ACEM PRIMARY VIVA D Thursday Morning Session 1 ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: A 70 year old man presents to ED as he has become jaundiced following his return from a trip to India			
Stem: Here are his blood results.			
Question 1 **Clinical Building Block:** (hepatic and renal failure)	Please interpret these biochemistry results Prompt: what is the pattern of the liver enzyme abnormality?	Bicarb 6 - Metabolic acidosis eGFR 31 mL/min Creatinine 151 µmol/L - Moderate-Severe **renal impairment** Bilirubin 32 µmol/L (reduced excretion) Albumin 22 g/L (reduced synthesis) - Mild hepatic impairment ALT 1778 U/L AST 5314 U/L ALP 272 U/L GGT 471 U/L **-Abnormal liver enzymes c/w hepatitis**	Must recognise renal failure and hepatitic LFTs to pass. **(bold to pass)**
Stem: Moving onto Pathology. You suspect Hepatitis A .			
Question 2 Hepatitis A **Subject:** Path LOA: 2	What is the causative agent of Hepatitis A? How is hepatitis A transmitted? How do the clinical outcomes of Hepatitis A differ from Hepatitis B? (Prompt- How are the long term outcomes different?) How is Hepatitis A diagnosed serologically?	**Hep A virus** – small unenveloped single stranded RNA picornavirus, icosahedral capsid **Faecal oral spread** Self-limiting illness no carrier state no chronic state no association with hepatocellular Ca rarely leads to fulminant disease low fatality rate of 0.1% **Acutely IgM-anti- HAV**, followed by appearance / persistence of IgG-anti HAV	Bold to pass Bold to pass 3/6 to pass Bold to pass

Stem: Moving onto Pharmacology. His regular medications include hydrochlorothiazide			
Question 3 Thiazide diuretics **Subject:** Pharm LOA: 2	Describe the mechanism of action of thiazides?	**Inhibition of Na/Cl transporter in the distal convoluted tubule** leading to increased NaCl excretion and **diuresis**	**Bold to pass**
	What are the major clinical indications for thiazide diuretic use?	**Hypertension** **Heart failure** Nephrolithiasis Nephrogenic Diabetes Insipidus Generalised oedema Nephrotic syndrome cirrhosis	**2 bold to pass**
	What are the potential adverse effects of thiazide diuretics?	**Hypokalaemia** **Dehydration/post hypotension/hypovolaemia** **Hyponatraemia** Metabolic alkalosis Hyperuricaemia Hyperlipidaemia Allergic Reactions – x- reactivity with sulphonamides Impaired carbohydrate tolerance – Hyperglycaemia Hypercalcaemia Pancreatitis	**2 bold plus 1 other**

Stem: Moving onto Anatomy. A CT abdomen is done to exclude renal obstruction as the cause of his renal failure			
Question 4 CT abdomen **Subject:** Anatomy LOA: 2	Identify the structures on this CT. (Axial image)	**Liver**, portal vessels, **R Kidney** (top), **aorta** **L kidney**, spleen, splenic vein (not tortuous), bowel loops, **pancreas**, IVC, Vertebra, ribs, paravertebral muscles, intercostal and abdominal wall muscles, fat, skin.	**5 Bold** + 2 others
	Describe the course of the ureters	Originate at renal hilum (PUJ) – approx. L2 Run inferiorly lying across psoas Near tips of transverse process of lumbar vertebra (L3 – L4) Cross over pelvic brim Cross anterior to bifurcation of common iliac artery Lie on lateral wall of pelvis Travel medially to bladder Short intramural path at VUJ	4/8 points to pass
	What are the 3 narrowest points of the ureters?	PUJ VUJ Pelvic brim	2 of 3

Stem: Moving onto Physiology.			
Question 5 GFR **Subject:** Phys LOA: 1	What is the definition of the glomerular filtration rate?	The amount of fluid (plasma filtrate) filtered by the glomerulus per unit time	**Concept of filtration and time to pass.**
	What is the normal GFR?	Usually 125mL/min (180L/day) 10% less in women.	**+/- 20 % to pass (either per min or per day)**
	What are mesangial cells? (Prompt – Where are mesangial cells found? What do mesangial cells do?) (Prompt if "in nephron" stated – where in nephron?)	**Contractile cells** that help to **regulate GFR**. Located between the basal lamina and the endothelium, **in the glomerulus** Common between neighbouring capillaries, and in these locations the basal membrane forms a sheath shared by both capillaries Also secrete the extracellular matrix, take up immune complexes, and are involved in the progression of glomerular disease.	**Bold to pass**
	What factors influence GFR?	Age Afferent arterial (renal artery) pressure (however autoregulation keeps this stable between about 90-210mmHg) Afferent arteriolar pressure Efferent arteriolar pressure Efferent venous pressure Intra-renal (interstitial) pressure (obstruction, oedema) Oncotic pressure Glomerular filtration fraction	**Any 3 to pass**
	What substances act on mesangial cells to change GFR? (Prompt - What substances act on mesangial cells to alter their function?)	Glomerular filtration fraction (mesangial cell function) – influenced by: Increased – ANP, dopamine, PGE2, cAMP Decreased – noradrenaline, vasopressin, AII, PGF2, endothelins, TXA2, Leukotrienes	**BONUS!**

