

# Ask Weber session 5

## 29-04-2021

**Weber Liu**

**Aerobic function**

Circulatory system

Respiratory system





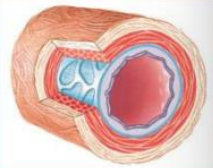




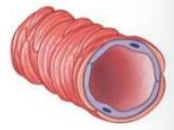














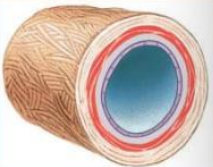


# **L1: Human circulatory system**

- 1. In the lecture, Dr. Cameron notes that the human has a 'double circulatory system' – what are the names of the two circulatory systems? And what are their functions?**

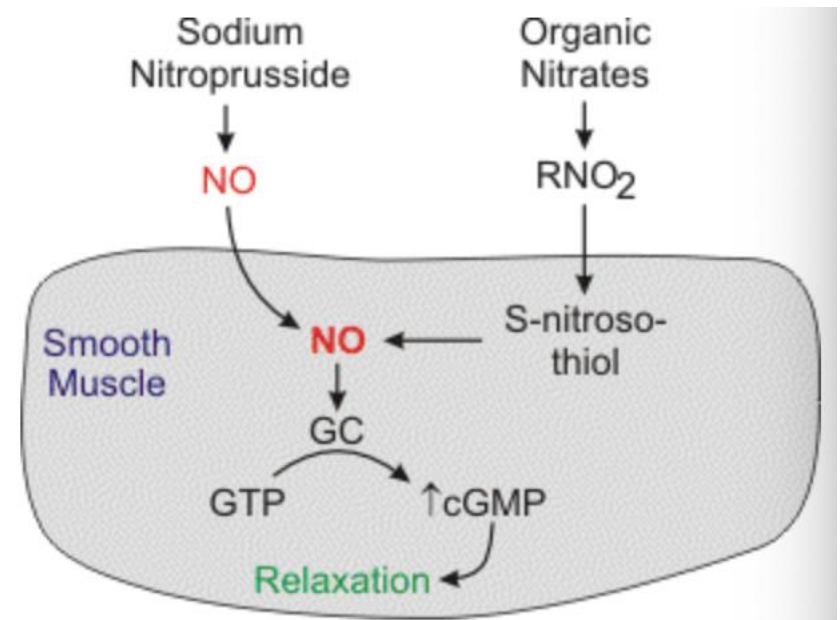
# L1: Vessels

1. Which artery is most likely responsible for the majority of systemic vascular resistance?

	Diameter	Mean wall thickness	Endothelium	Elastic tissue	Smooth muscle	Fibrous tissue	
Artery	0.1–10+ mm	1.0 mm					
Arteriole	10–100 $\mu\text{m}$	6.0 $\mu\text{m}$					
Capillary	4–10 $\mu\text{m}$	0.5 $\mu\text{m}$					
Venule	10–100 $\mu\text{m}$	1.0 $\mu\text{m}$					
Vein	0.1–100+ mm	0.5 mm					

## L1: Vessels

**Q: When individuals with unstable angina (i.e. chest pain due to atherosclerosis of their vessels) experience a bout of chest pain, one of the drugs given to them in the ED is GTN (glyceryl trinitrate). The reason the chest pain is experienced is because a narrowing coronary vasculature results in myocardial ischemia (thus pain). What do you think GTN does?**



<https://cvpharmacology.com/vasodilator/nitro>

## **L1: Vessels**

**Q: Individuals with Raynaud's phenomena experience a cyanosis of their fingertips due to local peripheral vasoconstriction. One of the treatments is a dihydropyridine calcium channel blocker (E.g. nifedipine). How do you think CCBs work?**

# L1: Blood

**Q: When rugby players play 'away' games in higher altitudes, they tend to perform poorly compared to the players who have already trained there.**

**However when these away players also train there for a few weeks/months, they perform better as well. Why is this?**

- By playing in a higher altitude, there is a lower oxygen partial pressure and so they don't have the required Hb to carry O<sub>2</sub> to the muscles. Players who already reside there have adapted through the production of EPO (kidneys can sense O<sub>2</sub> concentrations and produce hormonal EPO) which results in increased Hb production

## Erythropoiesis

- Controlled by the glycoprotein **erythropoietin (EPO)** + some cytokines
  - Produced by the kidneys

