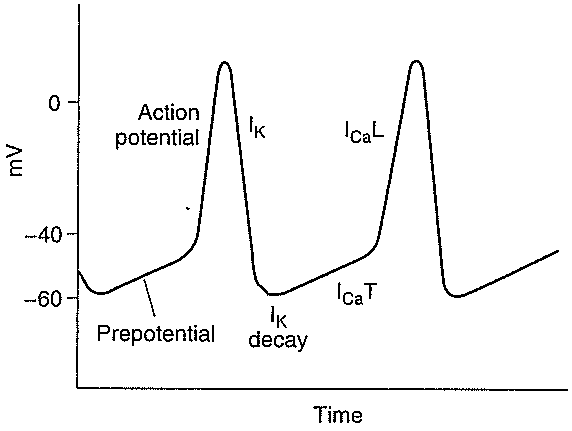
ACEM PRIMARY VIVA B **Thursday Morning Session 1** ***Candidate Number*:** **AGREED MARK:**

Stem: A 70 year old man with a history of Atrial Fibrillation presents with sudden onset of painful index and middle fingers in his left hand. Examination reveals these fingers to be pale and cold. We will start with Anatomy.

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Hand (photo) **Subject:** Anat LOA: 1	1. Please describe the arterial supply of the hand. (without photo) Prompt: What happens to these arteries in the palm? How are they arranged? How do they terminate?	The **Ulnar and Radial Arteries supply all of the blood supply to the hand**. **Radial Artery- Deep Palmar Arch.** Lies deep to long flexor tendons and sits across the metacarpals just distal to their bases. Branches: (of Deep Palmar arch) • 3x Palmar metacarpal arteries • Princeps pollicis artery • Radialis indicis artery **Ulnar Artery-** Two terminal branches Deep Palmar Branch (24) anastomoses with the Radial Artery via the Deep Palmar arch. **Superficial Palmar Arch** is main terminal branch. • 3 Common Palmar Digital Arteries arise. • Each divides into a pair of Proper Palmar digital arteries that run along adjacent sides of 2-4th digits.	Bold + 1 branch of each arch to pass
	2. Can you identify the Ulnar artery on the picture?	**Ulnar Artery (23),**	Bold to pass
	3. Describe the sensory innervation to the index and middle fingers?	**Median nerve (16) & Radial to dorsum to DIP** **1st** (7) **and 2nd** (22) **Lumbricals.** Muscles of the Thenar eminence: Opponens pollicis, **Abductor pollicis brevis (2) and Flexor pollicis brevis (13)**.	Bold to pass Thumb, Index, Middle and half of the ring finger (palmar aspect). Dorsal tips (nail beds) of the thumb, 2nd and 3rd fingers.
	4. Can you identify on the picture muscles in the hand and forearm that are innervated by the median nerve?	Forearm: **Flexor carpi radialis (8),** Palmaris longus, **Flexor digitorum superficialis(12), Radial half Flexor digitorum profundus (2nd and 3rd digits) (11). Flexor pollicis longus (14),** Pronator quadratus	4 of 8 bold to pass FPL and PQ supplied by the Anterior Interosseous nerve (a branch of the Median)

Stem: We will now move on to Pathology.			
Question 2 Embolism (pp 125-127) **Subject:** Path LOA: 1	1. What is an embolus?	A **detached intravascular** solid/liquid/gas **mass** that is carried by the blood stream from its site of origin to a **distant site**.	Bold to pass
	2. Name the different types of embolus?	• **Thromboembolus** **1. Venous: pulmonary** **2. Arterial: systemic** • Fat embolus: from bone marrow • Gas embolus: eg air/nitrogen • Amniotic fluid embolus • Tumour fragment embolus • Foreign body embolus eg catheter	Bold + 2 to pass
	3. What is systemic thromboembolism?	**Definition: Emboli in arterial circulation**	Bold to pass
	4. From where do they arise and where do they lodge?	<u>Sources:</u> 80% from **intracardiac mural thrombi** (2/3 L vent wall infarcts, ¼ L atrial dilation/AF) Other sources: aortic aneurysms, ulcerated atherosclerotic plaques, valvular vegetation, paradoxical emboli, unknown <u>Lodgement Sites:</u> Lower limbs (75%), brain(10%), Other: intestine, kidneys, spleen, upper limbs	Bold + 2/4 sources and 2/4 sites to pass
	Bonus Question Describe the process of infarction from arterial occlusion. Prompt: What are the features that influence the development of an infarct?	Area of ischaemic necrosis: dominant histologic characteristic is ischaemic necrosis - White infarcts occur in solid organs with end-arterial circulation - Acute inflammation happens within hours; reparative response follows - Factors influencing infarct development: nature of vascular supply (end artery vs presence of collateral blood supply), rate of occlusion, vulnerability to hypoxia, oxygen content of blood, calibre of occluded vessel,	