Stem: A 30 year old man has sustained a fractured femur in a motor bike accident. Starting with anatomy.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Femur - Bone **Subject:** Anat LOA: 1	Describe the bony features of the middle and lower end of the femur.	Linea aspera Medial supracondylar ridge/line - inferior continuation of the medial lip of the linea aspera, interrupted to allow passage of the femoral artery, ends in the adductor tubercle Lateral supracondylar ridge/line- descends to the lateral epicondyle. Medial condyle Lateral condyle Intercondylar fossa Adductor tubercle Attachment of the medial ligament	**5/8 to pass**
	Which muscles attach to the linea aspera?	Vastus medialis, vastus lateralis, adductor brevis, adductor longus, adductor magnus, and short head of the biceps femoris	**3/6 to pass**
	Which artery is most likely to be damaged by a fracture of the midshaft of the femur?	**Profunda femoris**	**Bold**
	(Prompt : what is the course of the femoral artery through the thigh?)	(use this as supplemental question for better candidates)	

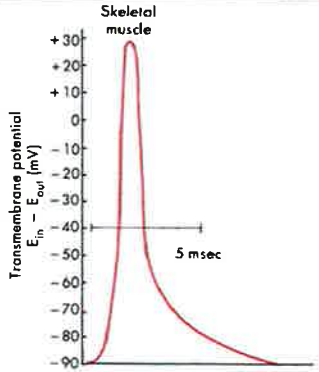
Stem: Moving onto Pathology. He has a head injury and a CT brain reveals gross cerebral oedema.			
Question 2 Cerebral Oedema and raised ICP **Subject:** Path LOA: 1	Describe the pathological mechanisms which cause cerebral oedema. (prompt if specific examples used – can you describe the difference between vasogenic and cytotoxic oedema?)	**Vasogenic.** BBB disruption, increased vascular permeability. Fluid shift intravascular to intercellular spaces of brain May be generalised or localised (inflammation or neoplasm) **Cytotoxic.** Increased intracellular fluid due to neuronal, glial, or endothelial injury eg generalised hypoxic/ ischaemic insult or metabolic damage Interstitial or ependymal oedema around (lateral) ventricles due to the high pressure of hydrocephalus	**Bold to pass or basic understanding of two mechanisms**
	What are the morphological findings of generalised cerebral oedema. (Prompt: What would be the CT findings?)	Flattened gyri, narrowing of sulci, compression of ventricles and/or basal cisterns, herniation	3 of 4 to pass
	Describe the major herniation locations associated with raised intracranial pressure	**Subfalcine** herniation- Asymmetric expansion of cerebrum displaces the cingulate gyrus under the falx cerebri **Transtentorial** or Uncal herniation -Medial aspect of the temporal lobe is compressed against the free margin of the tentorium **Tonsillar herniation**- Displacement of the cerebellar tonsils through the foramen magnum.	2 of 3 bold plus correct description

Stem: Moving onto Physiology. He is becoming progressively hypertensive and bradycardic.			
Question 3 Cerebral Circulation **Subject:** Phys LOA: 1	What are the factors that determine cerebral blood flow?	**Intracranial pressure** **Mean arterial pressure** **Mean venous pressure** at brain level Blood viscosity Local constriction/dilation of arterioles	**Bold** and 1 other to pass
	Describe the autoregulation of cerebral blood flow (Prompt: what happens to cerebral blood flow when blood pressure changes?)	**Maintains CBF at constant rate** (~750ml/min) **across a range of perfusion pressures** (MAP 65-140mmHg)	**Bold** to pass
	The patient's bradycardia and hypertension is caused by the head injury. Describe the mechanism responsible.	Cushing reflex – **increased ICP compromises blood flow to medulla** → **sympathetic outflow** from vasomotor centre → increases BP in attempt to restore medullary flow → **stretch of baroreceptors** → vagal stimulation→ bradycardia	**Bold** to pass Vagal stimulation OK instead of stretched baroreceptors

Stem: Moving onto Pharmacology. He is given Mannitol.			
Question 4 Mannitol **Subject:** Pharm LOA: 2	Why is mannitol used in the management of head injury?	Mannitol is used to **reduce intracranial pressure** after head injury.	**Bold** to pass
	What is the mechanism of action of mannitol?	Mannitol is an osmotic diuretic, it alters Starling forces as it does not cross the intact blood-brain barrier and thus **draws water out of cells** and **reduces intracellular volume** (hence reduces intracranial volume and intracranial pressure)	**Bold** to pass
	What are the other clinical effects?	Reduces intraocular pressure Diuresis / dehydration / hypovolaemia Hypernatraemia Hyperkalaemia	2/4 to pass
	Supplemental Question; What is an appropriate dose of mannitol in this clinical situation?	1-2g/kg as an IV bolus over 15 mins (**0.25-2g/kg IV bolus**).	