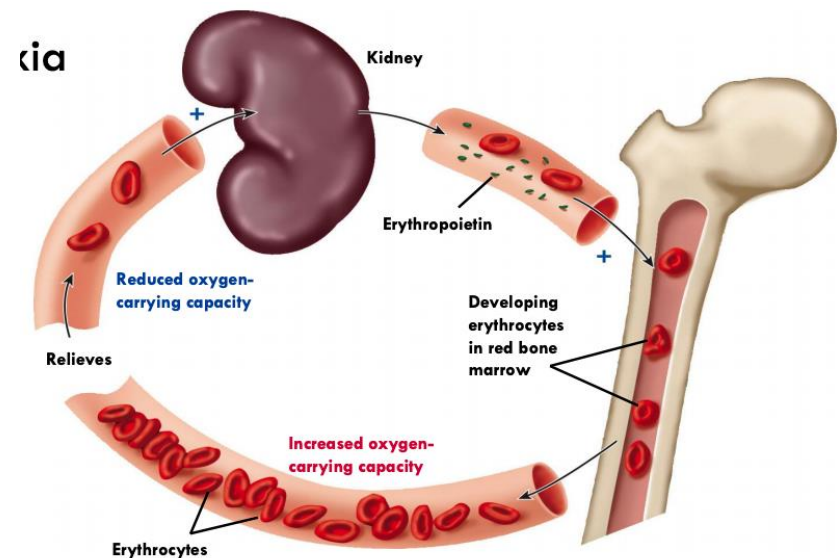
# L1: Blood

**Q: What is the function of EPO?**

**A: EPO is produced in response to hypoxia – it tells the bone marrow to increase erythropoiesis (i.e. production of RBC)**

**Q: What is the difference between hypoxia and hypoxaemia?**

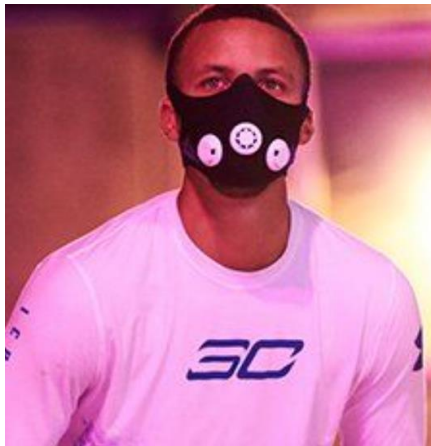
**A: hypoxia is low O<sub>2</sub> in tissues, hypoxaemia is low O<sub>2</sub> in the blood**



## **L1: Blood**

**Q1: Steph Curry was in an ad once where he was wearing an O<sub>2</sub> restriction mask. The idea was to wear this while training.**

- 1. What do you think the purpose of this mask was?**
- 2. Why do you think this mask would/would not work?**



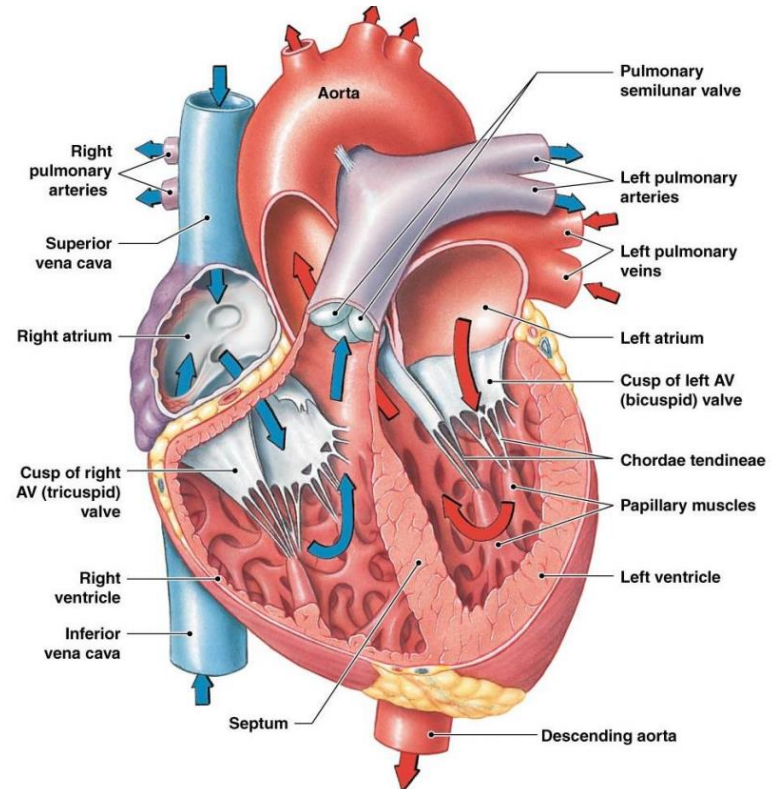
**Q2: Lance Armstrong was a record breaking cyclist who was caught blood doping.**