Stem: We will now move on to Pharmacology. A heparin infusion is commenced.			
Question 3 Heparin (pp 604-607) **Subject:** Pharm LOA: 1	1. How does heparin act?	Heparin binds endogenous **antithrombin and enhances its activity. Antithrombin inhibits factors IIa, IXa and Xa** by complexing with them and inducing a conformational change.	Bold to pass
	2. How may heparin be administered?	**IV vs SC.** Continuously (following bolus) vs intermittent. Therapeutic vs prophylactically	Bold to pass
	3. What are the potential adverse effects?	**Bleeding,** allergy, alopecia, osteoporosis, **HIT,** mineralocorticoid deficiency	Bold + 1 to pass
	4. What are the advantages of low molecular weight heparins compared to unfractionated heparin?	Have equal efficacy, increased SC bioavailability, require less frequent dosing, and less monitoring. Shorter chain heparin with less effect on thrombin (IIa).	Demonstrates understanding

Stem: We will now move on to Physiology. The patient has reverted to sinus rhythm.			
Question 4 **Subject:** Phys LOA: 1	1. Describe the normal sequence of electrical excitation of the cardiac conduction system and cardiac muscle?	Normal sequence of depolarisation: **SA Node** **Atria** **AV Node** Bundle of His **Major bundles (Right and left)** Purkinje fibres **Ventricular muscle**	Bold to pass.
	2. What are the common mechanisms that cause abnormalities of cardiac conduction?	Abnormal pacemakers Re-entry circuits Conduction deficits Prolonged repolarisation Accessory pathways Electrolyte disturbance	4 to pass
	3. Please draw and explain the action potential of a cardiac pacemaker cell Prompt: Which electrolytes are responsible for each phase of the action potential?	Pre-potential is initially due to a decrease in K+ efflux, then completed by Ca2+ influx through CaT channels. The action potential is due to influx of Ca2+ via CaL channels. Repolarisation is due to K+ efflux  	To pass: Correct shape of graph Know ion fluxes: • Pre-potential decrease K+ efflux/Ca2+ influx • Action potential Influx Ca2+ • Repolarisation K+ efflux

Stem: 85 year old man presents to your ED in urinary retention, the day after a prostate biopsy. On PR examination, his prostate is extremely tender and you suspect prostatitis. We will start with Pathology.

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Acute inflammation – questions to focus on acute inflammation not prostatitis specifically (as this is an LOA 3 topic) (pp 48-56) **Subject:** Path LOA: 1	1. What are the three major components of acute inflammation?	1. **Dilation of small vessels** leading to increase blood flow. 2. **Increased permeability of the microvasculature** enabling plasma protein and leucocytes to leave the circulation. 3. **Emigration of leucocytes from the microcirculation to the site of injury**.	Bold to pass Neutrophils predominate in the early inflammatory (6 – 24 hours) infiltrate and are later replaced by monocytes and macrophages (24 – 48 hours).
	2. How are leucocytes delivered to the site of injury? PROMPT: What are the three processes that leucocytes undergo to move from the blood to the site of injury?	This is a multistep process mediated and controlled by adhesion molecules and chemokines. 1) **Margination:** Occurs when leucocytes adopt peripheral position along the epithelium. Rolling (transient adherence mediated by selectins), activation and firm attachment (mediated by integrins) to the endothelium. 2) **Transmigration** (diapedesis): across the endothelium. Migration through interendothelial spaces typically in post capillary venules. 3) **Chemotaxis**: Leucocytes move toward the site of injury along a chemical gradient of chemoattractants, which can be exogenous or endogenous.	Bold to pass Polymerisation of actin at the leading edge of the cell establishes a “front wheel “ drive in the direction of the injury
	3. Name some of the chemoattractants responsible for chemotaxis?	Most common exogenous agent **Bacterial products**. Endogenous: IL-8, **C5a**, and Leukotriene B4. All bind to specific receptors and promote polymerisation of actin.	Bold + 1
	4. What chemical mediators are responsible for pain, fever and tissue damage?	**IL-1,** TNF, Prostaglandins, Bradykinin, Neutrophil and Macrophage Lysosomal enzymes, Oxygen metabolites, NO.	Bold + 1

