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

Stem: A 40 yo man presents with extensive burns to the lower half of his body. A CVC is inserted. **We are starting with Anatomy**			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Photo – Anterior Triangle of Neck (McMinn's p39) **Subject:** Anat LOA: 1	What are the boundaries of the anterior triangle of the neck? SCM has been removed in this photo. Where is the internal jugular vein? Describe its course. What major structures are at risk during insertion of an IJ line.	**SCM, midline, mandible** 22 Internal jugular vein IJV – continuation of sigmoid sinus Contained in **carotid sheath**. Lies lateral and ventral to artery. Goes **deep to SCM** and 2 heads of SCM – sternal and clavicular heads **Joins subclavian vein** posterior to **sternal end of clavicle**. Forms **brachiocephalic vein.** external carotid artery (11) common carotid artery (8) vagus (63) , other nerves, lung, trachea, scm, thyroid, thoracic duct	All 3 to pass Need to identify Concept. 4/5 Bold to pass. 2 to pass
Stem: We are now moving to Pathology			
Question 2 Thermal Injury (Robbins pp 421-422) **Subject:** Path LOA: 1	How are thermal burns classified? What are the potential complications of thermal burns? How do you determine the extent of burns?	According to depth of injury: • **Superficial** – confined to epidermis • **Partial thickness** – extends to dermis • **Full thickness** – involves subcutaneous tissue **Early:** • Hypovolaemic shock (especially with >20% BSA) • Compartment syndrome (circumferential LL burn) • Associated injuries (eg inhalational burn, CO poisoning) • Airway compromise • Hypermetabolic state **Late:** • Infection / sepsis (Pseudomonas) • ARDS • Multi organ failure • Skin grafting, scarring / cosmetic • Psychological TBSA calculation notoriously inaccurate. Does not include superficial burns • Wallace "rule of nines"/Lund & Browder diagram	Bold required 2 early and 2 late Mention 1 method

Stem: We are now moving on to Physiology			
Question 3 Venous Pressure and flow (Ganong 24th ed pp 582-584) **Subject:** Phys LOA: 1	1. Describe the mechanisms of venous return to the heart	a) **Thoracic pump:** inspiration resulting in negative pressure in the thorax and positive pressure in the abdomen. Blood flow towards the heart because of venous valves b) Effect of heart beat: during systole, AV valves are pulled downward → increase the capacity of the atria c) Muscle pump: contraction of muscles around the veins in the limbs during activity d) Differential resistance: resistance of the large veins near the heart is less than peripheral veins	Thoracic pump plus one other
	2. What factors might effect the CVP of this patient?	a) **Decrease CVP**: Fluid loss; blood loss b) **Increase CVP**: Excessive fluid replacement; other pre-existing conditions eg CCF; positive pressure ventilation; increased thoracic pressures	1 example from each bold category
	3. What is the value of mean CVP in normal individuals	4.6-5.8 mmHg or 6-8 cm H2O	Reasonable value
Stem: We are now moving to Pharmacology. He is resuscitated with Hartmann's solution			
Question 4 Compound Sodium Lactate (MIMs & product information) Constitution, Indications, Adverse effects. Comparison to other crystalloids and colloids **Subject:** Pharm LOA: 1	(a) How does Hartmann's solution differ from normal saline?	Addition of **Sodium Lactate, Potassium** Chloride**, Calcium** Chloride (+pH adjustment) Na 131, K 5, Cl 112, Ca 2, Lactate/Bicarb 28 mmol Compare Normal Saline Na 150 Cl 150)	Bold
	(b) What are the potential advantages of Hartmann's solution in resuscitation?	Closer to physiologic – potassium, calcium **Less Hyperchloraemia** **Effective bicarbonate – some (slow) good effect on acidosis** (proof of superiority lacking)	Bold
	(c) What are the potential complications of IV fluid therapy?	overload/under resuscitation, hypothermia, extravasation, acidosis, electrolyte abnormalities, osmo changes, air embolism, infection, cerebral oedema, haemodilution	Bonus

