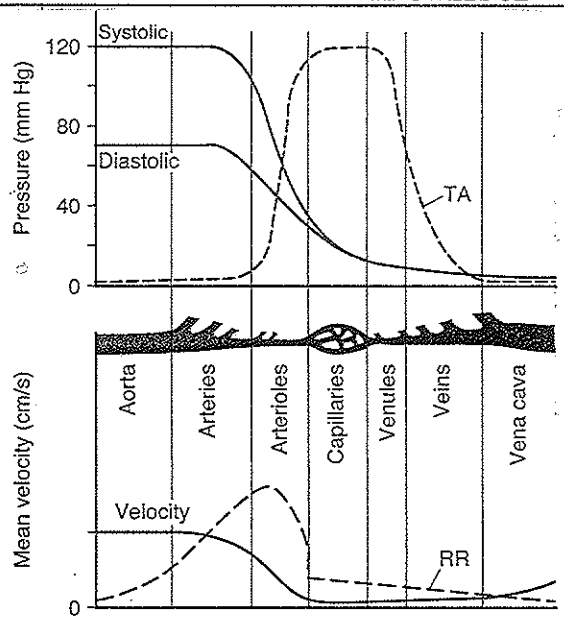
QUESTION	KNOWLEDGE	PASS CRITERIA
1. a) Please draw a diagram of the changes in systolic and diastolic pressure as blood flows through the systemic circulation. -suggest: pressure on y-axis, and label the various parts of the systemic circulation on the x-axis. 1 b) How does the total cross-sectional area of vessels change through the systemic circulation?	 **FIGURE 32–27 Diagram of the changes in pressure and velocity as blood flows through the systemic circulation.** TA, total cross-sectional area of the vessels, which increases from 4.5 cm^2 in the aorta to 4500 cm^2 in the capillaries (Table 32–9). RR, relative resistance, which is highest in the arterioles. TA = total cross-sectional area of vessels RR = relative resistance (highest in arterioles) • pressure falls very slightly in large and medium-sized arteries because resistance to flow is small • pressure falls rapidly in small arteries and arterioles, which are main sites of peripheral resistance against which heart pumps • magnitude of pressure drop along arterioles varies depending on whether constricted or dilated 1. small pressure change in large and medium-sized arteries 2. rapid fall in pressure in small arteries and arterioles 3. **mean** pressure at end of arterioles is 30-38mmHg 4. **Pulse pressure** 5mmHg at ends of arterioles TA – maximal in capillaries and venules (about 10 x that in arterioles)	Need to pass: 1. Correct shape of pressure diagram 2. Rapid fall in pressure in arterioles 3. TA is maximal in capillaries, and venules

2 a). What are the principal actions of insulin?	Storage of carbohydrate, prot and fat, varies with tissues Rapid- seconds. Glc, amino acids and K+ into insulin sens cells Intermediate- minutes. Stimulates prot synthesis, inhibits prot degradation, activates glycolytic enzymes & glycogen synthase, inhibits phosphorylase and gluconeogenic enzymes. Delayed- Hrs. increase in mRNA for lipogenic & other enzymes	Glc and K from rapid. 2 others Answer must reflect understanding of effects on carbohydrate, protein and fat
2 b) What happens when insulin binds to its receptor?	• Binds to a cell membrane-based stereospecific insulin receptor on insulin-sensitive cells • Insulin binding triggers tyrosine kinase activity of β subunits → autophosphorylation of β subunits on tyrosine residues • The above reaction → phosphorylation and de-phosphorylation of proteins that are effectors and secondary mediators.	Binding results in activation of secondary protein effectors (tyrosine kinase activity) and mediators (phosphorylation)
3 What are the factors which keep fluid out of the alveoli?	a)Starling's Law (Theoretical Concept, exact values of pressures unknown) 1. Hydrostatic pressure (of column of blood) a. In the capillaries (positive thus outwards) = Pc b. In the interstitium (probably negative and thus also outwards) = Pi 2. Colloid osmotic pressure (of proteins in blood) a. In the capillaries (inwards) = πc b. In the interstitium (outwards) = πi Net pressure probably slightly outward Net fluid out = K[(Pc-Pi)-σ(πc-πi)] K = Filtration Coefficient σ = reflection coefficient (capillary wall barrier) b)Lymphatic drainage c)alveolar epithelial cells	Demonstrate understanding of hydrostatic pressure & colloid osmotic pressure One other

