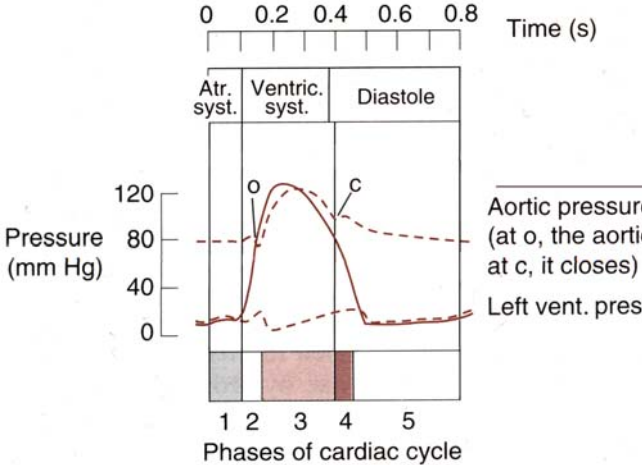
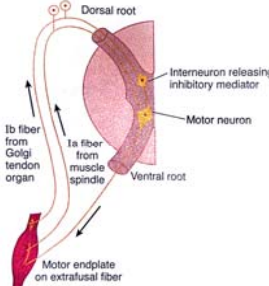


TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
<p>Question 1: Mechanical events/phases of cardiac cycle.</p>	<p>List in order, the mechanical phases of the cardiac cycle</p> <p>Please draw the pressure changes in the ventricle that occur during the cardiac cycle</p>	<ul style="list-style-type: none"> • Atrial systole. • Isovolumetric ventricular contraction. • Ventricular ejection. • Isovolumetric ventricular relaxation. • Ventricular filling. 	<p>Three out of five phases to pass this subsection. Basic diagram and correct pressure</p>
<p>Question 2</p>	<ol style="list-style-type: none"> 1) What is normal GFR 2) what factors affect GFR 	<ol style="list-style-type: none"> 1. @ 125ml/min in 70kg male, 10% less in female 2.a) Net filtration pressure= hydrostatic pressure in capillaries and bowmans capsule AND colloid osmotic pressure -afferent – efferent pressure- under control of – autoregulation, sympathetic, AgIII, dobutamine, PGs, serum Na - renin Pressure in bowmans capsule – renal obstruction 2. b)capillary filtration coefficient Relates to surface area and permeability of capillaries Surface area controlled by mesagial cells under control 	<ol style="list-style-type: none"> 1) reasonable value 2) 3 to pass 3) 1 of 2

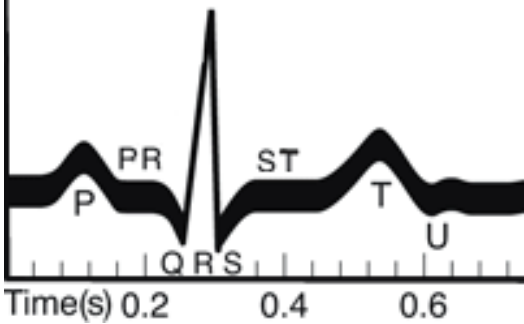
	<p>3)As well as filtration , by what other means does the kidney regulate the composition of urine –</p>	<p>of AgtII, ADH, NA, PGs (constrict) ANP, dopamine, cAMP, PGE2(relax)</p> <p>3.secretion and resorption</p>	
<p>Question 3 Vasopressin</p>	<p>(a) What stimuli influence vasopressin (ADH) secretion?</p> <p>(b) How does vasopressin exert its anti-diuretic effect?</p>	<p>Increased by</p> <ul style="list-style-type: none"> • decreased ECF volume • increased effective plasma osmotic pressure • angiotensin II <p>• nausea & vomiting; stress, exercise, • clofibrate, carbamazepine;</p> <p>Decreased by</p> <ul style="list-style-type: none"> • increased ECF volume • decreased effective plasma osmotic pressure • alcohol <p>(b) <i>It increases permeability of the collecting ducts to water resulting in renal retention of water.</i> activation of V2 receptors, causing insertion of proteins called water channels (aquaporins) into apical (luminal) membranes of the principal cells of the collecting ducts.</p>	<p>Need – volume, osmolarity and aquaporin</p>
<p>Question 4 Monosynaptic reflex</p>	<p>Please describe a monosynaptic stretch reflex</p>	 <p>Muscle spindle and its reflex connections are involved in proprioception</p>	<p>Essential to pass Monosynaptic, sensory organ, effector</p>

