

TOPIC	QUESTIONS	KNOWLEDGE (essential in bold)	NOTES
<p>Question 1 PATHOLOGY</p> <p>LOA: 1</p>	<p><i>“An elderly man presents with an acute exacerbation of COPD.”</i></p> <p>What is the definition of emphysema?</p> <p>Describe the pathogenesis of emphysema.</p> <p>Prompt: What is the mechanism of the destruction?</p> <p>What are the possible complications of emphysema?</p>	<ul style="list-style-type: none"> • 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 B₄, 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”) 	<p>BOLD TO PASS</p> <ul style="list-style-type: none"> • Irreversible • Destruction <p>TWO EFFECTS</p> <ul style="list-style-type: none"> • Chronic inflammation • High protease activity • Reactive oxygen species <p>THREE COMPLICATIONS</p>
<p>Question 2 PHYSIOLOGY</p> <p>LOA: 1</p>	<ol style="list-style-type: none"> 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. 	<p>Hypoventilation Diffusion limitation Shunt V/Q mismatch</p> $PAO_2 = PIO_2 - \frac{PACO_2}{R} + F$ <p>Difference between the measured and the predicted paO₂.</p>	<p>Need 2 /4 to pass or a good understanding of the concepts</p> <p>Numbers ok</p> <p>Need the basic concept</p>
<p>Question 3 PHARMACOLOGY</p> <p>LOA: 1</p>	<p><i>“Moving on. He is treated with a cephalosporin.”</i></p> <ol style="list-style-type: none"> 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 ? 	<ol style="list-style-type: none"> 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 	<ol style="list-style-type: none"> 1. Bold to pass 2. Beta-lactams 3. 4 Generations 4. Concept of increasing activity against gram –ves and example of 2 classes

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

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<p>Question 1 PATHOLOGY</p> <p>LOA: 1</p>	<p>“A patient presents with chronic inflammatory arthritis.”</p> <p>1. What are the characteristics of chronic inflammation?</p> <p>2. Why does macrophage accumulation persist in chronic inflammation?</p> <p>3. What are the causes of chronic inflammation? (prompt can you give an eg. of each)</p>	<ul style="list-style-type: none"> • 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 <p>Continued recruitment of monocytes (continued expression of adhesion molecules and chemotactic factors) Local proliferation of macrophages Immobilisation of macrophages</p> <ul style="list-style-type: none"> • Persistent infection- TB, syphilis • Autoimmune-RA, MS, IBD, SLE • Prolonged exposure to an agent: exogenous-silica->silicosis , FB, persistent trauma endogenous-lipid->atherosclerosis 	<p>¾ Bold to pass</p> <p>Bold</p> <p>2/3 bold with examples</p>
<p>Question 2 PHYSIOLOGY</p> <p>LOA: 1</p>	<p>Question 2 - Physiology</p> <p>1. List the physiological effects of glucocorticoids</p> <p>2. What are the vascular effects of abruptly stopping long term glucocorticoids?</p> <p>Bonus: What is the benefit of elevated glucocorticoid levels in stress?</p>	<p>a) Inc protein catabolism.</p> <p>b) Inc hepatic glycogenolysis and gluconeogenesis, inc Glu-6-phosphatase → inc plasma glucose</p> <p>c) Antiinsulin effects on peripheral tissues</p> <p>d) Inhibit ACTH secretion</p> <p>e) Controls vascular reactivity to NAd and Ad</p> <p>f) Control ability to excrete water load</p> <p>g) Increased neutrophils/ plts/ RBC and dec eosinophils/ lymphocytes/ basophils</p> <p>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</p> <p>Effect on vascular activity to catecholamines plus necessary for catecholamines to mobilise FFA for emergency energy source</p>	<p>2 bold and 2 others</p> <p>Must have general concept</p>
<p>Question 3 PHARMACOLOGY</p> <p>LOA: 1</p>	<p>1. Moving on to pharmacology. What is the mechanism of action of the non steroidal anti – inflammatory drugs (NSAIDs)?</p> <p>2. How does aspirin differ from other NSAIDs in its action on COX?</p>	<p>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.</p> <p>Cyclo – oxygenase (COX) is the key catalyst for arachidonic acid conversion to prostaglandins. NSAIDs inhibit COX, thus inhibiting this conversion.</p> <p>Aspirin (original NSAID) irreversibly inhibits COX, whilst the newer NSAIDs (ibuprofen, diclofenac) reversibly inhibit COX.</p>	<p>Pass criteria</p> <p>Inhibit COX, thus decrease prostaglandin synthesis – and in so doing the response to inflammation is modulated. Irreversible vs reversible</p>