- 1. What are 3 ways in which he could have blood doped**
- 2. What is the overall reason for blood doping?**



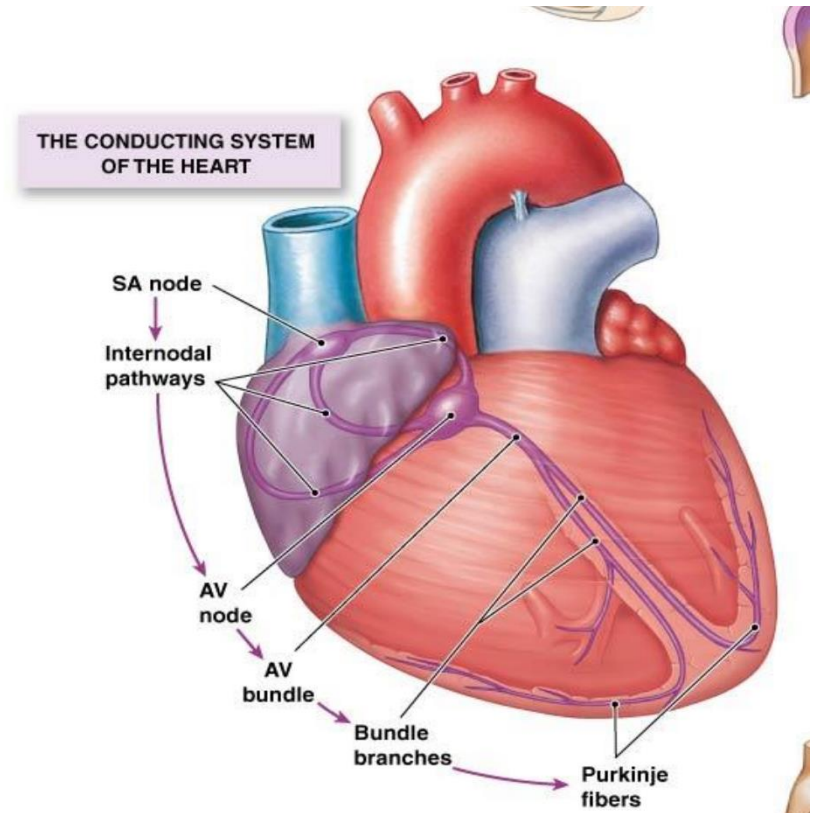
## L2: Anatomy of the heart

1. Name the two vascular structures which bring blood back to the heart from systemic circulation
2. Name the structure which brings blood back to the heart from pulmonary circulation
3. Name the main structure which carries blood out into systemic circulation
4. Name the main structure that carries blood into pulmonary circulation



## L2: The heart

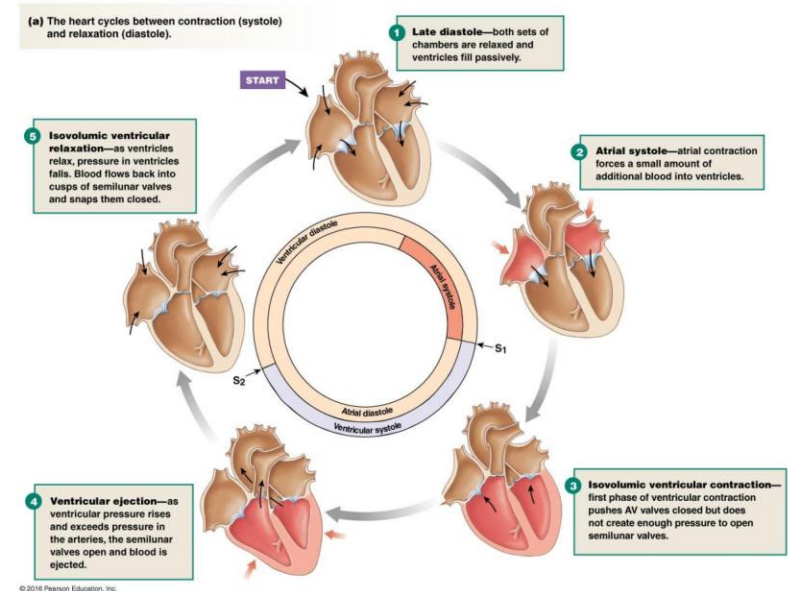
1. In order, name the conducting systems an electrical depolarization has to pass through in the heart
  - SA node, AV node, Bundle of His, Purkinje fibres of myocardium
2. Does the left or right atrium contract first in the heart?
  - Right atrium contracts first – think about the fact that the SA node is in the RA



