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Question 1

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES & MARKS
Question 1	1.1 Describe the normal sequence of electrical excitation of the cardiac conduction system and cardiac muscle?	Normal sequence of depolarisation: **SA node** **Atria** (pathways) **AV node** Bundle of His **Major bundles** (Right & Left) Purkinje fibres **Ventricular muscle** Left side of IV septum first Spread down septum to apex Then up to AV grooves Spread from endocardial to epicardial surfaces	All of bold to pass
	1.2 What are the common mechanisms which cause abnormalities of cardiac conduction?	**Abnormal pacemakers** **Re-entry circuits** **Conduction defects** Prolonged repolarisation Accessory pathways	2/3 bold to pass
	1.3 What are the possible clinical consequences of these conduction abnormalities? (Flexibility between 1.2 & 1.3)	Abnormal pacemakers • **ectopic beats** • pacemaker failure (sinus arrest) • **fibrillation (atrial or ventricular)** Re-entry circuits • leading to tachyarrhythmias Conduction delays • **heart block** • bundle branch blocks Prolonged repolarisation • Long QTc Accessory pathways • WPW or LGL	2 bold + 2 others to pass

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Question 2

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES & MARKS
Question 2 Pulmonary blood flow	2.1 Describe the normal distribution of pulmonary blood flow.	2.1 Influenced by gravity-3 main zones Zone 1(apex) PA>Pa>Pv – least blood flow Zone 2(mid) Pa>PA>Pv Zone 3(base) Pa >PA>Pv – Most blood flow	Posture/Hydrostatic pressure and describe 3 zones to pass
	2.2 How is the distribution of pulmonary blood flow actively controlled?	2.2 Hypoxic pulmonary vasoconstriction-alveolar hypoxia constricts pulmonary arteries, directs blood away from poorly ventilated diseased lung areas. Mechanism-NO, endothelin-1, TXA2, low pH, autonomic system	Hypoxic pulmonary vasoconstriction to pass
	2.3 Please explain how cardiogenic pulmonary oedema occurs.	2.3 Starling's Law- differences in capillary and interstitial hydrostatic and colloid osmotic pressures. Significant increases in net outward pressure of Starling equation results in interstitial oedema especially at perivascular and peribronchial spaces. Further increases of outward pressure results in fluid entering alveolar spaces.	Basic description of Starling forces

Question 3

Question 3:	3.1 What are the principal buffering systems in the body? PROMPT: How about in other fluid compartments?	Blood: Bicarbonate, Protein and Haemoglobin Interstitium: Bicarbonate Intracellular: Protein, Phosphate Urine: also uses ammonia	3 buffering systems to pass 2 fluids to pass
	3.2 Outline how the body responds to a metabolic acid load.	a) Buffering in blood, interstitial and intracellular spaces b) Respiratory response: H_2CO_3 converted to H_2O and CO_2, CO_2 expired via lungs through increased minute ventilation. c) Renal: • Renal mechanisms operate to compensate for metabolic acidosis and return the serum pH towards normal • Anions that replace HCO_3^- are filtered at the glomerulus along with corresponding cations (mainly Na^+) • Renal tubule cells secrete H^+ into tubular fluid in exchange for Na^+ and HCO_3^- • Buffering in the urine gives greater capacity to this system (otherwise limiting pH of 4.5 would stop further H^+ secretion) • Buffering systems include: Bicarbonate, Phosphate, Ammonia	Buffering in blood CO_2 expiration via lungs Acid secretion in kidney + buffering in urine All 3 to pass

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Question 4

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES & MARKS
Question 4 Thermoregulation	4.1 What mechanisms does the body use to regulate temperature? PROMPT: What mechanisms are activated by cold? PROMPT: Are any voluntary?	a) **Activated by cold:** Shivering, Hunger, Increased voluntary activity, adrenaline and noradrenaline secretion, decreased heat loss, cutaneous vasoconstriction, curling up, horripilation	4 to pass
		b) **Activated by heat:** Increased heat loss, cutaneous vasodilation, sweating, increased respiration, decreased metabolic heat production, anorexia, apathy & inertia	4 to pass
	4.2 How are these temperature regulating mechanisms controlled?	Reflex responses activated by cold controlled from posterior **hypothalamus** Those activated by warmth are controlled primarily from the anterior **hypothalamus**	Bold to pass