4 a).Outline the steps in the synthesis of catecholamines	**TyOHylase** **Tyrosine→ DOPA** **↓ DOPA** **Decarboxylase** **Dopamine** **↓ Dopamine** **βhydroxylase** **Adrenaline← Nor Adrenaline** **PNMT (adrenal medulla, some central)** **Adrenaline/Noradrenaline**	**Tyrosine to dopamine to noradrenaline, plus one of the synthesis enzymes**
4 b).What happens to noradrenaline after it is released into the synaptic cleft?	**Removed by post-synaptic and pre-synaptic binding, reuptake and catabolism** **IC) MAO ↓ COMT (EC)** **VMA**	**Three out of four processes**
5. Describe the renal response to metabolic acidosis.	• Renal mechanisms operate to compensate for metabolic acidosis and return the serum pH towards normal • Anions that replace $HCO3^-$ 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 futher H^+ secretion) • Buffering systems include: Bicarbonate, Phosphate, Ammonia Prompts: (i) What prevents H^+ secretion stopping when urine pH falls to 4.5? (ii) Can you name any of the buffers that operate?	Compensatory mechanisms identified Must know H^+ secreted into tubular fluid in exchange for Na^+ Must know about buffering and give two buffers

ACEM PRIMARY 20010.1 Physiology VIVA Thursday 25 March pm Candidate Number:..... Mark.....

QUESTION	KNOWLEDGE	PASS CRITERIA
1. (a)Describe how tissues regulate their own blood flow. Prompt- What are the proposed mechanisms of this process?	Most vascular beds have intrinsic capacity to compensate for moderate changes in perfusion pressure by changing vascular resistance and therefore maintaining constant bld flow. 1. Myogenic theory of autoregulation: Intrinsic contractile response of smooth muscle to stretch As pressure rises: blood vessels are distended → vascular smooth muscle surrounding vessels contract Law of Laplace: maintenance of given wall tension, as pressure rises, requires a decrease in radius 2. Metabolic theory of autoregulation Vasodilator substances tend to accumulate in active tissues when blood flow decreases → dilatation When blood flow increases → washed away Hypox, inc Co2, Inc H+, Inc lactate, inc K+, inc temp, histamine, adenosine	**1. Myogenic theory of autoregulation** **2. Metabolic theory of autoregulation** To pass must demonstrate understanding of both
2 a). What are the causes of hypoxaemia in a person breathing room air	Hypoventilation, diffusion limitation, shunt, V/Q inequality	Need 3/4
2 b). Explain why ventilation-perfusion inequality causes a reduced arterial P_{O_2} while arterial P_{CO_2} remains relatively normal	Basically due to the differences in their dissociation curves. If one could in isolation cause V/Q inequality then gas exchange would deteriorate with hypoxia and hypercapnia. But the chemoreceptors act to increase ventilation. P_{CO_2}- The **CO_2 dissociation curve is linear** at the working range. The increased ventilation is able to correct the P_{CO_2}.by increased CO_2 output, particularly in units with high V/Q ratios P_{O_2} –the **oxygen dissociation curve is not linear**. So high V/Q areas can only boost their P_{O_2} a little with increased ventilation. Conversely very low V/Q areas have proportionally lower P_{O_2} (close to mixed venous). Overall P_{O_2} is low.	Bold plus demonstrate understanding

