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

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 PATHOLOGY LOA: 1	*"An elderly man presents with an acute exacerbation of COPD."* What is the definition of emphysema? Describe the pathogenesis of emphysema. Prompt: What is the mechanism of the destruction? What are the possible complications of emphysema?	• A condition of the lung characterised by **irreversible enlargement** of the airspaces distal to the terminal bronchiole accompanied by **destruction of their walls** without obvious fibrosis. • **Mild chronic inflammation (neutrophils + macrophages)** - **mediator release** (e.g. leukotriene B_4, IL-8, TNF) – causes damage and sustains inflammation • **Protease-antiprotease imbalance – destructive effect of high protease activity** in pts with **low anti-protease activity** - 1% of pts with emphysema have alpha1-antitrypsin deficiency (inhibits proteases, including elastase, secreted by neutrophils) • **Oxidant-antioxidant imbalance – abundant reactive oxygen species** (superoxide dismutase, glutathione)**in smoke depletes antioxidant mechanisms,** incite tissue damage • Bullous lung disease • Expiratory airflow limitation • Infection • Respiratory failure • Pneumothorax • Cor pulmonale, congestive heart failure ("pink puffers")	**BOLD TO PASS** • Irreversible • Destruction **TWO EFFECTS** • Chronic inflammation • High protease activity • Reactive oxygen species **THREE COMPLICATIONS**
Question 2 PHYSIOLOGY LOA: 1	1. What are the possible physiological causes for hypoxemia in this man? 2. What is the alveolar gas equation ? 3. Explain the concept of the A-a gradient.	Hypoventilation Diffusion limitation Shunt V/Q mismatch $PAO_2 = PIO_2 - \frac{PACO_2}{R} + F$ Difference between the measured and the predicted paO_2.	Need 2 /4 to pass or a good understanding of the concepts Numbers ok Need the basic concept
Question 3 PHARMACOLOGY LOA: 1	*"Moving on. He is treated with a cephalosporin."* 1. What is the mechanism of action of cephalosporins? 2. What class of antibiotics do they belong to? 3. How are they classified and give an example of each class ?	1. **Inhibit bacterial cell wall synthesis**, cell division and growth (similar to penicillins) Bactericidal Work best in rapidly dividing cells 2. Beta-lactams 3. Generations – First through Fourth 4. **1st Generation**: very active against GPC, E. coli, K. pneumoniae, Proteus OK but Pseudomonas not. Anaerobic cocci sensitive. Cephalexin, Cephazolin	1. Bold to pass 2. **Beta-lactams** 3. **4 Generations** 4. Concept of increasing activity against gram –ves and example of 2 classes

	Prompt: How does the spectrum of microbiological activity differ between the different generations?	**2nd Generation**: active against those by 1st generation but added GN coverage – Klebsiella, some anaerobe cover. NO Pseudomonas. Cefaclor, Cefuroxime **3rd Generation:** expanded GN coverage and cross BBB. Less active vs Staph. Effective against against B- lactamase producing Haemophilus and Neisseria. Ceftazadime works vs Pseudomonas. Ceftriaxone, Ceftazidine, Cefotaxime. **4th Generation:** more resistant to B- lactamases, extended coverage against enteric GNR, pseudomonas, enterobacteriaceae, S pneumonia, S aureus, Haemophilus and Neisseria. Cross BBB. Cefipime.	
Question 4 ANATOMY LOA: 1	*"Moving on, the patient has limitation of shoulder movement."* What muscles are called the "rotator cuff muscles?" Demonstrate or describe the origins and insertions of the rotator cuff muscles. Note that the model has no rotator cuff muscles. What are the actions of the rotator cuff muscles?	**Subscapularis** Origin – Medial 2/3 costal surface of scapula Insertion – fuses with capsule of shoulder joint and into lesser tuberosity of humerus Nerve – Upper and lower subscapular **Teres minor** Origin – Dorsal surface axillary border of scapula Insertion – Lower facet greater tuberosity humerus Nerve – Posterior branch axillary N **Supra spinatus** Origin – medial2/3 supraspinous fossa scapula Insertion – Upper part of greater tuberosity humerus Nerve – Suprascapular nerve C5,6 **Infraspinatus** Origin – Medial 2/3 infraspinous fossa and deep surface infraspinous fascia which covers muscle. Insertion – Central facet greater tuberosity humerus Nerve – Supra scapular **Supraspinatus** – **initiates abduction** and other muscles hold humeral head down **Subscapularis** – **medial rotation** of humerus **Infraspinatus** and **teres minor** –**lateral rotators** of humerus Supraspinatus – **abducts shoulder** All muscles **stabilise the shoulder joint** by bracing humeral head against glenoid (tendons fuse with capsule)	Must know all 4 to pass Must have knowledge about origins, insertions and actions of 2/4.

