ACEM PRIMARY 2009/1 PHYSIOLOGY VIVA 2nd April Thurs Session (am) *Candidate Number*..... AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: Isovolumetric contraction and relaxation Ganong 565-8	In the heart: i) Describe the pressure and volume changes in the ventricles at the onset of systole. <u>Prompt</u>: describe the events that occur around the isovolumetric relaxation phase of systole. ii) Describe the pressure and volume changes in the ventricles at the onset of diastole <u>Prompt</u>: describe the events that occur around the isovolumetric relaxation phase of diastole iii) <u>Additional / offer as an option if candidate struggles with the question</u>: Can you draw the pressure-volume loop of the left ventricle?	i) At the start of **systole mitral and tricuspid valves close**. Ventricular muscle initially shortens very little and pressure rises sharply. AV valves bulge into atria causing a small but sharp rise in atrial/venous pressure. **Isovolumetric contraction** lasts about 0.05 secs until left pressure exceeds aorta (80mmHg), right pressure exceeds pulmonary artery (10mmHg) and so **aortic and pulmonary valves open.** ii) When the momentum of ejected blood is overcome by arterial pressure the **aortic and pulmonary valves close** (setting up transient vibrations). Ventricular pressures drop rapidly until they fall below atrial pressures – **isovolumetric relaxation**. Then **AV valves open** to start ventricular filling. iii) Pressure-volume loop.P566	Core knowledge in bold
Question 2: Role of surfactant and applied Laplace's Law (West 99-104)	i) In the lung, what is surfactant and how does it work? ii) What are the physiological advantages of surfactant ?	a) Surfactant is a phospholipid. Dipalmitoyl phosphatidylcholine (DPPC) is an important constituent) b) Produced in type 2 alveolar cells. Lamellated bodies within them are extruded into the alveoli and transform into surfactant. c) Fast synthesis with rapid turnover d) Formed relatively late in foetal life. e) With surfactant present, surface tension changes greatly with surface area. It falls to very low values when area is small f) Molecules of DPPC are hydrophobic at one end and hydrophilic at the other. When aligned on the surface, their **repulsive forces** oppose the normal attractive forces between the liquid surface molecules. i) **Reduction in surface tension** is greatest when film is compressed and molecules of DPPC are closest together. ii) **Lower surface tension** in the alveoli **increases lung compliance** and decreases work of breathing	Core knowledge in bold

		iii) **Promotes alveolar stability** (reduces tendency for small alveoli to empty into large alveoli) iv) 3) **Keep the alveoli dry** (surface tension "sucks" fluid into alveolar spaces from capillaries, by reducing hydrostatic pressure in the tissue)	
Question 3: Acid secretion & absorption in kidney Ganong pp 720-1	i) How is H+ ion secreted in the proximal tubule of the kidney?	a) **Secondary active transport** (The renal tubular cells secrete H^+ into the tubular fluid in **exchange for Na^+;** and for each H^+ secreted, one Na^+ and one HCO_3^- are added to the blood) b) Linked to **Na+/K+ ATPase**	
	ii) Outline the buffer systems that act to bind H+ ion in the tubular fluid	i) 3 systems – **HCO3, HPO4, NH3** ii) Major role of carbonic anhydrase/HCO3 system	To pass: 2/3, must have bicarbonate
	iii) What is the importance of H+ buffering systems in the urine ?	**Limiting pH (~4.5) would rapidly be reached unless free H+ is buffered**	
Question 4: Temperature regulation Ganong 251-5	i) How does the body generate heat?	a) Heat production: **basal metabolic processes, muscular activity**, food intake	Core knowledge in bold
	ii) How does the body lose heat?	b) Heat loss: conduction, convection and **radiation** (70%), **sweat vaporisation** (27%), respiration (2%), urine and defaecation (1%)	
	iii) What is the thermo-regulatory response to cold?	i. **Increased heat production** such as shivering, muscular activity, hunger (eating), hormonal increase in adrenalin/ NA. **Decreased heat loss** with cutaneous vasoconstriction, curling up, horripilation (goose pimples – erection of hairs to ii. Decrease conduction/convection.	
Question 5: Serotonin (Ganong pp 106-107, 262-263)	i) What are the functions of serotonin?	a) Regulation of **vomiting reflex** b) Regulation of **mood** c) Control of respiration d) Platelet aggregation and smooth muscle contraction e) Facilitate GI secretion and peristalsis f) Regulation of circadian rhythms	Core knowledge in bold
	ii) What are the steps in synthesis and catabolism of serotonin?	i) Hydroxylation and decarboxylation of tryptophan to form serotonin ii) Released serotonin from serotonergic neurones is recaptured by an **active re-uptake mechanism and inactivated by MAO** to form 5HIAA iii) (5-hydroxyindoleacetic acid) iv) 5HIAA is excreted as a urinary metabolite	Core knowledge in bold