Question 5	<p>a. Please explain the concept of compliance as it relates to the lung</p> <p>b. What factors affect compliance</p>	<p>a. Volume change per unit pressure change (slope of pressure volume curve) $\approx 200\text{ml/cm H}_2\text{O}$ Depends on lung volumes and demonstrates hysteresis, may draw compliance curve, point out that inc compliance at low volumes Depends on structural proteins and surface tension</p> <p>b. Decrease – fibrosis, pulmonary oedema, not ventilated, inc pulmonary venous pressure Increase – emphysema, asthma surfactant</p>	<p>Basic concept</p> <p>1 of each</p>
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TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: Myocardial contractility	Please draw the Starling curve What factors influence myocardial contractility?	<ul style="list-style-type: none"> • Positively Inotropic: <ul style="list-style-type: none"> ○ Sympathetic stimulation via nerves or circulating catecholamines; ○ Post-extrasystolic potentiation; ○ Increased heart rate (small effect); ○ Drugs such as xanthines, glucagon, cardiac glycosides, adrenergic agents; ○ Increased myocardial mass (chronic). • Negatively Inotropic: <ul style="list-style-type: none"> ○ Parasympathetic stimulation (small) ○ Hypercapnoea, hypoxia, acidosis; ○ Drugs such as calcium channel blockers, beta-blockers, quinidine, barbiturates; ○ Cardiac failure (intrinsic myocardial depression); ○ Cardiomyopathy or infarction. 	Five of the factors listed with at least two each positively or negatively inotropic to pass this subsection.
Question 2: Thyroid hormones	1) Outline the physiological effects of thyroid hormones 2) Describe the mechanism regulating thyroid hormone	(a) Heart: chronotropic; inotropic (↑ number of B-adrenergic receptors; ↑ response to catecholamines; ↑ proportion of α-myosin heavy chain); Adipose tissue: catabolic (stimulated lipolysis); Muscle: catabolic (↑ protein breakdown); Bone: developmental (promote normal growth (Cretin) and skeletal development); Nervous system: promote normal brain development; Gut: metabolic (↑ CHO absorption); Lipoprotein: metabolic (formation of LDL receptors); other – calorogenic (↑ metabolic rate, ↑ stimulation O ₂ consumption) (b) Negative feedback effect of T ₄ and T ₃ on hypothalamus and pituitary to inhibit TRH and TSH secretion respectively. Cold stimulates	3 to pass incl cardiac

		thyroid hormone secretion, stress and glucocorticoids inhibit.	
Question 3: Renal blood flow	1) what is the normal RBF 2) How is renal blood flow regulated	1) RBF @ 1250ml/min, 25% CO 2) a) neuroendocrine; NA constrictor, DA dilator, Angio2 constrictor, PG's (incr cortical decrease medulla), ACH dilates b) autoregulation, probably vessel wall stretch reflex as occurs in denervated isolated vessels c) renal nerves.	1) approximate value 2) Must have 2 of DA, NA, AgtII, autoreg
Question 4 Glycogen	1) What are the physiologic actions of glucagon? 2) What factors affect glucagon secretion?	<ul style="list-style-type: none"> • Glycogenolysis in liver – not muscle • Gluconeogenesis from aa's only at very high levels • Lipolysis – • Ketogenesis • +ve inotropic effect on heart • Inc blood flow to kidneys • • Stimulates secretion of GH, insulin and somatostatin <p>2) Stimulators beta adrenergic stimulants Cortisol, protein meal, vagal stimulation, starvation, stress, exercise CCK gastrin theophylline</p> <p><u>Inhibitors</u> Glucose ----most important, insulin Somatostatin FFA Ketones α Adrenergic stimulators. GABA Phenytoin</p>	3 bulleted – essential to pass. Rest additional –3 to pass from each Rest additional

