ACEM Primary Examination Vivas > Physiology > The Respiratory System

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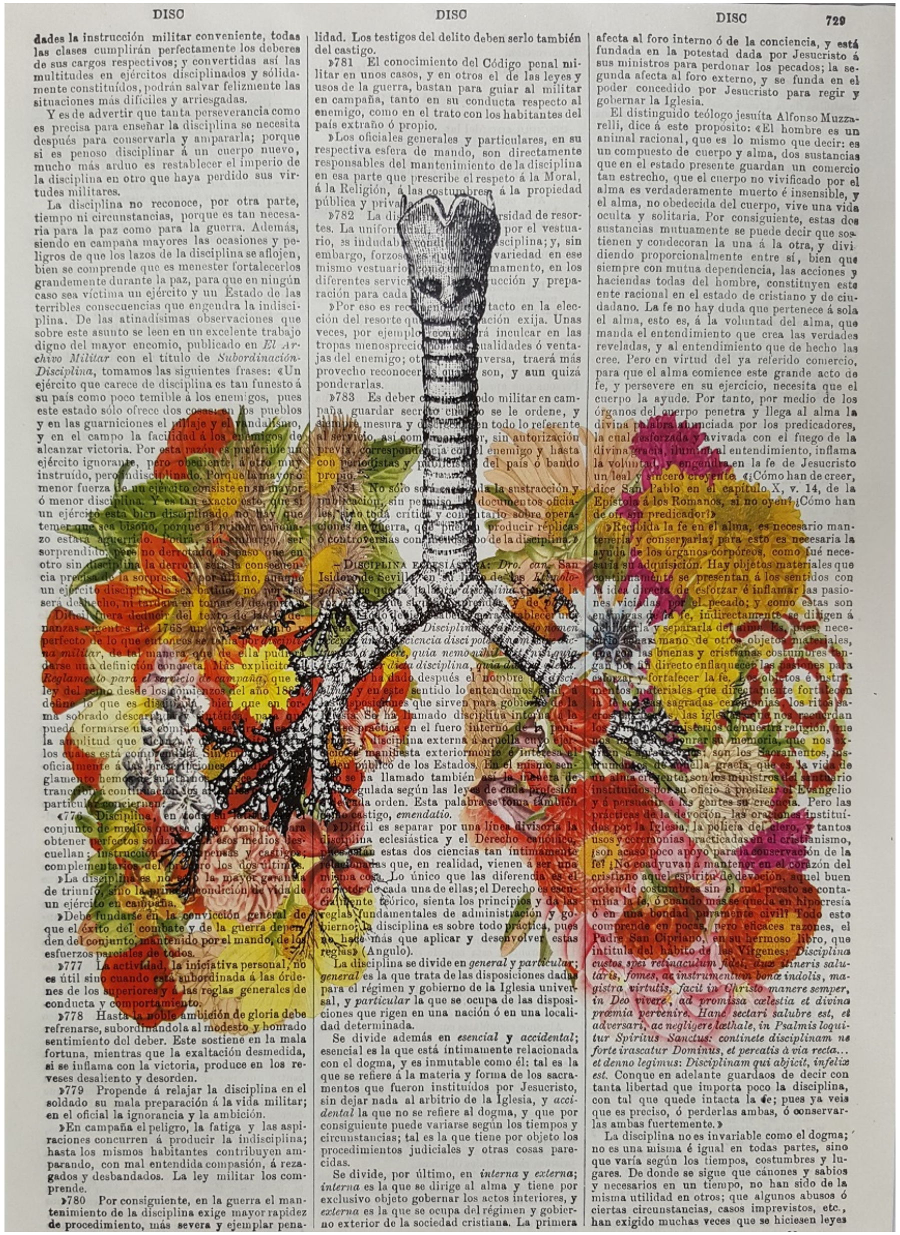
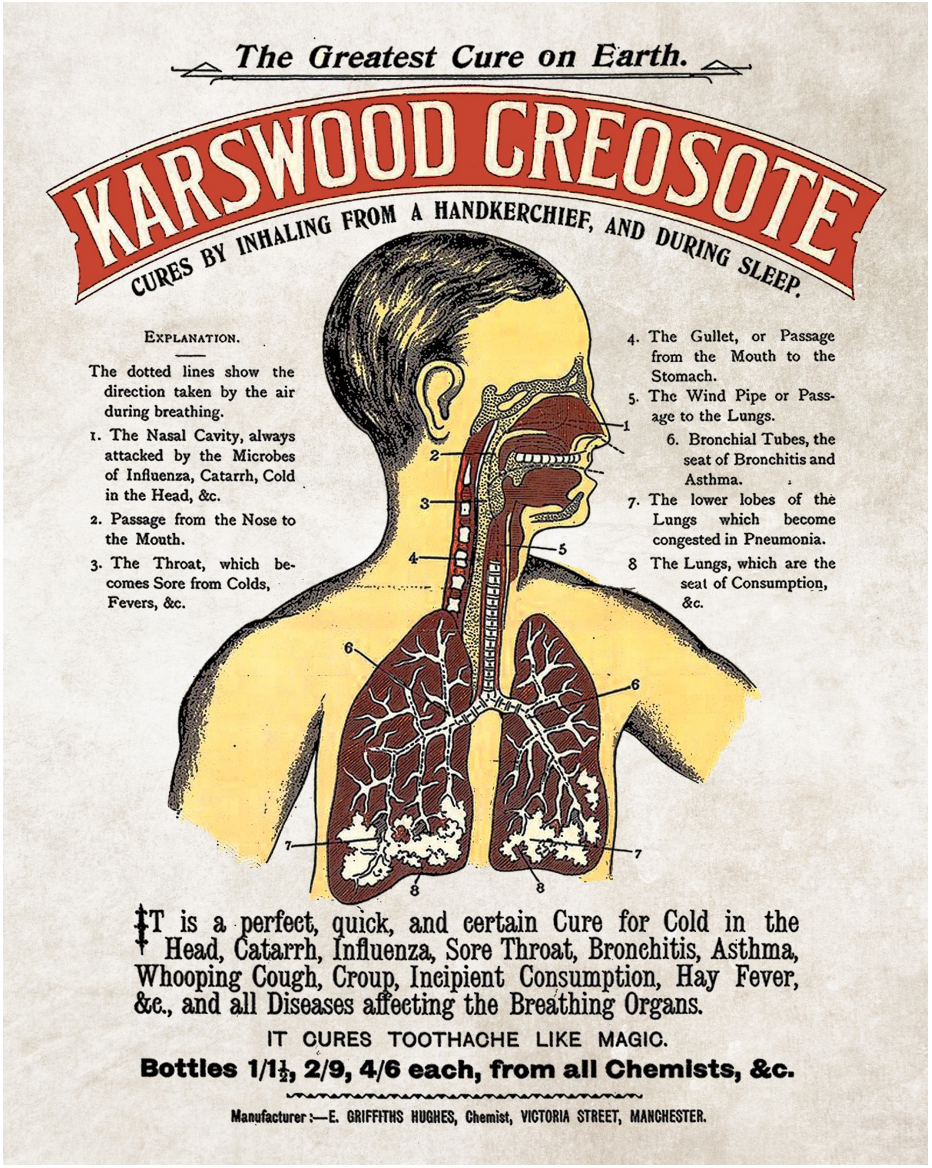




Acid Base Changes 2005-2

1.2 Respiratory compensation for acid base changes Davenport	Describe how respiration compensates for acid-base changes What clinical conditions might cause metabolic acidosis? / metabolic alkalosis?	**CO2 + H2O = H2CO3 = H + HCO3. Rapid responder.** Respiratory Centre responds to H, mainly at peripheral chemoreceptors, also transferred to CSF by CO2. **Metabolic acidosis –> inc ventilation, dec CO2 -> dec H, dec HCO3** ('base deficit'). **Metabolic alkalosis –> dec ventilation, inc CO2 -> inc H, inc HCO3** ('base excess'). In reality often no compensation. Davenport Diagram. - DKA; hypoxia -> lactic acid - Vomiting ->loss of acid.	

Airway resistance 2017-1-D

Stem: Moving to Physiology			
Question 2 Airway resistance **Subject**: Phys LOA: 1	a) Describe factors affecting airway resistance **PROMPT**: What is Poiseulle's law?	• In laminar flow, resistance is proportional to **the length** of the tube and **viscosity**, and inversely proportional to fourth power of the radius of the tube. (**Poiseuille's Law**: R = 8 x Length x Viscosity/n x **radius to 4th power**) • Turbulent flow is most likely to occur at high Reynolds numbers, that is, when inertial forces dominate over viscous forces. (Reynolds No. = density x Diameter x Velocity /Viscosity) • Highest in the medium-sized bronchi; low in the very small airways • Airway resistance decreases as lung volume rises because the airways are then pulled open by radial traction • Bronchial smooth muscle is controlled by the autonomic nervous system; stimulation of β-adrenergic receptors causes bronchodilation. Reduced alveolar PCO2 causes increased resistance.	Must understand Poiseuille's law (bolded) List at least 2 other factors. (West, 9th ed. 108-119)
	BONUS: Define dynamic compression of airways and its effects on flow	Intrapleural pressure > alveolar pressure causing airway compression. Dynamic compression of airways limits airflow during forced expiration.	Concept

Airway resistance 2013-1

Question 2 Pulmonary resistance & Compliance LOA: 1	a. Describe the factors that determine the airway resistance in the lung. Prompt: when would airway resistance increase?	<u>Airway resistance</u> • Decreases with stimulation of β-adrenergic receptors causing bronchodilatation. • Increases with parasympathetic nerve stimulation causing bronchoconstriction. • Increases with histamine • Increases when Lung volume reduces • Increases when pCO2 decrease • Increases with increase density & viscosity of gas	Need 3 factors to pass Poiseuille's Law: Resistance = 8 x viscosity x length / radius^4 x π
	b. With regard to lung **<u>compliance</u>** give examples of diseases that reduce compliance. Prompt define compliance: volume change/unit pressure.	b. Pulmonary fibrosis, pulmonary oedema, pulmonary haemorrhage, atelectasis, loss of surfactants such as respiratory distress syndrome.	Need 3 examples (may be others not listed that are acceptable)

Airway resistance 2008-1

2.1 Airway Resistance West pp 106-112	What factors impact on resistance in airways?	• Size of airway: R highest in medium sized bronchi, low in very small airways. • Lung volume: R decreases with expansion as airways pulled open • Bronchial smooth muscle tone: controlled by B sympathetics • Gas density: eg heliox -> low R • Forced expiration: intrathoracic pressure compresses airways = 'dynamic compression'	
	What factors cause turbulent flow in airways	Expressed by Reynold's number; **(3 out of 4)** Where: ρ is the fluid density; D is the diameter of the tube; V is the velocity of flow; η is the viscosity of the fluid. $\mathbf{Re} = \frac{\rho DV}{\eta}$ Laminar flow only in small airways, transitional most areas, turbulent in trachea (rapid breathing)	/2

Airway resistance 2006-1

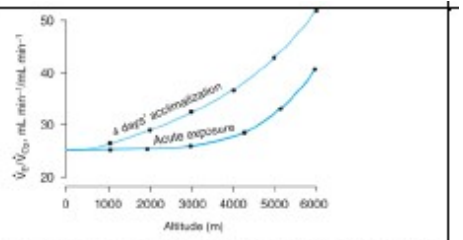
TOPIC: Airway resistance NUMBER: 2

OPENING QUESTION	Discuss the factors that determine airway resistance.	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1. Flow resistance R = 8 X viscosity X length / pieX r^4		Need to say that radius is the most important determining factor, 2/3 to pass.
	2. Directly proportional to viscosity & length. Inversely proportional to radius to the power of 4 (ie: half the radius increases resistance 16 fold).		
SECOND QUESTION (if needed)	What factors affect the radius of the airway?		Need 2 to pass.
POINTS REQUIRED	1. Bronchial smooth muscle tone: sympathetic and parasympathetic activity		
	2. Lung volume		

Altitude 2017-1-A

Stem: Moving on to Physiology. His fall occurred at high altitude.			
Question 4 High Altitude **Subject:** Phys LOA: 1	a) What are the initial physiological responses at high altitude?	1. Hyperventilation : decreases CO_2 > O2 2. Alkalosis : limited by movement of bicarbonate from CNS (1-2 days) and renal excretion HCO_3 3. Increased 2,3-DPG – R shift O_2-Hb dissociation curve (early), then left shift at higher altitudes due alkalosis 4. Alveolar hypoxia induces pulm vasoconstriction, then pulmonary HTN 5. Decreased work of breathing	3/5 to pass
	b) What are the longer-term physiologic effects of altitude exposure?	• Polycythaemia (incr EPO) • Incr viscosity of blood • Increased 0_2 carriage • Pulm HTN resulting in RVH • More capillaries • Increased oxidative enzymes. • Increased mitochondria	3 to pass

