

Stem: An 80 year old man who is on warfarin is brought in following a motor vehicle accident in which he sustained multiple injuries. On arrival in ED, his blood pressure is 80/40 and pulse rate is 130 / minute. A chest X-ray is done.

TOPIC	QUESTIONS	KNOWLEDGE (essential in bold)	NOTES
<p>Question 1 Clinical Building Block:</p>	<p>Please describe the abnormalities on this CXR</p>	<p>Surgical emphysema, Pneumothorax, RML changes ? consolidation or contusion</p>	<p>Bold to pass</p>
<p>Moving on to Physiology</p>			
<p>Question 2 Frank- Starling Curve Subject: Phys LOA: 1</p>	<p>1. Please draw the Frank Starling curve as it relates to human cardiac muscle</p> <p>Prompt: What effect does EDV have on SV?</p> <p>2. What factors influence the Frank-Starling curve?</p>	<div data-bbox="1048 561 1563 933" data-label="Diagram"> </div> <p>2. Circulating catecholamines; inotropes, hypoxia, hypercarbia, acidosis, pharmacol depressants; loss of myocardium; intrinsic depressing; symp NS & PSym, fluid status</p>	<p>Q2.1 – to pass must be able to draw the FS curve including the hump and correctly label axes (SV or Pressure on y axis)</p> <p>Q2.2 – 4 factors with correct influence</p>

Stem: Moving onto Pathology. He has multiple facial lacerations which are bleeding			
Question 3 Haemostasis Subject: Path LOA: 1	1. What are the sequence of events in haemostasis after a vascular injury? Prompt: Is there any particular sequence to the events? 2. What laboratory tests are used to assess the function of the different pathways of the coagulation cascade? Prompt: Which one is vitamin K dependant	a. Vasoconstriction: arteriolar, reflex neurogenic, enhanced by endothelin b. Primary haemostasis: extracellular matrix exposed, pl adherence/activation - - pl aggregates & forms plug c. Secondary haemostasis: Tissue factors exposed, Fac III, thromboplastin, Fac VII, platelet plug consolidated - thrombin/fibrin generated d. Thrombus & antithrombotic effect – fibrin polymerises to form permanent plug, tPA regulates Prothrombin time – extrinsic pathway factors VII, X, II, V, fibrinogen (including vit K dependent factors) Partial thromboplastin time – intrinsic pathway factors XII, XI, IX, VIII,X, V, II, fibrinogen	Q3.1 – to pass identify 3/4 steps of hemostasis (bold) in correct sequence Q3.2 – to pass identify test, what pathway it is testing and identify which one is vit K dependant
Stem: Moving onto Pharmacology. It is decided to reverse his anticoagulation and Vitamin K is administered.			
Question 4 Vitamin K and warfarin Subject: Pharm LOA: 2 and 1	1. What is vitamin K? 2. Please describe its mechanism of action in reversal of warfarin anticoagulation Prompt: How long does it take for the onset of action	Fat-soluble substance in leafy vegetables; usually synthesised by gut bacteria. Vit K1(food) & K2(bact) Warfarin – coumarin anticoagulant, prevents reductive metabolism of inactive vit K to active form so produces biologically inactive VII, IX, X, prothrombin, protein C&S Vit K1 confers biologic activity upon prothrombin and factors VII, IX, X by participating in their postribosomal modification. Onset of action 6 hours , complete by 24 hours	Q4.1 Bold to pass Q4.2 to pass need concept of warfarin producing biologically inactive factors, vit K overcoming this, & delayed onset of action

Stem: Moving onto Anatomy. Following stabilisation, a secondary survey is undertaken and his facial wounds are closely inspected.