Stem: We will now move on to Anatomy.			
Question 2 Male pelvis model (Model No: MS2) **Subject:** Anat LOA: 2	1. Name the parts of the male pelvis visible on this model. Prompts: What are the… • Skeletal features • Organs of the urogenital system • Vascular structures	1 **Pubic bone**, 2 **Sacrum**, 3 Coccyx, 4 **Urinary bladder** (a. apex, b. fundus, e. ureteral orifice, f. trigone), 5 **Prostate**, 7 Seminal vesicles, 8 Spermatic duct, 9 **Ureter**, 10 Urethral corpus cavernosum, 11 **Penis**, 12 Glans penis, 13 External urethral orifice, 14 Ischio-cavernosus muscle, 15 **Testicle**, 16 **Epididymis**, 17 Pampiniform plexus, 18 Testicular artery, 19 Cremaster muscle, 20 **Rectum**, 21 **Common iliac artery,** 22 **Common iliac vein**, 23 Peritoneum, 24/25 **Inguinal ligament**, 26 Femoral canal	Bold to pass
	2. Name the parts of the urethra	- pre-prostatic part (surrounded by internal urethral sphincter - **prostatic part** - **membranous (intermediate) part** (surrounded by external urethral sphincter) - **penile (spongy) part**	Bold to pass
	3. What is the innervation of the urethra?	- **prostatic nerve plexus** to first 3 parts above - **dorsal n. of the penis** (from pudendal n.) to penile part	1 of 2 bold to pass
	Bonus Describe the anatomy of the prostate	• Surrounds prostatic part of the urethra (about the size of a walnut). • base sits near the neck of the urinary bladder • Apex is next to the urogenital diaphragm. • Covered in a thick fibrous capsule, which houses the prostatic plexuses of nerves and veins. • 5 lobes: anterior, middle, posterior, & 2 lateral • Arterial supply via inferior vesical, internal pudendal, and middle rectal arteries. • Venous drainage via the prostatic venous plexuses, which is located around the base and sides of the prostate.	

Stem: We will now move on to Pharmacology. Treatment with Ciprofloxacin is commenced.			
Question 3 Fluoroquinolones (Chp 46) **Subject:** Pharm LOA: 2	1. What class of drug is Ciprofloxacin?	**Fluoroquinolone**	Bold to pass
	2. What is its mechanism of action?	**Blocks DNA synthesis** by inhibiting bacterial topoisomerase II and IV	Bold to pass
	3. What is its antimicrobial spectrum?	**Excellent Gram neg activity and moderate Gram positive activity**. Methicillin susceptible strains of S *Aureus* are susceptible, but methicillin resistant Staphylococci are resistant. Also active against agents of atypical pneumonia – Mycoplasma and Chlamydiae Intracellular pathogens such as Legionella and Mycobacterium. Ciprofloxacin the drug of choice for anthrax.	Bold + 1 to pass MIC for Gram neg are 1-2 mcg/mL.
	4. What are the potential adverse effects of Fluoroquinolones?	• **Prolonged QT** (with some), • Nausea, vomiting, diarrhoea (inc. C difficile) • Rash • Abnormal LFTs • Photosensitivity • Hyperglycemia in diabetics, • Growing cartilage damage (not routinely recommended for < 18 yo or pregnancies) • Tendonitis • Allergy	Bold + 2 dot points