Altitude 2011-2

Question 2 LOA: 1	(a) Describe the ventilatory response that occurs as you acclimatise to high altitudes. (b) Outline other compensatory responses to high altitudes.	(a) **Hyperventilation: Inc altitude – hypoxic stimulation of peripheral chemoreceptors - inc ventilation.** Resulting low CO2 / alkalosis inhibits response; but bicarb shift from CSF and renal excretion bicarb – corrects pH to near normal, allowing further incr ventilation. (b) • Moderate altitude – right shift in O2 dissoc curve (due to incr 2,3 DPG) – dec O2 affinity to Hb (unload O2 at tissues); Higher altitude – left shift O_2 dissoc curve (due to decr CO2) – incr O2 affinity to Hb (load O2 in lungs) • Inc erythropoietin – polycythaemia • Inc mitochondria to facilitate O_2 transport into tissues • Inc cellular oxidative enzymes (cytochrome oxidase) • Inc capillaries in periph tissues	 **(a) Bold to pass** **(b) 2 to pass**

Altitude 2009-2

Question 2:	**What are the physiological changes that allow survival at high altitude ?**	1) Hyperventilation > decreases CO_2, > O2 2) Increased Hb (> EPO), 3) Alkalosis moderated by movement of bicarbonate from CNS (1-2/7) and renal excretion 4) Increased 2,3,DPG - R shift, 5) Pulm hypertension (due to alveolar hypoxia inducing pulm vasoconstriction) - 6) RV hypertrophy – not really an "adaptation" 7) Decreased work of breathing	All hypoxia driven, > viscosity helpful as pick up more difFic (3 of 7 to pass)

Altitude 2007-2

QUESTION: 1. Effect of high altitude on respiration

Question	Required response [Key items marked with*]	To Pass
Describe the effect of high altitude on respiration. *Prompt if required:* *Explain the mechanism underlying hyperventilation at altitude.*	*Hyperventilation Most important factor in acclimatisation to altitude. *Mechanism: Hypoxic stimulation of peripheral chemoreceptors [carotid bodies, aortic bodies]. Low pCO2 and alkalosis work against this but CSF pH 'normalised' by movement of bicarbonate out of CSF [~1-2 days] and renal excretion of bicarbonate [2-3 days] 'normalises' arterial pH taking this brake off. Sensitivity of carotid bodies to hypoxia increases during acclimatisation.	Hyperventilation plus mechanism
What other processes are involved in acclimatisation to high altitude?	*Polycythaemia [hypoxia, erythropoietin] Shifts in the O2 dissociation curve Right at moderate altitude 2° 2,3 DPG favouring unloading in tissues; left at high altitude 2° respiratory alkalosis favouring loading in lungs. Changes in capillary numbers/ density Changes to oxidative enzymes in cells Increased maximum breathing capacity	Polycythaemia with mechanism One other

Altitude 2005-1

Effect of altitude on respiration	What are the acute respiratory adaptations to altitude? What are the longer term physiologic effects of altitude exposure?	Hyperventilation. (Oxygen-haemoglobin curve shifts right or left.) Polycythaemia; one of increased O_2 carriage and viscosity; RVH; more capillaries; increased oxidative enzymes.	

TOPIC: Altitude on respiration _____ **NUMBER:**

OPENING QUESTION	What is the initial effect on respiration with ascent to 6000 metre	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1. (where the ambient pressure is about half the atmospheric pressure)	1	2/2
	2. hyperventilation	2	
	3. Shift of the oxygen dissociation curve to the right	3	
	4.	4	
SECOND QUESTION (if needed)	If the person remains at the same altitude for 6 months, what additional changes would occur?		2/5
POINTS REQUIRED	1. Polycythaemia	1	
	2. Increase in 2,3 DPG	2	
	3. Increase in the number of capillaries in peripheral tissues	3	
	4. Increase maximal breathing capacity	4	
	5. Pulmonary vasoconstriction resulting in pulmonary hypertension and right ventricular hypertrophy	5	
	7		
THIRD QUESTION (if needed)	Describe the symptoms of acute mountain sickness.		
POINTS REQUIRED	1. headache, fatigue, dizzy, palpitations, nausea, loss of appetite & insomnia.	1	
	2.	2	

Alveolar Gas Equation 2009-1

Question 2: Alveolar gas equation and its use in a clinical setting West pp 58, pp170	i) What is the alveolar gas equation? ii) How do you calculate the alveolar-arterial gradient? iii) What is the physiological significance of the A-a gradient?	i) **(4 out of 4)** $$PAO2 = PIO2 - \frac{PACO2}{R}$$ Where: • PAO2 is the alveolar oxygen partial pressure • PIO2 is the oxygen partial pressure of inspired air • PACO2 is the alveolar CO2 partial pressure. • R is the respiratory quotient; CO2 production/O2 consumption, typically 0.8 Note that a small correction factor F of 2mmHg has been omitted from the equation. ii) **Difference between PAO2** (alveolar) and **PaO2** (arterial). iii) **V/Q mismatch** (eg: shunting or dead space)	

Chemoreceptors 2008-2

OPENING QUESTION	**What is the role of central chemoreceptors in control of ventilation?**	**COMMENTS**
POINTS REQUIRED	1. Located near ventral surface of medulla	
	2. Rise in blood CO2 increases CO2 in CSF	
	3. CSF has poor buffering capacity so pH changes rapidly	Bolded to pass
	4. Liberated H^+ ions stimulate chemoreceptors (increasing pH has reverse effect)	
	5. Efferents stimulate medullary respiratory centre to increase ventilation and return CO2 to normal.	
	6. Chronic CO2 elevation gives normal CSF pH and insensitivity	
PROMPTS	What happens in the brain when the blood CO2 level rises?	
SECOND QUESTION (if needed)	**What is the role of the peripheral chemoreceptors?**	
POINTS REQUIRED	1. Located in carotid and aortic bodies that have high blood flow	
	2. Respond mostly to decrease in O2 below 100mmHg	
	3. Impulses transmitted to respiratory centre to increase ventilation	3 of 5 to pass
	4. Responsible for all of the ventilatory response to hypoxaemia	
	5. Also responsible for small but rapid response to rise in CO2 and decrease in pH (carotid bodies)	
PROMPTS	How is hypoxaemia detected?	

COMMENTS

CO2 2016-2-A

Question 4 CO_2 transport **Subject:** Physiology LOA: 1	Q1) How is CO_2 transported in the blood? *Prompt:Are there any other mechanisms?* Q2) What is the most important mechanism? *Prompt:What proportion does each mechanism contribute.* Q3) What is the role of Red Blood Cells in CO_2 transport?	**Diffusion, Carbamino-proteins and CO_2 to Bicarbonate buffering** Diffusion 5 (a)-10 (v)% Carbamino 5 (a)-30%(v) Bicarbonate 60 (v)-90 (a)% a) Carbonic anhydrase only found significantly in Red cells, major buffer for CO_2 and H+ <u>b) Haldane Effect</u> -Hb (partic de-oxy) is also major H+ buffer allowing ->/faster H+/HCO3dissociation -<u>Chloride shift</u> (allowing 70% HCO_3- diffusion into plasma maintaining ionic neutrality/ enhanced diffusion) is mediated by Band 3 Cl- transporter in RBC membrane c) - Hb protein is the major carbamino protein (better when deoxyHb as more negative charge)	2/3 bold required Bic is the most important 1/3 for pass

CO2 2015-1-B

Stem: An 80 year old man is sent to ED from his nursing home after a fall. He has multiple injuries and has been given IV morphine by the ambulance officers. On arrival in ED he is hypoventilating. We will start with Physiology.

TOPIC	QUESTIONS	KNOWLEDGE (**essential in bold**)	NOTES
Question 1 CO_2 transport **Subject:** Phys LOA: 1	Q1. How is CO2 carried in the blood?	CO2 is carried in the blood in 3 forms: 1. dissolved - approx 5-10% **2. as bicarbonate - approx 90%** 3. combined with proteins as carbamino compounds, approx 5-10%	Bold + 1 other
	Q2. How is bicarbonate formed in the blood? Prompt: can you write an equation	CA CO2 + H2O ←→ H2CO3 ←→ H+ + HCO3- The 1st reaction is very slow in plasma but fast within the red blood cell because of the presence there of an enzyme **Carbonic Anhydrase (CA).** The 2nd reaction, ionic dissociation is fast without an enzyme.	Talk through equation and Bold.
	Q3. What is the chloride shift?	HCO3- diffuses easily out of the cell. H+ doesn't because the **cell membrane is relatively impermeable to cations**. Therefore to **maintain cell neutrality** Cl- diffuses from the plasma into the cell.	At least 1 bold
	Q4. What is the Haldane effect?	H+ + HbO2 <–> H+-Hb + O2 The Haldane effect: DeoxyHb binds more H+ than oxyHb and forms carbamino compounds more readily. Binding of O2 to Hb reduces its affinity for CO2 1. **Enhances the removal of CO2** from O2 consuming **tissues** (eg muscles) into the blood. CO2 can bind to amino groups on Hb to form carbaminoHb. CarbaminoHb is the major contributor to the Haldane effect. 2. Promotes the **dissociation of CO2 from Hb** in the **presence of O2** (eg the lungs) which is vital for alveolar gas exchange	Additional question if time permits

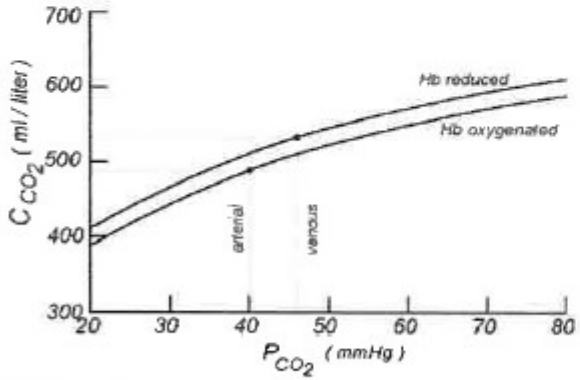
CO2 2014-1-D

<table>
<tr><td colspan="4">Stem: Moving onto PHYSIOLOGY</td></tr>
<tr>
<td>Question 4
CO2 carriage and dissociation curve

Subject: Physiology
LOA: 1</td>
<td>(a) How is carbon dioxide transported from the tissues to the lungs?

(b) Draw and explain the carbon dioxide dissociation curve

(c) What is meant by the term ‘chloride shift’?</td>
<td>(a) In plasma:
• Dissolved
• Carbamino compounds with plasma proteins
• Hydration – H+ buffered – HCO3- in plasma
In RBC:
• Dissolved
• Formation of carbamino-Hb
• Hydration – H+ buffered – 70% of HCO3- enters plasma
Each 49ml CO2/dL arterial blood – 5% dissolved, 5% in carbamino compounds, 90% hydrated as HCO3

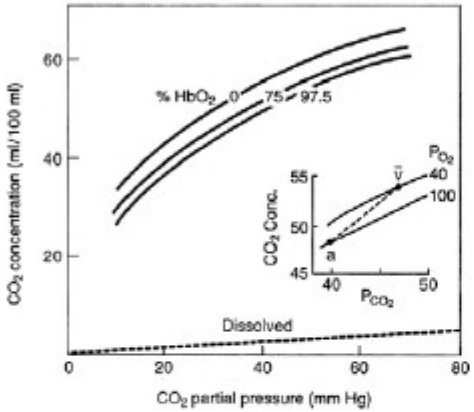
 

(c) 70% of HCO3- formed in red cells enters the plasma in exchange for chloride – exchange is the chloride shift</td>
<td>(a) Bold to pass

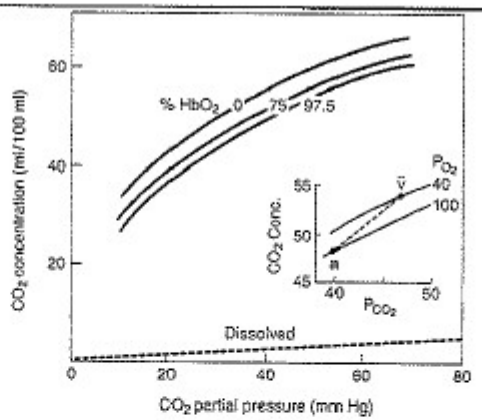
(b) Concept to pass

(c) Reasonable definition to pass</td>
</tr>
</table>

CO2 2012-1

Question 2	2.1 In what forms is carbon dioxide transported in the blood?	• Dissolved. • As carbamino compounds with proteins, especially Hb. • Hydrated in red cells – H^+ is buffered and 70% of HCO_3^- enters the plasma.	**Two of three to pass.**
	2.2 Please draw the carbon dioxide dissociation curve for normal arterial blood. Prompt: "Draw a graph showing the relationship between the pressure of carbon dioxide and the total carbon dioxide content in arterial blood."	 **Figure 6-6.** CO_2 dissociation curves for blood of different O_2 saturations. Note that oxygenated blood carries less CO_2 for the same P_{CO_2}. The *inset* shows the "physiological" curve between arterial and mixed venous blood.	**Reasonable shape of the curve indicating the near linearity in the physiological range.** Prompt if necessary.
	2.3 Where will the curve lie for venous blood and why?? Prompt: "Does the curve move up or down and why??"	• The **graph moves upwards** indicating greater CO_2 content per unit pressure. • Deoxygenated haemoglobin binds more H^+ and forms more carbamino compounds than oxyhemoglobin so venous blood carries more CO_2 than arterial blood. • This is known as the Haldane effect.	**The candidate must understand that venous blood is able to carry proportionately more CO_2 than arterial blood.**

CO2 2009-1

Question 2: Carbon Dioxide Transport West pp 80-3	i) How is carbon dioxide transported in the blood?	 Figure 6-6. CO_2 dissociation curves for blood of different O_2 saturations. Note that oxygenated blood carries less CO_2 for the same P_{CO_2}. The *inset* shows the "physiological" curve between arterial and mixed venous blood. • **Dissolved**. • As **carbamino compounds** with proteins, especially Hb. • Hydrated in red cells — H^+ buffered — **HCO_3^-** in plasma.	To pass: 2/3
	ii) How does venous blood carry more CO2 than arterial blood?	• Deoxygenated haemoglobin binds more H^+ and forms **more carbamino compounds** than oxyhemoglobin so venous blood carries more CO_2 than arterial blood. • This is known as the Haldane effect.	Does the curve move towards the left or the right, and why??

