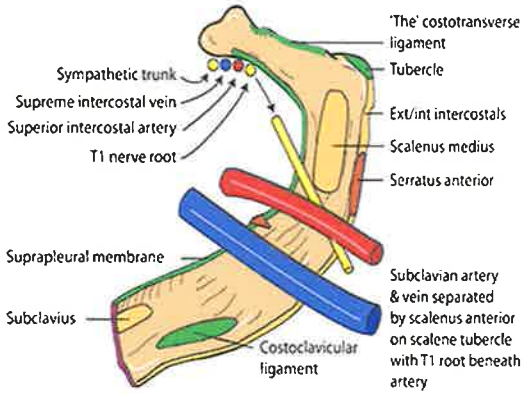
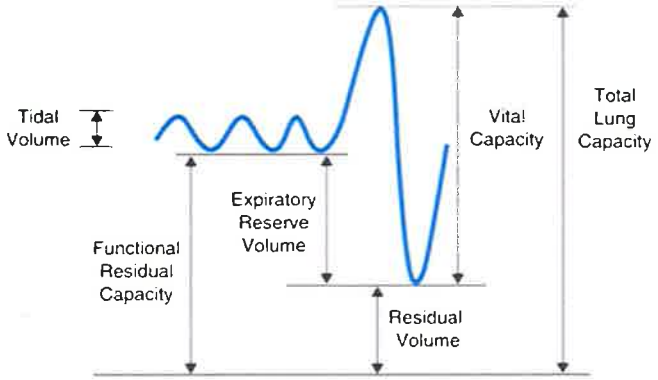
Stem: A 20 year old motor cyclist is brought to the ED with chest injuries. He is asthmatic.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: Let's start with Pharmacology. He has been given Salbutamol nebulisers for his wheeze.			
Question 1 Salbutamol **Subject:** Pharm LOA: 1	1. What is Salbutamol? 2. Describe the **pharmacokinetics** of salbutamol? (Prompt for t1/2) 3. Describe the **pros and cons** of the different **routes** of delivery of salbutamol? Prompt: Is there any other route? (non-inhaled)	Salbutamol is a selective B2 agonist and used as a Bronchodilator 1. **Absorption – Fast and complete (inhaled)** a. GIT – rapidly absorbed b. Inhaled – Bronchodilation maximal within 15-30 min and persists for 3-4 hours. 2. Metabolism – 50% 1st pass. Sulphated in the liver and metabolites excreted in the kidneys (also excreted unchanged in renal. No metabolism in lungs 3. t1/2 – 3-6 hours **1. Inhaled** a. Spacer/inhaler Pro: Targeted, low dose, minimise systemic side effects. As effective as nebulised. No 1st pass metabolism Con: Coordination and education required b. Nebulised Pro: Less coordination required and minimal education Con: Larger particles and hence dose required, noisy (children get frightened), higher incidence of systemic SE 2. Oral Pro: Easier in very young/disabled. Longer t1/2 Cons: Big doses, high SE profile (tachycardia, tremor, nervousness and weakness). Minimal advantage to inhaled. 50% first pass metabolism 3. IV/IMI/SC – useful in severe asthma Pro: No first pass metabolism Con: Needle, painful, higher cost and SE profile	**A selective B2 agonist** **1. Absorption – Fast or complete (inhaled).** **2. Rapid onset of action** **3. t1/2 3-6 hours** **(2 of 3 to pass)** **Need to describe pros and cons of Inhaled plus 1 other route**

Stem: Arterial blood gases are done as part of his initial trauma work up.			
Question 2 **Clinical Building Block:**	Please **describe** this ABG. On O2 – Fi02 60% P02 85 pCO2 123 pH 6.99 HCO3 28	Primary respiratory acidosis with CO2 retention and hypoxia	**Primary respiratory acidosis with CO2 retention and hypoxia**
Stem: Moving onto Anatomy. Chest X-ray shows multiple rib fractures.			
Question 3 First rib **Subject:** Anat LOA: 1	1. Please identify this bone and demonstrate its features **(bold to pass)** Prompt: What's this? (scalene tubercle) 2. What are the important relations?	**First rib** Head/neck/shaft/ tubercle (articulates with TP of T1) /articulation with costal cartilage to manubrium **/groove for subclavian vein** (ant) **and artery** (posterior to **scalene tubercle**) **Apex of lung** **Subclavian vessels**, intercostal vessels & ns Sympathetic trunk Lower trunk of brachial plexus (sup.) Scalenus ant/ medius Intercostals, Serratus anterior, Subclavius	**RELATIONS AND ATTACHMENTS OF LEFT FIRST RIB**  The under surface of the 1st rib is smoother. When the rib is laid on a flat surface, the head touches the flat surface when the rib is the correct way up
Stem: Moving onto Physiology.			
Question 4 Lung Volumes and Curves **Subject:** Phys LOA: 1	1. **Draw** a diagram that demonstrates the components of total lung volume.	Should correctly include TLC, VC, FRC, TV, RV, ERV  	TLC, VC, FRC, TV, RV, ERV **(3/6 to pass)**

