STEM: Following administration of anti-venom for a snakebite, a 60 yr old man is noted to be hypotensive. We will begin with Physiology....

TOPIC	QUESTIONS	KNOWLEDGE (essential in bold)	NOTES
Question 1 **PHYSIOLOGY** **Cardiac Output** LOA: 1 (Ganong 24th ed p545-552)	What is cardiac output?	**Output of the heart per unit time.** **HR x SV**	**Bold**
	What factors determine cardiac output?	SV is related to the **preload (degree of stretch prior to contraction) and afterload** (resistance to flow) of the heart and the **intrinsic contractility** of the myocardial cells. HR- Sympathetic vs parasympathetic stimulation.	
	What methods can be used to measure cardiac output?	**Direct Fick method or indicator (or thermal) dilution** Can also measure by **Doppler U/sound** techniques Fick principle; amount of substance taken up by organ per unit time = (A-V conc difference) x blood flow. In the heart can use O_2. LV output =O_2 consumption ml/min/[A_{O2}]-[V_{O2}] (both in ml/L) Indicator dilution; substance injected IV and serial sampling in arterial blood performed, log plotted and extrapolated to find circulation time (indicator must not be lost from circulation)	*2 to pass*
	What causes of decreased cardiac output could be causing this man's hypotension?	1) variation in heart rate due to induction of **arrhythmias or heart block** (too fast or too slow) 2) **Reduced preload** (venodilatation with reduced venous return due to anaphylaxis) 3) Increased afterload (not too likely in this case **4) Reduced contractility** (i.e. ischaemia, venoms, drugs)	

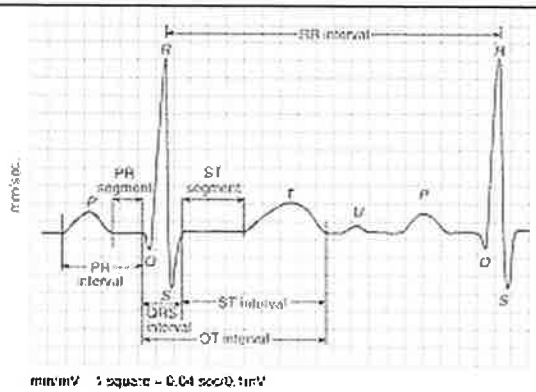
The patient develops airway obstruction and is going to be intubated. We are now moving to Pharmacology.			
Question 2 **PHARMACOLOGY** **PROPOFOL** LOA: 1 (Katzung 12th ed p 438-440)	1. Describe the pharmacokinetics of propofol.	**1. Distribution half life 2-4 minutes** **Elimination half life 4-23 minutes** **Rapid onset and recovery**. Termination of drug effect due to **redistribution** from brain to sk muscle and then fat (rather than metabolism). Duration of action 3-8min Rapidly metabolised in liver and extrahepatic sites (lungs). Water soluble metabolites excreted in urine.	**Bold**
	2. What is the usual induction dose of propofol?	**2. 1-2.5mg/kg adults,** 2.5-3.5mg/kg in kids	**Bold**
	3. What clinical effects are expected after this dose of propofol is administered.	**3. Anaesthesia / Sedation**. Respiratory depression. Transient **apnoea**. **Decreased blood pressure** through vaso and venodilation (most pronounced of induction drugs). Does NOT have analgesic properties Anti-emesis, Metabolic acidosis, Pain at injection site	**Bold**
	4. List some drug interactions of propofol important in the setting of sedation/anaesthesia	**4.** Opioids – enhance respiratory depression Benzodiazepines - enhanced sedation and respiratory depression	**1 of 2**

We are now moving to Anatomy			
Question 3 **ANATOMY** **Model – Larynx (Full model with tongue in situ)** LOA: 1 Model - Tongue & Airway (Somso upper airway models)	1. a) Identify the structures in the upper airway that could lead to airway obstruction b) What other structures are visible 2. What are the bony and cartilaginous components of the larynx 3. What is the innervation of the larynx?	**1.** **Tongue** Tonsils, **pharynx** **Epiglottis,** glottis **Hyoid** bone, floor of mouth - **mylohyoid (Prompt)** **Mandible** Buccal muscles, cheek. Medial pterygoid muscles ary-epiglottic folds & vallecula, Piriform fossa **2. Cartilage:** **Cricoid, Thyroid,** Arytenoids **and Epiglottis** Bone: Hyoid **3. Motor:** **Recurrent laryngeal N** (inferior laryngeal-terminal branch of Rec Laryngeal) except for Cricothyroid which is External Laryngeal N (tenses cords). Both from CN X **Sensory:** **Above cords: Internal Laryngeal N** (branch of superior laryngeal N) **Below cords: Recurrent Laryngeal N** (Inferior laryngeal branch) (Br of CrN X)	**bold 5/6 total** **2 underlined** **All bold** **Bold**

One week later he develops serum sickness. We are now moving to Pathology			
Question 4 **PATHOLOGY** **Type 3 Hypersensitivity** LOA: 1 (Robbins pp 204-205)	1. What is the pathogenesis of serum sickness? Prompt (if required): How is the tissue damage caused?	**1. Type 3 hypersensitivity** Phase 1: **Formation** of **Immune complexes.** Protein Ag, 1/52 -> Ab -> blood -> Ag-Ab complexes Phase 2: **Deposition** of immune complexes. Medium size, Ag excess most pathogenic High pressure filtration , glomeruli, joints Phase 3: **Tissue injury** caused by immune complexes **Acute inflam reaction** ~ day 10 IgG & IgM (C' fixing Ab) bind to leukocyte Fc receptors. Leuk recruitment and activation - release proteases/lysozymal enzymes ->damage. Deposition, activation and Consumption of C' and decreased C3 levels -> inflam reaction and tissue damage	Bold 3 Phases
	2. What are some clinical features?	2. Fever, urticaria, arthralgia, LN enlargement, proteinuria	3 of 5
	3. What are some other examples of Type III hypersensitivity?	3. Acute: post strep G-N, reactive arthritis, Arthus reaction Chronic: SLE, PAN, other vasculitides, possibly membranous G-N,	3 examples