Question 5

Question 5.1	Please outline the different ways in which a substance can cross a cell membrane	Passive • Diffusion • Facilitated diffusion Active • Endo/exocytosis • Ion channels –ligand, voltage, mechanical gated • Active transport Primary and secondary	3/5 methods to pass
Question 5.2	Can you please explain the process of secondary active transport? PROMPT: Give a clinical example	The movement of an ion down its electrochemical gradient provides energy to transport another substance against its electrochemical gradient. Example – Na/glucose, Na/ aminoacids	Basic concept or clinical example to pass

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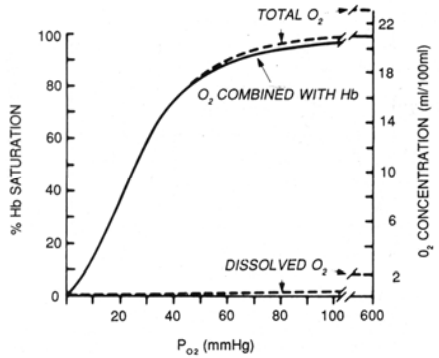
Question 1

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES & MARKS
Question 1	1.1 How are cardiac stroke volume and cardiac output related?	CO = SV X HR	Need to know equation to pass
	1.2 What is cardiac preload?	Degree of stretch of cardiac muscle compared to resting length Equivalent to end diastolic volume	Definition to pass
	1.3 What factors affect preload? PROMPT - What are the causes of reduced end diastolic volume (preload)?	**Blood volume** **Change in driving pressure** (pericardial (tamponade), intrathoracic (tension pneumothorax, IPPV)) **Venous return** Sympathetic tone Muscle pump Loss of atrial contraction Venous compression (eg uterus in pregnancy) Reduced cardiac compliance Diastolic dysfunction / infiltrative diseases	2/3 bold to pass

Question 2

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES & MARKS
Question 2: Oxygen Dissociation Curve (west 77-80)	2.1 Please draw and describe the features of the haemoglobin-oxygen dissociation curve	 (a) May need prompt for SO_2 at various PO_2. (b) Describe importance of: • Flat upper portion of curve- Hb uploading of O_2 unaffected unless PAO_2 falls significantly; • Steep lower part of curve – large amounts of O_2 unloaded at peripheral tissues for only small drop in capillary PO_2.	To pass: Label the axes Draw an approximately correctly shaped curve Locate at least two points Standard points are 27 mmHg to SO_2 50%; 40 mmHg to SO_2 75%; 56 mmHg to SO_2 90%; 80 mmHg to SO_2 95% and 90 mmHg to SO_2 97%.

	2.2 What factors cause shift in the curve?	2.2 To Right: Increased Temperature, Increased PCO2, Increased H+ (decreased ph) and 2-3 DPG To Left: Reverse of above (and CO as below)	Temp, CO2, pH essential
	2.3 What are the effects of carbon-monoxide on haemoglobin oxygen transport capacity	2.3 CO has 240 times the affinity of O2 for Hb, hence oxygen saturation greatly reduced and Hb oxygen carrying capacity reduced. CO also shifts O2 dissociation curve to left, interfering with unloading of O2	Reduced O2 carrying capacity for both with adequate explanation

