Stem: An 80 year old woman presents to ED following a fall secondary to an episode of melaena.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Anaemia (pp 639-665) **Subject:** Path LOA: 1	1. How are the causes of anaemia classified? Prompt if use RC morphology: How are the causes classified by mechanism? Prompt for example if not volunteered.	1. **Blood loss**: acute, chronic 2. **Increased RC destruction** Inherited genetic: H Spherocytosis, G6PD, Thal, Sickle cell Acq genetic: Parox noct hemo. Ab mediated: transfusion, drugs, Rh disease. Mech trauma: HUS, DIC, TTP, cardiac valves, runners. Infx: malaria; Toxic: envenom, clostridia, Pb. 3. **Decreased RC production** Inherited genetic: Fanconi's, thalassemia. Nutritional: B12/folate, iron. Erythropoietin deficit: renal failure, chronic dis. Immune: aplastic anaemia.	Bold main headings & 1 example of each to pass.
	2. Describe the pathogenesis of iron deficiency anaemia.	Causes: Chronic blood loss, poor diet, impaired absorption, incr reqs Iron stores used up first – **ferritin** haemosiderin. Once reserves depleted **serum iron & transferrin** decr. Erythroid activity increases, no iron in marrow macrophages. RCs become **hypochromic & microcytic.**	Bold to pass.
	3. (Please give examples of anaemias that are more common in specific ethnic groups.) Ask if there is time.	Hereditary spherocytosis: northern Europe G6PD: 10% African American, Africa, Middle East, Med Sickle cell: African descent, up to 30% Thalassemia trait: Africa, Asia, Med, India Pernicious: Scandinavian, Caucasian.	1 correct with example.
Stem: Initial treatment included commencement of a Pantoprazole infusion			
Question 2 Proton Pump Inhibitors (pp 1085-1089) **Subject:** Pharm LOA: 2	1. Describe the MOA of PPIs	**Irreversibly inactivates H^+K^+ATPase**, blocking the proton pump-inhibiting >90% acid secretion, for up to 24 hrs (time taken for synthesis new enzymes).	Bold to pass.
	2. Why is an IV infusion preferred to a single bolus dose?	**Only inactivates actively secreting acid pumps** (<10% in fasting patients). Hence single dose only decreases acid secretion for a few hours.	Bold to pass.
	3. Regarding oral formulations of proton pump inhibitors, please describe strategies used to increase their bioavailability and activity.	Taken as **inactive pro-drugs**, Begin as **acid resistant enteric coated** to prevent gastric elimination. Take on **empty stomach** as food decreases bioavailability. Weak bases so pass into acidified parietal cells, where concentrated 1000x, ecomes activated and binds to H^+K^+ATPase**. Take 1 hour prior to meal** so peak dose drug occurs when most pumps are active.	2 concepts.

Stem: Her BP is low.			
Question 3 Renal response to hypovolaemia (pp 701-706) **Subject:** Phys LOA: 1	1. Explain how hypotension activates the renin-angiotensin system. 2. How does the renin-angiotensin system contribute to the restoration of the blood volume? 3. What other factors increase renin secretion?	1. Hypotension leads to **reduced perfusion pressure of the afferent glomerular arteriole**, stimulating release of **renin** by the juxtaglomerular cells. 2. Renin **converts angiotensionogen to angiotensin I. Angiotensin converting enzyme** converts AG1 to **angiotensin II.** Ang II acts on the adrenal cortex's zona glomerulosa cells to **release aldosterone**. Aldosterone acts on the renal distal tubules to **retain Na and water**, thus increases intravascular volume. Ang II also a potent arteriolar constrictor and contributes to a rise in blood pressure. 3. Renin (protease) release is stimulated by increases in: **catecholamines, sympathetic activity** through renal nerves, **prostaglandins**, low Na states: cardiac failure, liver failure and Na depletion.	1. Bold to pass. 2. 4/5 bold to pass. 3. 1/3 bold to pass.
Stem: Following resuscitation, she complains of a painful hip and X-rays show a fractured neck of femur.			
Question 4 Femur (bone) **Subject:** Anat LOA: 2	a) Identify this bone and the significant bony features at the proximal end (Fig 5.7 p 517). **b)** What is the blood supply of the head and neck of the femur? c) You plan to do a femoral nerve block. What structures does the femoral nerve supply? (Supplementary question if time remaining)	Femur, appropriate side/orientation **Head**, fovea, **neck, greater & lesser trochanters, intertrochanteric crest** & line, quadrate tubercle, pectineal line, gluteal tuberosity. Med and lat **circumflex** femoral aa Usu branches of deep art of thigh (**profunda femoris**) Branch to form **retinacular** aa (from med >lat), feed under post unattached capsule (med) or through iliofemoral lig (lat). Artery to head of femur – br of obturator (less important). **Anterior thigh muscles** (quadriceps) Pectineus, Sartorius, iliacus **Articular branches to hip** and knee joints **Cutaneous branches** to anteromedial thigh Terminal cutaneous branch is saphenous nerve to anteromedial knee, leg, foot.	Bold structures to pass. Bold to pass Need to show understanding of dual supply, and relative contributions (circumflex aa > art to head of femur). 2/3 bold to pass.