OPENING QUESTION	How is carbon dioxide transported from the tissues to the lungs?	COMMENTS
POINTS REQUIRED	1. In plasma: • **Dissolved** • **Carbamino compounds with plasma protein.** • **Hydration - H^+ buffered - HCO_3^- in plasma.** 2. In red blood cells: • Dissolved. • Formation of carbamino–Hb. • Hydration - H^+ buffered - 70% of HCO_3^- enters plasma. 3. Of the approximately 49 mL of CO_2 in each decilitre of arterial blood, 2.6 mL (5%) is dissolved, 2.6 mL (5%) is in carbamino compounds, and 43.8 mL (90%) is in HCO_3- 4. In the tissues, 3.7 mL of CO_2 per decilitre of blood is added; 0.4 mL (10%) stays in solution, 0.8 mL (20%) forms carbamino compounds, and 2.5 mL (70%) forms HCO_3^-. The pH of the blood drops from 7.40 to 7.36.	Bolded
PROMPTS	Which is the most important? Anywhere else ? (other than plasma)	
SECOND QUESTION (if needed)	**What is meant by the term 'chloride shift'?**	
POINTS REQUIRED	1. **About 70% of the HCO_3^- formed in the red cells enters the plasma in exchange for Cl^-. The exchange is called the chloride shift.** 2. This process is mediated by Band 3, a major membrane protein and is essentially complete in 1 second. 3. Note that for each CO_2 molecule added to a red cell, there is an increase of one osmotically active particle–either an HCO_3^- or a Cl^-–in the red cell. Consequently, the red cells take up water and increase in size.	Bolded
PROMPTS		

COMMENTS

CO2 2003-1

TOPIC: Carbon dioxide transport _____ **NUMBER:** _____

OPENING QUESTION	How is carbon dioxide transported in the blood?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1. CO2 is carried in the blood as dissolved, as bicarbonate, and in combination with proteins as carbamino compounds.	1	2/3
	2.	2	
SECOND QUESTION (if needed)	Draw and label the carbon dioxide dissociation curve.		
POINTS REQUIRED	1.	1	
	2.	2	

Compliance 2017-1-C

Stem: Once intubated, she is difficult to ventilate. Moving on to Physiology.			
Question 5 Lung compli-ance **Subject**: Phys LOA: 1	a) What is pulmonary compliance?	Compliance = **volume change/pressure change (ΔV/ΔP)**, maximal in mid inspiration, lower at extremes, approx. 200ml/cm H20	Bold with concept
	b) What factors decrease or increase pulmonary compliance?	**Decreased:** alveolar oedema, pulmonary fibrosis, pulmonary venous hypertension, un-ventilated lung, **Increased:** age, emphysema	3 examples 1 example
	c) What are the physiological effects of surfactant of the lung?	i. Increased lung compliance ii. Reduced work of breathing iii. Improved stability of alveoli iv. Keeps alveoli dry	2 of 4

Compliance 2010-2

Question 5	a.Please explain the concept of compliance as it relates to the lung	a. Volume change per unit pressure change (slope of pressure volume curve) =~200ml/cm H2O Depends on lung volumes and demonstrates hysteresis , may draw compliance curve, point out that inc compliance at low volumes Depends on structural proteins and surface tension	Basic concept
	b.What factors affect compliance	b. Decrease – fibrosis, pulmonary oedema, not ventilated, inc pulmonary venous pressure Increase- ,emphysema, asthma surfactant	1 of each

Compliance 2010-1

QUESTION	KNOWLEDGE	PASS CRITERIA
1a). What is Pulmonary Compliance?	a) Compliance =Volume Change/Pressure Change, proportional to slope the pressure volume curve of the lung. Within normal range (-2 to –10 cm H20) of expanding pressures, lung is very compliant. At higher expanding pressures, compliance is smaller. Normal human lung compliance =200 ml /cm H2O) Specific Compliance = "compliance per unit volume of lung"	a) delta V/delta P
b) What are the factors that decrease and increase pulmonary compliance?	c) Reduced = pulm venous hypertension, unventilated lung (espec at low lung volumes i.e. atelectasis), pulm fibrosis and alveolar oedema of any type. Increased = increasing age and emphysema	b) Three factors which decrease Compliance and both the factors which decrease it c)two factors which increase it
c) What are the physiological effects of surfactant on the lung?	d) What are the physiological effects of Surfactant? i) increases lung compliance ii) reduces work of breathing iii) improves stability of alveoli iv) keeps alveoli dry	d) Three of four

Compliance 2008-1

3.1 Elastic Properties of the Lung West pp 96-106	Define lung compliance?	• Change in volume / change in pressure (Slope of pressure-volume curve) (Lung "stiffness")	
	What factors influence lung compliance?	**(3 out of 6)** • Fibrosis • Alveolar oedema • Elastic tissue • Emphysema / age • Volume / Size of lung • Surface tension in alveoli (Surfactant)	
	What else does surfactant do?	**(2 of 3)** • Reduces WOB Prevents collapse Keeps alveoli dry	/2

Compliance 2007-1

TOPIC: Elastic properties of the lung _____ **NUMBER:** _____

OPENING QUESTION	What is thoracic compliance?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 Change in lung volume per unit change in airway pressure (DV/DP)		Essential
	2 Measure of elastic recoil of lungs and chest wall	What is it a measure of?	
	3 Normally 200 mL/mmHg in intact thorax		
SECOND QUESTION	What are the main determinants of compliance of the thorax?		2 to pass
POINTS REQUIRED	1 Surface tension of the alveoli (2/3rds)		
	2 Elastin/collagen fibres (1/3rd)		Allow tissue properties
	3 Alveolar surface tension depends on alveolar pressure, alveolar radius, surfactant (Law of Laplace – P=2(or4) x T/R)		
THIRD QUESTION (if needed)	How does compliance vary throughout the upright lung?		Must say that base > apex
POINTS REQUIRED	1 Higher at base than apex because apex is already more distended		

Compliance 2006-2

TOPIC: Elastic Properties of the lung _____ **NUMBER: 1** _____

OPENING QUESTION	What is pulmonary compliance and what are the factors that influence it?	**PROMPTS COMMENTS**	
POINTS REQUIRED	1 Volume change per unit pressure change.	1	1 – 3 to pass
	2 Elastic recoil of the lung especially the geometry of the elastin fibres	2	
	3 Surface tension in the alveoli	3	
	4 Disease such as fibrosis, oedema decrease compliance	4 Ask about the effects of disease and ageing if not volunteered	Additional information
	5 Ageing and emphysema increase compliance	5	Additional information
SECOND QUESTION (if needed)	What are the physiological advantages of surfactant?		2/3 to pass
POINTS REQUIRED	1 Increases compliance and reduces work of breathing	1	
	2 Prevents small alveoli collapsing P = 2 x Tension / Radius	2 Prompt for La places law if doing well	Law is additional information
	3 Reduces transudation	3	
THIRD QUESTION (if needed)	Can you draw the pressure volume curve of a normal lung?		2/3 to pass
POINTS REQUIRED	1 Hysteresis	1	
	2 Closing volume	2	
	3 Lung becomes stiffer at higher volumes	3	

TOPIC: Relationship of intrapleural pressure and lung volume and regional differences in ventilation _____ **NUMBER:** _____ 5b

OPENING QUESTION	What is the relationship between intrapleural pressure and lung volume?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 Sigmoid curve of IP pressure vs volume, does not reach 0% lung volume	1	
	2 Shows lung volume is higher during deflation than inflation for any given pressure = Hysteresis	2	Pass if describes relationship, or draws curve with features
	3 Shows that lung contains residual air, without any expanding pressure (due to airway closure)	3	3 OF 4 POINTS
	4 Shows that compliance decreases at higher lung volumes – lung becomes stiffer due to reaching limits of elasticity	4	
	3	3	
SECOND QUESTION (if needed)	**What variables affect pulmonary compliance?**		
POINTS REQUIRED	• Slightly greater during deflation than during inflation as noted above; • Lung volume – at very low and very high volumes compliance is reduced; • Increased when tissue elasticity is reduced, as in emphysema; • Decreased by increased tissue mass - fibrosis or pulmonary congestion; • Decreased by loss of surfactant.	1	2 of 4 to pass
THIRD QUESTION (if needed)	Describe how regional differences in intrapleural pressure affect the ventilation		
POINTS REQUIRED	1 states that the intrapleural pressure is higher at the apex than at the base of the lung – to keep the lung expanded against its own weight	1	Extra mark for correct answer here
	2 Increased compliance at base, hence better ability to ventilate base compared with apex	2	Bonus if gives values 10cm H2O at apex, 2.5cm at base
	2	2	
	3		

Control of Ventilation 2017-2-A

Stem: Moving onto Physiology. She is tachypnoeic.			
Question 4 Control of ventilation **Subject:** Physiology LOA: 1	a) What parts of the brain control respiration? b) How are chemoreceptors involved in the control of ventilation? c) What other sensors are involved in the control of ventilation?	a) Voluntary - **Cerebral cortex**. Automatic **– Medulla** (Pacemaker cells in pre-Botzinger complex). Pons – pneumotactic centre modifies medulla activity. b) **Chemoreceptors – central and peripheral** Central (ventral surface medulla) - **sensitive to changes in H+.** **CO2** readily penetrates BBB and enters CSF and brain interstitial fluid. Increased CO2 causes increased H+ in CSF, stimulating ventilation. Peripheral (carotid and aortic bodies) – fast response to **decreasing O2**, stimulating bodies. Decreased pH causes carotid response only. Minor response to CO2. c) Pulmonary - Stretch receptors in lungs, muscles, joints, Irritant receptors in airways, J receptors. Irritant receptors in nose and upper airways Baroreceptors (arterial, atrial. Ventricular, pulmonary) – stimulation may cause reflex hypoventilation Pain/temperature – may cause initial apnoea, then hyperventilation Proprioceptors (muscle spindles from intercostals and diaphragm, other muscles/tendons/joints.	Bold to pass Bold and concept for response to CO2/O2. 3 examples

Control of Ventilation 2016-2-C

Stem: Moving onto Physiology. Her respiratory rate falls.			
Question 5 Control of ventilation **Subject:** Phys: LOA: 1	1. What parts of the brain control respiration? 2. What stimuli affect respiration? [Prompt for Chemical or non-chemical control]	1. -**Cerebral cortex** for voluntary control -**Medulla** for automatic control (driven by pacemaker cells in the pre-Bötzinger complex) 2. <u>Chemical control</u> : chemoreceptors -**CO_2** (readily penetrates the BBB and enters CSF and brain interstitial fluid H^+ concentration), promptly hydrated and H+ ions increase - chemoreceptors in **medulla** sensitive to changes in H^+ Increase in H+ conc in CSF stimulates ventilation Some CO_2 regulation via carotid/aortic bodies -**O_2 / H^+** (via **carotid/aortic bodies**) ↓ O_2 results in ↑ glomus cell activity in carotid/aortic bodies Fast response to decreased O2 Dec pH causes response in carotid body only. <u>Nonchemical control</u> -vagal afferents from pulmonary stretch receptors -afferents from pons/hypothalamus/limbic system -afferents from proprioceptors in mm, tendons, jts -afferents from baroreceptors: arterial, atrial, ventricular, pulmonary	Bold Bold + concept for CO2/O2 regulation + 1 non-chemical control
	3. How does hypoventilation affect respiration? [Bonus Question] Prompt: What is the role of H+ ions?	3. BBB is permeable to CO_2; relatively impermeable to HCO_3^- ↑ blood pCO_2 → ↑CSF pCO_2→↑ H^+ in CSF→ stim vent ↑ H^+ in CSF stimulates ventilation ↓ H^+ in CSF inhibits ventilation; causes cerebral vasodilatation → enhance diffusion of pCO_2 into CSF	General concept of bolded

