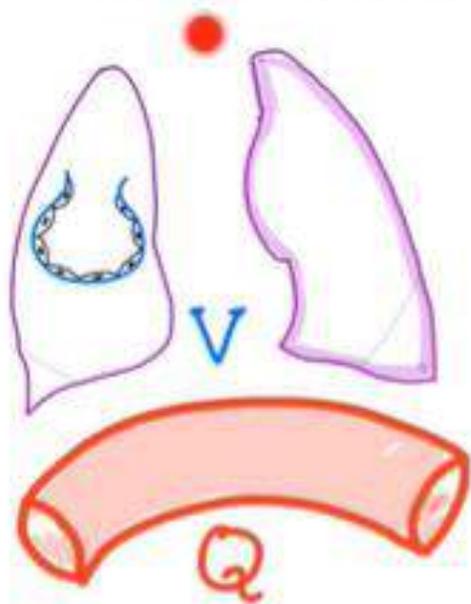
In the alveoli:  $P_A O_2 = 105 \text{ mmHg}$

In the arterial blood:  $P_a O_2 = 100 \text{ mmHg}$

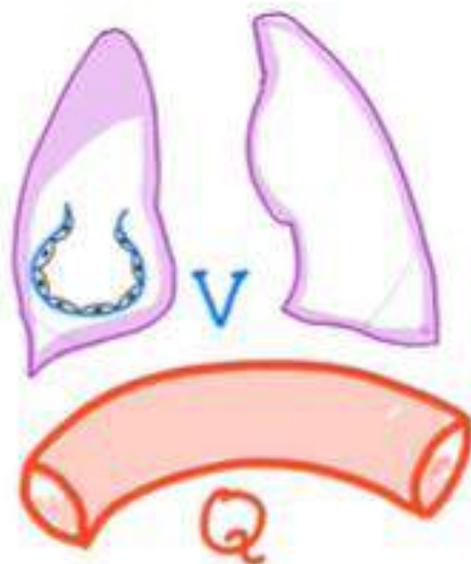


} A-a gradient

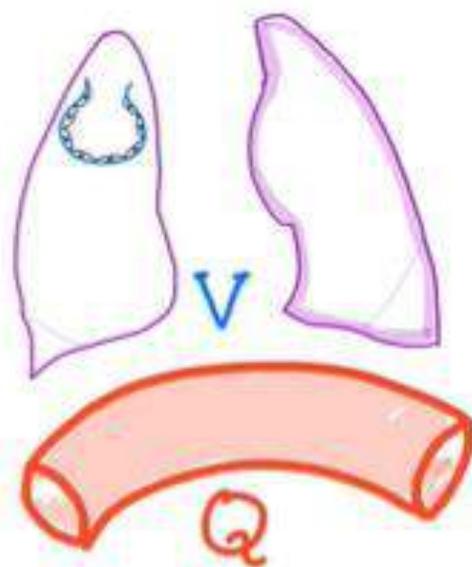
Normal "overall"



Normal base



Normal apex



$\frac{V}{Q}$  ratio 80%

$\uparrow \frac{V}{Q} < 80\%$   
 $\uparrow \uparrow Q$  "0.6"