## L2: The heart

### 1. What are the stages of mechanical heart contraction?

1. Late diastole – passive filling of the atrium and ventricles
2. Atrial systole – ‘atrial kick’, whereby atrium contraction fills up the last ~20% of the ventricle
3. Isovolumic ventricular contraction – ventricles contract, generate a high pressure, **lock shut the AV valves**, but the pressure isn’t high enough to stent open the aortic/pulmonary valves yet
4. Ventricular ejection – pressures generated can now stent open the pulmonary/aortic valves, blood leaves ventricles
5. Isovolumic ventricular relaxation – pressures drop, the afterload **locks shut the pulmonary valves**, but pressures aren’t low enough to open the AV valves yet

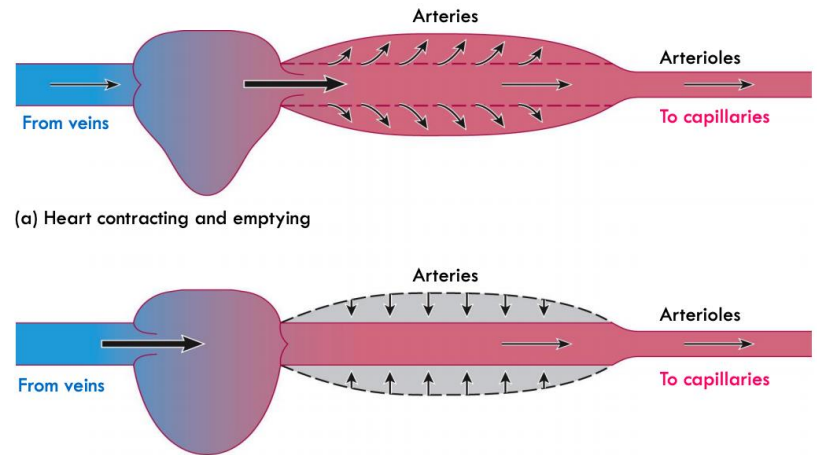


## L2: Physics of cardiology

- **What is the formula for cardiac output?**
  - $CO = SV \times HR$
- **What is approximate stroke volume of the heart?**
  - $\sim 70\text{mL/beat}$
- **What is the normal physiological range of resting human heart rate?**
  - $\sim 60\text{-}80\text{bpm}$
- **What is the expected cardiac output?**
  - $\sim 5.4\text{L/min}$  (if  $70\text{mL/beat}$  and  $72\text{bpm}$ )
- **What is the effect of the sympathetic nervous system on heart rate?**
  - Increases HR
- **You will eventually come across a variation of Ohm's law to calculate mean arterial pressure:**
  - $MAP = CO \times TPR$
- **You can put these formulas together to make estimates on TPR, etc. as well as interpret clinical findings with regards to deranged physiologies**

## L3: Blood flow

1. When blood is pushed out of the heart (through a ventricular contraction), there is obviously blood flowing **OUTWARDS**, however the reason that the aortic valves close is because there is a **backwards force** of the blood back onto the valves (otherwise they wouldn't close!). That would imply the retrograde movement of blood, which would also imply that blood can remain static. How then, does the body maintain unidirectional **AND** continuous blood flow in the body?
  - Windkessel effect



## **L3: Vascular physiology**

**Q: Last Friday, after I donated blood, I realized that while standing up, I would feel a bit faint (or my vision would get a bit blurry/dark). What do you think is the reason for this phenomena?**