	2. What are the typical volumes? Optional: Which of these volumes can be measured in the ED?	TLC ~7000ml, VC ~4500 to 5000 mL, RV ~1200 mL, FRC ~2400 mL, TV ~500mL FEV1, FVC or TV.	**2/4** (reasonable approximations)
Stem: Moving onto Pathology.			
Question 5 Asthma **Subject:** Path LOA: 1	1. What are the pathological features of acute asthma? 2. What is the underlying mechanism of atopic asthma? Prompt: What may trigger an exacerbation? 3. What happens in the early-phase reaction in atopic asthma?	1. **Increased airway responsiveness; episodic bronchoconstriction; bronchial wall inflammation; increased mucus** 2. **IgE mediated type 1 hypersensitivity**; **Environmental allergens/triggers** (eg dust, pollens, foods, drugs) 3. **Allergen exposure** produces IgE a. re-exposure triggers mast cell degranulation/cytokines b. bronchoconstriction c. mucus production d. vasodilation/incr vasc permeability	**3/4 to pass** **Bold and one trigger** Bold & concept

Stem: A 60 year old lady presents to ED with a painful arm following a fall.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: She has significant pain and is given morphine			
Question 1 Morphine **Subject:** Pharm LOA: 1	1. What is the mechanism of action of morphine?	**Act on receptors: mu**/delta/kappa **Reduce presynaptic neurotransmission (esp glutamate)** **Inhibit post-synaptic neurons** Central (thalamic action)	Mu + 1 other mechanism of action to pass
	2. Why do opiates cause respiratory depression?	**Inhibition of brainstem respiratory controls** allowing less response to hypercapnoea	Bold to pass
	3. How is morphine metabolised?	**Conjugated in liver** (morphine-3-glucuronide = most) Small amount (10%) morphine-6-glucuronide = increased analgesic potency Renal excretion	Bold to pass
Stem: Here is her xray.			
Question 2 **Clinical Building Block:** (# humerus)	Describe the abnormality	**Spiral/oblique fracture mid-shaft L humerus** with displacement.	
	What structure may be injured in this fracture?	**Radial nerve**	
Stem: Moving on to normal Anatomy.			
Question 3 Humerus X-ray **Subject:** Anat LOA: 1	1. Identify the features of the **humerus** on this x-ray	Prox: **Head**, Anat and **Surg neck, Shaft** **Gt tuberosity**/Lesser Tuberosity Distal: **Medial + Lateral** epicondyles, Trochlea, Capitulum, Lateral and medial supracondylar ridges	6 bold to pass
	2. What are the rotator cuff muscles and describe their actions	**Subscapularis** medial rotation of humerus **Supraspinatus** initiates abduction and abducts shoulder **Infraspinatus** and **teres minor** – lateral rotators of humerus All 4 muscles stabilise shoulder joint	4 muscles + 1 action to pass

Stem: Moving on to Pathology.			
Question 4 Fracture Healing **Subject:** Path LOA: 1	1. How do fractures heal? Prompt: What are the stages of fracture healing?	1. **Haematoma** 2. **Influx of Inflammatory cells, platelets, fibroblasts, new vessels and osteoprogenitor cells = procallus** 3. **Fibrocartilagenous callus**: mesenchymal cells + new cartilage along fracture line undergoes **endochondral ossification =** 4. **Bony Callus** 5. **Remodelling**	3/5 to pass + detail
	2. What factors inhibit fracture healing	Inadequate immobilisation/severe displacement/poor reduction/soft tissue Vascular compromise Infection, foreign body Systemic – nutrition, osteoporosis etc.	3 to pass
Stem: Moving on to Physiology.			
Question 5 Calcium metabolism **Subject:** Phys LOA: 1	1. How is plasma calcium regulated?	**1,25-dihydroxycholecalciferol (from Vit D) incr Ca absorption from gut & kidneys.** **PTH mobilises Ca from bone**, incr Ca reabs in kidneys, incr 1,25 DHCC formation in kidneys **Calcitonin** (from thyroid) **inhibits bone resorption**, incr Ca excretion in urine.	2/3 to pass
	2. How is the synthesis of 1,25-dihydroxycholecalciferol (vit D) regulated?	**1,25-DHCC formed in kidneys by 1α-hydroxylase.** **High Ca/high PO4 inh 1,25-DHCC** (incr inactive 24,25-DHCC instead). **Low Ca incr PTH which stimulates 1α-hydroxylase** (low PO4 directly stimulates 1α-hydroxylase).	Bold to pass

