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

Stem: An 18 year old woman presents to the ED, 3 days following a self-inflicted wrist injury. She complains of numbness in her hand. We will start with Anatomy			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Wrist dissection (photo) **Subject:** Anat LOA: 1	(a) Identify the structures seen in this image	**Median nerve** (16) + branches (17, 18, 19) **Ulnar nerve** (25) + branches (24, 26, 27, 28) Ulnar artery (23) Thenar muscles – APB (2), FPB (13) Adductor pollicis (4), Lumbricals (7, 22) FCR (8), FCU (9), BR (5), FDS (12). FDP (11)	Median nerve, ulnar nerve, AND 6 out of 10 structures
	(b) Describe the median nerve supply in the hand	Motor – **LOAF (1st & 2nd lumbricals, OP, APB, FPB** [superficial head]) Sensory – **palmar surface of lateral 3½ digits** & dorsum of distal halves of these digits	Both motor (LOAF, 2 out of 4 muscles needed to pass) and sensory
	(c) How would you clinically test the median nerve function in the hand?	Motor – **thumb opposition** (OP) or **thumb abduction** (APB) [thumb flexion not reliable as deep head of FPB supplied by ulnar nerve] Sensory – sensation over volar aspect of **lateral 3½ digits** [sensation over thenar eminence preserved as supplied by palmar cutaneous branch of median nerve]	Both motor and sensory
Stem: Moving onto Physiology.			
Question 2 Resting Membrane Potential **Subject:** Phys LOA: 1	(a) Define resting membrane potential of a neuron	**Potential difference** across cell at rest, as a result of separation of positive and negative electronic charges across cell membrane (**inside negative** relative to outside of cell). Normal RMP of neuron = -70mV	Bold
	(b) Explain how resting membrane potential is created Prompt: Why is RMP negative on the inside of a cell?	Main ions involved – **Na^+ & K^+** **Na^+-K^+-ATPase pump** creates **electrochemical gradient** by pumping out 3 Na^+ for every 2 K^+ pumped in **Na^+ & K^+ diffuse down concentration gradient** across permeable cell membrane (K^+ diffuses from inside to outside of cell; opposite for Na^+) Cell membrane **more permeable to K^+** at rest → that's why RMP is close to equilibrium potential for K^+ RMP represents an equilibrium state; driving force for ions down concentration gradient = driving force down electrical gradient	Bold
	(c) Why is a cell more excitable in hyperkalaemia	RMP moves **closer to threshold potential for eliciting action potential** (becomes less negative on the inside of cell).	Bold

Stem: Moving onto Pathology. On examination there is a purulent discharge coming from the wound.			
Question 3 Staph aureus **Subject:** Path LOA: 1	(a) Name some common bacteria that cause wound infections	**Staphylococcus aureus** **Streptococcus pyogenes** Clostridium perfringens Aerobic Gram negative bacilli Pseudomonas aeruginosa Clostridium tetani	Staph aureus, Strep and 1 other
	(b) What diseases are caused by Staphylococcus aureus?	Skin / soft tissue : cellulitis, impetigo, abscess (furuncle, carbuncle), folliculitis, paronychia, felon, lymphadenitis, necrotising soft tissue infection, scalded skin syndrome Pneumonia Endocarditis Osteomyelitis / septic arthritis Food poisoning Toxic shock syndrome	3 skin and 3 non-skin infections
	(c) Describe the clinical features of Staph. Aureus toxic shock syndrome.	Hypotension (shock), renal failure, coagulopathy, liver disease, respiratory failure, generalised erythematous rash, soft tissue necrosis at site of infection	4 out of 7 (must have specific organs)
Stem: Moving onto Pharmacology. Prior to surgery for debridement, Flucloxacillin is administered			
Question 4 Flucloxacillin **Subject:** Pharm LOA: 1	(a) What micro-organisms are susceptible to flucloxacillin Prompt: is fluclox active against all Staph?	**Staphylococci** (including B lactamase producing**), streptococci** (not active against enterococci, anaerobes, Gram negatives, MRSA)	Bold
	(b) What is the mechanism of action of flucloxacillin Prompt : how does penicillin work	Inhibits bacterial growth by binding to active site of PBPs, **interfering with transpeptidation of bacterial cell wall synthesis** → cell death **(bactericidal)**	Bold
	(c) Why is oral flucloxacillin given before meals	It is acid labile (inactivated by gastric acid), and binds to food proteins (decreasing absorption)	1 out of 2
	(d) What are the important side effects of flucloxacillin?	**Liver (cholestasis),** GI upset (Nausea, vomiting, etc), renal interstitial nephritis, neutropenia/thrombocytopenia, **allergy/anaphylaxis**, serum sickness.	Both bold to pass
	Extra question: What is the frequency of cross allergenicity between flucloxacillin and cephalosporins	Around 5-10%	Any % in range to pass