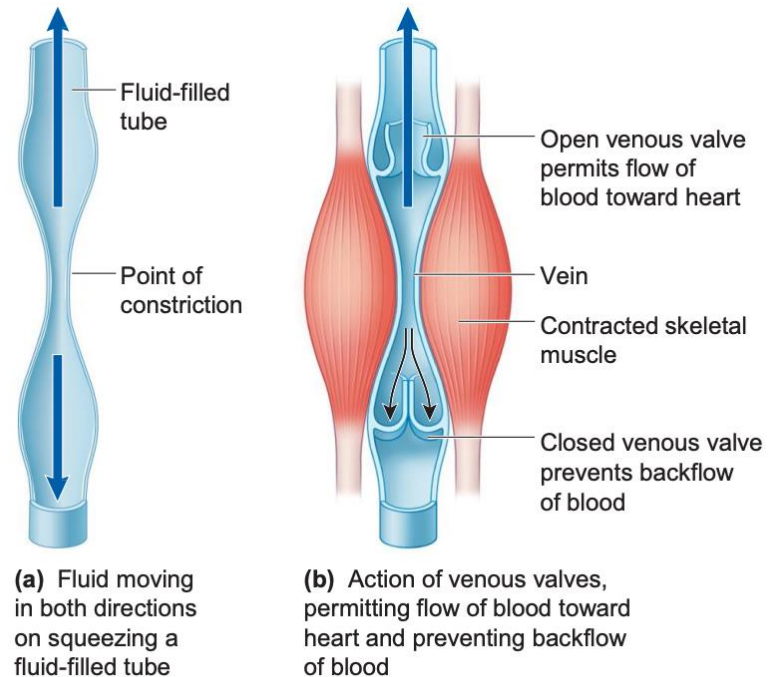
**Q: My friend has to wear compression stockings when she is on a long haul flight (that being said, anyone on a long haul flight I would recommend to wear compression stockings). She notes that if she doesn't, her legs get puffy. What do you think is the reason for this?**

## L3: Vascular physiology

**Q: How do the veins stop blood from flowing backwards?**

**- Veins have valves within them which prevents retrograde blood flow**

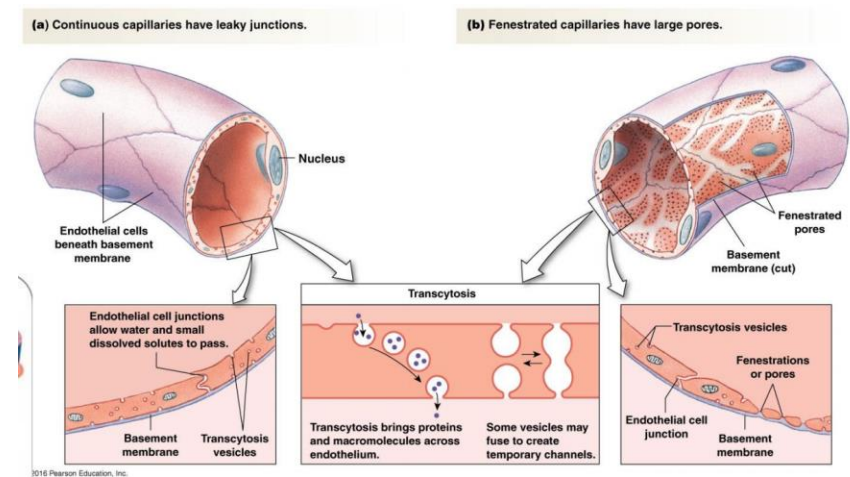
**Q: Normally, when an individual stands up very quickly, the blood 'falls out' of the heart into the IVC due to gravity – as a response, the heart compensates the drop in blood return by increasing blood pressure. In orthostatic/postural hypotension, the person faints, blacks out, loses consciousness or become dizzy. Why is this?**



## L4: Vascular transport

**Q: What are the 3 main mechanisms of exchanging nutrients in the vessels?**

- Paracellular movement
- Transcytosis
- Bulk flow





## **L4: Lymphatics**

**In individuals with suspect breast cancer, axillary lymph nodes may be removed. This has been shown to be extremely detrimental to a patient's quality of life – why?**

- Removal of lymphatic vessels as well compromises fluid return back to the circulatory system. This will result in something known as 'lymphoedema'
- Their arms will become puffy

**What other functions does the lymphatic system play?**

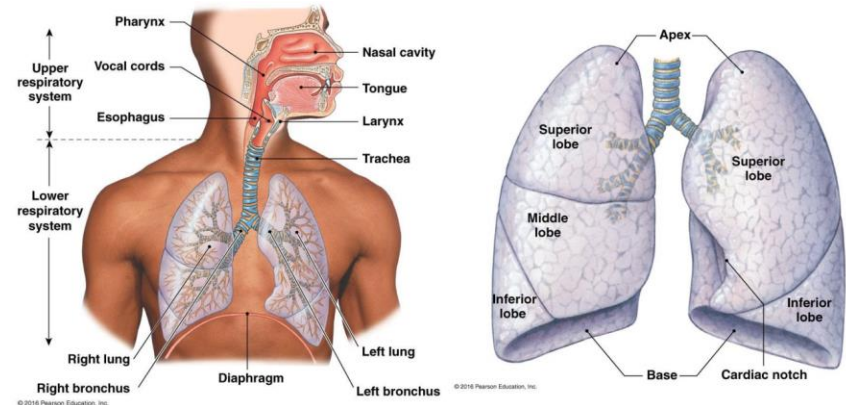
- Fat resorption from gut
- immunosurveillance