Stem: We will now move on to Physiology.			
Question 4 Micturition (pp 693-695) **Subject:** Phys LOA: 2	1. Describe the neurological pathways involved in normal micturition.	**Spinal reflex mediated by S2, S3 and S4 nerve roots. Facilitated and inhibited by higher centres; subject to voluntary control.** - First urge to void at 150ml. Marked fullness at 400ml - sudden rise in intra-vesical pressure triggers reflex contraction. **Micturition reflex:** - Stretch receptors in bladder wall. Afferent limb in pelvic nerves. **Parasympathetic efferent fibres** (via same pelvic nerves) mediate contraction of detrusor muscle. **- Pudendal nerve (S2, S3 and S4)** permits voluntary contraction of perineal muscles/external urethral sphincter, to slow or halt flow. - Sympathetic nerves to bladder play no role in micturition	To Pass: Spinal Reflex Parasympathetic control higher centre control
	2. Describe the muscles involved in micturition.	**1. Bladder:** smooth muscle arranged in spiral, longitudinal and circular bundles. Circular bundle is called the **detrusor muscle. Contraction of detrusor is responsible for involuntary emptying.** **2. External urethral sphincter – skeletal muscle sphincter of the membranous urethra. Relaxes during micturition. This is voluntarily controlled.** 3. Perineal muscles. Relaxes during micturition. Also voluntarily controlled. 4. In males, urine left in urethra expelled by several contractions of bulbocavernosus muscle. 5. Contraction of abdominal wall muscles aids expulsion of urine. NB: Internal urethral sphincter (smooth muscle bundles passing on either side of urethra) plays no apparent role in micturition.	Bold to pass
	3. What prevents vesico-ureteric reflux?	**Oblique passage of ureters through bladder wall** keeps ureters closed except during peristaltic waves.	Bold to pass

Stem: A 70 yo woman with metastatic lung cancer presents with polydipsia and polyuria. We will start with Pathology.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Lung Tumours (pp 721-731) **Subject:** Path LOA: 2	1. What are recognised aetiological factors in lung cancer? Prompt for detail: Are you aware of any environmental factors that place you at greater risk for lung cancer?	**Tobacco smoking -** 87% of cancers in recent or current smokers- 10x increase in risk**,** Statistically associated with daily amount; inhalation tendency; duration of habit**,** Histologic changes in respiratory epithelium in smokers **Industrial Hazards** Ionising radiation, Uranium, Asbestos **Air pollution -** Radon **Molecular genetics -** Familial clustering **Precursor lesions -** Squamous dysplasia and CIS, Atypical adenomatous hyperplasia, Diffuse idiopathic pulm neuroendocrine cell hyperplasia	Tobacco smoking and 2 other bold to pass
	2. What are the most common presenting symptoms of lung cancer?	**Cough** (75%), Loss of weight(40%), Chest pain (40%), Dyspnoea (20%), Haemoptysis	3 to pass
	3. What are the clinical effects of local lung tumour spread?	• **Airway obstruction ->pneumonia, abscess, lobar collapse**, Lipoid pneumonia, • **Obstruction of SVC** leading to SVC syndrome • **Pleural effusion**, • **Pericarditis or tamponade**, • **Hoarseness** (r/c laryngeal n), • **Dysphagia** (oesophagus), Rib destruction, • **Diaphragmatic paralysis** (phrenic nerve) • **Horner syndrome** (sympathetic ganglia)	5 of 8 bold to pass
	4. What paraneoplastic syndromes are associated with lung cancer? PROMPT: What hormones might be produced?	Clinically significant in 1-10% of patients ACTH- **Cushing's** (predominantly small cell) ADH—**hyponatraemia** (predominantly small cell) PTH, PTH related peptide, PGE and some cytokines**- hypercalcaemia** (predominantly small cell/squamous cell)**,** Calcitonin-hypocalcaemia, Gonadotrphins-	2/3 bold + 1 other to pass

		gynaecomastia, 5HT and bradykinin- wheeze/flushing	

Stem: We will now move on to Physiology. She has a raised corrected calcium level.			
Question 2 Calcium metabolism (pp 377-378) **Subject:** Phys LOA: 1	1. Where in the body is Ca2+ stored?	**Bone:** 99%, **Plasma – bound to protein**, **Plasma – unbound (free/ionised**) - important second messenger and is required for coagulation, nerve function and muscle contraction.	Bold to pass
	2. How is the plasma Ca2+ level regulated? Prompt: What hormones increase or decrease plasma Calcium?	**Parathyroid Hormone**: Increases plasma Ca2+ by mobilising Ca2+ from bone. Increases Ca2+ reabsorption in kidney. Increases formation of 1,25 DHCC in the kidney. **1, 25 DHCC** (from Vit D) increases Ca2+ absorption from intestine and kidneys. **Calcitonin** (from thyroid) lowers circulating Ca2+ levels. Effect by inhibition of bone reabsorption. It also increases Ca2+ excretion in urine Glucocorticoids – decrease plasma Ca2+ by inhibition osteoclast formation and activity. Oestrogens – inhibit stimulatory effects of cytokines on osteoclasts Growth Hormone – increases Ca2+ excretion in urine & absorption in intestine. Net balance may be positive. Hypercalcaemia is a complication of cancer. Raised Ca2+ from either: - bone erosion (local osteolytic hyperCa2+ - elevated Parathyroid hormone related protein (PTHrP)	Bold and their effects on plasma Ca2+ (increase / decrease)
	3. How does bone resorption occur	**Osteoclasts** are monocytes that develop from stromal cells under influence of RANKL. • Attach to bone via integrins in sealing zone of the membrane. • Hydrogen dependent proton pumps move into cell and acidify the area. • Acid dissolves hydroxyapatite and acid proteases break down collagen.	Osteoclasts + 1 other RANKL – receptor activator of nuclear factor kappa B ligand