Question 5	What are the metabolic functions of the lung? (Prompt – What substances are metabolised in the lung?)	Metabolism of vasoactive amines a. Activation of Angiotensin 1 → AT 2 (ACE in capillary endothelium) b. Inactivation of bradykinin (ACE); PGs E/F c. Uptake & storage of Serotonin d. Arachadonic acid metabolites → leukotrienes / SRS-A & Prostaglandins Synthesis of a. Surfactant b. IgA c. Phospholipids d. Proteases (collagen/elastin breakdown)	2 of each
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TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES												
<p>Question 1:</p> <p>Normal ECG and cardiac membrane polarisation changes/ECG findings in MI</p>	<p>Please draw a normal ECG tracing</p> <p>Describe the cardiac events that relate to each of the intervals</p> <p>What is the electrophysiological basis for ST elevation in acute MI?</p>	 <p>The ST segment represents the plateau (Phase 2) of the AP and the T wave is repolarisation (Phase 3).</p> <p>ST segment elevation concave upwards.</p> <table border="1" data-bbox="981 895 1619 1294"> <thead> <tr> <th>Polarisation anomalies in infarcted Cells</th> <th>Current flow</th> <th>ECG Change in leads over the Infarct</th> </tr> </thead> <tbody> <tr> <td>Rapid repolarisation</td> <td>Out of infarct</td> <td>ST segment elevation</td> </tr> <tr> <td>Decreased resting membrane potential</td> <td>Into infarct</td> <td>TQ segment depression (manifested as ST segment elevation)</td> </tr> <tr> <td>Delayed depolarisation</td> <td>Out of infarct</td> <td>ST segment elevation</td> </tr> </tbody> </table>	Polarisation anomalies in infarcted Cells	Current flow	ECG Change in leads over the Infarct	Rapid repolarisation	Out of infarct	ST segment elevation	Decreased resting membrane potential	Into infarct	TQ segment depression (manifested as ST segment elevation)	Delayed depolarisation	Out of infarct	ST segment elevation	<p>Recognisable shape with QRS</p> <p>Name intervals.</p> <p>Describe the ST segment representing plateau (Phase 2) to pass this subsection.</p> <p>One of three</p>
Polarisation anomalies in infarcted Cells	Current flow	ECG Change in leads over the Infarct													
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<p>Question 2: Insulin</p>	<p>(a) What metabolic effects does insulin have on the liver?</p> <p>B, What are the effects of insulin deficiency on the body over</p>	<p>(a) ↓ketogenesis; ↑protein synthesis; ↑lipid synthesis; ↓glucose output due to ↓gluconeogenesis, ↑glycogen synthesis, ↑glycolysis</p> <p>(b) 1. Decreased cellular glucose uptake – ; total body dehydration and acidosis.</p>	<p>2 to pass</p>												

<p>Question 3</p>	<p>Describe the Physiological process of Micturition</p> <p>Prompts: What muscles and nerves are involved?</p>	<ul style="list-style-type: none"> • Spinal reflex facilitated and inhibited by higher centres • First urge to void at 150ml • Marked fullness at 400ml • During micturition, the Detrusor muscle contracts and perineal muscles/external urethral sphincter relax • Parasympathetic(S2,3,4) afferents respond to stretch receptors in bladder wall to initiate reflex contraction via parasympathetic efferents. • Pudendal nerve to External Urethral Sphincter causes relaxation. • Spinal reflex integrated in sacral portion of spinal cord • Sympathetic (L1,2,3) play no role in micturition but only in prevention. EUS and perineal muscles can be controlled voluntary for a period of time but eventually void reflex overcomes voluntary control. 	<p>To Pass Spinal Reflex Parasympathetic control Voluntary Control</p>
<p>Question 4 thermoregulation</p>	<p>What factors are responsible for heat production and heat loss?</p> <p>Describe the body's adaptive response to a cold environment</p>	<p><u>Heat Production</u></p> <p>Basic metabolic process Specific dynamic action of food Muscular activity</p> <p><u>Heat is lost by</u></p> <p>Radiation and conduction Vaporisation of sweat Respiration Urination and defaecation</p> <p><u>Mechanisms activated by cold</u></p> <p>Shivering</p>	<p>To Pass 2 of each</p> <p>3 to pass</p>

		<p>Hunger</p> <ul style="list-style-type: none"> ↑ Voluntary activity ↑ Secretion of adrenaline/nor adrenaline ↓ Heat loss <p>Cutaneous vaso constriction Curling up Horripilation (Controlled from posterior hypothalamus)</p>	
Question 5	<p>a. What factors influence the rate of transfer of oxygen from the alveolus into a pulmonary capillary</p> <p>b. Could you give some clinical examples of when these may be affected</p>	<p>A) Process is passive diffusion (Fick's law of diffusion) Affected by – surface area, membrane thickness, gradient of p O₂ (O₂ in alveolus and O₂ binding capacity of Hb) Also – constant –solubility and MW $V = A/T \times D \times (P_1 - P_2)$ $D = Sol/MW^{1/2}$</p> <p>b) exercise alveolar hypoxia and thickening of blood gas barrier</p>	2 to pass