ACEM PRIMARY VIVA A Thursday Afternoon Session 2 ***Candidate Number:*** **AGREED MARK:**

Stem: An 80 year old man is brought by ambulance to ED following a syncopal episode with a head injury. You suspect a base of skull fracture.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Bone – Base of skull **Subject:** Anat LOA: 2	1. What are the major bony compartments within the Base of Skull and what are the major bones forming them?	**Anterior cranial fossa – frontal bone** (ant), ethmoid (mid) and lesser wing of Sphenoid (post). **Middle cranial fossa – Sphenoid** plus Squamous Temporal laterally, contains Sella Turcica. **Posterior cranial fossa – Occipital Bone** plus dorsum sella of Sphenoid anteriorly.	Needs skull model as prop Must identify all 3 fossae plus identify major bone in each.
	2. Identify the various foramina in the Base of Skull.	ACF: Cribriform Plate – Olfactory N, MCF: Optic Canal – Optic N, Ophthal A Superior Orbital Fissure – CN III, IV, VI Foramen Lacerum – Int Carotid A plus associated sympathetic Internal acoustic meatus – CN VII, VIII plus labyrinthine a Foramen Rotundum – V2 Foramen Ovale – V3, accessory meningeal A Foramen Spinosum – middle meningeal A Groove for Petrosal N and Petrosal Br Middle Meningeal A	Must identify 5 foramina.
	3. What structures pass through the foramen magnum?	PCF: Foramen Magnum – **Medulla/Brainstem**, plus vert a, XI Jugular Foramen – CN IX, X, XI, sup bulb of IJV Hypoglossal Canal – CN XII Condylar Canal – emissary veins (sigmoid sinus) Mastoid Foramen – Mastoid emissary vein.	Bold to pass
Stem: His heart rate is 40 beats per minute and he takes metoprolol.			
Question 2 **Subject:** Pharm Metoprolol / Beta blockers (Ch 10) LOA: 1	1. Describe the pharmacokinetics of metoprolol **Prompt** what is its bioavailability and why?	Oral or IV, Vd – large, T ½ 3 – 4 hrs, Metabolised in liver Bioavailability 50% due to 1st pass effect.	**Oral & IV & 1st pass** Or **3/5**
	2. How does metoprolol differ from propranolol in its action at beta receptors?	Beta 1 – full agonist Beta 2 - 50 – 100 fold less potent	**B1 Selective**
	3. How do BB control hypertension?	Negative **inotropic and chronotropic** effects Slow a-v node conduction Antagonises release of renin/not fully understood.	**Negative inotropic & chronotropic effect**

<table>
<tr><td colspan="4">Stem: Now we will move on to Physiology</td></tr>
<tr><td>Question 3
Pressure Volume Loop (pp 540-550)
Subject: Phys
LOA: 1</td><td>1. What is the stroke volume in a normal adult at rest?

2. Please draw and label the pressure volume loop of the left ventricle.

Prompt: Describe the changes in pressure and volume that occur during systole and diastole.

 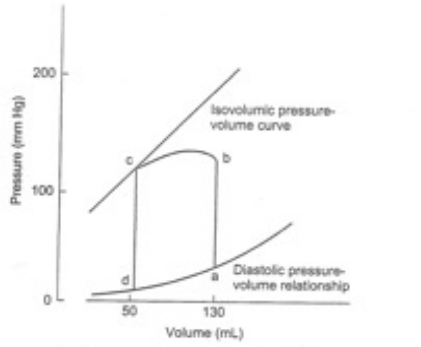

FIGURE 30–2 Normal pressure–volume loop of the left ventricle. During diastole, the ventricle fills and pressure increases from d to a. Pressure then rises sharply from a to b during isovolumetric contraction and from b to c during ventricular ejection. At c, the aortic valves close and pressure falls during isovolumetric relaxation from c back to d. (Reproduced with permission from McPhee SJ, Lingappa VR, Ganong WF (editors): Pathophysiology of Disease, 6th ed. McGraw-Hill, 2010.)</td><td>Stroke vol – 70-90ml

A. Start of systole: mitral (and Tric) valves close
Isovolumetric contraction til LVP > Aortic P (80mmHg)
Aortic (and Pulmonary) valves open.
B. Ventricular ejection (rapid at first) peak pressure 120mmHg
End systole: momentum of ejected blood overcome by aortic pressure.
C Aortic valve closes. ESV – 50ml

C-D. Isovolumetric relaxation
LVP drops below atrial pressure – mitral valve opens – ventricle begins to fill (rapidly at first)
EDV – 130ml</td><td>Bold to pass

Correct graph needed to pass. Need to demonstrate reasonable understanding of the loop.</td></tr>
<tr><td colspan="4">Stem: The patient has aortic stenosis.</td></tr>
<tr><td>Question 4
Calcific Aortic Stenosis (pp 561-563)
Subject: Path
LOA: 2</td><td>1. What are the predisposing factors for calcific aortic stenosis?