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

Stem: A 65 yr old man presents with an inferior myocardial infarction
We are starting with Physiology

TOPIC	QUESTIONS	*KNOWLEDGE* ***(essential in bold)***	NOTES
Question 1 **PHYSIOLOGY** **ECG including MI changes** LOA: 1 Ganong 24th ed pp 524-529, 534-537	1. Draw and describe an ECG tracing of a single normal heart beat Prompt: What produces the waves and segments?	 **P wave- atrial depolarization, PR AV conduction** **QRS- ventricular depolarization, ST- plateau of Vent depolarization, (QT- Ventricular Action potential),** **T wave- Vent repolarization**	Bold 5/6
	2. What features would appear different in this patient's ECG?	**2. ST segment elevation in inferior leads** **ST segment depression in the reciprocal leads**	both
	3. At the myocardial cell membrane level, what causes these changes?	3. Abnormally rapid depolarisation in early phase (accelerated opening of K+ channels) Decreased resting membrane potential (due to loss of intracellular K+) Slowed depolarization of affected cells (cf normal cells)	1 of 3 to pass

We are now moving to Pharmacology			
Question 2 **PHARMACOLOGY GTN** LOA: 1 Katzung 12th ed Chapter 12) MoA, principles of tachyphylaxis	1. By what routes can GTN be administered?	1. **Sublingual, transdermal, IV, oral,** buccal, inhaled	Bold 3/4
	2. Why are parenteral routes favoured?	2. To avoid the **hepatic first pass** effect which significantly decreases bio-availability	bold
	3. What is meant by the term tachyphylaxis as it relates to Glyceryl Trinitrate (**GTN**)	3. Continuous exposure to nitrates – smooth muscle may develop tolerance. Particularly seen with continuous IV infusion or long acting preparations. (oral, transdermal)	Understand concept
	What is the implication of this for the dosing and administration of GTN	Concept of "drug-free" interval – at least 8h between doses	concept
	What is the theoretical basis for this phenomenon? (bonus)	(a) Diminished release of nitric oxide resulting from reduced bioactivation secondary to depletion of tissue thiol compounds, decreased tissue sulphydryl groups, increased generation of O2 free radicals , decreased availability of CGRP. (b) Systemic compensation – after > 1 day of therapy salt and water retention reverse favourable hemodynamic change	for better candidates
	4. When should GTN be used with caution?	4. **hypotension**, those on sildenafil, inferior&posterior MI/RV infarct, Fixed cardiac output (AS, tamponade etc), raised ICP, significant tachy/brady cardia, allergy	Bold +2

We are now changing to Anatomy			
Question 3 **ANATOMY** LOA: 1 Heart model assembled	1. Identify the arterial supply of the heart	1. L+R coronary arise from corresponding aortic sinuses above AV **R coronary** courses inf in AV groove. Gives off branches to SA node, Marginal, Post interventric, and AV nodal **L coronary** bifurcates into **Circumflex and LAD** (anterior I – V art), then Cx gives off Marginal branch, and LAD gives off diagonals.	**bold** to pass
	2. What does the R Coronary artery supply?	2. R atrium, most of RV, Diaphragmatic surface LV Post 1/3 septum, 60% SA, 80% AV	3 out of 6 to pass
(Take the model back!)	3. Describe the venous drainage of the heart	3. Major drainage is via the **Coronary sinus** 3 main tributaries are: Great cardiac vein (accompanies LAD, then Cx) Middle (accompanies PIV) Small cardiac veins (accompanies R marginal). Oblique vein L atrium marks start of sinus. Ant cardiac vn's start ant surface RV, drain straight into R atrium Smallest cardiac vn's (venae cordis minimae) drain direct into chambers	bold +2
	4. Describe the major components of the conducting system	4. SA Node junction of SVC & RA AV Node near coronary sinus-postero-inferior interatrial septum AV Bundle R & L Bundles	3 of 4

We are now changing to Pathology			
Question 4 **PATHOLOGY** **Healing post MI** LOA: 1 Robbins pp 551-553, 102-106	1. What are the consequences and complications of a myocardial infarction	1. Contractile dysfunction/CCF, Arrhythmias, Myocardial rupture, Pericarditis, R vent infarction & RHF, infarct extension, Infarct expansion, Mural thrombus (=>embolism), Ventricular aneurysm, Papillary muscle dysfunction, Progressive late HF, Remodelling, death	6
	2. What are the main cardiac rupture syndromes	2. Free wall -> tamponade (most common of 3 occurs at 1-10 days) Septum -> VSD and L->R shunt Papillary muscle dysfunction -> severe Mitral Regurg	1 of 3
	3. What changes occur in ventricular remodelling	**3. Hypertrophy** and **dilatation**, increased oxygen demand -> **ischaemia** & **depressed cardiac function**, **scar formation** -> **stiffening** and hypertrophy.	3
	4. What systemic factors affect infarct healing?	4. Nutritional: **protein, Vit C** Metabolic: **diabetes** Circulatory: **arterial** or **venous** Hormonal: **glucocorticoids**	3