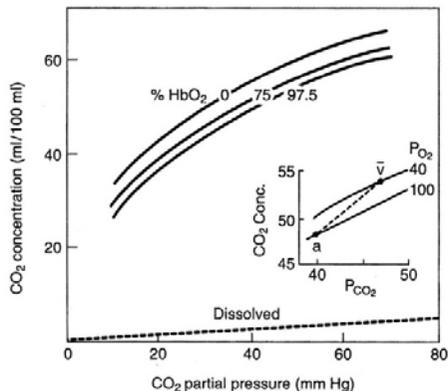


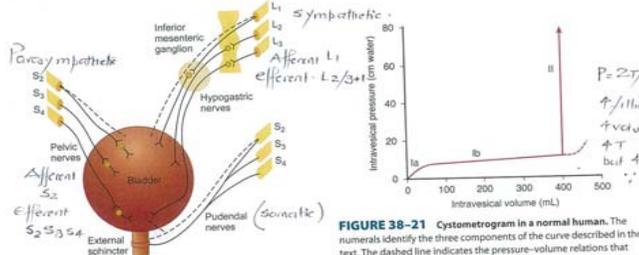
TOPIC	QUESTIONS	KNOWLEDGE (essential in bold)	NOTES
Question 1	<p>1.1 Describe what happens to Cardiac Output during exercise. <i>Prompt: By what mechanisms?</i></p> <p>1.2 What are the local mechanisms that maintain a high blood flow in exercising muscles?</p>	<p>Increases ($CO = SV \times HR$) \uparrow venous return and hence \uparrow end diastolic volume, \uparrow myocardial contractility, so \uparrow stroke volume. \uparrow sympathetic drive and heart rate</p> <p>\downarrow in tissue PO_2, \uparrow tissue PCO_2, and accumulation of K^+ and other vasodilator metabolites, \uparrow temperature in active muscle</p>	<p>Increases + one mechanism stroke vol + one mechanism heart rate</p> <p>Need 3 to pass.</p>
Question 2	<p>2.1 In what forms is carbon dioxide transported in the blood?</p> <p>2.2 Please draw the carbon dioxide dissociation curve for normal arterial blood.</p> <p>Prompt: "Draw a graph showing the relationship between the pressure of carbon dioxide and the total carbon dioxide content in arterial blood."</p> <p>2.3 Where will the curve lie for venous blood and why??</p> <p>Prompt: "Does the curve move up or down and why??"</p>	<ul style="list-style-type: none"> • Dissolved. • As carbamino compounds with proteins, especially Hb. • Hydrated in red cells – H^+ is buffered and 70% of HCO_3^- enters the plasma.  <p>Figure 6-6. CO_2 dissociation curves for blood of different O_2 saturations. Note that oxygenated blood carries less CO_2 for the same PCO_2. The inset shows the "physiological" curve between arterial and mixed venous blood.</p> <ul style="list-style-type: none"> • The graph moves upwards indicating greater CO_2 content per unit pressure. • Deoxygenated haemoglobin binds more H^+ and forms more carbamino compounds than oxyhemoglobin so venous blood carries more CO_2 than arterial blood. • This is known as the Haldane effect. 	<p>Two of three to pass.</p> <p>Reasonable shape of the curve indicating the near linearity in the physiological range. Prompt if necessary.</p> <p>The candidate must understand that venous blood is able to carry proportionately more CO_2 than arterial blood.</p>

Question 3	<p>3.1 By what mechanism is H⁺ secreted in the distal tubules and collecting ducts of the kidney?</p> <p>3.2 In H⁺ secretion, what is the limiting urine pH?</p> <p>3.3 Describe the principal urinary buffers and what is their role?</p>	<p>ATP driven proton pump. Aldosterone acts on this pump to increase H⁺ excretion. Abundant carbonic anhydrase in the cells numerous tubulovesicular structures. Pumps in the vesicles H – K⁺ ATPase</p> <p>A urine pH of 4.5 is the maximal H⁺ gradient against which transport mechanisms can secrete H⁺</p> <p>HCO₃ buffer system particularly in the proximal tubules HPO₄²⁻ in the distal tubules NH₃ in the proximal and distal tubules</p>	<p>ATP driven proton pump</p> <p>pH 4-5</p> <p>2 examples + increased capacity to excrete H+</p>
Question 4	<p>4.1 What is normal serum osmolality?</p> <p>4.2 What substances contribute to serum osmolality?</p> <p>4.3 How does plasma differ in composition to intracellular fluid?</p>	<p>~ 290mOsmol/L</p> <p>Principally (all but 20mOsmols) the ions (Na, K, Cl, HCO₃). Rest is other cations & anions, urea, glucose. Much less so proteins (due to high MW). Possibly alcohols or mannitol.</p> <p><i>Intracellular</i> K⁺ and proteins high, many more 'miscellaneous' phosphates Na⁺, Cl & HCO₃ low, (Figure 1-1 page 3)</p>	<p>Within the range 280-300</p> <p>Na+, Cl- and one other</p> <p>Na, K, protein differences</p>