3a). What is the basis of the resting membrane potential?	a). Potassium – more open Potassium channels at rest therefore intracellular/extracellular Potassium concentrations are prime determinants of resting membrane potential b).Sodium c). Separated by the cellular membrane 1.Na actively transported out of cells 2.K actively transported into cells 3.Activity of Na-K ATPase pump	Na, K, ATPase and correct directions.
3b). Describe the ionic fluxes during the action potential	Voltage gated Na channels open (short lived), Na channels overwhelm K once threshold reached. Memb potential approaches Na (+60mV) . Na stops as short open phase, then they close/become inactivated, then resting state again. Also electrical gradient reversed. Then opening of volt gated K channels-slower and more prolonged than Na. Slow return to closed state causes after hyperpolarization	Na and K and sequence.
4 a). What is the normal range of osmolality of ECF 4b). How is this maintained? 4 c). What other stimuli affect Vp secretion? Prompt- Anti-diuretic Hormone	285-295mosm/L Maintained by **Vasopressin-secreting** and **thirst** mechanisms If osm ↓, thirst is ↓, Vp secretion is ↓, resulting in **urinary loss of free water** If osm ↑, thirst is ↑, Vp secretion is ↑, leading to **renal reabsorption of free water** in renal collecting ducts/pyramids VP secretion ↑ by: (↑ effective plasma osm pressure) ↓ **ECF volume** (via low pressure receptors) **Pain, emotion, exercise, stress (eg surgery)** **Nausea & vomiting** **Angiotensin II** Standing Clofibrate, carbamazepine VP secretion ↓ by: (↓ effective plasma osm pressure) ↑ **ECF volume** **Alcohol**	Accept 280-300 3/4Bold 2/4 Bold 1 of 2 Bold

5 a) Name the principal pancreatic enzymes and the substances upon which they act.	• Trypsin – proteins, polypeptides • Chymotrypsin – proteins, polypeptides • Elastase – elastin and some other proteins • Carboxypeptidase A & B – proteins, polypeptides • Colipase – fat droplets • Pancreatic lipase – triglycerides • Bile salt-acid lipase – cholesterol esters • Pancreatic α-amylase – starch • Ribonuclease – RNA • Deoxyribonuclease – DNA • Phospholipase A2 – Phospholipids	Must be able to give trypsin, lipase, amylase plus 1 other & their appropriate substrate group (protein, fat, carbohydrate)
5 b) Describe the regulation of pancreatic juice secretion	• Primarily under hormonal control • Secretin acts on the duct to cause production of copious amounts of very alkaline pancreatic juice poor in enzymes. • As flow of pancreatic juice increases it becomes more alkaline because exchange of HCO_3^- for Cl^- in the distal duct is inversely proportional to flow • CCK acts on acinar cells to cause of release of zymogen granules and pancreatic juice rich in enzymes • Acetylcholine also stimulates release of zymogen granules (minor effect ?basis of vagally-mediated pancreatic juice secretion in response to sight/smell of food). Prompt: Do you know any hormones involved in secretion of pancreatic juice?	Must give Secretin and CCK and know that secretin causes mainly alkaline fluid and CCK mainly enzymes.

ACEM PRIMARY 20010.1 Physiology VIVA Friday 26 March Session am Candidate Number:..... Mark.....

QUESTION	KNOWLEDGE	PASS CRITERIA
1a). What is Pulmonary Compliance?	a) Compliance =Volume Change/Pressure Change, proportional to slope the pressure volume curve of the lung. Within normal range (-2 to –10 cm H20) of expanding pressures, lung is very compliant. At higher expanding pressures, compliance is smaller. Normal human lung compliance =200 ml /cm H2O) Specific Compliance = "compliance per unit volume of lung"	a) delta V/delta P
b) What are the factors that decrease and increase pulmonary compliance?	c) Reduced = pulm venous hypertension, unventilated lung (espec at low lung volumes i.e. atelectasis), pulm fibrosis and alveolar oedema of any type. Increased = increasing age and emphysema	b) Three factors which decrease Compliance and both the factors which decrease it c)two factors which increase it
c) What are the physiological effects of surfactant on the lung?	d) What are the physiological effects of Surfactant? i) increases lung compliance ii) reduces work of breathing iii) improves stability of alveoli iv) keeps alveoli dry	d) Three of four
2 a). What **chemical** factors regulate coronary blood flow	Increases in blood flow occur secondary to coronary vasodilation due to:. • Hypoxia, • Locally increased CO2, • H+, • K+, • lactate, • PGs, • adenine nucleotides, • adenosine	Hypoxia plus 3 others
2 b). Describe the **neural** regulation of coronary blood flow	Alpha–adrenergic receptors mediate vasoconstriction Beta–adrenergic receptors mediate vasodilation Vagal nerve stimulation dilates coronaries. Noradrenaline constricts coronaries (although noradrenergic nerves cause increased HR and contractility, with resultant metabolite prod and vasodilation- this is the effect with hypotension that maintains coronary flow)	Alpha–adrenergic – Vc, and beta–adrenergic – Vd effects.