Control of Ventilation 2015-2-B

Stem: A 50 year old alcoholic presents with a GCS of 7 and respiratory depression. Starting with Physiology			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Control of Ventilation **Subject:** Phys LOA: 1	(a) What are the receptors involved in the control of ventilation?	**Central chemoreceptors, Peripheral chemoreceptors,** Pulmonary stretch receptors, Irritant receptors, J receptors, Bronchial C fibres, Nose and upper airway receptors, Joint and muscle receptors, Gamma system, Arterial baroreceptors, Pain & temperature receptors	Bold & 3 others to pass
	(b) Where are the central chemoreceptors located?	200-400 μm below ventral surface of **medulla.**	Medulla must be stated
	(c) How do these receptors function? Prompt : How do H^+ ions affect their function	BBB permeable to CO_2; relatively impermeable to HCO_3^- **↑blood pCO_2 → ↑CSF pCO_2 → ↑H^+ in CSF** **↑H^+ in CSF stimulates ventilation** ↓H^+ in CSF inhibits ventilation; causes cerebral vasodilation → enhance diffusion of pCO_2 into CSF CSF pH 7.32. Less buffering than blood, CSF pH changes more for given pCO_2 Prolonged pH changes compensated by HCO_3^- transport across BBB. (Chronic CO_2 retention has near normal CSF H^+)	Bold concepts to pass

Control of Ventilation 2013-2-A

In the ED, she becomes progressively tachypnoeic			
Physiology: Control of ventilation	What are the major components of the control of ventilation (or respiration)	**Voluntary versus automatic** **Medulla pacemaker cells,** **Pons** pneumotactic centre modifies the medulla actvity **Higher centres** hypothalamus, limbic system, cerebral cortex **Vagal afferents** from lung **Central chemoreceptors** CSF (medulla, floor 4th ventricle)– ⇧H^+, **Peripheral chemoreceptors carotid and aortic bodies** – pO_2, pH ⇩, pCO_2⇧ Integrated response: $PaCO_2$, PO_2, pH **Lung receptors** – stretch, irritant, bronchial C fibres (J receptors),	5 of 7 bold
	How does a rise in CO_2 affect ventilation?	Direct effect on **central and peripheral chemoreceptors**, due to both **high CO_2** and **lower pH.** **Increase in rate and depth of ventilation.**	4 of 5 bold

Control of Ventilation 2011-2

Question 2 LOA: 1	(a) Describe the function of the central chemoreceptors in the regulation of ventilation. (b) Describe the function of the peripheral chemoreceptors in the regulation of ventilation.	Chemoreceptors – respond to changes in chemical composition in blood or fluid around them (a) **Central chemoreceptors** - situated in the ventral surface of the medulla, **regulates ventilation in response to CSF pH** (CO_2 in blood changes pH in CSF – **incr CO2 -> incr H+ in CSF -> incr ventilation).** (a) **Peripheral chemoreceptors** – located in the **carotid & aortic bodies**. They contain glomus cells with high conc dopamine, high blood flow. **Respond to a dec in PaO_2 and pH, inc in $PaCO_2$. . Responsible for all the inc ventilation in hypoxia** - max response occurs PaO_2<50 mm Hg. Also rapid response to sudden changes in $PaCO_2$, while carotid body responds to a fall in pH.	Adequate description of function for (a) and (b) - bold

Control of Ventilation 2011-1

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES & MARKS
Question 2: Integrated responses to O2,CO2, pH	2.1 What sensors are involved in the control of ventilation?	a) Central chemoreceptors in medulla-respond to CSF pH (via CO_2) b) Peripheral chemoreceptors in carotid and aortic bodies respond to O_2, pH, CO_2 (minor) c) Pumonary receptors: o Stretch receptors in lungs, muscles, joints o Irritant receptors in airways o J receptors-engorged lung capillaries and alveolar wall interstitial fluid d) Nose and upper airway receptors - irritant receptor e) Joint and muscle receptors - muscle spindles in the intercostal muscles and diaphragm f) Arterial baroreceptors - stimulation may cause reflex hypoventilation g) Pain and temperature receptors - may cause initial apnoea and then hyperventilation	Central and peripheral chemoreceptors and pulmonary receptors
	2.2 Describe the ventilatory response to metabolic acidosis	2.2 Low arterial pH stimulates peripheral chemoreceptors to increase ventilation. Central chemoreceptors or respiratory centre itself may be stimulated in severe cases	Peripheral chemoreceptors dominate response

Control of Ventilation 2007-1

TOPIC: Sensors in control of ventilation _____ **NUMBER:** _____

OPENING QUESTION	What sensors are involved in control of ventilation?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 Chemoreceptors and mechanical receptors	What senses changes in blood gases?	
	2 Central chemo in medulla respond to H^+		Need peripheral and central
	3 Peripheral chemo in carotid and aortic bodies respond to O2, CO2, H^+		
	4 Stretch receptors in lungs, muscles, joints		
	5 Irritant receptors in airways		
	6 J receptors respond to engorged capillaries		

Control of Ventilation 2005-1

Control of ventilation.	What are the basic elements of the respiratory control system? What inputs are there into the respiratory control system? Explain the function of the central chemoreceptors	Sensors; Central Controller; Effectors. Cortical Central and peripheral chemoreceptors, Lung and other receptors. Respond to changes in H^+ concentration. CO_2 regulates ventilation by effects on pH	

TOPIC: Control of ventilation _____ **NUMBER:** _____

OPENING QUESTION	Where are the peripheral chemoreceptors involved in control of ventilation?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 Carotid bodies near carotid bifurcations	1 O^2 sensor?	
	2 One or more near arch of aorta	2	
	3 (Carotid bodies more important)	3	
	4	4	
	5	5	
SECOND QUESTION (if needed)	What is their role?		
POINTS REQUIRED	1 Sense O^2, CO^2, H^+ concentrations	1 Tell us more about the specific substances that stimulate chemoreceptors?	
	2 Feedback to medullary respiratory centre	2	
	3 O^2 sensor critical to response to hypoxia	3	
	4 pO^2 less than 60 gives inc ventilation	4	
	5 CO^2 less important (but faster than central)	5	
	6 H^+ (carotid b) gives inc vent if pH falls	6	
	7		
THIRD QUESTION (if needed)	Where are the central receptors involved in ventilation and what do they respond to?		
POINTS REQUIRED	1 Medulla	1 Brain sensors?	
	2 Respond to pH of CSF	2	
	3 Indirectly respond to CO^2	3	
	4	4	
	5	5	
	6	6	
	7		

Dead Space 2017-2-B

Stem: A 75-year-old male with metastatic lung cancer presents acutely short of breath. We will start with Physiology.			
TOPIC	**QUESTIONS**	**KNOWLEDGE**	**NOTES**
Question 1 Dead space **Subject:** Physiology *West's Respiratory Physiology 10th edition pages 19 - 21*	(a) What is the anatomical dead space?	a) The anatomical dead space refers to the airway volume with **ventilation and no blood flow.** The conducting airways (to division 16) **take no part in gas exchange**. Vol = approx. 150mls.	Bold to pass
	(b) How does it differ from physiological dead space? *Prompt: What happens in normal vs diseased patients?*	b) Anatomical dead space is determined by morphology of the airways and lung. Physiological dead space is the volume of airways and lung that does not eliminate CO2. The two dead spaces of volume are **almost the same in normal subjects**, but the physiological dead space is **increased in many lung disease**s due to inequality of blood flow and ventilation in the lung. (VQ mismatch)	Bold to pass
	Bonus Q: How are these different dead spaces measured?	Measurement: Fowler's = Anatomic Dead Space Bohr = Physiological Dead Space *(bonus)*	

Dead Space 2016-1-D

Stem: Moving onto Physiology. The patient is short of breath			
Question 3 Physiological vs anatomical dead space **Subject:** Physiology LOA: 1	What is "dead space"? What types of DEAD SPACE are there?" PROMPT: Explain difference between the two types How is it measured? (bonus)	**Portion of the tidal volume that does not participate in gas exchange.** VT = VD + VA **ANATOMICAL** • **Volume of conducting airways** (without alveoli) – trachea, bronchi, terminal bronchi. • About 150mls of 500ml VT • Determined by: Increased diameter of airways during inspiration and Size & posture of individual **PHYSIOLOGICAL** • **Volume of gas that does not eliminate CO2/not equilibrating with blood** • Same as anatomical DS in normal individuals • **Increased in lung disease** because of inequality of blood flow and ventilation within the lung Anatomical – Fowlers method, Physiological - Bohr method	Demonstrate principle **in bold to pass** Two types of dead space and describe what it is. May mention VQ mismatch Will accept either

Dead Space 2014-2-D

Stem: Moving on to Physiology			
Question 4 Dead Space (pp 19-21) **Subject:** Phys LOA: 1	What is DEAD SPACE? 2. What types of DEAD SPACE are there?" Prompt explain difference between the two types 3. How is it measured? (bonus)	Portion of the tidal volume that **does not participate in gas exchange** $V_T = V_D + V_A$ 1. **ANATOMICAL** • Volume of **conducting airways** – trachea, bronchi, terminal bronchi (16 gen) • About 150mls of 500ml V_T • Measured by Fowler's method • Determined by: o Increased diameter of airways during inspiration o Size & posture of individual 2. **PHYSIOLOGICAL** • Volume of gas that **does not eliminate CO_2** • Same as anatomical DS in normal individuals • **Increased in lung disease** because of inequality of blood flow and ventilation within the lung Measured by Bohr method	Demonstrate principle of bold to pass Two types dead space and describe what it is

TOPIC: Dead space in the lung _____ **NUMBER:** _____

OPENING QUESTION	What is the definition of dead space in the lung?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 Volume of lung not involved in CO^2 $elim^n$	1 Can you describe anatomical and physiological dead space?	
	2 Anatomical and physiological	2	
	3 Vols nearly same (150 ml or 0.2-0.35 V_t) in health	3	
	4	4	
	5	5	
	6	6	
	7	7	
	8		
SECOND QUESTION (if needed)	How can the physiological dead space be measured?		
POINTS REQUIRED	1 Bohr's method calculates fraction of tidal volume by measurement of mixed expired CO2 and arterial CO2	1 Direct?	
	2 $V_D = V_T \times (Pa_{CO2} - PE_{CO2}) / Pa_{CO2}$	2	
	3	3	
	4		
THIRD QUESTION (if needed)	What will lead to increased physiological dead space?		
POINTS REQUIRED	1 V/Q mismatch= Non-perfused alveoli and Alveoli with excessive ventilation	1 Can you give clinical examples	
	2	2	
	3	3	

Exercise 2012-1

Question 2 LOA: 1	2.1 What are the effects of exercise on the respiratory system? Prompt(s): "What are the effects on: gas exchange; OR ventilation; OR pulmonary blood flow."	• **Gas exchange:** o ↑Respiratory uptake and consumption of O_2 (VCO_2) and production and excretion of CO_2 (VCO_2) - increases by 10-20 times; o ↑Lung diffusing capacity due to ↑diffusing capacity of the membrane and the pulmonary blood volume; o ↓**Ventilation**–perfusion inequality; • Ventilation: o ↑Respiratory rate; o ↓Functional residual capacity (FRC); o ↑Tidal volume (TV); o ↑Minute ventilation. • **Pulmonary blood flow:** o Distension and recruitment of pulmonary vessels increases total cross-sectional area of the pulmonary vasculature; o ↑Total pulmonary blood volume; o ↑Cardiac output and pulmonary blood flow; o ↑Pulmonary vascular pressures; o ↓Pulmonary vascular resistance. • Other respiratory effects: o ↑Respiratory exchange ratio (R) from 0.8 to 1.0 due to carbohydrate metabolism and may exceed 1.0 due to anaerobic glycolysis; o The Hb-O_2 dissociation curve shifts to the right in the tissues and back to the left in the lungs; o Additional capillaries open in peripheral tissues;	One effect from each **bolded** section and at least six to pass.
	2.2 What changes occur in blood gases during exercise?	• Arterial blood gases are little affected by moderate exercise but at high workloads pH falls due to lactic acidosis, $PaCO_2$ often falls to compensate for the acidosis and PaO_2 rises; • Arteriovenous pH, PaO_2 and $PaCO_2$ differences increase.	Basic understanding of the effects on blood gases.