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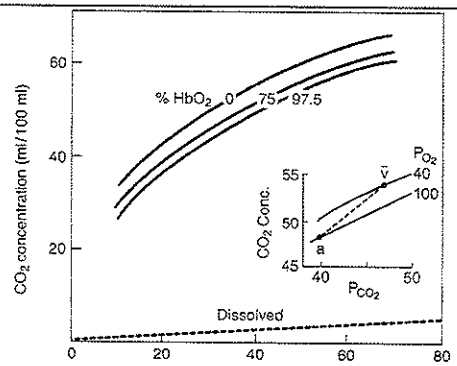
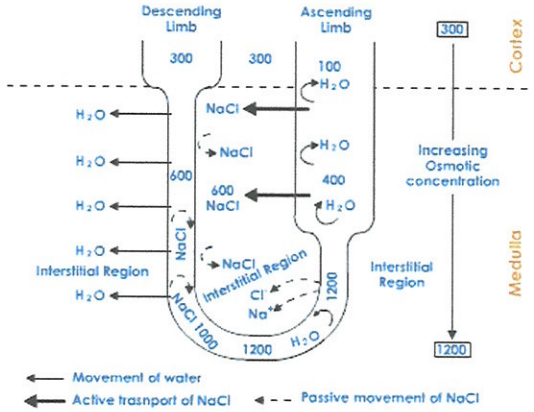
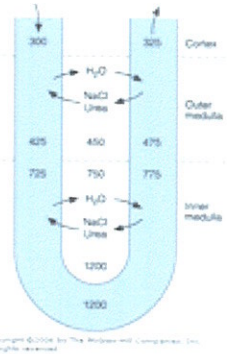
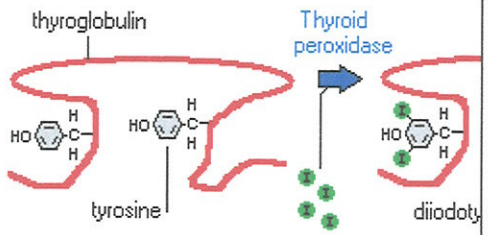
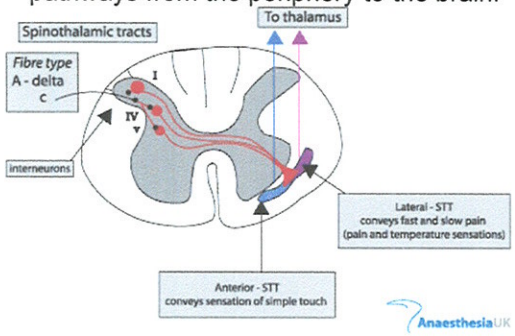
TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: Cerebral blood flow & its regulation. Ganong pp 611-620	i) What factors determine cerebral blood flow? Prompt: What about pressure or vascular factors? ii) Additional: Describe the process of autoregulation.	**(1) Pressures:** · **MAP** · **Intracranial pressure** / extra-cranial venous pressure (whichever is greatest). *Intracranial pressure is determined by intracranial blood volume, CSF volume, tissue oedema, SOL.* **(2) Cerebral arteriole tone** · **Autoregulation** Maintains normal CBF at MAPs of 65-140 mmHg (stretch response[myogenic], local [metabolic]); *Autoregulation may be lost/impaired by brain injury* · **pCO2** (effect on both arteriole tone and intracranial blood volume) · pO2 (at extremes) (3) Blood viscosity	Must get **bold** to pass.
Question 2: Carbon Dioxide Transport West pp 80-3	i) How is carbon dioxide transported in the blood?	 **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 P_{CO_2}. The *inset* shows the "physiological" curve between arterial and mixed venous blood. • **Dissolved**. • As **carbamino compounds** with proteins, especially Hb. • Hydrated in red cells — H^+ buffered — **HCO_3^-** in plasma.	To pass: 2/3
	ii) How does venous blood carry more CO2 than arterial blood?	• 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.	Does the curve move towards the left or the right, and why??