<p>Question 5 Face Dissection (model) Subject: Anat LOA: 2</p>	<p>Demonstrate on the model the arterial supply to the face?</p> <p>You are concerned about injury to his facial nerve. Using the model can you demonstrate the branches of the facial nerve? Prompt: Start from the parotid gland</p> <p>What is the function of the facial n.?</p> <p>What is the sensory nerve supply of the face? Prompt: What are the branches?</p>	<p>Facial artery (61) – arises External Carotid a. – contacts submandibular gland, hooks up over mandible anterior to masseter m. then a tortuous course to the medial angle of the eye. Transverse facial artery(62)- anastomoses with above</p> <p>Facial n. (66) – motor supply of the face 5 branches – Temporal n(67), Zygomatic n (68), Buccal n (69), Marginal Mandibular n (70), Cervical n (Exits BOS at stylomastoid foramen)</p> <p>Motor supply to the face Muscles of facial expression Taste anterior 2/3 rds tongue</p> <p>Trigeminal n. (5th Cranial n) - 3 branches: Ophthalmic , maxillary, mandibular NB – not on model – but candidate can map sensory division supply</p>	<p>Bold to pass and demonstrate on model</p> <p>Bold + 3/5 branches</p> <p>Bold</p> <p>Bold + 2/3 branches</p>
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Stem: An 80 year old woman is noted to be in heart failure. Starting with Pathology			
TOPIC	QUESTIONS	KNOWLEDGE (essential in bold)	NOTES
<p>Question 1 Heart Failure</p> <p>Subject: Path LOA: 1</p>	<p>What is heart failure?</p> <p>Please classify the types of heart failure? Prompt: Examples?</p> <p>What are the clinical features of heart failure? Prompt: What symptoms or signs from other organ systems might occur with heart failure?</p>	<p>When cardiac function is impaired and/or the heart is unable to maintain a cardiac output sufficient for the body's metabolic needs.</p> <p>Pump failure: Systolic dysfunction (Contractile dysfunction) eg myocardial contractile dysfunction secondary to ischaemia, AMI, pressure or volume overload, dilated cardiomyopathy. Diastolic dysfunction (Inadequate filling) eg LV hypertrophy, myocardial fibrosis, amyloidosis, pericarditis. Others: arrhythmias, regurgitant flow eg MR, outflow obstruction eg AS, HOCM</p> <p>Left heart failure (IHD, HT, Valvular diseases eg AS, rheumatic heart disease, myocardial disease) Right heart Failure (eg secondary to left heart failure, PE, Pulmonary HT etc)</p> <p>Lung –breathlessness, orthopnoea, PND, APO, pleural effusions Cardiac – 3rd HS, gallop, displaced apex beat, AF, murmur, JVP elevation Renal – RAA activation – with fluid retention, pedal oedema, AKI Brain – confusion secondary to hypoxia Hepatic –engorgement, ascites, cirrhosis(late)</p>	<p>Bold to pass</p> <p>One of the classifications with examples</p> <p>3/5 organ system symptoms to pass</p>

Stem: Moving on to pharmacology. Her medications include digoxin.

<p>Question 4 Digoxin Subject: Pharm LOA: 1</p>	<p>What is digoxin's mechanism of action in heart failure</p> <p>Why are patients in heart failure prone to digoxin toxicity?</p> <p>What are the features of digoxin toxicity</p> <p>Prompt: Any features from other organ systems</p>	<p>Ca accumulation in cells (due Na- K+ ATP block, Na in cells drive Na/Ca exchange) leads to</p> <ul style="list-style-type: none"> a) increased contraction strength, b) > stroke vol/ CO per beat- with smaller EDSV, small heart, reduced Rht pressures/ volume c) slower HR- >er stroke volume (partic if AF), via effects on parasympathetic fibres/AV node <p>a) poor renal function from low C/O,</p> <ul style="list-style-type: none"> b) potential dehydration and/or other drug interactions (e.g. ACE/ diuretics/ spironalactone/ ca channel blockers) c) potential effects on effective vol of distribution d) low K+ from other ht failure meds esp diuretics (makes pts higher risk from dig/toxicity) e) poor cardiac reserve/ output, altered digoxin handling during acute HF/ fluid distribution changes/other major illnesses <p>a) high K (assocd strongly with mortality)</p> <ul style="list-style-type: none"> b) yellow/ green (or other) colour vision c) GI- D and V, nausea/ malaise-anorexia/ d) arrhythmias from > automaticity and also Av node block (partic brady but R on T as well) e) severe heart blocks- partic if previous blocks , worsening failure, low BP f) CNS, tiredness -lethargy- headaches, paraesthesias, <p>Candidate may differentiate acute vs chronic</p>	<p>2/3 bold + one other</p> <p>To pass 2 including 1 bold</p> <p>To pass hyperkalaemia + at least 2 others from 2 different groups.</p>
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Stem: Moving on to anatomy			
<p>Question 5 Heart Model Subject: Anat LOA: 1</p>	<p>1. Using the model identify the great vessels and branches which enter and exit the heart</p> <p>2. Identify the main coronary arteries and their branches</p> <p>3. Which areas of the heart is supplied by the LCA?</p> <p>4. (If required) Describe the position of the heart in the left hemithorax</p>	<p>Superior vena cava - R brachiocephalic v, L brachiocephalic v Inferior vena cava Ascending aorta - brachiocephalic trunk, L common carotid artery, L subclavian artery Pulmonary trunk and pulmonary arteries Pulmonary veins</p> <p>RCA LCA Circumflex LAD/ant interventricular Marginal</p> <p>Most of the left atrium Most of left ventricle Part of right ventricle Intraventricular septum AV bundle (SA node in 40%)</p> <p>Inferior border lies on the diaphragm Apex is in the 5th ICS Base is against the Thoracic vertebrae T6 to T9</p>	<p>(bold to pass)</p> <p>4/5 to pass</p> <p>Bold +2 to pass</p>

