

Stem: A patient presents with a penetrating eye injury. He has been given oxycodone. Commencing with pharmacology			
TOPIC	QUESTIONS	KNOWLEDGE ( <b>essential in bold</b> )	NOTES
<p><b>Question 1</b></p> <p>Oxycodone (p 558)</p> <p><b>Subject:</b> Pharm LOA: 1-2</p>	<p>Describe the pharmacokinetics of oxycodone?</p> <p>Prompt: Describe the pharmacokinetics of opiates.</p> <p>What adverse effects might you anticipate?</p> <p>When prescribing oxycodone what prescribing strategies may help in reducing the development of, dependence.</p>	<p><b>Oral commonly</b> Good oral absorption High Vd <b>Low first pass metabolism CW others</b> 10 morphine = 4.5mg oxycodone duration 3-4h, longer if CR formulation. Hepatic met</p> <p>Sedation/Respiratory depression/N+V/hypotension/dysphoria/biliary colic/pruritis/caution in renal failure</p> <p><b>Smaller doses at longer intervals/establish goals at start of Rx/limit doses/use of other analgesics/frequent evaluation of ongoing need/use of modified CR formulations</b></p>	<p>Bold plus one more</p> <p>N+V a particular concern in context of penetrating eye injury</p> <p>3 to pass</p> <p>2 to pass</p>
Stem: Moving on to physiology You assess his visual acuity as 6/24.			
<p><b>Question 2</b></p> <p>Eye / Acuity / Vision (pp 178 -183)</p> <p><b>Subject:</b> Phys LOA: 2</p>	<p>How is visual acuity measured? What does the fractions of a VA of 6/24 represent?</p> <p>What factors influence visual acuity?</p> <p>Why is the fovea important for visual acuity</p>	<p>Measurement from <b>Snellen chart</b> viewed at a distance of <b>6m</b> or 20 feet; <b>6/24 indicates reduced VA</b></p> <p><b>Optical factors</b> The state of the image forming mechanisms/sharpness of focus <b>Retinal factors</b> the state of the cones <b>Stimulus factors</b> (illumination, brightness of the stimulus, contrast between stimulus and background, length time exposed to stimulus); sensitivity and interpretative ability of the brain Resolving power of the eye, property of the cones</p> <p>fovea is the point where <b>VA is greatest</b>; fovea is the centre of the macula, a thinned out rod free portion of the retina where the <b>cones</b> are densely packed &amp; each synapses on a single bipolar cell</p>	<p>numerator is the distance at which the chart is read; the denominator is the <b>smallest line</b> that can be read; 6/6 indicates normal vision;</p> <p>2/3 to pass</p> <p>One of bold</p>

<b>Stem: Moving on to anatomy.</b> He has abnormal eye movement			
<p><b>Question 3</b></p> <p>Eye (Model) – (model no. F 13)</p> <p><b>Subject:</b> Anat LOA: 2</p>	<p>Identify the muscles responsible of eye movement.</p> <p>Describe their actions.</p> <p>What nerves supply these muscles?</p> <p>How are the actions of these muscles tested clinically? <i>Prompt: Why is the “H” pattern used?</i></p>	<p><b>Recti: Superior (elev, add, med rot); Inferior (dep, add, lat rot); Medial (add); Lateral (abd)</b></p> <p><b>Obliques: Superior (dep, abd); Inferior (elev, abd)</b></p> <p><b>Oculomotor (III) N</b> to all, except Abducent (VI) N to Lateral R, and Trochlear (IV) N to Sup Obl</p> <p>In <b>Abd</b> (LR): Elev (SR) and Dep (IR) In <b>Add</b> (MR): Elev (IO) and Dep (SO)</p>	<p>All Bold to pass</p> <p>Bold plus one to pass</p> <p>abd and add isolates recti and obliques to pass</p>
<b>Stem: Moving on to pathology:</b> Six weeks later ne develops sympathetic ophthalmia, which is a <b>Type IV</b> hypersensitivity reaction			