**Q: If blood simply flows from arteries to veins and back to the heart again, why is there extra fluid that needs to be absorbed by lymphatic vessels? Where does this fluid even come from?**

# Respiratory system

# L1: Respiratory system – quick fire questions

1. How many lobes in left lung, how many in right?
2. Does the trachea sit in front or behind of the oesophagus?
3. If I were to swallow a foreign body whilst unconscious, which lung would it likely end up in?
4. What is the function of respiration?
5. In passive breathing, what are the two most important sets of muscles we rely on?
6. When we breathe, does the diaphragm flatten, or curve more?
7. Do we normally fill our alveoli when we breathe in?

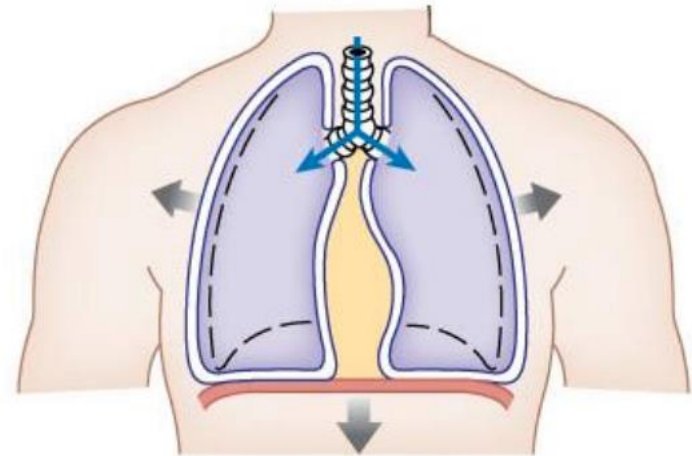


# L1: Conceptually challenging respiration

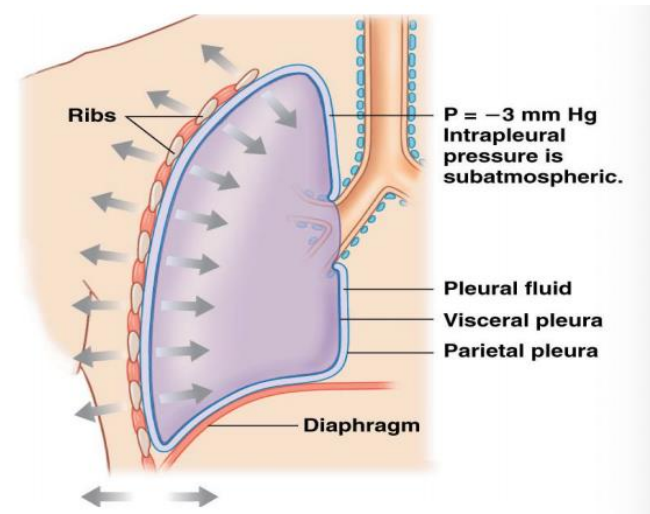
**Q: How does expansion of the chest cavity actually cause a negative pressure?**

