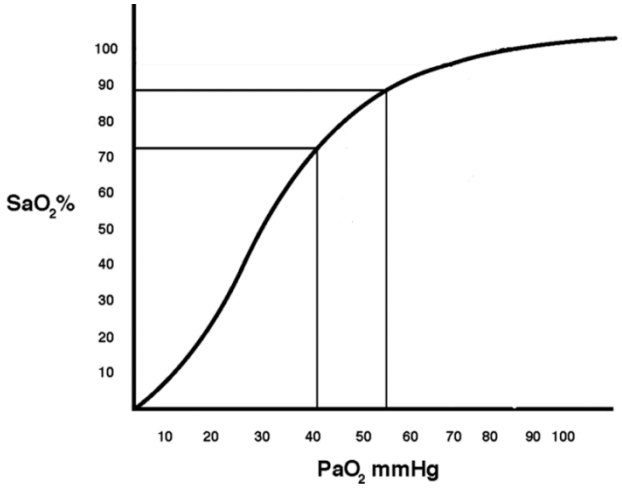
Stem: A 20 year old woman presents with a rash and dyspnoea. Her oxygen saturation is low. **We are starting with physiology**			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 VQ mismatch (West Chp 5) **Subject:** Phys LOA: 1	1. What are the causes of hypoxemia in a patient breathing room air? 2. How does the ventilation/perfusion ratio change in different regions of the lung? 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?	1. Hypoventilation 2.Diffusion limitation 3. Shunt 4. Ventilation/perfusion (V/Q) inequality **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 **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)	**3 of 4 to pass** **BOLD + general concepts to pass** **BOLD + demonstrates understanding**
Stem: We are now moving to anatomy. A CXR is performed.			
Question 2 CXR including understanding of pleural reflections **Subject:** Anat LOA: 2	1. Demonstrate the lobes of the lungs 2. What are their immediate relationships (if not answered in Q1) Prompt: what are the boundaries of the lobes	**Right superior mediastinum to apex ; right upper lobe** RUL: apex -horizontal fissure /upper right mediastinum medially **Right heart border; right middle lobe** RML: right heart border & horizontal fissure (superior border 4th rib) to 6th costal cartilage	**Demonstrate all 5 lobes**

		Left upper mediastinum to apex; left upper lobe LUL: Apex- 4LICS parasternal line,6th LICSMCL & 5th LICS AAL **Left heart border ; Lingula** lobe : left heart border **Lower lobes posteriorly, sit over domes of diaphragms** rise as high as 3rd intercostal space posteriorly R & L lower lobes: from Obliques fissures (T2 spinous process-6th costal cartilage anteriorly) to level T10 spinous process posteriorly , 10th ribs at scapular line & 8th ribs in MAL.	
	3. Describe the surface anatomy of the parietal pleura	supraclavicular fossa, medially follow the middle of the sternum to the level of the 6th intercostal cartilage, deviates laterally reaching MCL at 8th rib, MAL at the 10th rib, paravertebral line 12th rib. Notch on Left.	**Reasonable description**
Stem: We are now moving to pathology. The rash is diagnosed as Varicella Zoster			
Question 3 Varicella Zoster (p 353) **Subject:** Path LOA: 1	1. What are the 2 clinical conditions caused by this virus	Chicken pox and shingles	**Both**
	2. Describe the pathogenesis and clinical course of infection with this virus Prompt: start with how the virus is transmitted	Starts with aerosol or direct contact spread →haematogenous dissemination → vesicular skin lesions → vesicles rupture, crust over then heal Some virus lies dormant in dorsal root ganglia and reactivated later with immunosuppression	**Reasonable sequence**
	3. What are the complications of chicken pox	Lung → interstitial pneumonia Nervous system - encephalitis, transverse myelitis Skin and mucous membranes → shingles, bacteria superinfection Gut – necrotising visceral lesions	**3 to pass**

Stem: . We are now moving to pharmacology. Treatment is commenced with Acyclovir			
Question 4 Acyclovir (pp 862-864) **Subject:** Pharm LOA: 2	1. What are the indications for acyclovir in the ED?	**HSV – encephalitis**; VZV, patients with HIV, genital herpes	**Bold**
	2. Describe the mechanism of action of acyclovir.	**Inhibition of viral DNA synthesis** • Irreversible binding to viral DNA polymerase. • Incorporation in to viral DNA with termination Specificity for virus-infected cell (virus-specific thymidine kinase).	**Bold**
	3. Describe the pharmacokinetics of acyclovir?	**Short half life 2.5 hrs** (5xdaily dosing oral); low oral bioavailability; mostly excreted unchanged in urine; CSF 20-50% of plasma; wide distribution	**Bold + 1 other**
	4. Name some side effects of acyclovir	Nausea, vomiting, diarrhoea, headache, reversible renal toxicity Neuro – tremor, delirium, seizures	**2 to pass**