<p><b>Question 4</b></p> <p>Type 4 hypersensitivity reaction (pp 205-208; 1356)</p> <p><b>Subject:</b> Path LOA: 1</p>	<p>1. Describe the sequence of events that lead to this reaction.  Prompt: what cells are involved?</p> <p>2. What tissue changes would occur</p> <p>3. Name other examples of Type IV hypersensitivity reactions.</p>	<p>Injury</p> <p><b>Initiated by antigen</b> sensitised CD4+ or CD8+ <b>T cells</b></p> <p>Retinal antigens may be transported in the lymphatics of the damaged eye</p> <p>Reaction may occur in both eyes causing a Pan Uveitis.</p> <p>CD4+ predominate in autoimmune disease CD8+ in post infectious (esp viral) reactions</p> <p><b>Can be cytokine</b> (CD4+ Th1 or TH17 cells involved) <b>or direct cellular</b> (Cytotoxic lymphocyte) <b>mediated</b> tissue injury (either satisfactory).</p> <p>Perivascular cellular infiltrates, tissue oedema, granuloma formation, cell destruction.</p> <p>Type I diabetes Multiple sclerosis Rh arthritis Inflammatory bowel disease Guillain Barre Contact sensitivity dermatitis Tuberculin reaction Granulomatous diseases Viral hepatitis</p>	<p>Requires antigen and either cytokine or direct cellular mechanisms to pass</p> <p><b>(2/4 to pass)</b></p> <p><b>(2 examples to pass)</b></p>

Stem: An elderly woman is brought to your ED hypothermic and unconscious. She is intubated. Commencing with pharmacology			
TOPIC	QUESTIONS	KNOWLEDGE (essential in bold)	NOTES
<p><b>Question 1</b> Biotransformation – Phase 1 and 2 reactions with an emphasis on Suxamethonium (Chp 4) <b>Subject:</b> Pharm  LOA: 1</p>	What is drug biotransformation?	Drug metabolism to allow drugs to become <b>inactive or by increasing excretion</b> by making them more hydrophilic, or by metabolising them to less active agent.	Bold
	Describe phase 1 and phase 2 reactions?	Phase 1 – <b>unmasking functional group</b> (-OH, -NH <sub>2</sub> , -SH) to become more polar metabolite. Includes oxidation, deamination, hydrolysis, reductions Phase 2- <b>conjugation with endogenous substrate</b> to become highly polar conjugate	Bold
	How is Suxamethonium metabolised?	<b>Rapid phase 1 hydrolysis</b> by <b>butyrylcholinesterase and pseudocholinesterase</b> in liver and plasma Genetically deficient in BCHE so slowed metabolism	One of the bold
	Why may a patient have a prolonged paralysis following Sux		
<b>Stem: Moving on to physiology</b>			
<p><b>Question 2</b> Hypothermia / thermoregulation (pp 316-320) <b>Subject:</b> Phys LOA: 1</p>	By what processes does the body lose heat?	<b>Radiation &amp; Conduction</b> (70% of loss at 21 °C) <b>Vaporization of sweat</b> (27%) Respiration (2%) Urination & defecation (1%)	Bold to pass
	How does the body produce heat?	<b>Basal metabolic processes</b> Food intake <b>Muscular activity</b> Shivering Hunger Increased voluntary activity Increased secretion of Adr + NorAdr	Bold to pass
	What temperature-regulating mechanisms are activated by the cold?	Decreased heat loss mechanism Cutaneous vasoconstriction Curling up Horripilation	4 to pass
	What part of the brain controls the reflex responses activated by cold?	The posterior <b>hypothalamus</b>	bold
<b>Stem: Moving on to pathology.</b>			