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

Stem: A 50 year old alcoholic presents with a GCS of 7 and respiratory depression. Starting with Physiology			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Control of Ventilation **Subject:** Phys LOA: 1	(a) What are the receptors involved in the control of ventilation?	**Central chemoreceptors, Peripheral chemoreceptors,** Pulmonary stretch receptors, Irritant receptors, J receptors, Bronchial C fibres, Nose and upper airway receptors, Joint and muscle receptors, Gamma system, Arterial baroreceptors, Pain & temperature receptors	Bold & 3 others to pass
	(b) Where are the central chemoreceptors located?	200-400 μm below ventral surface of **medulla.**	Medulla must be stated
	(c) How do these receptors function? Prompt : How do H^+ ions affect their function	BBB permeable to CO_2; relatively impermeable to HCO_3^- **↑blood pCO_2 → ↑CSF pCO_2 → ↑H^+ in CSF** **↑H^+ in CSF stimulates ventilation** ↓H^+ in CSF inhibits ventilation; causes cerebral vasodilation → enhance diffusion of pCO_2 into CSF CSF pH 7.32. Less buffering than blood, CSF pH changes more for given pCO_2 Prolonged pH changes compensated by HCO_3^- transport across BBB. (Chronic CO_2 retention has near normal CSF H^+)	Bold concepts to pass
Stem: He requires intubation and vecuronium is administered. Moving on to pharmacology.			
Question 2 Vecuronium **Subject:** Pharm LOA: 1	(a)What is the mechanism of action of vecuronium	**Non depolarising neuromuscular blockade** **Competitive antagonist** for acethylcholine at **nicotinic receptors** of **neuromuscular junction** Large doses will enter ion channel's pore directly → more intense blockade Also blocks prejunctional Na channels → interfere with Ach mobilization at nerve endings	Must mention blockade type, & either receptor type or ACh.
	(b) Describe the pharmacokinetics of vecuronium Prompt : what is its onset time, what is its duration of action, how is it eliminated	Highly polar/ionic Poorly absorbed from GIT Given IV **Onset within 1 min;** **Max effect at 3-5 mins** **Duration of action : 20-35 mins** **Short half life** **Rapidly distributed** to extracellular space Small volume of distribution (~blood vol), Plasma protein binding : 60-90%, **Eliminated by liver** (75-90%), rest by kidney	4 of 6 bold to pass

Stem: Following intubation he requires inotropic support and a central line is inserted. Moving onto Anatomy			
Question 3 Anterior Neck Photo **Subject:** Anat LOA: 1	(a) Identify the **venous** structures in this photo	SVC (26), right brachiocephalic v (18), left brachiocephalic v (13), subclavian v (24), internal jugular v (8), inferior thyroid v (7)	4 to pass
	(b) Identify the **nerves** in this photo	Phrenic nerve (17), right vagus nerve (22), right recurrent laryngeal nerve (20), left vagus nerve (15), sympathetic trunk (28)	3 to pass
	(c) What is the difference between the course of the right and left recurrent laryngeal nerve	Right : hooks around subclavian artery Left : hooks around aorta After looping, they ascend in trachea-oesophageal groove to supply intrinsic muscles of larynx (except cricothyroid)	Both to pass
Stem: These are his coagulation blood results.			
Question 4 **Clinical Building Block:** Coagulopathy	What is the abnormality on this coagulation profile	Delayed clot formation in both the extrinsic (PT / INR) and intrinsic (APTT) systems. Fibrinogen low. Consistent with a consumptive coagulopathy/DIC	Must state coagulopathy / DIC with one example of possible cause
	What could cause this	Sepsis, liver failure, malignancy, trauma, envenoming (Brown / Tiger / Taipan) etc	
Stem: Moving onto Pathology			
Question 5 Cirrhosis **Subject:** Path LOA: 1	(a) What types of liver disease may result from chronic excessive alcohol consumption	Hepatocellular steatosis (fatty change) – reversible Alcoholic hepatitis – reversible Cirrhosis – non reversible Hepatocellular carcinoma – non reversible	1 reversible and 1 non-reversible
	(b) What are the morphological features of cirrhosis Prompt : what happens to liver cells when chronically exposed to toxins or injurious agent	Occurs **diffusely** throughout the liver, **parenchymal nodules (regenerating hepatocytes)** surrounded by dense **bands of fibrous scar, disorganised architecture**, variable degrees of **vascular / portosystemic shunting,** elements of progression and regression	3 out of 5 bold to pass
	(c) What are the possible sequelae of cirrhosis	**Portal Hypertension**, GIT Bleeding, Hepatic Failure, Coagulopathy, Hepatocellular Ca, Hepatorenal Syndrome, Hepatopulmonary Syndrome, Encephalopathy, Infection	Bold plus 3 others