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

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 PATHOLOGY LOA: 1	*"A patient presents with chronic inflammatory arthritis."* 1. What are the characteristics of chronic inflammation? 2. Why does macrophage accumulation persist in chronic inflammation? 3. What are the causes of chronic inflammation? (prompt can you give an eg. of each)	• Inflammation for a **prolonged period** (week or more). • Characterised by **macrophages**, lymphocytes and plasma cells • With simultaneous-active inflammation/ **tissue destruction** and **attempts at repair** by connective tissue, fibrosis **Continued recruitment** of monocytes (continued expression of adhesion molecules and chemotactic factors) Local proliferation of macrophages Immobilisation of macrophages • **Persistent infection**- TB, syphilis • **Autoimmune**-RA, MS, IBD, SLE • **Prolonged exposure to an agent**: exogenous-silica->silicosis , FB, persistent trauma endogenous-lipid->atherosclerosis	¾ Bold to pass Bold 2/3 bold with examples
Question 2 PHYSIOLOGY LOA: 1	Question 2 - Physiology 1. List the physiological effects of glucocorticoids 2. What are the vascular effects of abruptly stopping long term glucocorticoids? Bonus: What is the benefit of elevated glucocorticoid levels in stress?	a) **Inc protein catabolism.** b) Inc hepatic glycogenolysis and gluconeogenesis, inc Glu-6-phosphatase → **inc plasma glucose** c) Antiinsulin effects on peripheral tissues d) Inhibit ACTH secretion e) **Controls vascular reactivity to NAd and Ad** f) Control ability to excrete water load g) Increased neutrophils/ plts/ RBC and dec eosinophils/ lymphocytes/ basophils Vascular smooth muscle becomes **unresponsive to NAd and Ad** Capillaries dilate and inc permeability Failure to respond to NAd **impairs vascular compensation** for hypovolaemia and promotes **vascular collapse** Effect on **vascular activity to catecholamines** plus necessary for catacholamines to mobilise **FFA** for emergency energy source	2 bold and 2 others Must have general concept
Question 3 PHARMACOLOGY LOA: 1	1. Moving on to pharmacology. What is the mechanism of action of the non steroidal anti – inflammatory drugs (NSAIDs)? 2. How does aspirin differ from other NSAIDs in its action on COX?	NSAIDs serve to suppress inflammation chiefly by **inhibiting prostaglandin synthesis.** In so doing they decrease the sensitivity of vessels to bradykinin and reverse the vasodilation of inflammation. Cyclo – oxygenase (COX) is the key catalyst for arachidonic acid conversion to prostaglandins. **NSAIDs inhibit COX,** thus inhibiting this conversion. **Aspirin** (original NSAID**) irreversibly inhibits COX**, whilst the **newer NSAIDS** (ibuprofen, diclofenac) **reversibly** inhibit COX.	**Pass criteria** Inhibit COX, thus decrease prostaglandin synthesis – and in so doing the response to inflammation is modulated. **Irreversible vs reversible**

	2. What are the adverse effects of NSAIDs?	2 types of COX exist – COX 1 is expressed in most cells, and COX 2 is inducible, its expression varies depending on stimulus. Selective COX 2 inhibitors (celecoxib) do not affect platelet function at usual doses, whilst the other NSAIDs do inhibit platelet aggregation. **GI EFFECTS** – GI irritation, ulcers, abdominal pain, N and V **BLEEDING** – secondary to platelet effects **RENAL** – nephrotoxicity, hyperkalaemia ALLERGY – rash, pruritis **CARDIOVASCULAR** – Selective COX 2 inhibitors - implicated in increased risk of c'vasc thrombotic events, - fluid retention, oedema, hypertension CNS – headaches, tinnitus, dizziness, stroke PULMONARY – asthma HAEM - rare – t'cytopaenia, neutropaenia HEPATIC – abnormal LFTs	**¾ Bold plus one other to pass** – namely – GI effects, bleeding, and renal effects...plus any one of the others
Question 4 ANATOMY LOA: 2	Moving on to anatomy a. This is a photograph of the gluteal region. Identify the labelled structures. Point to piriformis if not identified. b. Describe the actions of the gluteus maximus muscle. c. Describe the course of the sciatic nerve in the gluteal region and leg.	**23. Sciatic nerve** – tibial part 1 – common fibular part **2. gluteus maximus** 3. gluteus medius 4. gluteus minimus 5. greater trochanter of femur 6. inferior gamellus 7. inferior gluteal artery 8, 21, 22. inferior gluteal nerve 9. internal pudendal a 10. ischial tuberosity 11. nerve to obturator internus 13. obturator externus 14. obturator internus **15. piriformis** 16. posterior femoral cutaneous nerve 17. pudendal nerve 18. quadratus femoris 19. sacrotuberous ligament 20. superior gamellus **Straightens the leg at the hip** during walking, running, climbing. Assists in raising from a sitting position. Lower part acts as adductor and external rotator of lower limb. Tensor of the fascia lata and by its connection with the iliotibial band, steadies the femur on the tibia during standing when the extensor muscles are relaxed. Enters the gluteal region via the greater sciatic foramen **inferior to piriformis** and **deep to gluteus maximus**; descends in midline of the posterior thigh deep to biceps femoris; **bifurcates into tibial and common fibula nerves at apex of popliteal fossa.**	**All three bolded** to pass and at least two other named structures. **Bold to pass** **Two of three bolded to pass**