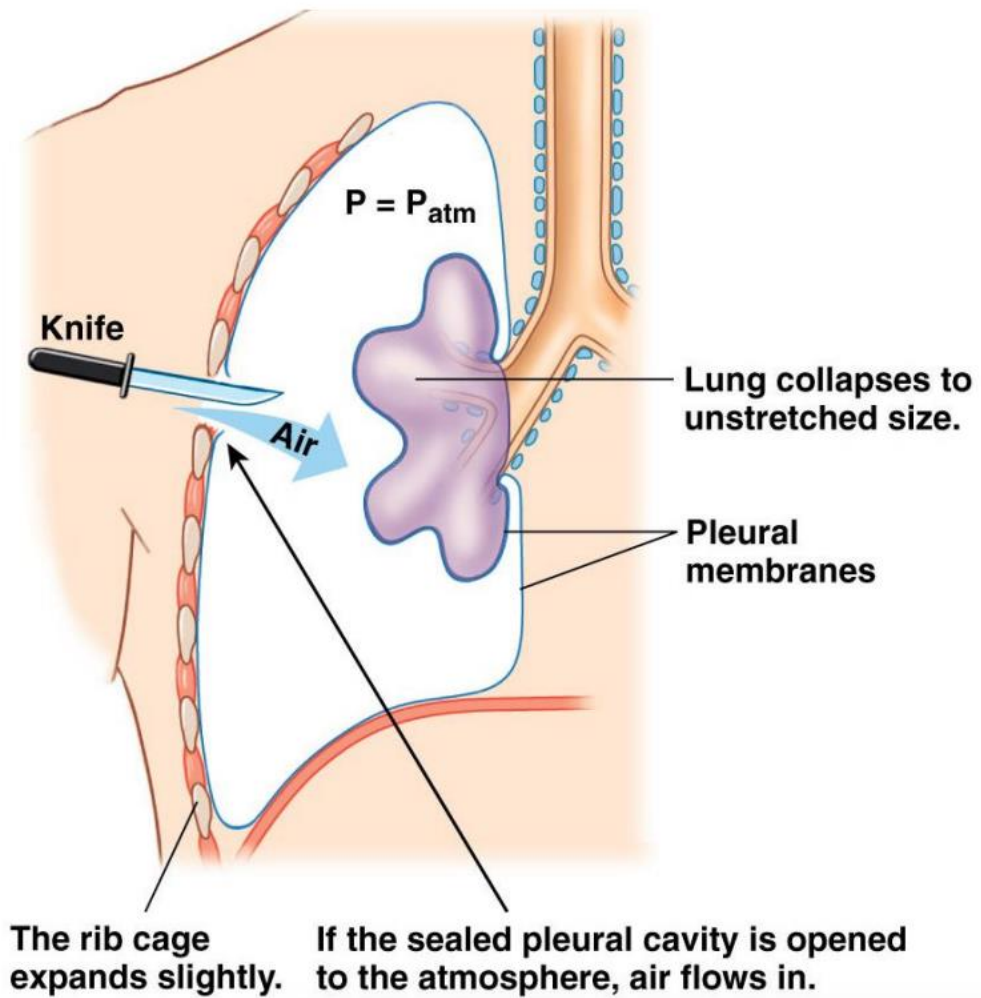
**Q: How is this related to a pneumothorax?**