Stem: A 25 year old man has ruptured his Achilles tendon. Starting with Anatomy.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Model – lower leg **Subject:** Anat LOA: 1	Identify the muscles of the posterior compartment of the leg	Superficial posterior compartment: **24a,b Gastrocnemius m.** **24c Soleus m.** **24 Plantaris m.** Deep posterior compartment: **26. Popliteus m.** **27 Flexor digitorum longus m.** **28 Tibialis posterior m** **29 Flexor hallucis longus m**	24 a,b,c triceps surae muscle 51 popliteal a. 56 posterior tibial a **Must get 6/8 bold**
	Which muscles form the Achilles tendon	**gastrocnemius and soleus**, +/- plantaris	**Bold** to pass
	Where does the Achilles tendon insert?	supero-posterior aspect of the **calcaneus**	**Bold** to pass
	Can you identify the nerve supply of these muscles?	**tibial n.**	
	Can you identify the structures posterior to the medial malleolus?	Anterior to posterior: Tibialis posterior Flexor Digitorum Longus Posterior Tibial Artery Tibial Nerve Flexor Hall Longus	2/3 muscles and neurovasc bundle to pass
	Supp: What is the blood supply of these mm.?	gastrocnemius - sural a. (branch of popliteal a.); soleus - posterior tibial a. and peroneal a.)	

Stem: Moving onto Pathology.			
Question 2 Repair by healing, scar formation and fibrosis **Subject:** Path LOA: 2	What is the sequence of events for tissue healing by scar formation?	1) Blood Clot (stop bleeding, create scaffold) 2) Granulation tissue (angiogenesis, migration and proliferation of fibroblasts) 3) Cell Proliferation and Collagen Deposition (extracellular matrix (ECM) deposition) 4) Scar formation (blanching, increased collagen: type 3 then type 1) 5) Wound contraction (myofibroblasts) 6) Connective tissue remodelling (ECM synthesis and degradation) 7) Recovery of tensile strength	**5/7 to pass**
	How do skin wounds recover tensile strength?	**Increase in collagen synthesis** (type 1) **and reduction in collagen degradation** (first 2/12) then **structural modification** of collagen with cross linking & increased fibre size	**Bold to pass**
	What is the approximate time frame for recovery of tensile strength in skin wounds? (prompt : what is the strength of skin wounds when sutures are removed?)	Skin wound has **10% tensile strength at 1/52,** and continues to improve over next 3 weeks and **plateaus at ~3/12 when tensile strength is 70-80%.** May never recover to 100%	**Concept that very weak at time of suture removal and months to attain plateau phase**

Stem: Moving onto Physiology.			
Question 3 Skeletal Muscle action potential **Subject:** Phys LOA: 1	Draw a skeletal muscle action potential **(**Prompt if draw cardiac musc AP**)**		**Correct shape, axes, resting membrane potentials and durations (+/- 25%).**
	What is the sequence of events in the contraction of a skeletal muscle fibre, starting at the motor end-plate?	1.Discharge of motor neuron 2.Release of transmitter (acetylcholine) at motor endplate 3.Binding of ACh to Nicotinic Ach receptors 4.Increased Na^+ and K^+ conductance in end plate membrane 5.Generation of end plate potential 6.Generation of action potential in muscle fibers 7.Inward spread of depolarisation along T tubules 8.Releases of Ca^{2+} from terminal cisterns of sarcoplasmic reticulum and diffusion to thick and thin filaments 9.Binding of Ca^{2+} to troponin C, uncovering myosin-binding sites on actin 10.Formation of cross-linkages between actin and myosin and sliding of thin on thick filaments, producing movement	**5/10** to pass
	What is the sequence of events in the relaxation of a skeletal muscle fibre?	1.**Ca^{2+} pumped back into sarcoplasmic reticulum** 2.Release of Ca^{2+} from troponin 3.**Cessation of interaction between actin and myosin**	**Bold to pass**

Stem: Moving onto Pharmacology. He is given Ondansetron for nausea.			
Question 4 Ondansetron **Subject:** Pharm LOA: 1	What is the mechanism of action of Ondansetron? Prompt- Where are these receptors found?	**5-HT$_3$ receptor antagonist**; Effect brought about at peripheral (Gut) > central receptors (chemoreceptor trigger zone and vomiting centre)	**Bold,** plus 1 receptor location
	What are the doses and routes of administration of Ondansetron ?	**4-8mg** SL , PO, IV , SC, IM	**Bold**, plus 3/5
	What are the adverse effects of Ondansetron?	Constipation, headache, dizziness, QT prolongation	**1/4** to pass
	In which disease state would you need to modify the dosing?	**Hepatic failure** Not with renal failure or age	**Bold**
	What are some other classes of antiemetic drugs? (ask for drug class if just name a drug)	Phenothiazines Antihistamines Cannabinoids Benzodiazepines Butyrophenones (Droperidol) Benzamides (eg Metoclopramide) Neurokinin receptor antagonists Corticosteroids	**3/8** to pass