<p><b>Question 3</b> Cerebrovascular Disease (pp 1290-1295) <b>Subject:</b> Path LOA: 1</p>	<p>What are the types of cerebral ischemic injury? Prompt: Describe the patterns cerebral ischemic injury</p> <p>What are the causes of focal cerebral infarction? Prompt: Give examples</p> <p>What are the pathological effects of hypertension on the brain?</p>	<p><b>Global cerebral ischemia</b> (ischemic/ hypoxic encephalopathy) when there is a <b>generalised reduction</b> of cerebral perfusion <b>Focal cerebral ischemia</b> follows reduction of blood flow to a <b>localised</b> area of the brain</p> <p><b>Embolic</b> (from cardiac mural thrombi; thromboemboli from arteries, esp. carotid; paradoxical assoc with cardiac anomalies; tumour, fat or air), <b>thrombotic arterial occlusion/</b> in situ thrombosis (large vessel disease); <b>Vasculitis</b> (small vessel disease) infectious (immunosuppression and aspergillus, CMV encephalitis, syphilis, TB); non-infectious eg PAN, primary angiitis; <b>Others</b> eg amphetamines, cocaine, heroin; dissecting aneurysm extracranial arteries; hypercoaguable states</p> <p>Lacunar infarcts (in lenticular nucleus, thalamus, internal capsule, deep white matter, caudate nucleus, pons); slit haemorrhages; hypertensive encephalopathy; massive intracerebral haemorrhage)</p>	<p>Both types and description</p> <p>3 causes plus 1 example of each</p> <p>4 out of 4</p>
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**Stem: Moving on to anatomy** She has a swollen right elbow.

<p><b>Question 4</b> Elbow X-ray <b>Subject:</b> Anat LOA: 1</p>	<p>Identify the bony features on this Xray</p> <p>What factors determine the stability of the elbow joint? Prompt – What are the ligaments of the elbow</p>	<p>Medial / Lateral epicondyles, capitulum, olecranon, radius – head/neck, olecranon fossa, coronoid fossa, trochlea, proximal radio-ulnar joint, coronoid process of ulna</p> <p><b>Bony factors – shape of trochlea / olecranon fossa</b> Joint capsule – fibrous joint capsule weak <b>Ligaments</b> – <b>radial collateral ligament</b> – lateral epicondyle and blends with the <b>annular ligament</b> of the radius (which holds the radial head in the radial notch of the ulna). - medial <b>ulnar collateral ligament</b> from medial epicondyle to the coronoid process and olecranon of the ulna - 3 bands 1. Anterior – strongest 2. Posterior – weakest 3. oblique – deepens trochlear notch Muscles – biceps, brachialis,(BR), triceps RCL &amp; UCL &amp; annular ligament</p>	<p>6 to pass</p> <p>Bone and ligaments 3 of 4 bolded</p>
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Stem: A 50 year old woman is brought to the ED with an amitriptyline overdose. Commencing with Pharmacology.			
TOPIC	QUESTIONS	KNOWLEDGE (essential in bold)	NOTES
<p><b>Question 1</b> Tricyclics including Volume of distribution (Chp 30) <b>Subject:</b> Pharm  LOA: 1</p>	<p>Which factors determine the volume of distribution of a drug?</p> <p>Describe the volume of distribution of tricyclic antidepressants How does this influence their toxicity?</p> <p>What therapies for tricyclic toxicity might reduce their tissue distribution?</p>	<p><b>Drug factors;</b> lipid solubility (high in TCA), pKa, pH, protein binding (high in TCA). <b>Patient factors;</b> age, gender, comorbid disease (eg. Oedema or ascites), body fat, blood flow to tissues. TCAs have a <b>large Vd</b> (5-30L/kg), tissue <b>concentrations are high</b> especially in well perfused organs such as <b>the brain and heart.</b></p> <p><b>Alkalinisation</b> (Bicarbonate or hyperventilation) increases <b>plasma protein binding of the free drug</b> removing it from the tissues reducing its tox</p>	<p>At least 2 from each group</p> <p>bold</p> <p>bold</p>
Stem: Move onto Anatomy. You insert a femoral venous line.			