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

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: A motor bike accident victim is transferred from a rural ED to a trauma centre. A chest X-ray is performed post intubation			
Question 1 **Clinical Building Block:** CXR- Pul contusions	Describe the positive findings in this CXR. What is the likely cause?	Portable supine CXR, ETT insitu (2cm above carina), **increased opacities in both lungs** (interstitial & alveolar) – increased opacity in RLL & obliteration of right hemidiaphragm **Pulmonary contusion** (+/- haemothorax)	Must be able to describe CXR, opacities. Pneumothorax difficult to exclude on supine film. Must say pulmonary contusion
Stem: He is hypoxic. Moving on to physiology			
Question 2 Oxygen uptake along the pulmonary capillary **Subject:** Phys LOA: 1	(a) In an alveolus, what factors affect oxygenation (b) Describe the oxygen uptake along a pulmonary capillary (c) How does hypoxia affect oxygenation	**Ventilation, perfusion, diffusion across the blood gas barrier** and alveolar-pulmonary capillary pO_2 gradient **Alveolar pulmonary capillary O_2 gradient** (Alveolar pO_2 = 100mmHg, pulmonary capillary pO_2 = 40mmHg), blood gas barrier thickness 0.3 microns, **RBC transit time = 0.75s** Under normal circumstances, **O_2 uptake is perfusion-limited** (complete in 0.25s) **& alveolar end capillary O_2 difference is minimal.** Rate of rise of end capillary pO_2 is steep – O2-Hb dissociation curve **Alveolar pulmonary capillary O_2 gradient is decreased, O_2 diffusion is decreased** & rate of rise of pO_2 for given O_2 concentration in blood is less	3 Bold to pass Must have knowledge of 3 of 4 concepts in bold. Numbers not required to pass. Can draw graph to explain (West pages 28-29)
Stem: He is quadriplegic and hypotensive. Moving onto Pharmacology. A Noradrenaline infusion is commenced			
Question 3 Noradrenaline **Subject:** Pharm LOA: 1	(a) What receptors do NA act on (b) How does NA increase blood pressure Prompt : what is the effect of NA on blood vessels (c) How does NA affect the heart rate?	**Predominantly α 1 receptor** → vascular smooth muscle constriction Also α 2 receptor (presynaptic) – inhibits NA release (negative feedback) Some effect on β 1&2 receptors (more potent effect on β 1) α 1 activity → vasoconstriction → **↑ total peripheral resistance** → ↑DBP β 1 activity → **↑ myocardial contractility** → ↑ SBP Overall rise in both DBP & SBP β 1 activity ↑heart rate. However compensatory baroreflex causes reflex bradycardia → **therefore minimal change in HR**	Need to mention predominant α 1 and one other receptor. Bold Bold

Stem: You suspect that he must have a cervical spinal cord injury. Moving onto Anatomy			
Question 4 Bones – C1, 2 **Subject:** Anat LOA: 1	(a) Identify this bone	**C2 (axis)**	Must say C2.
	(b) Describe its features	On C2; (1)body,**(2)dens**, (3)impression for alar ligament, (4)superior and inferior articular surface, (5)pedicle, (6)lamina, (7)bifid spinous process, (8)transverse process with foramen, (9)vertebral foramen	Bold + 4 other features
	(b) Name the ligaments that stabilize the atlanto-axial joint Prompt : how is the dens kept in place	**Transverse ligament** Superior & inferior longitudinal band } Cruciate lig Alar ligaments Tectorial membrane (continuation of post long lig) Anterior atlanto-axial membrane (continuation of ant long lig) Posterior atlanto-axial membrane (continuation of lig flavum) Apical ligament	Transverse lig & 2 others
	(c) What movement occurs at the atlanto-axial joint	**Rotation** around vertical axis	Bold
Stem: Moving onto Pathology			
Question 5 Spinal Cord injury including cellular injury as it relates to spinal cord **Subject:** Path LOA: 2 and 1	(a) What changes occur in the spinal cord after a traumatic injury	**Acute phase :** haemorrhage, necrosis, axonal swelling in the surrounding white matter at level of injury **Late phase :** area of neuronal destruction becomes cystic & gliotic, 2° wallerian degeneration involving long white matter tracts, liquefactive necrosis often seen in CNS	1 acute, 1 late
	(b) What are the features of irreversible injury at the cellular level	**Mitochondrial damage:** Failure of oxidative phosphorylation → **ATP depletion** → failure of energy dependent cellular functions **Membrane damage:** Plasma membrane → loss of osmotic balance Lysosomal membrane → **enzyme leakage** → cell necrosis	3 out of 4 bold
	(c) What are the acute clinical consequences of a cervical spinal cord injury Prompt: what happens in a high cervical level injury?	Complete or incomplete Spinal shock - **Quadriplegia**/flaccid paralysis, total anaesthesia, areflexia If above C4 → **respiratory compromise** (diaphragmatic paralysis) Neurogenic shock : hypotension, bradycardia, warm dry skin etc Incomplete syndromes, eg anterior cord, central cord etc	Bold