		• Products move across osteoclast into interstitial fluid.	

Stem: We will now move on to pharmacology. Treatment is commenced with normal saline and frusemide.			
Question 3 Frusemide (pp 258-260) **Subject:** Pharm LOA: 1	1. How does frusemide exert its action?	**Selectively inhibits Na+-K+-2Cl- transporter in thick ascending limb of loop of Henle thus preventing resorption of Na+ & Cl-** Abolishes counter-current concentrating mechanism leading to dilute urine. Increased prostaglandin synthesis -> inhibition of salt transport in thick ascending limb -> increased renal blood flow, decreased pulmonary congestion, decreased LV filling pressures	Bold to pass
	2. What are the pharmacokinetic properties of frusemide?	• Rapid absorption after oral admin • Oral bioavailability 50% (range 10 –100%) • Highly protein-bound (>95%) • 50% conjugated in kidney & 50% excreted in urine unchanged (tubular secretion) • Elimination t1/2 1.5 – 2 hours • Peak effect 30 minutes IV / 1 hour oral	List 3
	3. What are the potential adverse effects of frusemide? PROMPT: What are the electrolyte disturbances?	• Electrolyte disturbances • **hypokalaemia**, • hyponatraemia, • hypomagnesaemia, • hyperuricaemia • Postural **hypotension** & dizziness • Metabolic Alkalosis • Allergy - rash, eosinophilia, interstitial nephritis • Increased LDL & triglycerides, decreased HDL • Hyperglycaemia • Ototoxicity (high dose IV)	Bold plus 2

Stem: We will now move on to Anatomy. She has limited shoulder movement due to bony metastases.			
Question 4 Shoulder (bone /model) **Subject:** Anat LOA: 1	1. What are the articulating surfaces in the shoulder joint	Ball-and-socket synovial joint, Rounded **head of humerus**, Shallow **glenoid cavity of scapula**, deepened by labrum	Bold to pass
	2. What structures stabilise the shoulder joint?	Fibrocartilaginous glenoid labrum. Coraco-acromial arch, Anterior glenohumeral ligaments Coracohumeral ligament Transverse humeral ligament **Rotator cuff (SITS) muscles, Supraspinatus, Infraspinatus, Teres minor, Subscapularis**	Rotator cuff (3/4 muscles), plus 2 others to pass Need to show understanding that there are different elements that contribute to stability.
	3. What muscles are responsible for abduction and adduction of the shoulder joint?	Abduction -**Deltoid** (esp acromial part)**,** **Supraspinatus** (initiates), + upward movement of scapula Adduction -**Pec major** and **lat dorsi** acting in concert**, Teres major** and **long head of triceps** (synergists)	Bold to pass 2/4 Bold to pass
	Bonus What muscles are responsible for the other movements of the shoulder?	<u>Flexion -</u>Pectoralis major (clav head)**,** Deltoid (clav and anterior acromial parts)**,** Coraco-brachialis (synergist) <u>Extension -</u>Spinal part of deltoid**,** Lat dorsi, teres major, long head of triceps (synergists) <u>Medial rotation-</u> subscapularis, pec major, lat dorsi, teres major, deltoid-clavicular part (synergists) <u>Lateral rotation -</u>infraspinatus, teres minor, deltoid- spinal part (synergists) <u>Circumduction</u>	
	Bonus: Outline the bursae of the shoulder joint.	Subscapular bursa- located between neck of scapula and subscap tendon. Protects tendon. Subacromial (subdeltoid) bursa -Between acromion, CA ligament and deltoid superiorly, and supraspinatus tendon and joint capsule inferiorly Facilitates movement of supraspinatus tendon	