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

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: An elderly lady presents with acute abdominal pain. **We are starting with Anatomy.**			
Question 1 Photo of Abdominal wall (fig 258A) **Subject:** Anat LOA: 2	1 What structures in this photograph are potential sources of acute abdominal pain? 2 Identify the (other) vascular structures in this photograph (*if not already*) 3 Describe the arterial supply and venous drainage of the gut	**Aorta** (aneurysm), **Coeliac axis and SMA** (mesenteric ischaemic), **kidneys and ureters** (stones/infarcts), Splenic artery (aneurysm/dissection), Lymph nodes (adenitis/pressure), psoas (abscess or bleed) Landmarks and levels**: IVC, left renal vein, right renal vein. Aorta, Coeliac axis** (T12), **superior mesenteric artery** (L1). **Foregut (+hepatobiliary & spleen) - Coeliac axis**: common hepatic (->cystic, hepatic, right gastric, gastro-duodenal), splenic, left gastric (not shown); **Midgut (duodenum to transverse colon)-SMA**: inferior pancreaticoduodenal, jejunal/ileal branches, ileocolic, right and middle colic. **Hindgut-IMA** (small calibre + collaterals, therefore rarely blocked). Venous drainage – superior mesenteric vein (joins splenic vein to form portal vein), inferior mesenteric vein	Bold 4/6 bold Bold
Stem: We are now moving on to Pathology. She has ischaemic bowel.			
Question 2 Thrombosis **Subject:** Path LOA: 1	1. What factors predispose to thrombus formation in a vessel? 2. How are hypercoaguable states categorised? What are some examples of each type? 3. What are the possible outcomes for a vessel thrombus?	Virchows triad. **Endothelial injury; Alteration in blood flow** (stasis or turbulence); **Hypercoaguability** of blood **Primary (Genetic)** • Mutations - Factor V Leiden, Prothrombin • Increased levels - factors VIII, IX, XI, fibrinogen • Deficiencies - AT3, Protein C, S • Fibrinolysis defects, homozygous homocystinuria **Secondary (Acquired)** • Prolonged bed rest, immobilisation, MI, AF, Tissue injury (surgery, #, burn), cancer, prosthetic valves,, DIC, HITS, Anti phospholipid antibody syndrome • Cardiomyopathy, nephrotic syndrome, hyperoestrogenic states (pregnancy, post partum), OCP, sickle cell anemia, smoking • Note: often multifactorial **Propagation** (e.g. resulting occlusion); **Embolization; Dissolution; Organisation and recanalization** (e.g. to variable degree)	3/3 bold 2 categories plus Primary - 2 examples Secondary – 3 examples 2/4 categories

Stem: We are now moving to physiology. Arterial blood gases show a metabolic acidosis			
Question 3 Renal role in the handling of H+ ions **Subject:** Phys LOA: 1	1. Describe how the kidney responds to metabolic acidosis	**Renal tubule cells secrete H^+** into tubular fluid **in exchange for Na^+** **HCO_3^- is actively reabsorbed** into the peritubular capillary (for each H^+ secreted, $1Na^+$ and 1 HCO_3- are added into blood).	Bold
	2. What substances act as urinary buffers for the excretion of H^+	**NH_3** forms NH_4^+; **$HC0_3$-**forms CO_2 and H_20; **HPO_4^{2-}** forms H_2PO_4	2 of 3
	3. How else can the body compensate for a metabolic acidosis? Prompt: What other major system is involved in acidosis compensation?	The **respiratory system** responds by **increasing ventilation** which results in a decrease in PCO2 which causes increase in pH (this is a rapid response)	Bold to pass

Stem: We are now moving to Pharmacology. Her medications include captopril			
Question 4 ACE inhibitors **Subject:** Pharm LOA: 2	What is the mechanism of action of captopril?	**Angiotensin converting enzyme** (kininase II) **inhibitor**: inhibits hydrolysis of A1 to A2. Hence, **inhibits A2** effects (potent vasoconstrictor and increases Aldosterone secretion – salt and H2O retention) and decreases PVR, BP. Also, **inhibits bradykinin inactivation** to cause vasodilatation and decreased PVR, BP.	Bold to pass
	What are the adverse effects of captopril?	**Hypotension**, 1st dose esp. if hypovolaemic, diuretics, NaCl restriction, GI loss **ARF** esp. with bilateral RAS **HyperK+** esp. if renal insuff, DM **Cough, angioedema** (bradykinin, substance P), wheeze Fetal abnormalities (hypotension, anuria, renal failure – 2nd/3rd trim, increased teratogenesis – 1st trim) Altered taste, allergic skin rash, drug fever (10%)	3 of Bold to pass
	What drugs interact with captopril?	**K+ supplements, K+ sparing diuretics** – increase hyperK+ NSAIDs – impair BP reduction (block bradykinin) Other antihypertensives; haemaccel	Bold to pass