Exercise 2004-2

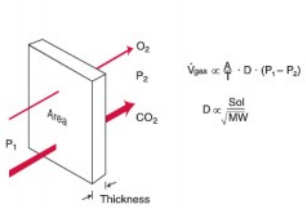
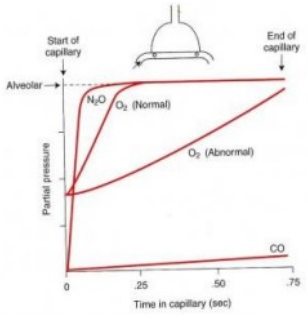
TOPIC: Respiratory system and exercise _____ **NUMBER:** _____

OPENING QUESTION	In the respiratory system, what changes occur with exercise?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 Inc O^2 demand and CO^2 production	1 Any changes?	
	2 Inc ventilation rate, tidal vol, minute vol	2	
	3 Inc pulmonary blood flow	3	
	4 More even V/Q ratios	4	
	5 Dec physiological dead space	5	
	6 Greatly inc O^2 uptake and CO^2 offloading	6	
	7 (Mean ABGs do not change until late)	7	
	8		
SECOND QUESTION (if needed)	What happens to the pulmonary circulation during exercise?		
POINTS REQUIRED	1 Flow increases	1 Vs arterial?	
	2 Distension, recruitment of vessels	2	
	3 Inc cross-sectional area	3	
	4 Thus pressures fall	4	
	5	5	
	6	6	
	7		
THIRD QUESTION (if needed)	What changes occur in venous gases during exercise?		
POINTS REQUIRED	1 Total CO^2 carried rises	1 Vs arterial?	
	2 Dec O^2 because inc extraction	2	
	3 Lactic acidosis	3	
	4	4	

Fluid Balance in the Alveoli 2010-1

3 What are the factors which keep fluid out of the alveoli?	a)Starling's Law (Theoretical Concept, exact values of pressures unknown) 1. Hydrostatic pressure (of column of blood) a. In the capillaries (positive thus outwards) = Pc b. In the interstitium (probably negative and thus also outwards) = Pi 2. Colloid osmotic pressure (of proteins in blood) a. In the capillaries (inwards) = πc b. In the interstitium (outwards) = πi Net pressure probably slightly outward Net fluid out = $K[(Pc-Pi)-\sigma(\pi c-\pi i)]$ K = Filtration Coefficient σ = reflection coefficient (capillary wall barrier) b)Lymphatic drainage c)alveolar epithelial cells	Demonstrate understanding of hydrostatic pressure & colloid osmotic pressure One other

Gas Diffusion 2017-2-B

Stem: Moving onto Physiology. He is dyspnoeic and his oxygen saturation is 90% on high flow oxygen			
Question 4 **Subject:** Physiology West's Respiratory physiology 10th edition. P 29 LOA:	1.. Explain Fick's law of diffusion. *Prompt :Fick's law of diffusion describes the factors influencing the diffusion of gases across the alveolar wall* *Prompt for diffusion constant: What factors determine the diffusion constant in Fick's law?*	1. Gases diffuse across a surface by passive diffusion. Fick's law says that the rate of diffusion is directly proportional to the area of the diffusion membrane, the pressure gradient across the membrane and the diffusion constant. It is inversely proportional to the thickness of the membrane. 	1**. Concept explained to pass.** Or adequately explains the formula – if written Dependent on the substances making up the membrane AND proportional to the solubility of the gas across the membrane - inversely proportional to the square root of the molecular weight of the gas.
	2. What is the difference between a diffusion limited and a perfusion limited gas? *Prompt – you may draw a graph to illustrate your answer*	2. <u>A perfusion limited gas</u> is one where the partial pressure on both sides of the membrane equilibrates rapidly such that no further diffusion into the blood can occur from the alveoli unless the blood perfusion rate increases. In the graph (see picture), there is no gap between the alveolar pp of the gas at the time blood leaves the pulmonary capillary. <u>A diffusion limited gas</u> is one where the partial pressure of the gas does not achieve equilibration in the time that blood spends in the pulmonary capillaries. In the graph, there is a gap between the pp of the gas at the end of the pulmonary capillary perfusion time.	2**. Concept explained to pass**. 

Gas Diffusion 2012-2

Question 2 LOA: 1	1.What factors influence the rate of oxygen transfer from the alveolus into the pulmonary capillary? 2. How do we measure diffusion capacity?	Passive diffusion Determined by **Ficks law of diffusion** **Vgas α $\frac{A}{T}$. D. (P1-P2)** (Affected by surface area (A), membrane thickness(T), Difference in partial pressures gas between alveolus (P1) and Capillary(P2), and diffusion constant(D) D α $\frac{\text{gas solubility}}{\sqrt{\text{Molecular weight gas}}}$ **Carbon monoxide** is used for measurement because **its uptake is diffusion limited**(not depend on amount blood available only on diffusion properties bld-gas barrier) (single breath method test can be used)	Need to know the **basic Fick equation** to pass. As bonus would need to explain why this is so – ie because the CO is so avidly taken up by Hb that the concentration gradient across the membrane never reduces, so membrane properties define flux

Gas Diffusion 2010-2

Question 5	a. What factors influence the rate of transfer of oxygen from the alveolus into a pulmonary capillary b. Could you give some clinical examples of when these may be affected	A)Process is passive diffusion (ficks law of diffusion) Affected by – surface area, membrane thickness, gradient of p O2 (o2 in alveolus and O2 binding capacity of Hb) Also – constant –solubility and MW ($V = A/T \times D \times (P_1-P_2)$ $D = Sol/MW^{1/2}$ b) exercise alveolar hypoxia and thickening of blood gas barrier	2 to pass

Gas Diffusion 2006-1

TOPIC: Diffusion across the alveolar-capillary membrane **NUMBER: 2**

OPENING QUESTION	What factors affect the diffusion of gases across the alveolar capillary membrane?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	Ficks law diffusion is proportional to tissue area and concentration gradient of gas and inversely proportional to the tissue thickness x R R = diffusion constant and relates to gas and tissue solubility		Need to mention diffusion, concentration gradient & thickness.

Gas Exchange 2003-2

TOPIC: Perfusion and diffusion limited gas exchange ____ **NUMBER:** _____ 5c

OPENING QUESTION	Describe the difference between diffusion limited and perfusion limited gas exchange in the lung.	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 Blood in pulmonary capillary has 0.75 seconds for gas exchange	1	
	2 Ability to reach partial pressure equilibrium depends on reaction with substances in the blood.	2	Pass if describes both adequately
	3 No reaction with substances in blood – gas dissolves only on plasma – rapid equilibrium reached, gas uptake limited by perfusion	3	
	4 Example of N2O as perfusion limited	4	
	5 Describes reaction of CO with Hb, such great affinity that PCO in capillary falls rapidly – slow equilibrium, diffusion limited		
SECOND QUESTION (if needed)	Explain how oxygen exchange is limited across the pulmonary capillary?		
POINTS REQUIRED	1 Perfusion limited	1	Must pass
	2 Describes O2 and Hb combination, and time frame of combination (0.3 sec)	2	Extra mark for correct answer here
	3	3	
THIRD QUESTION (if needed)	What would you expect to be the effect of heavy exercise on oxygen uptake in the pulmonary capillary?		
POINTS REQUIRED	1 Describes reduced time for combination with Hb (0.25 seconds), possible reduced O2Hb saturation	1	Bonus Points
	2 Describes possible effect of altitude	2	Bonus Points
	3		

Hypoxaemia 2016-2-C

Stem: Moving onto Physiology.			
Question 2 Alveolar gas equation **Subject:** Phys LOA: 1	What are the causes of hypoxaemia in general? [What are the lung related causes?] [can you give examples?] [How does that pathology cause hypoxaemia?]	**Hypoventilation**: Eg Drugs – Morphine/ barbiturates/ Chest wall damage/ Resp m. paralysis **Diffusion limitation**: Impaired diffusion process of oxygen across the Pulmonary capillary Eg Exercise/ thickened blood gas barrier state, Low O_2 mixture inhaled **Shunt**: Shunt refers to blood entering the arterial system without going through ventilated areas of the lung.Eg: Abnormal vascular connection (AV fistula/ CCHD defecet in R/L sides of heart). **Ventilation – Perfusion inequality** most common. Vent/Perfusion ratio determines gas exchange for any Resp unit. Regional variance exists. Hypoxaemia caused by V/Q mismatch cannot be eliminated with increased ventilation. Eg Pulmonary Embolism	3 of 4 Bold + 2 examples altogether
	What is the Alveolar gas equation? [Prompt: What is the calculation?]	Useful formula to measure the relationship between the fall in PO_2 and the rise in PCO_2 that occurs in Hypoventilation. Alveolar arterial difference (A/a gradient) – a useful measure of the V/Q inequality. **$PAO2 = PIO_2 - \frac{PACO_2}{R} + F$** Where: PAO_2 = Alveolar Oxygen partrial pressure PIO_2=Partial pressure Inspired (dry) Oxygen: FIO_2 less 47 mmHg Water Vapour (~ 149mmHg) when FiO2 21% and 760 mmHg. $PACO_2$ = Measured $PaCO_2$ R= Respiratory Quotient is the given CO2 production/ O2 consumption determined by the metabolism at steady state. Typically 0.8. Also called Respiratory Exchange ratio. F is a small correction factor for inert gases (typically 2mm Hg and can be ignored).	Describe formula Define all Bold terms.
	How is it used clinically? (Bonus)	A/a gradient calculated by subtracting the measured PaO_2 (arterial) from the calculated PAO_2.	Bonus

Hypoxemia 2013-2-B

Question 2 PHYSIOLOGY	1. What are the possible physiological causes for hypoxemia in this man?	Hypoventilation Diffusion limitation Shunt V/Q mismatch	Need 2 /4 to pass or a good understanding of the concepts
LOA: 1	2. What is the alveolar gas equation ?	$PAO_2 = PIO_2 - \frac{PACO_2}{R} + F$	Numbers ok
	3. Explain the concept of the A-a gradient.	Difference between the measured and the predicted paO_2.	Need the basic concept

Hypoxemia 2010-1

2 a). What are the causes of hypoxaemia in a person breathing room air	Hypoventilation, diffusion limitation, shunt, V/Q inequality	Need 3/4
2 b). Explain why ventilation-perfusion inequality causes a reduced arterial P_{O_2} while arterial P_{CO_2} remains relatively normal	Basically due to the differences in their dissociation curves. If one could in isolation cause V/Q inequality then gas exchange would deteriorate with hypoxia and hypercapnia. But the chemoreceptors act to increase ventilation. P_{CO_2}- The **CO_2 dissociation curve is linear** at the working range. The increased ventilation is able to correct the P_{CO_2}.by increased CO_2 output, particularly in units with high V/Q ratios P_{O_2} –the **oxygen dissociation curve is not linear**. So high V/Q areas can only boost their P_{O_2} a little with increased ventilation. Conversely very low V/Q areas have proportionally lower P_{O_2} (close to mixed venous). Overall P_{O_2} is low.	Bold plus demonstrate understanding

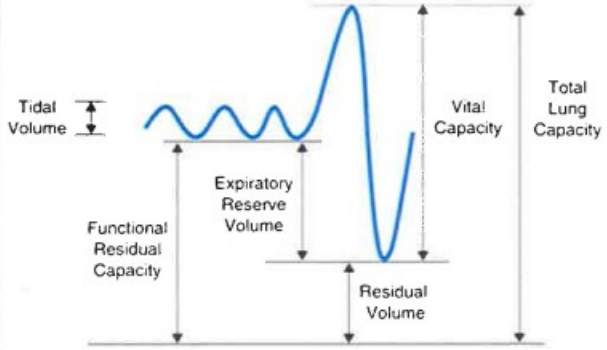
Hypoxia 2014-1-B

Stem: Moving now to your physiology question: He is noted to be hypoxic			
Question 2 Hypoxia **Subject:** Phys LOA: 1	1. Describe the different types of tissue hypoxia. Prompt: Hypoxia is deficiency of O2 at the tissue level	1. **Hypoxaemia (hypoxic hypoxia)** – arterial PO2 reduced 2. **Anaemic hypoxia** – arterial PO2 normal but Hb reduced 3. **Ischaemic/ stagnant hypoxia** – blood flow & O2 delivery decreased 4. **Histotoxic hypoxia** → because of toxin cells cannot use it	3 to pass
	2. Describe the respiratory mechanisms leading to hypoxaemia and give examples?	**Reduced ventilation** (asthma), **VQ mismatch (PE)**. Shunt (CHD), diffusion limitation (APO/LVF/pulmonary fibrosis)	2 mechanisms and correct example
	3. Describe the clinical effects of acute hypoxia	Disorientation, confusion, headache, LOC, Tachycardia +/- , hypertension, hypotension, AMI, arrest, diaphoresis, tachypnoea	2 to pass

Lung Defence Mechanisms 2005-2

3.1, Defence, Metabolic, Endocrine functions of lungs	Describe the lung defence mechanisms What are the metabolic and endocrine functions of lungs?	Cooling or warming air; hairs in nasal passages; NO in paranasal sinuses is bacteriostatic; lymphoid tissue in adenoids, tonsils; **secretory IgA** in bronchi; **ciliary escalator**; coughing reflex; release of PGE2 protects epithelial cells; alveolar **macrophages** are phagocytic; Synthesised: surfactant; fibrinolytic system for pulm.vessels; PGs; histamine, kallikrein. Inactivated: PGs, bradykinin, adenine, serotonin, noradrenalin, Ach. Activated: Angiotensin 1 to 11. Lung receives the entire circulation in one pass. ACE also inactivates bradykinin.	5/10

Lung Volumes 2015-2-C

Stem: Moving onto Physiology.			
Question 4 Lung Volumes and Curves **Subject:** Phys LOA: 1	1. **Draw** a diagram that demonstrates the components of total lung volume.	Should correctly include TLC, VC, FRC, TV, RV, ERV  	TLC, VC, FRC, TV, RV, ERV **(3/6 to pass)**
	2. What are the typical volumes? Optional: Which of these volumes can be measured in the ED?	TLC ~7000ml, VC ~4500 to 5000 mL, RV ~1200 mL, FRC ~2400 mL, TV ~500mL FEV1, FVC or TV.	**2/4** (reasonable approximations)