**Q: How is this related to a TENSION pneumothorax? What is the management for a tension pneumothorax?**



**Diaphragm contracts and flattens.**



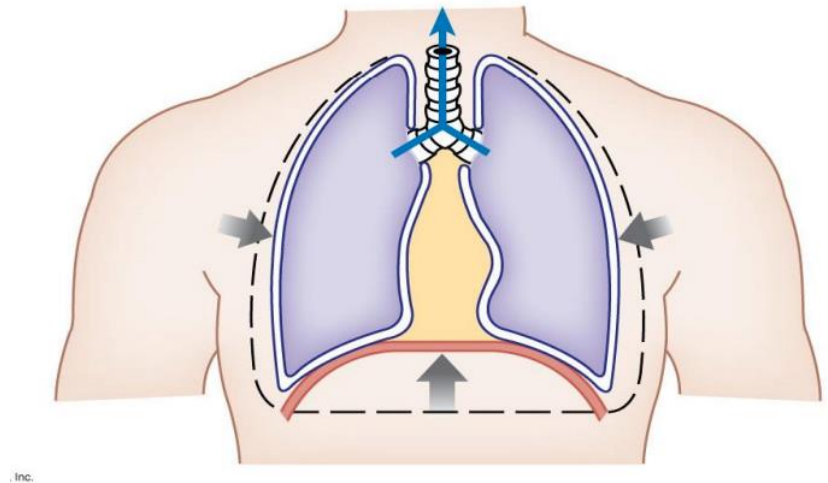


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# L1: Expiration

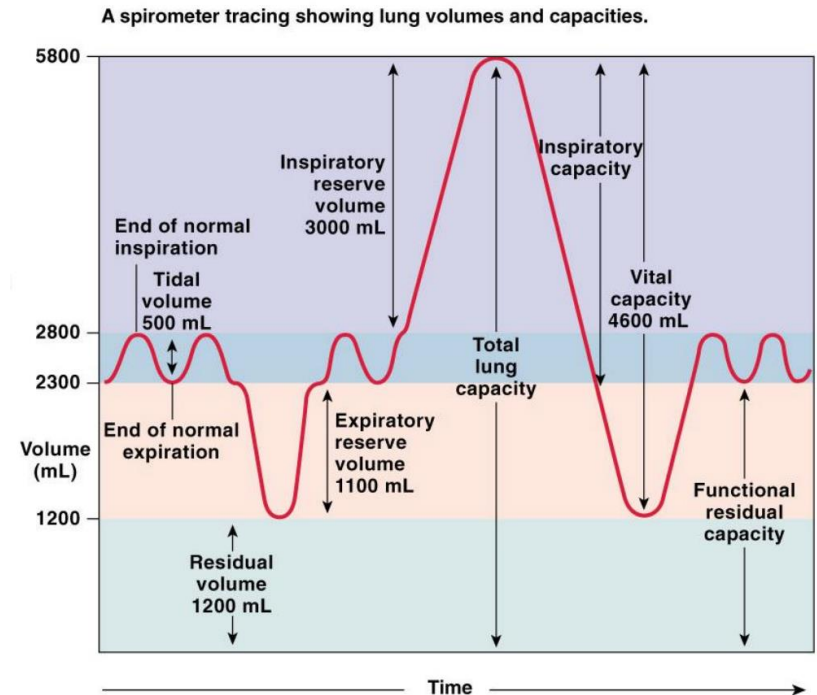
What structures do our lungs rely on to allow for **PASSIVE** expiration?

What about **ACTIVE/FORCED** expiration?



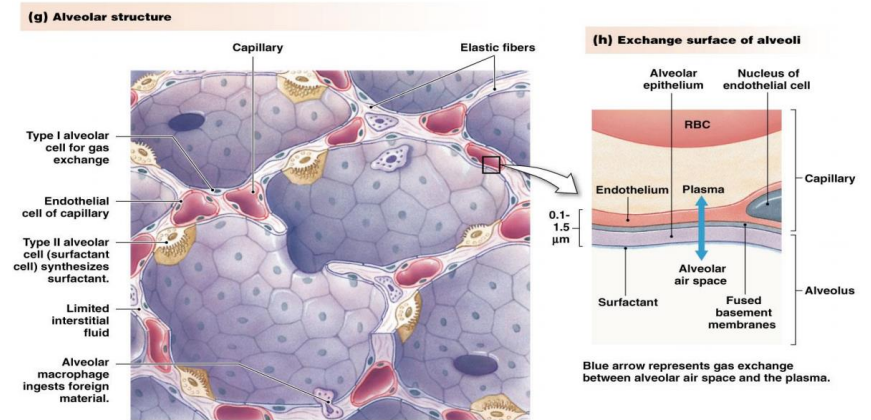
## Quick fire definitions

1. What is the tidal volume?
2. What is the inspiratory reserve volume?
3. What is the expiratory reserve volume?
4. What is the residual volume?
5. What is the vital capacity?
6. What is the functional residual capacity?



## L2: Gas exchange

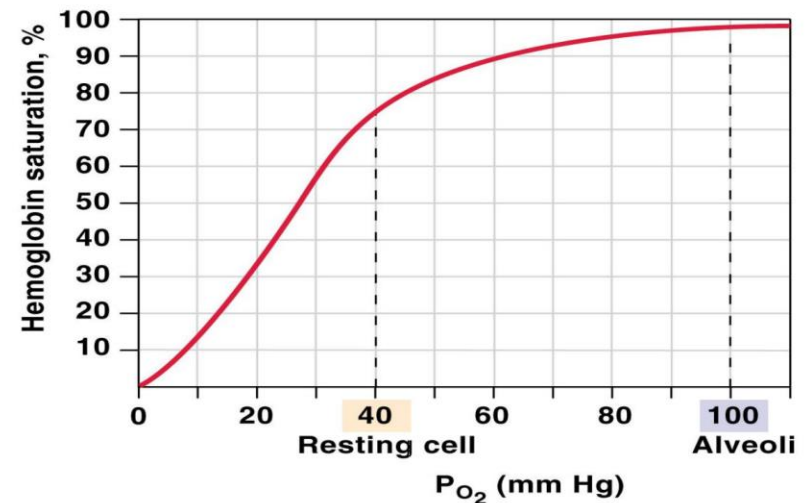
1. What structures does O<sub>2</sub> have to pass through to get into the RBC?
2. How is O<sub>2</sub> primarily transported in the body?
3. What is CO<sub>2</sub> transported?
  1. 7% plasma
  2. 23% bound to haemoglobin in RBC
  3. 70% bound to HCO<sub>3</sub><sup>-</sup> in RBC





## Quick fire

1. How many O<sub>2</sub> can bind each Hb?
2. How many chains is Hb made of?
3. What is normal Hb saturation?
4. Why do we say that a PaO<sub>2</sub> of <60mmHg is considered respiratory failure? Think of the oxyhaemoglobin dissociation curve



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