

**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 <b>PHYSIOLOGY</b>	What is cardiac output?	<b>Output of the heart per unit time.</b> <b>HR x SV</b>	<b>Bold</b>
<b>Cardiac Output</b> LOA: 1	What factors determine cardiac output?	SV is related to the <b>preload (degree of stretch prior to contraction) and afterload</b> (resistance to flow) of the heart and the <b>intrinsic contractility</b> of the myocardial cells. HR- Sympathetic vs parasympathetic stimulation.	
(Ganong 24 <sup>th</sup> ed p545-552)	What methods can be used to measure cardiac output?	<b>Direct Fick method or indicator (or thermal) dilution</b>  Can also measure by <b>Doppler U/sound</b> 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 <sub>2</sub> . LV output = O <sub>2</sub> consumption ml/min/[A <sub>O2</sub> ]-[V <sub>O2</sub> ] (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)	<i>2 to pass</i>
	What causes of decreased cardiac output could be causing this man's hypotension?	<ol style="list-style-type: none"> <li>1) variation in heart rate due to induction of <b>arrhythmias or heart block</b> (too fast or too slow)</li> <li>2) <b>Reduced preload</b> (venodilatation with reduced venous return due to anaphylaxis)</li> <li>3) Increased afterload (not too likely in this case)</li> <li>4) <b>Reduced contractility</b> (i.e. ischaemia, venoms, drugs)</li> </ol>	

**The patient develops airway obstruction and is going to be intubated. We are now moving to Pharmacology.**

<p>Question 2  <b>PHARMACOLOGY</b>  <b>PROPOFOL</b>          LOA: 1</p> <p>(Katzung 12<sup>th</sup> ed          p 438-440)</p>	<p>1. Describe the pharmacokinetics of propofol.</p> <p>2. What is the usual induction dose of propofol?</p> <p>3. What clinical effects are expected after this dose of propofol is administered.</p> <p>4. List some drug interactions of propofol important in the setting of sedation/anaesthesia</p>	<p><b>1. Distribution half life 2-4 minutes</b>  <b>Elimination half life 4-23 minutes</b>  <b>Rapid onset and recovery.</b> Termination of drug effect due to <b>redistribution</b> 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.</p> <p><b>2. 1-2.5mg/kg adults, 2.5-3.5mg/kg in kids</b></p> <p><b>3. Anaesthesia / Sedation.</b> Respiratory depression. Transient <b>apnoea</b>. <b>Decreased blood pressure</b> through vaso and venodilation (most pronounced of induction drugs).          Does NOT have analgesic properties          Anti-emesis, Metabolic acidosis, Pain at injection site</p> <p><b>4. Opioids – enhance respiratory depression</b>          Benzodiazepines - enhanced sedation and respiratory depression</p>	<p><b>Bold</b></p> <p><b>Bold</b></p> <p><b>Bold</b></p> <p><b>1 of 2</b></p>
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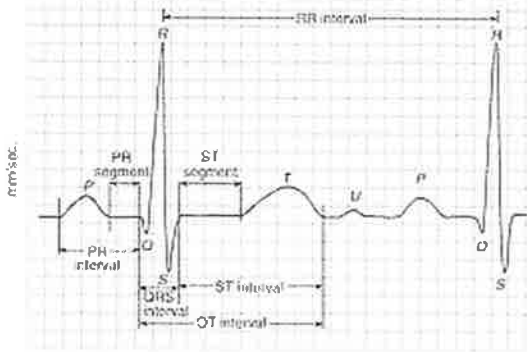
**We are now moving to Anatomy**