Lung Volumes 2015-1-D

Stem: During the procedure, he becomes hypoxic and requires assisted ventilation. We will now move to Physiology.			
Question 2 Lung volumes and curves (West pp 13-16) **Subject:** Phys LOA: 1	1. Draw a diagram that demonstrates the components of total lung volume.	Should correctly include TLC, VC, FRC, TV, RV, IRV, ERV	Bold to pass **TLC, VC, FRC, TV, RV** correct
	2. **In an adult**, what are the typical volumes of these components? [TLC, VC, RV, FRC and TV]	TLC ~7000ml, VC ~4500 to 5000 mL, RV ~1200 mL, FRC ~2400 mL, TV ~500mL	**2/4** (reasonable approximations)
	3. Which lung volumes can be measured in the ED? Extra:How are the other lung volumes measured?	Spirometer for FEV1 and FVC. TV on ventilator Helium dilution or body plethysmography for TLC, FRC and RV	**1/2 spiromoter**

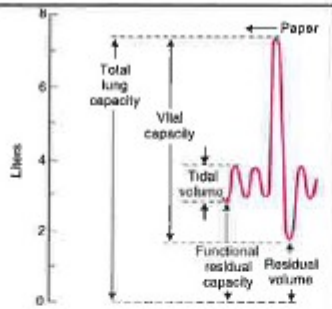
Lung Volumes 2013-1

Question 2 LOA: 1 Lung volumes and capacity	a. Please describe the components of total lung capacity? Prompt: What individual volumes or capacities are described in relation to the total lung capacity or volume.	**Tidal volume**: the volume of gas moved in and out of the lung during normal breathing (500ml) **Vital capacity**: the exhaled gas volume after a maximal inspiration (5.5-6 litres) **Residual volume:** the volume of gas remaining in the lung after maximal expiration (1.5-2 litres) **Functional residual capacity**: the volume of the gas in the lung after a normal expiration (3 litres)	Three of four volumes
	b. Name a method to measure each of these?	**Spirometer can measure tidal volume and vital capacity** Total lung capacity, functional residual capacity and residual volume may be measures by helium dilution or the body plethysmograph	Bold

Lung Volumes 2010-2

Question 5	a. Please draw a diagram showing static lung volumes b. How does physiological dead space differ from anatomical dead space?	a. TV 500ml b. DS 150 ml c. TLC 7L d. FRC 2L e. VC6L a. Anatomical dead space, conducting zones of lung 150 mls b. Physiological dead space- Parts of lung with ventilation but no perfusion	To Pass: Draw diagram Label diagram Prompt for both physiological and anatomical dead space Normally very nearly the same but physiological dead space much greater in disease because of V/Q mismatch, esp with increased airway pressures

Lung Volumes

Question 2 Lung Volumes LOA: 1	Please draw and label a diagram showing a spirometer tracing of static lung volumes.	▪ Tidal volume 500 mL ▪ Functional residual capacity 3L ▪ Residual volume 1.5-2.0 L ▪ Vital capacity 5.5-6L ▪ Total lung capacity 7-8 L 	**The candidate must be able to label the axes, draw a reasonable spirometer tracing and indicate three of the five major volumes.**
	What is residual volume and state a method or methods of measuring this volume?	▪ The residual volume is the volume of gas left in the lung after a maximal expiration. ▪ Residual volume may be measured by: o Helium dilution technique; o Body plethysmography; o Nitrogen washout and measurement. ▪ Helium dilution and nitrogen washout measure only the ventilated residual volume. The body plethysmograph measures the total volume of gas in the lung, including any that is trapped behind closed airways. ▪ In young normal subjects, these volumes are virtually the same, but in patients with lung disease, the ventilated volume may be considerably less than the total volume because of gas trapped behind obstructed airways.	**The candidate must be able to provide a satisfactory definition.**

Metabolic Functions of the Lung 2011-2

Question 2 LOA: 1	Outline the metabolic functions of the lung.	1. **Synthetic function:** synthesis of phospholipids such as dipalmitoyl phosphatidyl choline (**surfactant**), protein synthesis (collagen & elastin), carbohydrates - mucopolysaccarides of mucous 2. **Activation of Angiotensin I to II.** 3. **Inactivates bradykinin**, serotonin, PGE_1, PGE_2 and PGF_{2a},noradrenaline (30%) 4. Remove leukotrienes (e.g. SRS-A) 5. Secrete IgA	Prompt: 1.What substances can the lung produce? 2.What substances can the lung activate or inactivate? Bold to pass

Metabolic Functions of the Lung 2010-2

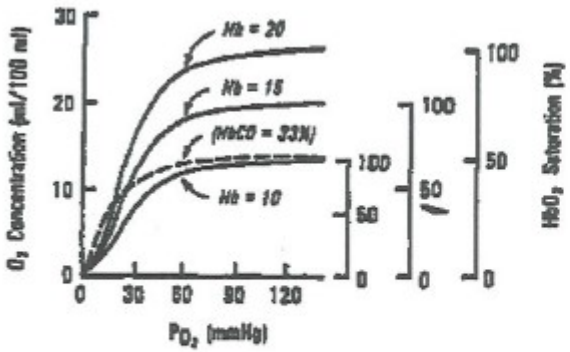
Question 5	What are the metabolic functions of the lung? (Prompt – What substances are metabolised in the lung?)	Metabolism of vasoactive amines a. Activation of Angiotensin 1 → AT 2 (ACE in capillary endothelium) b. Inactivation of bradykinin (ACE); PGs E/F c. Uptake & storage of Serotonin d. Arachadonic acid metabolites → leukotrienes / SRS-A & Prostaglandins Synthesis of a. Surfactant b. IgA c. Phospholipids d. Proteases (collagen/elastin breakdown)	2 of each

O2 Carriage 2006-1

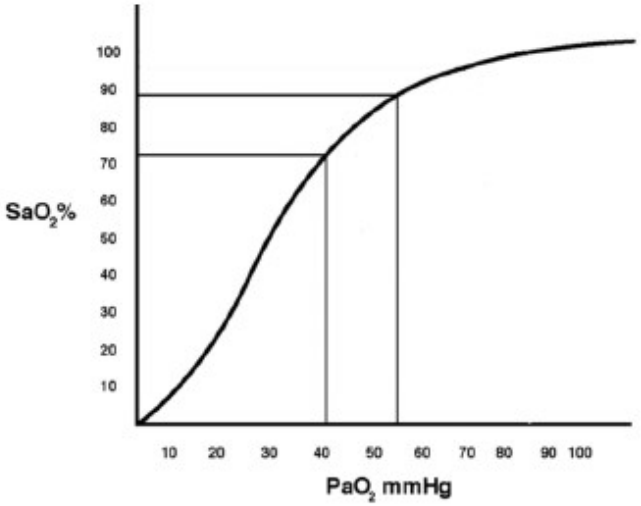
TOPIC: Oxygen & CO2 in the blood **NUMBER: 2**

OPENING QUESTION	How is oxygen transported in the blood?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	O2 dissolved (0.0003ml/100mlblood/mmHg), Heme protein		Need to name both.
SECOND QUESTION (if needed)	Describe the oxygen dissociation curve.		
POINTS REQUIRED	Name the axis Hb saturation and pO2 & name 50% saturation (pO2= 27 mmHg) or p40 = 75% saturation.		
THIRD QUESTION (if needed)	In what forms are carbon dioxide transported in the blood?		
POINTS REQUIRED	CO2 dissolved 10%, Bicarbonate (60%) Carbamino compounds (30%) deoxygenated blood is better at carrying CO2 = Haldane effect		Need to say all 3 pass.

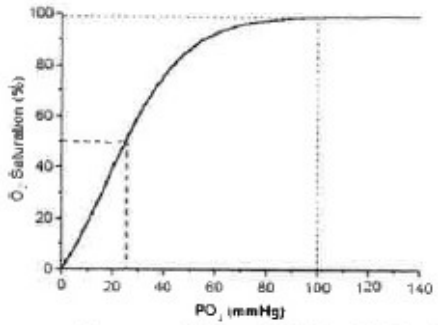
O2 Concentration Curve 2009-2

Question 2:	**Please draw the curve demonstrating the relationship between O_2 concentration v pO2** **How does this change in anaemic and polycythaemic individual?** **What is the effect of carbon monoxide on these curves?**	 1) decrease in effective Hb per percentage 2) COHb shift to Left	Prompt if draws saturation dissociation curve

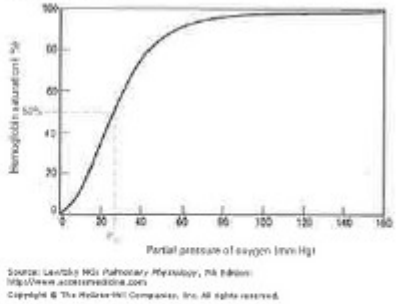
O2 Dissociation Curve 2014-2-C

Stem: We are starting with physiology. A 40 year old man with acute pancreatitis Is hypoxic.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Gas transport to the tissues (West Chp 6) **Subject:** Phys LOA: 1	How is oxygen carried in the blood? Prompt: Which dominates	**Dissolved**: amount dissolved proportional to partial pressure (Henry's law)-0.3ml O2/100ml blood at PO2 100mmHg **Most Combined with Hb**: 20.8 ml O2/100ml blood(at Hb level of 15g/dl)	**Bold**
	Draw and label the oxygen dissociation curve		**Draw correct shape and have 2 points of saturations** eg 27mmHg SaO2 50%, 30mmhg SaO2 60% 40mmHg SaO2 75%, 56mmHg SaO2 90%, 80mmHg SaO2 95%, 90mmHg SaO2 97%
	What are the implications of this curved shape? Prompt: what happens to the top + bottom	UPPER- If PO2 alveolar gas falls (eg ARDS in acute pancreatitis) loading of O2 little affected. LOWER- Steep lower part means large amounts of O2 unloaded at peripheral tissues for only small drop in capillary PO2	Explain **concept** of loading and unloading of oxygen
Stem: We are now changing to pathology			

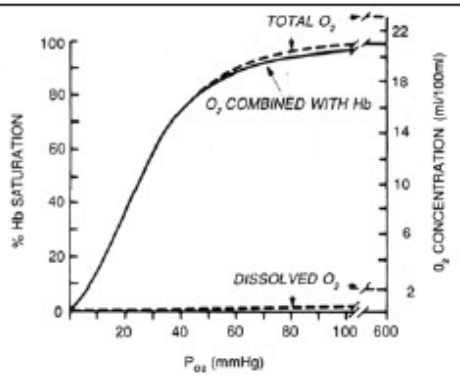
O2 Dissociation Curve 2014-1-B

Stem: Moving now to your physiology question: She is hypoxic with oxygen saturations of 90% on room air			
Question 2 Oxygen / Haemoglobin dissociation curve **Subject:** Phys LOA: 1	1. Please draw and label the oxygen dissociation curve.		Draw correct shape – have points of 90% (58-60) saturation.
	2. What factors can cause the curve to shift to the right (reduced affinity of Hb for O2)?	• Increased temp, PCO2, 2,3 DPG • Drop in pH (increased H+)	At least 3
	3. What are the physiological advantages of this curved shape?	(UPPER) If pO2 alveolar gas falls, loading of O2 little affected. Also, as RBC takes up O2 along pulmonary capillary, diffusion process hastened as large partial pressure difference maintained when most of O2 has been transferred. (LOWER) Steep lower part means peripheral tissues can withdraw large amounts of O2 for only small drop in capillary pO2	Concept of loading and unloading of oxygen being facilitated

O2 Dissociation Curve 2013-1

Question 2 O2 transport LOA: 1	a. Describe how oxygen is carried in the blood.	**Dissolved**: amount dissolved proportional to partial pressure (Henry's law) – 0.3 ml O_2/100 ml blood/100 mm Hg PO_2 **Combine with haemoglobin**: 20.8 mg/100 ml blood.	Need **bold**
	b. Please draw the Oxyhaemoglobin dissociation curve.	See diagram: draw graph to pass, 3 key points (2/3 accurate): examples P50 & 90/60 and 1 other.	
	c. Describe factors that can affect the oxygen dissociation curve.	**Shift to right** by inc H^+ conc, pCO_2, temp, 2,3 diphosphoglycerate to unload oxygen. **Shift to left** with the opposite changes.	2 factors

O2 Dissociation Curve 2011-1

Question 2: Oxygen Dissociation Curve (west 77-80)	2.1 Please draw and describe the features of the haemoglobin-oxygen dissociation curve	 (a) May need prompt for SO_2 at various PO_2. (b) Describe importance of: • Flat upper portion of curve- Hb uploading of O_2 unaffected unless PAO_2 falls significantly; • Steep lower part of curve – large amounts of O_2 unloaded at peripheral tissues for only small drop in capillary PO_2.	To pass: Label the axes Draw an approximately correctly shaped curve Locate at least two points Standard points are 27 mmHg to SO_2 50%; 40 mmHg to SO_2 75%; 56 mmHg to SO_2 90%; 80 mmHg to SO_2 95% and 90 mmHg to SO_2 97%.
	2.2 What factors cause shift in the curve?	2.2 To Right: Increased Temperature, Increased PCO2, Increased H+ (decreased ph) and 2-3 DPG To Left: Reverse of above (and CO as below)	Temp, CO2, pH essential
	2.3 What are the effects of carbon-monoxide on haemoglobin oxygen transport capacity	2.3 CO has 240 times the affinity of O2 for Hb, hence oxygen saturation greatly reduced and Hb oxygen carrying capacity reduced. CO also shifts O2 dissociation curve to left, interfering with unloading of O2	Reduced O2 carrying capacity for both with adequate explanation