Question 3

TOPIC	**QUESTION**	**ESSENTIAL KNOWLEDGE**	**NOTES & MARKS**
Question 3	3.1 Describe the factors influencing Angiotensin II production.	Is the effector protein in the renin-angiotensin system: integral to control of volume regulation So, principally those that influence renin secretion: Increased secretion due to: • Increased sympathetic activity • Increased circulating catecholamines • Prostaglandins (from Na^+ depletion, diuretics, hypotension, haemorrhage, dehydration, cardiac failure, cirrhosis, upright posture, renal artery and aortic constriction) Decreased secretion due to: • Increased Na^+ and Cl^- re-absorption across macula densa • Increased afferent arteriolar pressure • Vasopressin	Intravascular volume Renin + 2 others
	3.2 What are the physiological effects of Angiotensin II?	• Arteriolar constriction • Directly on adrenal cortex to increase aldosterone Facilitation release of norepinepherine release • Contraction of mesangial cells causing decreased GFR • Direct effect on renal tubules to increase Na+ re-absorption • On the brain to decrease sensitivity of baroreceptor reflex • On brain (circumventricular organs) to increase water intake and increase secretion vasopressin and ACTH	Vasoconstriction + 2 others

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Question 4

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES & MARKS
Insulin Deficiency	4.1 What are the effects of insulin deficiency? PROMPTS: What are the effects on the liver? What are the effects on other tissues?	**Decreased Peripheral Utilisation** (uptake) of glucose **Hyperglycaemia** but low intracellular glucose Derangement of the glucostatic function of the liver Hyperglycaemia with no decrease in gluconeogenesis Secondary osmotic diuresis with dehydration Electrolyte and calorie loss Catabolism of protein & fat due to low intracellular glucose **Contributes to ketosis – acidosis** Breakdown of amino acids for energy Increased Free fatty acids from breakdown of triglycerides Secondary Acidosis, Coma, raised cholesterol	3 bold essential

Question 5

Question 5	5.1 What are the different types of nerve fibres? PROMPT – What classifications are there? 5.2 What is the clinical relevance to emergency medicine?	• Diameter & speed of conduction • Function Large, fast – proprioception, conscious touch, somatic motor Small, slow – pain , temperature, autonomic • Gasser ABC (A – αβγδ) • Numerical Ia, Ib, II,III IV Pain fibres are smaller and better penetrated by local anaesthetic leading to loss of pain before loss of touch or proprioception	One system or concept of system One example

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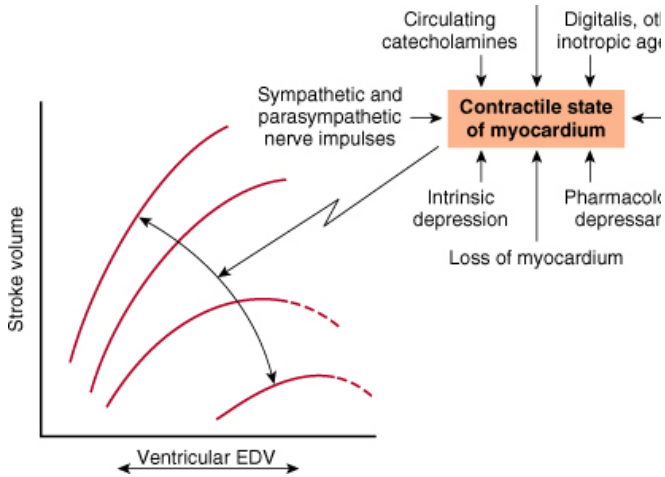
Question 1

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES & MARKS
Question 1	1.1 What factors may reduce myocardial contractility?	**Metabolic abnormalities** • Hypoxia • Severe acidosis • Hypercarbia **Reduced sympathetic tone** Increased parasympathetic tone **Blockade of circulating catecholamines** **Myocardial disease** (muscular dystrophies) or loss **Pharmacological depressants** (antiarrhythmics, Ca channel blockers) Intrinsic depression in heart failure. Hypothermia Abnormal myofibrils (actin, myosin or troponin) Reduced intracellular calcium Reduced levels of c AMP (reduced catecholamines or beta blockade)	3 of 5 of bold to pass
	1.2 How do changes in myocardial contractility alter the relationship between end diastolic volume and stroke volume?	Increasing contractility moves the curve upwards and to the left. Decreasing contractility moves the curve downwards and to the right.  	At least one of the two appropriate directions of movement of the curve to pass. Must get axes correct.