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 Draw and label the oxygen dissociation curve What are the implications of this curved shape? Prompt: what happens to the top + bottom	**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)  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	**Bold** **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% Explain **concept** of loading and unloading of oxygen
Stem: We are now changing to pathology.			
Question 2 Acute Pancreatitis (pp 893-896) **Subject:** Path LOA: 1	1. What are the potential causes of this man's pancreatitis? 2. What is the likely pathogenesis of acute pancreatitis?	1. **Gallstones, alcohol,** iatrogenic, viral, hyperlipoproteinaemia, hypercalcaemia, drugs, trauma, shock, vasculitis, genetic mutations, scorpion bite, atheroembolism, duct obstruction (tumour, parasites etc) **2. Autodigestion of the pancreatic substance by inappropriately activated pancreatic enzymes,** eg trypsinogen	**1. Bold plus 1** **2. Bold**

	3. What are the acute complications of severe pancreatitis?	Causes interstitial inflammation and oedema, proteolysis, fat necrosis and haemorrhage 3. Haemolysis, DIC, fluid sequestration, ARDS, diffuse fat necrosis. Peripheral vascular collapse; shock; acute renal tubular necrosis	**3. 3 answers to pass**
Stem: Another patient arrives with a stab wound to the back of his right thigh. **We will move now on to anatomy**			
Question 3 Posterior Thigh Muscles photo **Subject:** Anat LOA: 1	Identify the structures in this photo of the posterior thigh. What are the clinical features of a severed sciatic nerve in the upper thigh? Prompt: what does the sciatic nerve supply distal to this point.	**Sciatic nerve (19),** gluteus maximus (5), long head biceps (9), semitendinosus (22), semimembranosus (21), ischial tuberosity (8), gracilis (6), iliotibial tract (7), adductor magnus (1), popliteal artery and vein(16,17), quadratus femoris (18) **Motor**: SN supplies all posterior thigh muscles (depending on level of injury these may be affected), all leg and foot muscles **l**oss of hip extension and knee flexion**.** All ankle (**Flex/Ext, inversion, eversion**) and **toe movements** lost. **Sensory:** skin of most of leg and foot ->**posterior and lateral leg, sole** of **foot, lateral and dorsum of foot.**	**Bold + any 4 others** Prompt bold if required. **Motor 3 bold** **Sensory 3 bold**
Stem: We will now move onto pharmacology. He is agitated. You use Olanzapine to sedate him.			
Question 4 Olanzapine & atypical antipsychotics (Chp 29) **Subject:** Pharm LOA: 2	1. By what routes can Olanzapine be administered? 2. What dose, and route would you use in this situation? 3. What are the advantages of olanzapine over older "typical" antipsychotics? Prompt: e.g. chlorpromazine 4. What are some of its disadvantages? Prompt if needed – what about longer term effects	**1. Oral** (Tab or wafer); **Parenteral**- IMI, Depot IMI 2. Gives dose (10-20mg), same for each route 3. less hypotension; less tachycardia; **less extrapyramidal effect**; high clinical potency; less effect on prolactin; more effective vs neg&pos psychotic symptoms and cognition; multiple routes of admin 4. Anticholinergic effects; lowered seizure threshold; weight gain; DM; Hyperlipidaemia; expense	**Bold** **Reasonable answer** **Bold** **2 disadvantages**

ACEM PRIMARY VIVA C **Friday Morning Session 3** ***Candidate Number:*** **AGREED MARK:**