O2 uptake 2015-2-B

Stem: He is hypoxic. Moving on to physiology			
Question 2 Oxygen uptake along the pulmonary capillary **Subject:** Phys LOA: 1	(a) In an alveolus, what factors affect oxygenation	**Ventilation, perfusion, diffusion across the blood gas barrier** and alveolar-pulmonary capillary pO_2 gradient	3 Bold to pass
	(b) Describe the oxygen uptake along a pulmonary capillary	**Alveolar pulmonary capillary O_2 gradient** (Alveolar pO_2 = 100mmHg, pulmonary capillary pO_2 = 40mmHg), blood gas barrier thickness 0.3 microns, **RBC transit time = 0.75s** Under normal circumstances, **O_2 uptake is perfusion-limited** (complete in 0.25s) **& alveolar end capillary O_2 difference is minimal.** Rate of rise of end capillary pO_2 is steep – O2-Hb dissociation curve	Must have knowledge of 3 of 4 concepts in bold. Numbers not required to pass.
	(c) How does hypoxia affect oxygenation	**Alveolar pulmonary capillary O_2 gradient is decreased, O_2 diffusion is decreased** & rate of rise of pO_2 for given O_2 concentration in blood is less	Can draw graph to explain (West pages 28-29)

Pulmonary Blood Flow 2011-1

Question 2 Pulmonary blood flow	2.1 Describe the normal distribution of pulmonary blood flow.	2.1 Influenced by gravity-3 main zones Zone 1(apex) PA>Pa>Pv – least blood flow Zone 2(mid) Pa>PA>Pv Zone 3(base) Pa >PA>Pv – Most blood flow	Posture/Hydrostatic pressure and describe 3 zones to pass
	2.2 How is the distribution of pulmonary blood flow actively controlled?	2.2 Hypoxic pulmonary vasoconstriction-alveolar hypoxia constricts pulmonary arteries, directs blood away from poorly ventilated diseased lung areas. Mechanism-NO, endothelin-1, TXA2, low pH, autonomic system	Hypoxic pulmonary vasoconstriction to pass
	2.3 Please explain how cardiogenic pulmonary oedema occurs.	2.3 Starling's Law- differences in capillary and interstitial hydrostatic and colloid osmotic pressures. Significant increases in net outward pressure of Starling equation results in interstitial oedema especially at perivascular and peribronchial spaces. Further increases of outward pressure results in fluid entering alveolar spaces.	Basic description of Starling forces

Pulmonary Blood Flow 2008-2

OPENING QUESTION	**Describe the distribution of blood flow in the lung of an upright subject at rest.**	**COMMENTS**
POINTS REQUIRED	1. **Decreases linearly from base to apex**	Must identify 1, 2 ,3 and 5 to pass
	2. **Due to hydrostatic pressure,**	
	3. **Under normal conditions, flow almost ceases at apex**	
	4. Distribution more uniform with exercise	
	5. **Explanation of West's zones 1 – 3 +/- zone 4** 6. Zone 4 only at very low lung volumes	
PROMPTS	What are the zones of the lung described by West ?	
SECOND QUESTION (if needed)	**What are the main determinants of flow in these three zones ?**	
POINTS REQUIRED	1. **Zone 1 PA>Pa>Pv(not under normal conditions and is alv. dead space)** 2. **Zone 2 Pa>PA>Pv (recruitment)** 3. **Zone 3 Pa>Pv>PA (distension + recruitment)**	Must identify 3 pressures and their relationship to pass
PROMPTS	What pressure gradients determine flow in zones 1-3	
THIRD QUESTION (if needed)	**How does the distribution of blood change when the subject becomes supine?**	
POINTS REQUIRED	1. Blood flow from base to apex is almost uniform but flow in posterior segments exceeds that in anterior segments	
PROMPTS		

COMMENTS

Pulmonary Blood Flow 2007-2

QUESTION: 1. Factors that determine pulmonary blood flow

Question	Required response [Key items marked with*]	To Pass
What factors influence the distribution of pulmonary arterial blood?	1. **Alveolar Hypoxia** * 2 **Gravity** * :3 main zones Z1 (apical) PA>Pa>Pv Z2 (middle) Pa>PA.>Pv Z3 (basal) Pa>Pv>PA 3 vascular resistance pulmonary HT / PE 4 pulmonary disease : asthma /COAD / infection/ infarction/ cancer / fibrosis / pneumothorax / chest trauma 5 vasoactive substances * (NO, endothelin, prostaglandin) 6 low blood pH leads to pulm vasoconstriction 7 Sympathetic stimulation leads to stiff pulmonary arteries leads to vasoconstriction.	* To pass, plus 2 others
What **EXTRA-PULMONARY** factors influence pulmonary blood flow ?	1 blood volume 2 cardiac output 3 atmospheric pressure 4 temperature 5 pathology eg, anaemia, cancer, infection 6 exercise 7 posture	4 of 7

TOPIC: Pulmonary Blood Flow _____ **NUMBER: 1** _____

OPENING QUESTION	How would you calculate pulmonary vascular resistance?	**PROMPTS COMMENTS**	
POINTS REQUIRED	1 R = Change is pressure / Blood flow	1	Required for pass
	2 Normally very low	2 Prompt for comparison with systemic vascular resistance if necessary	Additional information
SECOND QUESTION (if needed)	What are the determinants of pulmonary vascular resistance?		2/4 = pass Others are additional info
POINTS REQUIRED	1 Increasing pressure as in exercise causes a reduction in resistance by recruitment and distension	1	
	2 Large lung volumes pull open extra-alveolar vessels but may narrow pulmonary capillaries so that resistance rises	2	
	3 Small lung volumes also cause increased resistance of extra-alveolar vessels because smooth muscle tone closes them if critical opening pressure is not reached	3	
	4 Hypoxic pulmonary vasoconstriction directs blood away from hypoxic lung	4	
THIRD QUESTION (if needed)	Describe Hypoxic Pulmonary Vasoconstriction?		2/4 = pass Others are additional information
POINTS REQUIRED	1 Alveolar hypoxia constricts pulmonary blood vessels	1	
	2 Direct effect of alveolar PO2 on smooth muscle	2	
	3 Important at birth	3	
	4 Directs blood away from hypoxic areas	4	

Pulmonary Blood Flow 2005-1

Regional differences in pulmonary blood flow	Describe the distribution of blood flow in the lungs	Linear increase from top to bottom 3 zones explained by hydrostatic pressures	
	Explain how V/Q matching varies from apex to base in the normal lung	Slow increase in ventilation from top to bottom but not as much as perfusion. Highest V/Q at apex	
	What factors effect pulmonary vascular resistance?	Hypoxia - arteriolar smooth muscle to contract One of low pH, autonomic or passive factors	

Pulmonary Compliance 2016-1-B

Stem: Moving onto Physiology.			
Question 3 Pulmonary Compliance LOA: 1	*a. What is lung compliance?*	**Change in lung volume per unit change in airway pressure (ΔV/ΔP) – measure of lung "distensibility"** Normally 200mls/cm H_2O. It occurs because of the opposing inward elastic recoil of the lungs and outward recoil of the chest wall. It is represented by the slope of the nonlinear lung pressure-volume curve.	Concept to pass
	b. What physiologic factors affect lung compliance?	Age, volume of the lung, phase of respiration (lower in deflation/expiration than inflation/inspiration), surfactant.	3 to pass
	c. How is lung compliance affected in emphysema?	**Compliance is increased** because of **loss of lung elasticity** / destruction of lung connective tissue & elastin (easy to inflate but reduced capacity to recoil). Patients have to force their expiration to expel air from lungs. Resultant increase in FRC.	Bold to pass
	d. What are the physiologic effects of pulmonary surfactant?	Lowers alveolar surface tension, increases lung compliance, reduces work of breathing, improves the stability of alveoli and keeps the alveoli "dry".	3 to pass

Pulmonary Vascular Resistance 2012-2

Question 2 [Pulmonary vascular resistance] LOA: 1	**2.1** What two mechanisms allow pulm vasc resistance to fall? (such as during exercise) **2.2** What other influences are there on pulm vasc resistance?	a. '**Recruitment**' of normally closed (non perfused) pulm capillaries b. '**Distension**' at higher vasc pressures, from near-flat to circular cross-section capillaries a. **Lung volume**: *when low*, pulm vasc resistance increased, due to smooth muscle and elastic tissue contraction: *when high*, again rises due to capil stretching and reduction in calibre b. **Hypoxia**: *increases* pulm vasc resistance from pulm vasoconstriction c. **Drugs**: *increased* by serotonin, histamine, norepi (contract vessel smooth muscle). : *decreased* by acetyl choline and isoprenaline (isoproterenolol)	Bold to pass Lung volume + one other.

Pulmonary Vascular Resistance 2009-2

Question 2:a) Score:	**What are the major factors that effect pulmonary vascular resistance in the normal lung?**	1) ↑Art or 2) Ven Pressure 3) Lung volume (U/J shaped curve) 4) Alveolar hypoxia > increased PVR via hypoxic vasoconstriction 5) Vascular Smooth Muscle Tone - response to endogenous/ exogenous factors 6)Area of lung (apex partic < base) 7) Position change 1	(A > V) (recruit – low P) (distension (high P) Low vol- collapsed ex-alveolar vessels Intermed Vol – vessels open High Vol – compressed alveol vessels (pulled open v normal elastic -cap 1st) (complex: Ipleural P < CO, alveolar P > capillary + caps squashed in alveoli) **Pass/Fail 3 of 6 , extra marks for detail in** eg nitrates, Ach, Isoprenaline, NO, decrease PVR; Increased sympathetic tone, serotonin, histamine and norepinephrine increase PVR, endothelin, thromboxane A2
b)	**Why is pulmonary flow so sensitive to pulmonary vascular pressures?**	) V low Pressure system – few resistance vessels 2) Easily distensible vessels 3) Recruitment 4) Only just enough P for normal gravity/ position to get apical flow 2/4 to pass	P just enough to reach only standing but (dependent lung may collapse) -due to < art pressure in low pressure system- partic if poor output V thin walls Vasc bed expands + geometry with alveolar expansion Surrounding IP/ alv P v significant effect on output Additional info 1/10th syst P (5-15 A-V diff) (low vol smooth muscle/high P and higher lung vol) (geometry-low P) (distension/ effects on cap) (due to v low P in system)

Pulmonary Vascular Resistance 2007-2

Question	Required response [Key items marked with*]	To Pass
Please describe the relationship between pulmonary vascular resistance and pulmonary vascular pressure? PROMPTS: What mechanisms are involved in the vascular response to rising pulmonary vascular pressure?	A low resistance system Capacity for resistance to DECREASE with INCREASE pressure* (both INCREASED pulmonary art & INCREASED pulm venous) Mechanisms: vascular 'recruitment' (with rises in pressure from low levels); vascular 'distension' (with rises in pressure at higher levels) * Diagram 	* to pass Drawing diagram not essential
How does lung volume influence pulmonary vascular resistance?	Vascular resistance initially decreases as lung volume increases, then rises (see diagram below) * At very low lung volumes (eg. lung collapse) must reach a 'critical opening pressure'(several cm H2O above downstream pressure) to enable any flow * Very high lung volumes, when alveolar pressure exceeds pulmonary capillary pressure, pulmonary vascular resistance will increase (pulm capillaries squashed). 	* to pass Drawing diagram not essential

Shunt 2007-1

TOPIC: Physiological shunt in the lung _____ **NUMBER:** _____

OPENING QUESTION	Explain the difference between alveolar and arterial oxygen concentrations in the healthy adult.	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 Physiological shunt of lung ($P_AO_2 > P_aO_2$)		Essential plus 1 reason.
	2 Blood enters arterial system without passing through a ventilated area of lung		
	3 Bronchial arterial blood flows to pulmonary veins		
	4 Coronary arterial blood flows to coronary veins then thebesian veins in left ventricle		
	5 Atelectasis in lung		

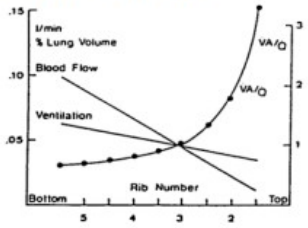
Surfactant 2009-1

Question 2: Role of surfactant and applied Laplace's Law (West 99-104)	i) In the lung, what is surfactant and how does it work?	a) Surfactant is a phospholipid. Dipalmitoyl phosphatidylcholine (DPPC) is an important constituent) b) Produced in type 2 alveolar cells. Lamellated bodies within them are extruded into the alveoli and transform into surfactant. c) Fast synthesis with rapid turnover d) Formed relatively late in foetal life. e) With surfactant present, surface tension changes greatly with surface area. It falls to very low values when area is small f) Molecules of DPPC are hydrophobic at one end and hydrophilic at the other. When aligned on the surface, their **repulsive forces** oppose the normal attractive forces between the liquid surface molecules.	Core knowledge in bold
	ii) What are the physiological advantages of surfactant ?	i) **Reduction in surface tension** is greatest when film is compressed and molecules of DPPC are closest together. ii) **Lower surface tension** in the alveoli **increases lung compliance** and decreases work of breathing iii) **Promotes alveolar stability** (reduces tendency for small alveoli to empty into large alveoli) iv) 3) **Keep the alveoli dry** (surface tension "sucks" fluid into alveolar spaces from capillaries, by reducing hydrostatic pressure in the tissue)	

Surfactant 2005-2

2.1 Alveolar surface tension and surfactant	Describe the relationship of pressure and wall tension in connected bubbles.	Law of Laplace: **P = 4T/r**. Two bubbles connected (same surface tension), the smaller with higher pressure will blow up the larger with lower pressure. Smaller bubble will collapse.	
	What are the effects of surfactant in alveoli?	**Surfactant reduces surface tension.** Alveolar bubbles are stable because of very low surface tension when small (on expiration). Hysteresis curve demonstrates very low pressures on expiration to small volumes = bubble stability. Increased **compliance** = ease of expansion. **Also keeps alveoli dry** = opposes transudation fluid into bubble.	
	How does surfactant achieve this?	**Bipolar molecules oppose the normal increasing attracting forces as molecules get closer in a smaller surface.** The ends of surfactant molecules repel each other and oppose collapse.	