Question 5	<p>5.1 What is the main hormonal factor that stimulates the release of cortisol from the adrenal cortex?</p> <p>5.2 What factors determine the rate of ACTH secretion?</p> <p>5.3 What happens to ACTH levels after prolonged treatment with high doses of glucocorticoids is stopped abruptly?</p> <p>5.4 How can this be avoided?</p>	<p>Adrenocorticotrophic hormone (ACTH)</p> <p>Increased by stress (pain, emotional), drive for circadian rhythm through the hypothalamus via release of CRH (corticotropin releasing hormone)</p> <p>Inhibited by circulating glucocorticoids and afferent from baroreceptors</p> <p>Slowly increases over weeks (the pituitary may not be able to secrete normal amounts of ACTH for as long as a month. Presumed to be secondary to diminished ACTH synthesis)</p> <p>This can usually be avoided by slowly decreasing the dose over a long period of time.</p>	

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<p>Question 1</p> <p>LOA: 1</p>	<p>1.1 What local factors can cause vasoconstriction or vasodilatation?</p> <p>1.2 What is autoregulation in relation to blood flow?</p>	<p>Vasodilatation: ↑ CO₂, ↑ lactate, ↑ adenosine, ↑ local temp; ↓ O₂ or ↓ pH</p> <p>Vasoconstriction: ↓ local temp, autoregulation.</p> <p>Autoregulation: blood flow remains constant by compensating pressure changes with peripheral resistance.</p> <p>1) Myogenic: as blood pressure rises, muscle fibres in the blood vessels contract. The muscles correspond to the wall tension which is maintained at fairly constant level. Wall tension is determined by the radius of the blood vessels (pressure x radius). So rise in pressure, leads to a reduction in the radius of the blood vessel.</p> <p>2) Metabolic: active metabolites cause local vasodilatation.</p>	<p>At least 4 to pass, and at least one in each group</p> <p>Need bold & some details to pass.</p>
<p>Question 2</p> <p>LOA: 1</p>	<p>2.1 What factors determine the work of breathing?</p> <p>2.2 What variables affect elastic workload?</p> <p>2.3 What variables affect viscous resistance?</p>	<ul style="list-style-type: none"> • Elastic forces of the lungs and chest wall • Viscous resistance of the airways and tissues <p>Larger tidal volumes increase elastic workload. Elastic workload is increased by reduced compliance due to:</p> <ul style="list-style-type: none"> ○ Lung volume - a person with only one lung has halved compliance. ○ Slightly lesser during inflation than during deflation. ○ Increased tissue mass - fibrosis or pulmonary congestion or chest wall restriction. ○ Loss of surfactant <ul style="list-style-type: none"> • Higher respiratory rates increasing flow rates • Decreased airway radius due to: Lower lung volumes; Bronchoconstriction; • Increased air density (eg SCUBA diving) • Increased air viscosity 	<p>Must understand both to pass. Prompt if necessary.</p> <p>Must understand both major points</p> <p>Must give at least two examples to pass.</p>

<p>Question 3</p> <p>LOA: 1</p>	<p>3.1 What are the essential features of the loop of Henle countercurrent multiplier?</p> <p>3.2 What is the role of urea in the countercurrent mechanism?</p> <p>3.3 How does urea reach the interstitium?</p>	<p>High permeability of the thin descending limb to water (via aquaporin-1) and active transport of Na^+ and Cl^- out of the thick ascending limb which is not permeable to water.</p> <p>A system in which Na^+ K^+ 2Cl^- are actively transported, and the inflow runs parallel to, counter to, and in close proximity to the outflow for some distance</p> <p>Contributes to the osmotic gradient in the medullary pyramids</p> <p>Transported by urea transporters, by facilitated diffusion Amount of urea depends on the amount filtered which is influenced by dietary protein</p>	<p>Either version</p> <p>Osmotic gradient</p> <p>Facilitated diffusion</p>