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

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: A patient presents with a Verapamil overdose.			
Question 1 Verapamil **Subject:** Pharm LOA: 1	1. Describe the mechanism of action of verapamil	**Block** voltage-gated L-type **Ca channels** (α1 subunit), reduced frequency of opening when depolarised, resulting in **decreased** transmembrane Ca current, and **Ca influx**: Vascular smooth muscle relaxation (< Dihydropyridines) **Cardiac – decrease AVN conduction, contractility**, CO	Bold to pass
	2. What are the toxic effects of verapamil?	CVS: bradycardia, AV block, cardiac arrest, heart failure, hypotension Minor: flushing, dizziness, nausea, constipation, peripheral oedema	3 to pass 1 to pass
	3. What antidotes can be used to treat verapamil toxicity?	Calcium iv, high-dose insulin (euglycaemia) therapy	1/2 to pass
Stem: Moving onto Anatomy. Intravenous access is obtained.			
Question 2 Cubital fossa/ forearm photo **Subject:** Anat LOA: 1	1. Describe the boundaries of the cubital fossa	**Lateral: Brachioradialis** (5), (extensors from lat epicondyle) **Medial: Pronator teres** (20), (flexors of forearm from CFO) Floor: Brachialis, supinator **Superior: Line between 2 epicondyles of humerus** Roof: Skin, deep fascia reinforced by bicipital aponeurosis (3)	Bold to pass
	2. Please identify the major veins that can be seen in the photo	Basilic vn (1); cephalic vn (6); median cubital vn (13); median forearm vn (14)	2/4 to pass
	3. Identify the major arteries that can be seen in the photo (Bonus: which is the larger terminal branch?)	**Brachial a** (4), radial a (21), ulnar a (22) Ulnar a	Bold to pass
Stem:			

Question 3 **Clinical Building Block:** Photo myoglobinuria and biochemistry	Interpret her biochemistry results? Why is her urine dark?	**Renal failure** likely Acute kidney injury in clinical context - elevated urea + Cr **Markedly elevated CK - rhabdomyolysis** Normal K^+ Rhabdomyolysis Breakdown of skeletal muscle -> **myoglobinuria**	Essential in bold
Stem: Moving onto Pathology			
Question 4 Acute Kidney injury / rhabdomyolsysis **Subject:** Path LOA: 2	1. Define Acute Kidney Injury 2. What are the causes of AKI (please give examples)?	Clinico-path entity, **acute reduction of renal function** with morphologic **tubular injury (usually).** Reversible. 1. **Ischaemia**/abnormal blood flow. Systemic – assoc with thrombosis (HUS, TTP, DIC) or hypovolaemia. Intra-renal – angiopathies, malignant HT 2. **Toxic injury to glomeruli/tubules** – myoglobin, drugs, contrast 3. Acute tub.int nephritis – hypersensitivity reaction to drugs, IgA nephropathy 4. Obstruction ("post-renal") – tumour, clot, stones	Bold Bold and 1 other category 1 example for each
Stem: Moving onto Physiology.			
Question 5 Cardiovascular Regulatory Mechanisms **Subject:** Phys LOA: 1	1. What are baroreceptors? 2. Where are they located? 3. What is their mechanism of action in hypotension?	**Stretch** receptors in the adventitia layer of vessels Located at **aortic arch** and **carotid sinus**, walls of right and left atria (SVC and IVC entrances) and pulmonary circulation. In response to hypotension, the arterial **baroreceptors are less stimulated** because they are less stretched. Reduced baroreceptor discharge travels via glossopharyngeal and vagus nerves to the medulla resulting in an **overall increase in sympathetic discharge** to increase heart rate and stimulate vasoconstriction and **reduce vagal drive**.	Bold Bold and 1 other Bold to pass and understand inhibitory concept