ACEM PRIMARY 20010.1 Physiology VIVA Friday 26 March Session am Candidate Number:..... Mark.....

<table>
<tr>
<td>3 a). What general mechanisms are involved in renal tubular reabsorption and secretion?</td>
<td>Mechanisms involved in re-absorption and secretion include endocytosis, passive diffusion and facilitated diffusion and active transport.</td>
<td>2 of Bold to Pass</td>
</tr>
<tr>
<td>3 b). How is Sodium reabsorped in the various parts of the nephron?</td>
<td>No sodium transport in Thin descending Loop of Henle.
In rest of system, sodium moves by co-transport, exchange or down concentration gradient.
Sodium pumped out of cell by Active Sodium-Cl-Potassium pump in basolateral membrane.
60% in PCT by Sodium-Hydrogen exchange.
30% in thick ascending Limb via Sodium –Potassium co-transport.
7% in DCT via Sodium-Chloride exchange
<table>
<tr><th>Site</th><th>Apical Transporter</th><th>Function</th></tr>
<tr><td>Proximal tubule</td><td>Na/. . . . . CT</td><td>Na$^+$ uptake, . . . uptake</td></tr>
<tr><td></td><td>Na$^+$/P, CT</td><td>Na$^+$ uptake, Pi uptak</td></tr>
<tr><td></td><td>Na$^+$ . . . . . CT</td><td>Na$^+$ uptake, ... uptake</td></tr>
<tr><td></td><td>Na/lactate CT</td><td>Na$^+$ uptake, lactate uptake</td></tr>
<tr><td></td><td>Na/H exchanger</td><td>Na$^+$ uptake, H$^+$ extrusi</td></tr>
<tr><td></td><td>Cl/base exchanger</td><td>Cl$^-$ uptake</td></tr>
<tr><td>Thick ascending limb</td><td>Na–K–2Cl CT</td><td>Na$^+$ uptake, Cl$^-$ uptake, K$^+$ uptake</td></tr>
<tr><td></td><td>Na/H exchanger</td><td>Na$^+$ uptake, H$^+$ extrusio</td></tr>
<tr><td></td><td>K$^+$ channels</td><td>K$^+$ extrusion (recycling)</td></tr>
<tr><td>Distal convoluted tubule</td><td>. . . CT</td><td>Na$^+$ uptake, Cl$^-$ uptake</td></tr>
<tr><td>Collecting duct</td><td>Na$^+$ channel (ENaC)</td><td>Na$^+$ uptake</td></tr>
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</td>
<td>Bold to pass, demonstrating reasonable understanding of different processes</td>
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4. What are the physiologic effects of the glucocorticoids?	1. Intermediary metabolism of carbohydrate, protein, fat* 2. Inhibit ACTH secretion* 3. Maintain reactivity of vascular (and bronchial) smooth muscle to catecholamines* 4. Allow excretion of a water load (mechanism unclear) 5. Blood - ↑ RCC, ↑ WCC (mainly PMNs), but ↓ Lymphocites and Lymph node size 6. CNS – irritability, apprehension, inability to concentrate (eg in exams) 7. "stress response" (Up to 3 specific prompts, eg "what are the vascular effects of glucocorticoids?")	3 asterisked
5. a) In the synapse, where can inhibition occur? b)What are the mechanisms involved?	Post-synaptic: direct or indirect (refractory periods, after-hyperpolarisations) Pre-synaptic: mediated by neurons that end on excitatory endings (axo-axonal synapses). i Increased Cl^- conductance – reduces Ca^{2+} influx and amount of excitatory transmitter released ii. Voltage-gated K^+ channels – K^+ also decreases Ca^{2+} entry iii. Direct inhibition of excitatory transmitter release, independent of Ca^{2+} influx	Must give pre-synaptic and post-synaptic Must give reduction in Ca^{2+} influx