	iii)	•	
Question 3: Counter-Current mechanism Ganong pp 716-8	i) Describe the counter-current mechanism in the kidney. Prompt: What is the role of the vasta recta?  **Countercurrent multipliers**	a) **Countercurrent multipliers** in the **LOH** through active transport of Na (& Cl^-) out of its thick ascending limb. Water moves out of the thin descending limb, with inflow of tubular fluid from the PCT. This increases the interstitial osmolarity. This results in hypotonic fluid flows into DCT, isotonic fluid flows into the asc thick LOH. The final result is a gradient conc from the top to the bottom of the LOH & **a gradient hyperosmolarity in the medulla interstitium**. b) **Vasta recta** as **countercurrent exchangers** in the kidney in which NaCl & urea diffuse out of the asc limb of the vessel & into the desc limb, while water diffuses out of the desc into the ascending limb of the vascular loop. As a result the solute remains in the medulla pyramid & maintain the interstitial conc.	 Countercurrent exchangers
Question 4: Thyroid hormone synthesis and effects. Ganong pp 319, 323-6	i) Describe the steps in synthesis of thyroid hormones. Prompt: What are thyroid hormones made from?  ii) What are the physiological effects of T4? Prompt: How do thyroid hormones alter metabolism?	i) Thyroid epithelial cells secrete **thyroglobulin** (comprising 134 tyrosines) and **iodine** into colloid. Iodide transport is via a symport with sodium (NIS). Thyroid peroxidise makes iodotyrosines (MIT and DIT) then combines them to **make T3 and T4**. Some reverse T3 (inactive) also made. Endocytosis and lysis of colloid releases free hormone. All steps TSH controlled. T3 also made peripherally by deiodination of T4. ii) Binds to intracellular thyroid receptors in the nuclei. Complex binds to DNA and alters gene expression. T3 more rapid and potent. **Incr metabolism and catabolism** of most cells (brain and others excluded). Lipid and carb mobilisation and usage. **Inc CVS** and CNS **activity**. Normal reproductive cycle and growth. Effects incr by catecholamines.	Core knowledge in bold. Subunits combine together

Question 5: Ganong pp 139-147	i) Describe the route followed by pain pathways from the periphery to the brain.  ii) What are the characteristics of the different types of pain fibres?	i) Primary efferent fibres - naked nerve endings peripherally - cell bodies in dorsal root ganglia (or equivalent in cranial nn.) - terminate on neurons in **dorsal horns** (Aδ fibres in laminae I and V, C fires in laminae I and II) Axons from dorsal horns travel in **anterolateral system** (**lateral spinothalamic tract**) to ventricular posterior nuclei (specific sensory relay nuclei of thalamus) and thence to cerebral cortex. ii) Aδ: **Myelinated** Large diameter (2-5 microm) **Fast** conduction rates (12-30 m/s) Modulate "fast" pain C: **Unmyelinated** Small diameter (0.4-1.2 microm) **Slow** conduction rates (0.5-2 m/s) Modulate "slow" pain	Core knowledge in bold.

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TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: Factors determining myocardial O2 demand Ganong pp 575-76	i) What factors determine myocardial oxygen demand?	i) **1) Heart Rate** **2)Intramyocardial Tension** **3)Contractile state of the myocardium** OR 1) **Stroke Volume** **2) MAP**	Core knowledge in bold. 2 out of 3
	ii) What effect does increase in preload and afterload have on myocardial O2 demand? Prompt: How does it work?	**i) Both increase** Ventricular work per beat correlates to O2 consumption Work = SV x MAP Stroke work LV is 7x that of RV Theoretically, volume changes and pressure changes should affect myocardial O2 consumption equally. HOWEVER, pressure work produces a greater increase in O2 consumption than does volume work. Reason not well understood Net result ; **Changes in afterload have greater effect than changes in preload.** Tension in the wall of a hollow viscus is proportional to the radius of the viscus. Myocardial fibres are stretched with increased stroke volume in a dilated heart. Increased radius of dilated heart increases wall tension which explains the increased oxygen consumption	Core knowledge in bold. Both increase Changes in afterload have greater affect than changes in preload
Question 2: Alveolar gas equation and its use in a clinical setting West pp 58, pp170	i) What is the alveolar gas equation? ii) How do you calculate the alveolar-arterial gradient? iii) What is the physiological significance of the A-a gradient?	i) **(4 out of 4)** $$PAO2 = PIO2 - \frac{PACO2}{R}$$ Where: • PAO2 is the alveolar oxygen partial pressure • PIO2 is the oxygen partial pressure of inspired air • PACO2 is the alveolar CO2 partial pressure. • R is the respiratory quotient; CO2 production/O2 consumption, typically 0.8 Note that a small correction factor F of 2mmHg has been omitted from the equation. ii) **Difference between PAO2** (alveolar) and **PaO2** (arterial). iii) **V/Q mismatch** (eg: shunting or dead space)	