Stem: A 40 year old woman presents with left loin pain and fevers. Urine microscopy is performed			
TOPIC	QUESTIONS	KNOWLEDGE (essential in bold)	NOTES
Question 1 Clinical Building Block: Urine Microscopy	Please describe the abnormalities. What is the most likely diagnosis?	High poly and RBC counts with +ve protein and blood (in the absence of epi-clean catch) indicates infection In the clinical context c/w pyelonephritis +/- stone	Bold to pass
Question 2 Pyelonephritis Subject: Path LOA: 2	What organisms cause acute pyelonephritis? Prompt: what are the most common? What steps are involved in ascending infection of the urinary tract? What conditions predispose to acute pyelonephritis?	G-ve bacilli (>85%), endogenous organisms E Coli, proteus, klebsiella, enterobacter, strep faecalis(enterococcus) Other: staph, fungi, (viruses in immunocompromised and renal transplant patients) 5 steps: 1. colonisation distal urethra 2. entry into bladder 3 . urinary tract obstruction / stasis of urine 4. vesicoureteric reflux 5. intrarenal reflux Urinary tract obstruction Instrumentation Vesico-ureteric reflux Pregnancy Female upto 50yrs Males >50 yrs Abnormalities- congenital/acquired DM, Immunosuppression	G-ve & 3 organisms pass Need to explain the concept clearly 4/9 to pass

Stem: Moving onto Physiology

<p>Question 3 Renal Circulation Subject: Phys LOA: 1</p>	<p>What is normal renal blood flow?</p> <p>What substances influence renal blood flow and how?</p> <p>How can renal blood flow be measured?</p> <p>Prompt: What substance can be used to measure renal plasma flow?</p>	<p>Renal blood flow = approx 1250 mL/min</p> <p>Noradrenaline-constriction, Dopamine, ACh -dilatation Angiotensin II – constricts afferent and efferent arterioles PGs-increase flow in cortex and decrease in medulla</p> <p>1. Fick principle (amount of a substance taken up per unit time divided by arterio-venous concentration difference) 2. PAH (or any substance that is excreted, not metabolised or stored, doesn't affect flow) is used to measure effective renal plasma flow (90% cleared) ERPF = Clearance of PAH = $UV/P = 630 \text{ mL/min}$ 3. Actual renal plasma flow = $ERPF/0.9 = 700 \text{ mL/min}$ 4. Renal blood flow = $RPF \times 1/1-Hct$ (Hct = 0.45)</p>	<p>Bold (accept 1000 – 1500)</p> <p>2/5 substances + correct action</p> <p>Concept/Principle</p>
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Stem: Moving onto Pharmacology. She is treated with Gentamicin			
Question 4 Gentamicin Subject: Pharm LOA: 1	1. Describe the mechanism of action of gentamicin	Irreversible inhibitor of protein synthesis. Binds 30S ribosome & inhibits protein synthesis by: 1) interfering with initiation complex of peptide formation 2) Inducing misreading of mRNA thus producing non functional protein; 3) causing break up of polysomes into non-functional monosomes <i>Additional information:</i> Enters cell by passive diffusion via porin channels across outer membrane, then enters cytoplasm by o2 dependant active transport process (transport coupled to a proton pump the transmembrane electrochem gradient supplies the energy) Low ecf pH & anaerobic conditions inhibit transport as reduces gradient; transport enhanced by cell wall active drugs eg penicillin, vancomycin.	Bold to pass
	2. What are the benefits of once daily dosing of gentamicin? <i>Prompt how does this improve clinical effectiveness?</i>	Concentration dependant killing (at increased conc kill increased no of bacteria at a more rapid rate); Post antibiotic effect (effect lasts longer than detectable serum levels); Reduced toxicity (as toxicity is time & conc dependant –time above critical level will be longer with multi dose than single dose schedule); less nursing time; OPD therapy possible; convenience	Bold to pass
	3. What micro-organisms is it effective against? Prompt: What group of organisms	Gram –ve bacteria – E. coli, Pseudomonas, Proteus, Klebsiella, Serratia Gram +ve- Staph, Strep- with beta lactams, vancomycin No anaerobic activity	Bold + 3 organisms

Stem: Moving onto Anatomy. A KUB X-ray is performed			
Question 5 AXR - ureters Subject: Anat LOA: 2	1. Could you point out on the xray the course of the L ureter	From hila of kidney L1-2 , along transverse processes, just medial to tips of transverse processes of lumbar vertebrae , on ant surface of psoas muscles, pass over pelvic brim around SI joint, run along lateral wall of pelvis till ischial spine, then medially to enter bladder	Bold
	2. Where in the ureters is a stone likely to lodge	PUJ, pelvic brim, VUJ	2/3
	3. Where else could a stone be present	Kidneys, bladder	1/2
	4. (only if required) What other structures can you identify on the xray (not required for pass)	Liver, Large Bowel, Lumbar spine, Pelvis, Femoral heads, Ribs, Psoas	