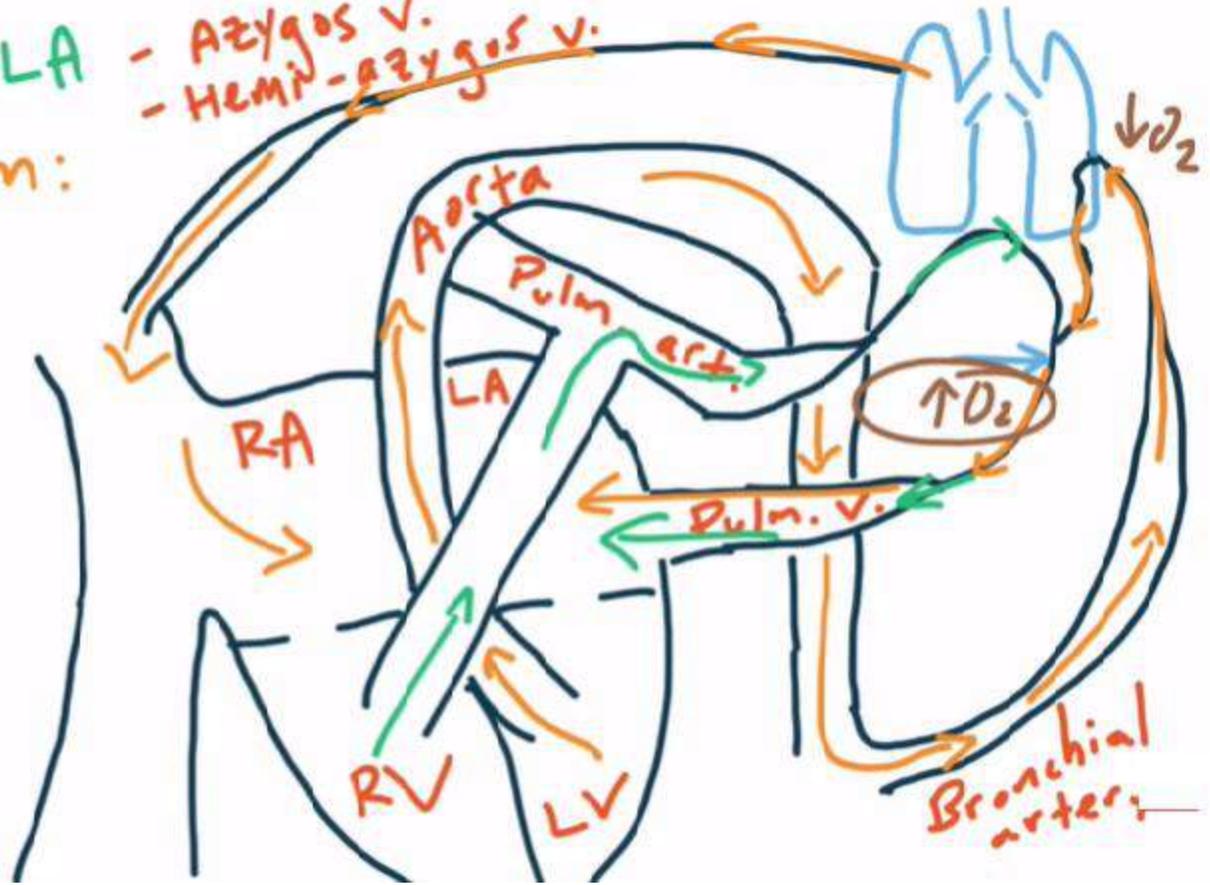
$\downarrow \frac{V}{Q} > 80\%$   
 $\downarrow Q$  "3"

Pulm: RV → LA

Bronchopulm:

LV → LA  
→ RA

- Azygos v.
- Hemi-azygos v.



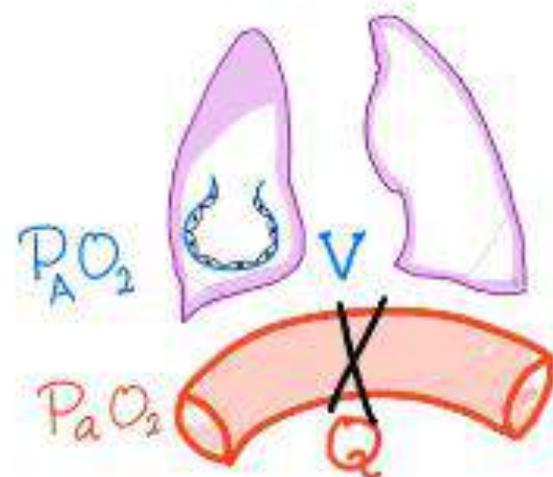
Normal



Ventilation defect



Perfusion defect



$$\frac{V}{Q} \text{ ratio} = \frac{4\text{L/min}}{5\text{L/min}} = 0.8 = 80\%$$

$$\Downarrow \frac{V}{Q} < 80\%$$

e.g. zero

$$\Downarrow \frac{V}{Q} > 80\%$$

e.g.  $\infty$

Ideally  $\frac{V}{Q} = 100\% = 1$   
 $V = Q$

	Normal	Decreased ventilation (e.g. Obstrutive lung disease) 	Decreased perfusion (e.g. pulmonary artery embolus) 
V/Q ratio	0.8	decrease "<0.8"	increase ">0.8"
PAO2	105 (alveolar air is like the arterial blood)	40 mmHg (alveolar air is like the venous blood) because no gas exchange happened, blood came and blood went "shunting"	152 mmHg (alveolar air became similar to atmospheric air) <b>Dead space</b>
PaO2	100	40 mmHg [Hypoxemia]	<100 mmHg (because there is no gas exchange) [Hypoxemia]
PvO2	40 mmHg	40 mmHg	40 mmHg
PACO2	40 (alveolar air is like the arterial blood)	46 mmHg	0 (alveolar air became similar to atmospheric air)
PaCO2	40	40	<40 (due to hyperventilation)
PvCO2	46	46	46
		In response: <ul style="list-style-type: none"> <li>• hypoxia → vasoconstriction → force the blood to the well ventilated areas.</li> <li>• i.e. when the bronchus is closed → close the vessel.</li> </ul>	In response: <ul style="list-style-type: none"> <li>• hypocapnia → alkalosis → vasoconstriction → to keep the CO2 in → acidosis (to try to compensate the alkalosis)</li> <li>• i.e. when the vessel is closed → close bronchus.</li> </ul>

#### Patient A "normal"

- RR = 12/min, TV = 500 mL
  - Pulmonary ventilation = respiratory rate x tidal volume =  $12 \times 500 = 6$  liters.
  - Alveolar ventilation = respiratory rate x (tidal volume - dead space) =  $12 \times (500 - 150) = 12 \times 350 = 4.2$  liters.