<p>Question 4</p> <p>LOA: 1</p>	<p>4.1 Describe the body's response to cold?</p> <p>4.2 Outline the pathogenesis of fever.</p>	<p>shivering, hunger, \uparrowvoluntary activity, \uparrowNA, A, \downarrow heat loss, curling up, behaviour change, cutaneous vasoconstriction, horripilation</p> <p>Toxins from infective agents act on monocytes, macrophages and Kupffer cells to produce cytokines which act as endogenous pyrogens (EPs),</p> <p><i>also IL-1β, IL-6, β-IFN, γ-IFN, TNF act on the OVLTL, which in turn activates pre-optic hypothalamus through local release of PGs.</i></p>	<p>Give 4</p> <p>EPs indirect action on hypothalamus to reset</p>
<p>Question 5</p> <p>LOA: 1</p>	<p>What is the sequence of events in skeletal muscle excitation contraction coupling?</p>	<p>Discharge of motor neuron.</p> <p>Release of transmitter (acetylcholine) at motor end-plate.</p> <p>Binding of acetylcholine to nicotinic acetylcholine receptors.</p> <p>Increased Na^+ and K^+ conductance in end-plate membrane.</p> <p>Generation of end-plate potential.</p> <p>Generation of action potential in muscle fibers.</p> <p>Inward spread of depolarization along T tubules.</p> <p>Release of Ca^{2+} from terminal cisterns of sarcoplasmic reticulum and diffusion to thick and thin filaments.</p> <p>Binding of Ca^{2+} to troponin C, uncovering myosin-binding sites on actin. ATP dependent</p> <p>Formation of cross-linkages between actin and myosin and sliding of thin on thick filaments, producing movement.</p>	<p>Need bold to pass</p>

TOPIC	QUESTIONS	KNOWLEDGE (essential in bold)	NOTES
<p>Question 1:</p> <p>LOA: 1</p>	<p>1.1 Describe the factors affecting Cardiac Output</p> <p>1.2 What are the physiological responses to moderate blood loss?</p>	<p>CO=SVxHR SV related to contractility, preload and afterload, HR controlled by intrinsic rate, autonomic, exogenous factors, heat, thyroid ↓venous return, stimulation of baroreceptors, inc catecholmine release, ↓ renal blood flow – activation of renin angiotensin system fluid shifts, hepatic synthesis of proteins, inc RBC production</p>	<p>Bold to pass + 2 mechanisms from each SV and HR</p> <p>Bold to pass</p>
<p>Question 2</p> <p>LOA: 1</p>	<p>2.1 What are the effects of exercise on the respiratory system?</p> <p>Prompt(s): “What are the effects on: gas exchange; OR ventilation; OR pulmonary blood flow.”</p> <p>2.2 What changes occur in blood gases during exercise?</p>	<ul style="list-style-type: none"> • Gas exchange: <ul style="list-style-type: none"> ○ ↑Respiratory uptake and consumption of O₂ (VCO₂) and production and excretion of CO₂ (VCO₂) - increases by 10-20 times; ○ ↑Lung diffusing capacity due to ↑diffusing capacity of the membrane and the pulmonary blood volume; ○ ↓Ventilation–perfusion inequality; • Ventilation: <ul style="list-style-type: none"> ○ ↑Respiratory rate; ○ ↓Functional residual capacity (FRC); ○ ↑Tidal volume (TV); ○ ↑Minute ventilation. • Pulmonary blood flow: <ul style="list-style-type: none"> ○ Distension and recruitment of pulmonary vessels increases total cross-sectional area of the pulmonary vasculature; ○ ↑Total pulmonary blood volume; ○ ↑Cardiac output and pulmonary blood flow; ○ ↑Pulmonary vascular pressures; ○ ↓Pulmonary vascular resistance. • Other respiratory effects: <ul style="list-style-type: none"> ○ ↑Respiratory exchange ratio (R) from 0.8 to 1.0 due to carbohydrate metabolism and may exceed 1.0 due to anaerobic glycolysis; ○ The Hb-O₂ dissociation curve shifts to the right in the tissues and back to the left in the lungs; ○ Additional capillaries open in peripheral tissues; • Arterial blood gases are little affected by moderate exercise but at high workloads pH falls due to lactic acidosis, PaCO₂ often falls to compensate for the acidosis and PaO₂ rises; • Arteriovenous pH, PaO₂ and PaCO₂ differences increase. 	