Stem: A 90 yo lady arrives by ambulance with confusion and agitation. She is hypotensive. **We will start with Physiology.**			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Baroreceptors / regulation of blood pressure (pp 589-592) **Subject:** Phys LOA: 1	1. What are baroreceptors and where are they located?	**1. Stretch receptors** in the walls (adventitia) of the heart & blood vessels, impt in control of BP (esp short term). Arterial- carotid sinus/Ao arch. Low pressure- Atria at entrance of IVC and SVC, Pulm veins and pulm circulation.	**Bold plus 2 locations.**
	2. What stimulates these receptors?	**2. Distension** of the structures above. More sensitive to pulsatile than constant pressure. Maximal firing at 150mmHg (@ Carotid sinus)	**Bold**
	3. What are their effects?	**3.** Inhibit tonic sympathetic drive& inc vagal drive=>**vasodilation, venodilation, hypotension, bradycardia (tachycardia in low pressure baroreceptors), ↓CO**. Allows rapid adjustments in BP in response to abrupt changes in posture, blood volume, cardiac output, or peripheral resistance	**3/5 end effects**
Stem: We will now move on to Pharmacology. Haloperidol is suggested for her agitation.			
Question 2 Haloperidol (pp 503-513) **Subject:** Pharm LOA: 2	What are the pharmacodynamics of haloperidol?	Butyrophenone- **high potency D2 receptor effects (dopamine antagonist)**, **high extra-pyramidal side effects**, **low sedative**, low hypotensive, minimal anticholinergic effects, minimal 5-HT and H1 blockade effects.	**2/3 Bold**
	How does olanzapine differ?	Thienobenzodiazepine- less D2 receptor effects, **high 5-HT receptor blockade effects**, **low extrapyramidal effects, medium sedative**, low hypotensive and anticholinergic effects, low H1 blockade effects	**2/3 Bold**

Stem: We will now move on to Anatomy. A recent CT brain is available.			
Question 3 CT brain **Subject:** Anat LOA: 1	1.Identify the intracranial structures visible on this CT (level of anterior & posterior horns lat ventricles)	Lobes: **frontal temporal parietal occipital** **Lat ventricle** : anterior and posterior horns 3rd ventricle, Caudate nucleus, choroid plexus Lentiform nucleus (putamen & globus pallidus) **Thalamus,** Septum pellucidum, **Falx** Anterior & posterior limbs of **internal capsule** Sylvian fissure	**Bold to pass** **Prompt if required**
	2. What arteries supply the main areas of the cerebral cortex? Prompt: point	**ACA** area anterior to anterior horns lat ventricle (**frontal and parietal lobes medially and superiorly**) **MCA** area between the ant & post horns LV (**most of lateral surface anterior, parietal, and temporal lobes**) **PCA** area posterior to posterior horn LV (**Inferior and medial aspects of occipital and temporal lobes**)	**Ant, Middle and Post CA** **Reasonable distribution**
	3. Describe the venous drainage of the cerebral hemispheres	3. Superior cerebral veins (superolateral surface of the brain) > **superior sagittal sinus**. Inferior and superficial middle cerebral veins (inferior, posterior and deep aspects of cerebral hemispheres) > straight, transverse and superior petrosal sinuses. **Great cerebral vein** (midline vein formed from the paired internal cerebral veins) >merges with inferior sagittal sinus to form the straight sinus . Eventually terminate in **Internal Jugular veins**	**2/3 bold**

Stem: We will now move on to Pathology. On examination she has a rigid, tender abdomen.			
Question 4 Ischaemic Bowel (pp 791-793) **Subject:** Path LOA: 1	What conditions can lead to infarction of bowel	**1. Acute arterial obstruction** Atherosclerosis, Aortic aneurysm, Hypercoagulable state, OCP use, Embolism **2. Intestinal hypoperfusion** cardiac failure, shock, dehydration, vasoactive drugs <u>systemic vasculitis</u> HSP, Wegeners granulomatosis <u>Mesenteric venous thrombosis</u> Hypercoagulable state, Invasive neoplasm, Cirrhosis, Trauma, Abdominal masses <u>Miscellaneous</u> Radiation, Volvulus, Stricture, Amyloid, diabetes	**BOLD to pass** **Minimum 2 from each bolded group** **2 from non-bolded**
	What are the clinical features of ischaemic bowel?	Severe **pain**, may be transient. Tenderness, peritonism, nausea, vomiting, bloody diarrhoea, melaena, shock, hyper/hypothermia, sepsis	**Bold + 3 features**
	What parts of the bowel are most susceptible to ischemic injury And why?	**Watershed zones** - Splenic flexure, sigmoid colon, rectum - Located at end of arterial supply Surface epithelium : Villi more at risk than crypts Intestinal capillaries run from crypts up villi to surface	**Must be able to explain why watershed zones are most at risk**