<p><b>Question 2</b> Femoral Triangle (photo)  <b>Subject:</b> Anat LOA: 1</p>	<p>Demonstrate the boundaries of the femoral triangle.</p> <p>What are the contents of the femoral triangle.</p> <p>What surface markings help would you look for when trying to locate the femoral vein? Which veins drain into the common femoral vein</p>	<p><b>Inguinal ligament</b> (11), medial border of <b>Sartorius</b> (23) and lateral border of <b>adductor longus</b> (1) form the triangle, <b>pectineus</b> (med) and <b>iliopsoas</b> (lat) form the floor. Contents = <b>femoral vein</b> (6), <b>artery</b> (4) and <b>nerve</b> (5) (med to lat) and deep inguinal lymph nodes.</p> <p><b>Artery is found below inguinal ligament, midway between ASIS and pubic tubercle, vein is just medial to artery</b> Continuation of the femoral vein, popliteal vein, receives <b>profunda femoris</b> and <b>great saphenous vein</b> (7), ends posterior to the inguinal ligament where it becomes the external iliac vein. Also receives superficial epigastric vein (27), superficial circumflex iliac vein (25) and superficial external pudendal vein (28).</p>	<p>3/5 to pass</p> <p>all content</p> <p>3/3 to pass</p> <p>bold</p>
Stem: Moving on to Pathology. She has a history of chronic alcohol abuse.			
<p><b>Question 3</b></p>	<p>1. Describe the pathological effects on the liver long-term alcohol ingestion.</p>	<p>1. <b>Steatosis:</b> fatty change, perivenular fibrosis</p>	<p>Bold with 3 morphologic features of each to pass.</p>

<p>Alcoholic Liver Disease (pp 857-860)  <b>Subject:</b> Path</p> <p>LOA: 1</p>	<p>PROMPT: please describe the morphological features</p> <p>2. Which of these conditions reversible?</p> <p>3. What are the possible sequelae of cirrhosis?  Prompt: Complications?</p>	<p>2. <b>Hepatitis:</b> liver cell necrosis, inflammation, Mallory bodies, fatty change, fibrosis</p> <p>3. <b>Cirrhosis:</b> extensive fibrosis, hyperplastic nodules</p> <p>4. (Hepatocellular carcinoma)</p> <p><b>Steatosis and Hepatitis are reversible.</b> Cirrhosis irreversible.</p> <p><b>Portal Hypertension,</b> GIT Bleeding, Hepatic Failure, Coagulopathy, Hepatocellular Ca, Hepatorenal Syndrome, Hepatopulmonary Syndrome, Encephalopathy, Infection</p>	<p>Bold to pass</p> <p>Bold plus 3</p>
<p><b>Stem:</b> Moving on to Physiology</p>			
<p><b>Question 4</b>  Dead Space (pp 19-21)</p> <p><b>Subject:</b> Phys</p> <p>LOA: 1</p>	<p>What is DEAD SPACE?</p> <p>2. What types of DEAD SPACE are there?"  Prompt explain difference between the two types</p> <p>3. How is it measured? (bonus)</p>	<p>Portion of the tidal volume that <b>does not participate in gas exchange</b> <math>V_T = V_D + V_A</math></p> <p><b>1. ANATOMICAL</b></p> <ul style="list-style-type: none"> <li>• Volume of <b>conducting airways</b> – trachea, bronchi, terminal bronchi (16 gen)</li> <li>• About 150mls of 500ml <math>V_T</math></li> <li>• Measured by Fowler’s method</li> <li>• Determined by: <ul style="list-style-type: none"> <li>○ Increased diameter of airways during inspiration</li> <li>○ Size &amp; posture of individual</li> </ul> </li> </ul> <p><b>2. PHYSIOLOGICAL</b></p> <ul style="list-style-type: none"> <li>• Volume of gas that <b>does not eliminate CO<sub>2</sub></b></li> <li>• Same as anatomical DS in normal individuals</li> <li>• <b>Increased in lung disease</b> because of inequality of blood flow and ventilation within the lung</li> </ul> <p>Measured by Bohr method</p>	<p>Demonstrate principle of bold to pass</p> <p>Two types dead space and describe what it is</p>