<p>One effect from each bolded section and at least six to pass.</p> <p>Basic understanding of the effects on blood gases.</p>

<p>Question 3</p> <p>LOA: 1</p>	<p>3.1 Describe the micturition reflex.</p>	<p>Spinal reflex, voluntary facilitation/inhibition from the higher centres. Micturition centre in the brain stem. Bladder innervation - sympathetic L1,2,3; parasympathetic S2,3,4; somatic S2,3,4.</p>  <p>FIGURE 38-20 Innervation of the bladder. Dashed lines indicate sensory nerves. Parasympathetic innervation is shown at the left, sympathetic at the upper right, and somatic at the lower right.</p> <p>FIGURE 38-21 Cystometrogram in a normal human. The numerals identify the three components of the curve described in the text. The dashed line indicates the pressure-volume relations that would have been found had micturition not occurred and produced component 2. (Modified and reproduced with permission from Taniguchi EA, McAninch JW. Smith's General Urology, 15th ed. McGraw-Hill, 2000)</p> <p><i>Spinal reflex & integration in higher centres = voluntary inhibition - micturition post-hydronephrosis</i></p> <p>Bladder muscle smooth and plastic (explanation) Initial urge at 150mls, fullness 400 mls. Detrusor muscle contracts. Perineal muscles/external urethral sphincter relax. In females aided by gravity; in males contraction of bulbocavernosus muscle</p>	<p>Need bold to pass – Innervation, sympathetic – inhibitory, parasympathetic – excitatory.</p> <p>Bladder distention, excitation of the mechanoreceptors, afferent projection to the brain stem and efferents via sympathetic, parasympathetic and somatic nerves.</p> <p>cystogram for additional marks</p> <p>Plastic – tension initially produced by filling (distension) is not maintained. $P = 2T/R$ as T increases so does R, i.e. filling and distension therefore P remains constant</p>
<p>Question 4</p> <p>LOA: 1</p>	<p>4.1 What factors stimulate glucagon release?</p> <p>4.2 What are the physiological effects of glucagon?</p>	<p>Hypoglycaemia; increased sympathetic drive to pancreas; vagal stimulation; protein load; amino acids oral or IV infusion; exercise; stress; starvation; CCK; gastrin; cortisol; theophylline.</p> <p>Gluconeogenesis; glycogenolysis (not in muscle); lipolysis; ketogenesis; calorogenic – through hepatic deamination of amino acids; +ve inotropic effect in large doses; stimulates secretion of GH, insulin and pancreatic somatostatin.</p>	<p>Must give hypoglycaemia + 2 others</p> <p>Gluconeogenesis + 1 others</p>
<p>Question 5</p> <p>LOA: 2</p>	<p>5.1 What is clonus?</p> <p>5.2 Why does ankle clonus occur with upper motor neuron lesions?</p> <p>5.3 What are the components of the stretch reflex?</p>	<p>Regular, repetitive, rhythmic contractions of a muscle subjected to sudden, sustained stretch.</p> <p>Loss of descending cortical input to inhibitory neurons called Renshaw cells, and therefore loss of inhibition of antagonists, resulting in repetitive sequential contractions of ankle flexors and extensors.</p> <p>Sensor, afferent nerve, Monosynaptic at spinal level, efferent nerve, effector</p>	<p>Bold to pass</p>