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

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: An 80 year old woman is transferred to your ED following a motor vehicle accident 12 hours ago, where she sustained serious chest injuries. **We are starting with Anatomy.**			
Question 1 Chest X-ray **Subject:** Anat LOA: 2	1. Outline the structures that make up the cardiomediastinal borders on this normal X-Ray	Right: **R Brachiocephalic v, SVC, R Pulmonary Artery, R Atrium, IVC** Left: **Aorta, L Pulmonary Trunk/Artery, L Atrium, L Ventricle**	6 Bold to pass
	2. Which parts of the lungs lie adjacent to the cardiomediastinum?	Right upper mediastinum: **R superior lobe** Right heart border: **R middle lobe** Left upper mediastinum: **L superior lobe** Left heart border: **Lingula segment of L superior lobe**	RML plus one other
	3. In this patient, what injuries may be seen on a CXR? (Prompt: the patient has sustained blunt trauma)	**Chest wall:** # ribs, clavicle, sternum **Lung:** pneumothorax, haemothorax, contusion, **Cardiovascular**: aorta, other vessels (widen mediastinum)	1 example from each bold category to pass
Stem: We are moving to Pathology. She has multiple wounds oozing blood due to DIC			
Question 2 DIC **Subject:** Path LOA: 2	1. On a full blood count and coagulation profile, what would you expect to find?	↓Hb (MAHA – microangiopathic haemolytic anaemia), ↑WCC, **platelets↓**, Fibrinogen↓, PT/INR↑, a/PTT↑ and **fibrin degradation products↑**	Bold to pass
	2. What are the pathological consequences of DIC?	DIC – major trauma releases tissue thromboplastins. Both sides of clotting cascade are activated. 2 major consequences – deposition of fibrin within microcirculation leading to **ischaemia/micro thrombosis** of vulnerable organs; and a **consumptive coagulopathy** - platelets and clotting factors leading to a **bleeding diathesis**.	Bold to pass 3/3
	3. What are the causes of DIC?	**Obstetric** – FDIU, amniotic fluid embolism, preeclampsia, **Sepsis** **Malignancy** – acute promyelocytic leukaemia, adenoca of lung, pancreas, stomach and colon **Trauma**- multi/burns/environmental/snakebite	Must get 3 categories

Stem: We are moving to Physiology. She is shocked			
Question 3 Circulatory Catecholamines **Subject:** Phys LOA: 1	1. Name the endogenous catecholamines? Where are they produced? (prompt to match catechol with source) 2. What are the physiological effects of adrenaline and noradrenaline?	**Adrenal Medulla: Adrenaline, Noradrenaline**, Dopamine. Intrinsic Cardiac Adrenergic Cells: Adrenaline. Sympathetic Nervous System Cells: Dopamine **Metabolic**- Glycogenolysis, increased metabolic rate, mobilisation of free fatty acids, increased lactic acid Cardiovascular- **vasoconstriction and dilation, increase heart rate and strength** α1:Constriction of blood vessels, smooth muscles (esp norad) α2:Mixed smooth muscle effects (esp adren) β1:Cardiac ionotropy and chronotropy, irritability (both) β2:Dilation blood vessels liver & muscle, other smooth muscle relaxation (adrenaline) β3: Lipolysis, detrusor relaxation (esp adren)	Bold One metabolic and bold cardiovascular Extra info only

Stem: We are now moving to pharmacology. You decide to use Bupivicaine as the local anaesthetic to insert a chest tube			
Question 4 Bupivicaine **Subject:** Pharm LOA: 1	1. What is the mechanism of action of bupivacaine? 2. How long will a bupivacaine block last? 3. What are the potential adverse effects from bupivacaine? 4. How can the risk of these effects be minimised in the ED?	1. **Blocks** voltage-gated **sodium channels in nerve.** Threshold for excitation increases, conduction slows, AP rise declines, AP generation abolished. If Na current blocked over length of nerve, propagation is ceased. 2. **3-6 hours** 3. **CNS toxicity** (sedation/light headedness/visual&auditory/tongue&mouth numbness/metallic taste/nystagmus/restlessness/ muscle twitches/seizure/resp depression), **Cardiac toxicity** (arrhythmias/cardiovascular collapse/cardiac arrest), Local toxicity (trauma/neurotoxicity) Allergy 4. Ask re Hx of allergy, Use safe max dose (<2mg/kg), withdraw pre injection, avoid vessels-anatomical consideration (above rib below) & use USS. Ask pt to flag Sx e.g. taste/tongue numb. Avoid hypoxia/acidosis.	Bold Approximate or long duration Bold Extra