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

<p>Question 4  <b>PATHOLOGY</b>  <b>Type 3 Hypersensitivity</b>          LOA: 1</p> <p>(Robbins pp 204-205)</p>	<p>1. What is the pathogenesis of serum sickness?</p> <p>Prompt (if required):          How is the tissue damage caused?</p> <p>2. What are some clinical features?</p> <p>3. What are some other examples of Type III hypersensitivity?</p>	<p><b>1. Type 3 hypersensitivity</b>          Phase 1: <b>Formation of Immune complexes.</b>          Protein Ag, 1/52 -&gt; Ab -&gt; blood -&gt; Ag-Ab complexes          Phase 2: <b>Deposition</b> of immune complexes.          Medium size, Ag excess most pathogenic          High pressure filtration , glomeruli, joints          Phase 3: <b>Tissue injury</b> caused by immune complexes  <b>Acute inflam reaction</b> ~ day 10</p> <p>IgG &amp; IgM (C' fixing Ab) bind to leukocyte Fc receptors.          Leuk recruitment and activation - release proteases/lysozymal enzymes -&gt;damage.          Deposition, activation and Consumption of C' and decreased C3 levels -&gt; inflam reaction and tissue damage</p> <p>2. Fever, urticaria, arthralgia, LN enlargement, proteinuria</p> <p>3. Acute: post strep G-N, reactive arthritis, Arthus reaction          Chronic: SLE, PAN, other vasculitides, possibly membranous G-N,</p>	<p><b>Bold</b>          3 Phases</p> <p>3 of 5</p> <p>3 examples</p>
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**Stem: A 65 yr old man presents with an inferior myocardial infarction**  
**We are starting with Physiology**

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

**We are now moving to Pharmacology**

<p>Question 2 <b>PHARMACOLOGY</b> <b>GTN</b> LOA: 1</p> <p>Katzung 12<sup>th</sup> ed Chapter 12) MoA, principles of tachyphylaxis</p>	<ol style="list-style-type: none"> <li>1. By what routes can GTN be administered?</li> <li>2. Why are parenteral routes favoured?</li> <li>3. What is meant by the term tachyphylaxis as it relates to Glyceryl Trinitrate (<b>GTN</b>)</li> </ol> <p>What is the implication of this for the dosing and administration of GTN</p> <p>What is the theoretical basis for this phenomenon? (bonus)</p> <ol style="list-style-type: none"> <li>4. When should GTN be used with caution?</li> </ol>	<ol style="list-style-type: none"> <li>1. <b>Sublingual, transdermal, IV, oral, buccal, inhaled</b></li> <li>2. To avoid the <b>hepatic first pass</b> effect which significantly decreases bio-availability</li> <li>3. Continuous exposure to nitrates – smooth muscle may develop tolerance. Particularly seen with continuous IV infusion or long acting preparations. (oral, transdermal)</li> </ol> <p>Concept of “drug-free” interval – at least 8h between doses</p> <p>(a) Diminished release of nitric oxide resulting from reduced bioactivation secondary to depletion of tissue thiol compounds, decreased tissue sulphhydryl groups, increased generation of O<sub>2</sub> free radicals , decreased availability of CGRP.</p> <p>(b) Systemic compensation – after &gt; 1 day of therapy salt and water retention reverse favourable hemodynamic change</p> <ol style="list-style-type: none"> <li>4. <b>hypotension</b>, those on sildenafil, inferior&amp;posterior MI/RV infarct, Fixed cardiac output (AS, tamponade etc), raised ICP, significant tachy/brady cardia, allergy</li> </ol>	<p><b>Bold 3/4</b></p> <p><b>bold</b></p> <p><b>Understand concept</b></p> <p><b>concept</b></p> <p>for better candidates</p> <p><b>Bold +2</b></p>
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**We are now changing to Anatomy**

<p><b>Question 3</b>  <b>ANATOMY</b>          LOA: 1</p> <p>Heart model assembled</p> <p>(Take the model back!)</p>	<p>1. Identify the arterial supply of the heart</p> <p>2. What does the R Coronary artery supply?</p> <p>3. Describe the venous drainage of the heart</p> <p>4. Describe the major components of the conducting system</p>	<p>1. L+R coronary arise from corresponding aortic sinuses above AV  <b>R coronary</b> courses inf in AV groove. Gives off branches to SA node, Marginal, Post interventric, and AV nodal  <b>L coronary</b> bifurcates into <b>Circumflex and LAD</b> (anterior I – V art), then Cx gives off Marginal branch, and LAD gives off diagonals.</p> <p>2. R atrium, most of RV, Diaphragmatic surface LV          Post 1/3 septum, 60% SA, 80% AV</p> <p>3. Major drainage is via the <b>Coronary sinus</b>          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</p> <p>4. SA Node                    junction of SVC &amp; RA          AV Node                    near coronary sinus-postero-inferior interatrial septum</p> <p>AV Bundle          R &amp; L Bundles</p>	<p><b>bold</b> to pass</p> <p>3 out of 6 to pass</p> <p><b>bold +2</b></p> <p>3 of 4</p>
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**We are now changing to Pathology**

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