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Question 2

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES & MARKS
Question 2: Integrated responses to O2,CO2, pH	2.1 What sensors are involved in the control of ventilation?	a) Central chemoreceptors in medulla-respond to CSF pH (via CO_2) b) Peripheral chemoreceptors in carotid and aortic bodies respond to O_2, pH, CO_2 (minor) c) Pumonary receptors: o Stretch receptors in lungs, muscles, joints o Irritant receptors in airways o J receptors-engorged lung capillaries and alveolar wall interstitial fluid d) Nose and upper airway receptors - irritant receptor e) Joint and muscle receptors - muscle spindles in the intercostal muscles and diaphragm f) Arterial baroreceptors - stimulation may cause reflex hypoventilation g) Pain and temperature receptors - may cause initial apnoea and then hyperventilation	Central and peripheral chemoreceptors and pulmonary receptors
	2.2 Describe the ventilatory response to metabolic acidosis	2.2 Low arterial pH stimulates peripheral chemoreceptors to increase ventilation. Central chemoreceptors or respiratory centre itself may be stimulated in severe cases	Peripheral chemoreceptors dominate response

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Question 3

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES & MARKS
Question 3	3.1 Describe how Na^+ is handled by the kidney.	• Glomerular filtration • Actively transported out of the tubule in all sections EXCEPT descending thin limb of loop of Henle. • 96-99% reabsorbed overall • Reabsorption influenced by changes in GFR and in tubular reabsorption (MAINLY in the 3% reaching collecting ducts) – circulating aldosterone, other adrenocoticoids, circulating ANP and other natriuretic hormones and rate of tubular secretion H+ and K+.	Filtration Active re-absorption Need to understand concepts
	3.2 How does aldosterone influence renal sodium handling?	• Increased tubular reabsorption of Na+, with secretion of K+ and H+ • Latent period of 10 – 30 minutes before effect (time delay due to need to alter protein synthesis via action on DNA) • Act principally on the collecting ducts to increase number of active epithelial sodium channels	Increased reabsorption

Question 4

Question 4 Osmosis/Tonicity	4.1 What is the difference between diffusion and osmosis?	**Diffusion** = Net flux of **solute** particles down a concentration gradient - from area of high to low concentration (Time for equilibrium proportional to square of distance) (Rate proportional to cross-sectional area & gradient [Fick]) **Osmosis** = Net flux of **solvent** across a membrane to an area of higher concentration of solute to which membrane impermeable (Osmotic pressure given by nRT/V – property of solution)	Bold to pass
	4.2 Define "Tonicity"	Osmolality of a solution **relative to plasma** (0.9% saline is "isotonic")	
	4.3 What is the genesis of the membrane potential?	Differences in **concentration gradient** and **electrical gradient** of major cations (Na, K), across the impermeable cell membrane Maintained by Na-K ATPase 3 Na out for each 2 K in so electrogenic	

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Question 5

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES & MARKS
Question 5	5.1 Please name the principal Ketone bodies.	Acetoacetate, β hydroxybutyrate, Acetone	2 out of 3 to pass
	5.2 How are the Ketone bodies produced and how are they metabolised?	Substrate – Fatty acids, AcetylCoA Site – mitochondria - Liver / Other tissues Mechanism – β oxidation of fatty acids and entry of AcetylCoA into CAC High energy yield process. AcetylCoA units condense to form AcetoacetylCoA. Liver – AcetoacetylCoA ⟶ Acetoacetate ⟶ β hydroxybutyrate and acetone which is excreted in the urine and the breath Tissues – SuccinylCoA ⟶ Acetoacetate ⟶ CO2 and H2O via CAC	Fatty acids AcetylCoA
	5.3 In which clinical situations do they accumulate in the body?	Ketosis – metabolic acidosis (Diabetes, Starvation, high fat low carbohydrate diet)	Diabetes Starvation
	What are the physiological and clinical consequences of excess ketones?		(Bonus)