2. What are the clinical consequences of aortic stenosis?

3. What are the potential complications of a congenital bicuspid aortic valve?</td><td>Age: normal valve 70-90 yrs, bicuspid 50-70
Bicuspid valve or other congenital abnormality
Wear and tear, chronic injury
Hyperlipidemia, hypertension, inflammation
Other factors associated with atherosclerosis

Gradual obstruction of LV outflow leads to concentric LVH – pressure overload
Ischaemia/angina
Can get systolic and diastolic dysfunction
CHF and syncope herald decompensation.

Calcification, stenosis, regurgitation, infective endocarditis, aortic dilatation, dissection</td><td>Bold and one other

3 out of 4 concepts in bold to pass

Bold and 2 other</td></tr>
</table>

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

Stem: A 60 yo man presents with fever and dyspnoea. He requires intubation. We will start with Anatomy.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Airway (model) (Somso Model) **Subject:** Anat LOA: 1	1.On the model, identify the structures of the larynx and upper airway (somewhat dependent on model – for this session we take half mandible off and the muscles at the back) 2.Describe the nerve supply to the intrinsic laryngeal muscles (muscles of vocalisation) 3.What is the results of an injury to the recurrent laryngeal nerve	Cartilages: **thyroid,** cricoid**, epiglottis**, arytenoids, corniculate, cuneiform Ligaments: **cricothyroid membrane**, thyrohyoid, **vocal cords** Muscles: cricothyroid muscle thyrohyoid,, cricoarytenoid Spaces & Folds: **vallecula** ,aryepiglottic folds All muscles supplied by branches of X All except cricothyroid supplied by recurrent laryngeal n, cricothyroid supplied by external laryngeal n. Hoarse voice, and if bilateral, stridor due to inability to abduct cords as posterior cricoarytenoids are only abductors.	Must name 4 of 5 bold and 2 others Must name rec laryngeal and X as its source Supplemental
Stem: A CXR shows pneumonia. We will now move onto Pathology			
Question 2 Community Acquired Pneumonia (pp 710-716) **Subject:** Path LOA: 1	1. What organisms cause community acquired pneumonia? **Prompts** What organisms cause atypical pneumonia, and what viruses may cause atypical pneumonia? 2. What are some potential complications of pneumonia? **Prompt** – pathological sequelae 3. How do the clinical features of atypical pneumonias differ from classic (typical pneumonias)?	1. **Bacterial – Step pneumonia**, H influenza, Moxarella catarrhalis, S.aureus, Kelbsiella, and pseudomonas 2. **Atypical orgs** Mycoplasma, chlamydiae spp, coxielle burnetti (Q fever), legionella pneum 3. **Viral** – RSV, parainf, influenza A and B, adenovirus, SARs, H1N1 Abscess formation, Empyema, Bacteraemia/bacterial dissemination (endocarditis, pericarditis, meningitis, kidney, brain abscess), sepsis, respiratory failure Moderate sputum, no physical findings of consolidation, only mod increase in WBC Cough not prominent, typical sx are fever, headache, myalgia. Lower mortality compared with classic pneumonia.	Bacterial – bold plus 2 others Atypical – 1 to pass Viral – 1 to pass 3 complications to pass 2 features to pass

Stem: Blood gases show an acidosis. We will now move onto Physiology			
Question 3 H+ handling in metabolic & respiratory acidosis (pp 711-712) **Subject:** Phys LOA: 1	1.Describe the renal response to acidosis Prompt – Describe the role of buffers in the kidney	Aims to return serum pH to normal by increasing H+ excretion. Kidney retains HCO3 by actively secreting H+ Renal tubule cells excrete carbonic anhydrase converting CO2 to H+ and HCO3, then tubule cells secrete H+ in exchange for Na+ Amount of secreted H+ limited by urinary pH >4.5 (limiting pH) Buffering in tubular fluid pH with HCO2, HPO4 and NH3 allows greater H+ secretion.	Must know that H+ actively secreted into tubular fluid in exchange for Na. Must know about buffering and name 2 buffers.
Stem: We will now move onto Pharmacology. He is a diabetic on metformin.			
Question 4 Metformin (p 757) **Subject:** Pharm LOA: 1	1. Describe the pharmacokinetics of metformin 2. Outline some common side effects of metformin 3. Contrast the mechanism of action of metformin (biguanide) and glipizide (sulfonylurea).	Well absorbed, not protein bound, not metabolised, elimination half-life 1.5 to 3 hours **Excreted by kidney as unchanged compound.** **GI most common (20%) – limits compliance with this drug.** HAGMA **(lactic acidosis)** esp in patients with coexistent renal disease, EtOH, chronic cardiopulmonary disease. Glipizide – **Increases insulin release from pancreas** (patients more prone to hypoglycaemia with glipizide compared with metformin) Decreases serum glucagon levels Metformin Mechanism unclear but: May reduce hepatic gluconeogenesis. **Not dependent on functioning pancreatic B cells – so doesn't influence insulin release from pancreas** May directly simulate glycolysis in tissues with increased glucose removal from blood Decreases glucose absorption in the gut	Bold and one other to pass. Bold to pass. Bold to pass.