#### Patient B "increased respiratory rate"

- RR = 30/min, TV = 200 mL
  - Pulmonary ventilation = respiratory rate x tidal volume =  $30 \times 200 = 6$  liters.
  - Alveolar ventilation = respiratory rate x (tidal volume - dead space) =  $30 \times (200 - 150) = 1.5$  liters.

#### Patient C "increased tidal volume"

- RR = 6/min, TV=1,000 mL
  - Pulmonary ventilation = respiratory rate x tidal volume =  $6 \times 1000 = 6$  liters.
  - Alveolar ventilation = respiratory rate x (tidal volume - dead space) =  $6 \times (1000 - 150) = 5.1$  liters.

Therefore, increasing the tidal volume is a better way to achieve more alveolar ventilation than increasing the respiratory rate.

- But, everything is good within limits.
  - if you increase the tidal volume too much → the alveoli will expand tremendously (inspiration) and then collapse (expiration)...This big difference, repeated over and over again, can lead to INFLAMMATION!

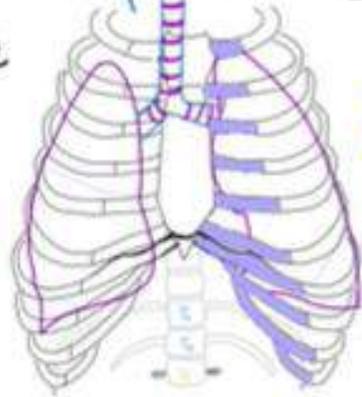
Cause of hypoxia	Example	A-a gradient	Effect of oxygen therapy (100% oxygen)
Atmospheric	High altitude	Normal	Excellent; Oxygen can COMPLETELY cure it. (100% effective therapy)
Hypoventilation	Decreased respiratory rate (e.g. Obstructive)	Normal	Very good; Oxygen therapy will help with the hypoxia (but won't cure the hypercapnia)
Diffusion defect	Pulmonary fibrosis, pulmonary edema	High	Very good: increase in PAO <sub>2</sub> → increase in PaO <sub>2</sub>
Hemoglobin defect	Anemia, methemoglobinemia	Normal	Ok: oxygen therapy will NOT increase the oxygen saturation or the hemoglobin saturation, but it can increase the dissolved oxygen (PaO <sub>2</sub> ) a little which can be the difference between life and death.
Ischemia	Thrombus, embolus, tissue edema, bad vessels in a peripheral organ (not lung).	Normal	Ok: oxygen therapy will NOT increase the oxygen saturation or the hemoglobin saturation, it won't remove the clot, but it can increase the dissolved oxygen (PaO <sub>2</sub> ) a little
Shunt	intrapulmonary shunt, or intracardiac (Rt-to-Lt) shunt	High	No effect.

Patient (A)

Spirometer



Chest wall disease  
Extrinsic Restrictive  
lung disease  
(extrathoracic)



S.O.B

A-a



ABG

$\downarrow PaO_2 \rightarrow \downarrow SaO_2$

$\downarrow$  lung volumes & capacities

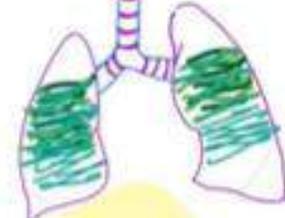
$\downarrow FEV_1, \downarrow FVC, \uparrow FEV_1 / FVC$  ratio  
or nl

Patient (B)

Spirometer



Pulmonary fibrosis  
Intrinsic restrictive lung  
disease  
(parenchymal)



Oximeter

$\downarrow PaO_2 \rightarrow \downarrow SaO_2$

$\downarrow$  lung volumes & capacities

$\downarrow FEV_1, \downarrow FVC, \uparrow FEV_1 / FVC$  ratio  
or nl

Spirometer

## FEV1/FVC ratio

- FEV1: the amount of air that you can quickly and forcibly exhale in 1 second after maximal inhalation (timed).
  - FVC: the amount of air that you can quickly and forcibly exhale after maximal inhalation (timed).
  - FEV1/FVC ratio: FEV1 divided by FVC
    - Normally, it's 80% (4L/5L).
-

In brief:

- low FEV1/FVC ratio → obstructive lung disease
- low TLC → restrictive lung disease.
- high FEV1/FVC ratio → restrictive lung disease
- low FEV1/FVC ratio and low TLC → mixed lung disease (obstructive + restrictive)