V/Q 2017-2-A

Stem: Moving on to Physiology.			
Question 2 V/Q inequality **Subject**: Physiology LOA: 1	a) Describe the normal relationship between ventilation and perfusion in an upright lung. *Prompt 1: How does gravity affect the ventilation & perfusion in the lung?* b) What conditions can increase V/Q mismatch? c) Which tests can be done in clinical practice to demonstrate a V/Q mismatch? *Prompt 3: Is there a calculation we can perform on the ABG we had earlier?*	a) Pulmonary circulation is affected by gravity. a. **Apex**: Less blood flow, larger alveoli, slightly less ventilation; **ventilation > perfusion**, **high V/Q ratio**. b. Middle: ventilation = perfusion, V/Q = 1 c. **Base**: More blood flow, smaller alveoli, more ventilation; **perfusion > ventilation**, **low V/Q ratio**. b) **Pulmonary embolism** (high V/Q ratio), pulmonary oedema, pneumonia, emphysema (low V/Q ratio). c) **A-a gradient** (also V/Q scan, CTPA)	Candidate may draw graphs in West, Chapter 5. Bold plus concept  b)Bold plus 1 to pass c) Bold to pass

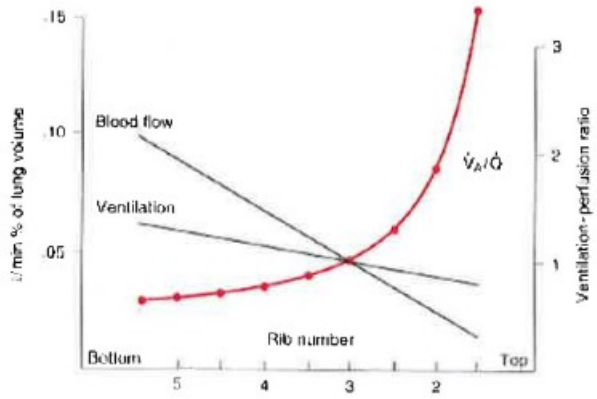
V/Q 2015-1-C

Stem: Moving onto Physiology in a NORMAL lung.			
Question 4 Regional Gas Exchange Subject: Phys LOA: 1	1. What happens to the V/Q ratio from top to bottom of the upright lung? Prompt: What happens to the relative values of ventilation and perfusion? 2. Explain the reasons for the normal Alveolar-arterial 0_2 difference? (Extra question = formula for A-a Gradient)	1. **Both ventilation and perfusion increase with blood flow (perfusion) (Q) increasing more than ventilation (V)** and this results in **V/Q ratio DECREASING down the lung.** 2. Normally 5-10 mmHg. A-a Gradient = measure of the difference between alveolar and arterial concentration of O_2 • Even though P Alv 0_2 at apex 40 mm Hg above base, **most of blood flow (Q) comes from base where P Alv 0_2 is low** -> decrease in P Art 02 • **Shunt:** Bronchial blood & coronary blood Also non-linear shape of 02 dissociation curve means that addition of small amount of shunted blood with low 0_2 concentration greatly decreases $P0_2$ of arterial blood and units with high $P0_2$ have little effect on 0_2 concentration because curve is flat at high 0_2 concentration $PA02 = PI02 - \frac{PAC02}{R} + F$	1. 3 of 3 bold to pass (be able to explain concept). 2. Bold.

V/Q 2012-2

Question 2 LOA: 1	a) What happens to normal ventilation, perfusion and the ventilation-perfusion ratio (V/Q) from top to bottom of the upright lung? b) Explain the reasons for the alveolar-arterial O_2 difference ?	a) **Both ventilation and perfusion increase** with **blood flow (perfusion) (Q) increasing more than ventilation (V)** and this results in **V/Q ratio DECREASING down the lung.** b) Normally 4 mmHg 1)Even though P Alv O_2 at apex 40 mm Hg above base, **most of blood flow (Q) comes from base where P Alv O_2 is low** → decrease in P Art O_2 2)Also **non-linear shape of O_2 dissociation curve means that addition of small amount of shunted blood with low O_2 concentration greatly decreases P O_2 of arterial blood** and units with high P O_2 have little effect on O_2concentration because curve is flat at high O_2 concentration	a) 3 of 3 bold to pass (know it all) b) 1 of 2 bold to pass OK Need to discuss both mechanisms

V/Q Mismatch 2016-2-C

Stem: Moving onto Physiology. She is hypoxic.			
Question 3 VQ mismatch **Subject:** Phys LOA: 1	What does Ventilation- Perfusion ratio mean?	The concentration of oxygen (PO_2) in any Respiratory unit is determined by the ratio of the amount of air getting to the alveolus (ventilation) and blood flow through the Pulmonary capillary (Perfusion). V/Q ratio 0.8 (4.2 litres gas flow/ 5.5L blood flow)	Definition
	In the upright lung, how does the V-Q ratio change? [Prompt: Can you graph the distribution of ventilation and Blood flow in the upright lung?] [Can you draw a diagram of the lung showing the V-Q Ratios?]	**Ventilation increases** slowly from top to bottom of the lung, and **perfusion increases more rapidly**. V/Q perfusion ratio DECREASES down the lung. It is **HIGH at the top** of the lung (where blood flow is minimal) and much **LOWER at the bottom** of the lung.  **Figure 5-8.** Distribution of ventilation and blood flow down the upright lung (compare Figures 2-7 and 4-7). Note that the ventilation-perfusion ratio decreases down the lung.	Bold If used graph must include V/Q relationship

V/Q Mismatch 2014-2-C

Stem: A 20 year old woman presents with a rash and dyspnoea. Her oxygen saturation is low. **We are starting with physiology**			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 VQ mismatch (West Chp 5) **Subject:** Phys LOA: 1	1. What are the causes of hypoxemia in a patient breathing room air?	1. Hypoventilation 2.Diffusion limitation 3. Shunt 4. Ventilation/perfusion (V/Q) inequality	**3 of 4 to pass**
	2. How does the ventilation/perfusion ratio change in different regions of the lung?	**V/Q ratio is high at apex (blood flow minimal) and decreases down the lung to the base.** PO2 highest at apex but blood flow is greatest at the base where PO2 is lowest (can be 40mmHg difference) Respiratory exchange ratio (CO2 output/O2 uptake) highest at apex where blood flow is lower	**BOLD + general concepts to pass**
	3. What is the effect of ventilation-perfusion inequality on arterial PO2 and arterial PCO2? *Prompt if required* Why does V/Q inequality cause reduced arterial PO2 while arterial PCO2 remains relatively normal?	**Much greater influence on PO_2 than CO_2.** **O2 dissociation curve nonlinear**. Areas with high V/Q ratio add relatively little O2 with increased ventilation. Whereas areas with low V/Q ratio have lower PO2 (close to mixed venous) overall PO2 is reduced **CO2 dissociation curve is linear** in the working range. Chemoreceptor stimulation increases ventilation and CO2 output especially in lung areas with high V/Q ratios. normal PCO2 (minimal change)	**BOLD + demonstrates understanding**

V/Q Mismatch 2008-1

Topic	Questions	Core Knowledge	Score
1.1 VP inequality (West pp 67-72)	Describe the relationship between ventilation and perfusion of the lung in a person while standing?	• Max ventilation 3-4x greater at apex • PO_2 40mmHg higher at lung apex • Max perfusion basally Q nearly 20x greater at base • Prompt: are there regional variations in either	
	What are the effects of V/Q inequality on gas exchange?	• V/Q inequality impairs uptake or elimination of all gases • Majority of blood returns from lung bases where the oxygen saturation is low • Results in blood PO2 being lower than that of mixed alveolar PO2	
	What effect does increasing ventilation to the lungs have on arterial PO2 and PCO2	• PCO2 reduces much more than PO2 increases	/2

TOPIC: Ventilation Perfusion Inequality_____ **NUMBER: 1** _____

OPENING QUESTION	What is the effect of ventilation perfusion inequality on gas exchange?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 Impedes exchange of oxygen and carbon dioxide	1 Both gases	Pass = 2/3
	2 Hypoxia which cannot be corrected by increased ventilation	2	
	3 Hypercapnia can be corrected by increased ventilation	3	
SECOND QUESTION (if needed)	Can increasing ventilation correct these problems?	Both gases	Pass = oxygen explanation Others additional information
POINTS REQUIRED	1 The oxygen dissociation curve is s shaped which means that increasing ventilation to units with high VQ ratios cannot compensate for the shunt caused by low VQ units	1	
	2 The carbon dioxide dissociation curve is more linear so that increasing ventilation will blow off CO_2 from lung units with both high and low VQ ratios	2	
THIRD QUESTION (if needed)	How can we determine the effect of VQ mismatch on oxygenation in clinical practice?	Ask about AA gradient if candidate does not volunteer it	
POINTS REQUIRED	1 Calculate the AA gradient = PAO_2-PaO_2	1	Prefer equation to pass
	2 PAO_2=PIO_2-$PaCO_2$/R	2	
	3 Give normal values for each	3	

V/Q Relationships 2016-1-A

Stem: Moving onto Physiology. The patient is hypoxic			
Question 4 Ventilation-perfusion relationships **Subject:** Phys LOA: 1	a) What are the physiological causes of hypoxaemia?	Hypoventilation, diffusion, shunt, ventilation-perfusion inequality.	Need 3 to pass
	b) How does ventilation-perfusion inequality result in hypoxaemia?	Lung units with low V/Q ratio have effluent blood with low pO2 (close to mixed venous). Units with high V/Q ratio have relatively high PO2 but **because of non-linear O2 dissociation curve add little to O2 concentration** (compared to the decrement caused by the low V/Q areas). Overall mixed return has lower O2.	Bold + demonstrate understanding of concepts
	c) How can ventilation-perfusion inequality be measured? (Prompt: is there a formula used to quantify V/Q inequality?)	Using the **alveolar-arterial PO2 difference (the A-a gradient)** – subtracting the (measured) arterial PO2 from the "ideal" alveolar PO2 as given by the alveolar gas equation.	Bold + understanding of concepts
		$PAO2 = PiO2 - PaCO2/0.8$ Normal is 5-10mmHg (increases with age)	
	Extra Q: Why does the PCO2 remain relatively normal in the setting of ventilation-perfusion inequality?	The CO2 dissociation curve is linear in the working range. An increase in PCO2 stimulates chemoreceptors → increase ventilation	

Work of Breathing 2012-1

Question 2 LOA: 1	2.1 What factors determine the work of breathing?	• **Elastic forces** of the lungs and chest wall • **Viscous resistance** of the airways and tissues	Must understand both to pass. Prompt if necessary.
	2.2 What variables affect elastic workload?	**Larger tidal volumes** increase elastic workload. Elastic workload is increased by **reduced compliance** due to: o Lung volume - a person with only one lung has halved compliance. o Slightly lesser during inflation than during deflation. o Increased tissue mass - fibrosis or pulmonary congestion or chest wall restriction. o Loss of surfactant	Must understand **both major points**
	2.3 What variables affect viscous resistance?	• Higher respiratory rates increasing flow rates • Decreased airway radius due to: Lower lung volumes; Bronchoconstriction; • Increased air density (eg SCUBA diving) • Increased air viscosity	Must give at least **two examples** to pass.

Work of Breathing 2003-2

TOPIC: Factors that determine the work of breathing. NUMBER: _____5a

OPENING QUESTION	**What factors determine the work of breathing.**	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 elastic forces of the lungs and chest wall		Must pass
	2 viscous resistance of the airways and tissues		Must pass
	3		
SECOND QUESTION	**What variables affect elastic workload?**		
POINTS REQUIRED	1 Larger tidal volumes	1	Must pass
	2 Reduced compliance due to: • lung volume - a person with only one lung has halved compliance; • slightly less during inflation than during deflation; • increased by increased tissue mass - fibrosis or pulmonary congestion or chest wall restriction; • loss of surfactant	2	2 of 4 to pass
	3	3	
THIRD QUESTION (optional)	**What variables affect viscous resistance?**		
POINTS REQUIRED	1 Higher respiratory rates increasing flow rates	1	
	2 Decreased airway radius due to: Lower lung volumes; Bronchoconstriction;	2	
	3 Increased air density (eg SCUBA diving)	3	
	4 Increased air viscosity	4	
	5	5	
	6	6	
	7		