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
<p>Question 1:</p> <p>Cardiac output and its measurement.</p>	<p>What are the parameters that define cardiac output?</p> <p>What are the factors that influence stroke volume?</p> <p>How can cardiac output be measured?</p>	<p>Cardiac output (CO) = heart rate (HR) X stroke volume (SV)</p> <ul style="list-style-type: none"> • Preload • Afterload • Myocardial contractility <p>The Fick principle states that the amount of a substance taken up by an organ (or by the whole body) per unit of time is equal to the arterial level of the substance minus the venous level (A-V difference) times the blood flow. The principle can be used to determine cardiac output by measuring the amount of O₂ consumed by the body in a given period and dividing this value by the A-V difference across the lungs.</p> $\text{Output of left ventricle} = \frac{\text{O}_2 \text{ consumption (mL/min)}}{[A_{O_2}] - [V_{O_2}]}$ <p>Whole body O₂ consumption is calculated by collecting expired gas in a spirometer and determining its O₂ content, which is then subtracted from the calculated O₂ content of inspired gas. The arterial O₂ content can be measured in an arterial sample and the mixed venous blood O₂ content is obtained from a pulmonary artery catheter.</p> <p>In the indicator dilution method, a known amount of a substance is injected into a vein and the concentration of the indicator in serial samples of arterial blood is determined. The output of the heart is equal to the amount of indicator injected divided by its average concentration in arterial blood after a single circulation through the heart. The cardiac output for that period is</p>	<p>Full equation to pass this subsection.</p> <p>3 to pass this subsection.</p> <p>Basic explanation of either principle.</p>

		<p>calculated and then converted to output per minute.</p> $\text{Flow} = \frac{\text{amount of indicator injected}}{\text{instantaneous concentration of indicator in arterial blood}}$ <p>The indicator must, of course, be a substance that stays in the bloodstream during the test and has no harmful or haemodynamic effects. A popular indicator dilution technique is thermodilution, in which the indicator used is cold saline.</p>	
Question 2	Can you draw a nephron and describe the functions of each part	<ul style="list-style-type: none"> • Glomerulus - filtration • Afferent arteriole (contains juxtaglomerular cells – secrete renin) then capillary tuft then efferent arteriole encapsulate in Bowman's capsule • PCT - resorption of most solute – Na, glucose, aa, reclaim HCO₃ • Desc limb of LOH – thin, water permeable • Thick Asc LOH – site of Na K 2 Cl – generates concentration gradient • DCT – site of Na K Cl pump • Proximal part is the macula densa forms juxtaglomerular apparatus - • CD- p cells - under control of ADH and aldosterone (water and Na resorption) • I cells – involved in H⁺ excretion 	To Pass: Draw basic shape and label 1 function of each part
Question 3 Mineralocorticoids	<p>(a) What is the physiological role of aldosterone</p> <p>(b) What conditions increase aldosterone secretion?</p>	<p>(a) Aldosterone causes Na⁺ and water retention, expanded ECF volume and shutting off the stimulus to increased renin secretion.</p> <p>(b) Primary: – stress hormone, low pressure/volume states secondary hyperaldosteronism: (eg. CCF, cirrhosis & nephrosis). Drugs:</p>	<p>Bold to pass</p> <p>To Pass: Primary and secondary</p>

<p>Question 4</p> <p>Regulation of serum calcium level</p>	<p>a. What hormones are involved in serum calcium regulation</p> <p>Outline the effects of PTH (Parathyroid Hormone)</p>	<p>PTH, Calcitonin, 1,25 DHCC</p> <p>↑ Parathyroid hormone secretion</p> <p>A Kidneys - ↑ Calcium reabsorption ↑ 1,25- (OH)2D formation ↓ Urinary excretion of Calcium</p> <p> ↑ Plasma 1,25- leads to (OH)2D levels cause –</p> <p>B Intestine - ↑ Calcium absorption</p> <p>C Bone – ↑ Resorption ↑ Release of Ca 2+ into plasma</p>	<p>To Pass 2 of 3</p> <p><u>Additional</u></p> <p>To Pass: 1 of each</p>
<p>Question 5</p>	<p>a. Please draw a diagram showing static lung volumes</p> <p>b. How does physiological dead space differ from anatomical dead space?</p>	<p>a. TV 500ml b. DS 150 ml c. TLC 7L d. FRC 2L e. VC6L</p> <p>a. Anatomical dead space, conducting zones of lung 150 mls b. Physiological dead space- Parts of lung with ventilation but no perfusion</p>	<p>To Pass: Draw diagram Label diagram</p> <p>Prompt for both physiological and anatomical dead space</p> <p>Normally very nearly the same but physiological dead space much greater in disease because of V/Q mismatch, esp with increased airway pressures</p>