Question 3: Renal regulation of sodium Ganong pp 709-10, 723-4	i) Where does sodium reabsorption occur in the nephron?	a) All parts of the nephron except thin part of the LoH (+Specify at least two of:) b) 60% **PCT primarily** by Na^+-H^+ exchange but also a range of cotransport (glc, Pi, AA, lactate) c) 30% thick ascending limb of LoH (Na^+-2Cl^--K^+ cotransporter) d) 7% DCT LoH (Na^+-Cl^- cotransporter) e) 3% collecting ducts through Na+ channels (ENaC)	Must get **bold** to pass.
	ii) What are the mechanisms of sodium re-absorption in the nephron?	• **Na/K ATPase active transport.** Moves (by gradient thus generated) across apical membranes from tubular lumen into cell via cotransport & exchanger proteins. Driven by active transport by Na-K ATPase (3Na/2K) from tubular cell into interstitium (mainly into lateral interstitial spaced)	
	iii) What mechanisms in the kidney reduce sodium excretion? Prompt (if they get it ar**-about-t**) *What mechanisms in the kidney cause the body to retain sodium by reducing sodium excretion?*	Multiple regulatory mechanisms (reflects importance of Na as the prime determinant of ECF volume) • **Reduced GFR** • Increased tubular reabsorption o ↑**adrenocortical hormones esp. aldosterone** - act primarily on collecting ducts (activation of ENaC) o ↓ANP (inhibit ENaC) o AT-II (PCT) o [↓secretion of K^+ and H^+]	Must get **bold** to pass.
Question 4: Regulation of plasma calcium levels. Ganong pp 382-95	i) How plasma calcium levels are regulated? Prompt: What increases or decreases plasma calcium?	a) **1,25-Dihydroxycholecalciferol (from Vit D)** incr Ca absorption from gut and kidneys. b) **Parathyroid hormone** mobilizes Ca from bone. c) **Calcitonin** (from thyroid) inhibits bone resorption. Glucocorticoids, GH, oestrogens and others also effect Ca. 95% in bone (some readily available). In plasma, some bound and some free (depends on plasma protein levels and pH). Incr phosphate decr Ca.	Need to list all 3 and discuss its effect on Ca (inc or dec).
	ii) Describe the regulation of parathyroid hormone levels. Prompt: What stimulates production of parathyroid hormone?	i) **Negative feedback** by Ca via a membrane Ca receptor and G protein. 1,25-Dihydroxycholecalciferol acts to decrease preproPTH mRNA. Incr phosphate incr PTH by decr Ca and 1,25 DHCC. Mg required for PTH secretion.	
		• **Reflex arc** consisting of **sensory** organ(s) | **afferent** limb(s) | central integrator(s) | **efferent** limbs | **effectors.** • Sensory organs are nociceptors in the skin or subcutaneous tissues, responding to noxious (usually painful) stimuli. • Afferent limb is/are sensory (pain) fibre(s) to the	

Question 5: Withdrawal reflex Ganong pp 135-6	i) Please describe the withdrawal reflex. Prompt: What happens if a painful stimulus is applied to the left lower limb of a normal person?	dorsal root, then dorsal horn of the spinal cord. • Central integrator consists of **polysynaptic** connections in the spinal cord. • Efferent limbs are motor nerves to effector muscles on the ipsi- and contralateral sides. • Effectors are muscles of the ipsi- and contralateral sides, which produce flexion and withdrawal of the ipsilateral limb and (crossed) extension of the opposite limb.	
	ii) What are the important characteristics of polysynaptic reflexes?	• The reflex effect becomes stronger and more prolonged the greater the stimulus. Due to: o The impulse arriving at effectors at different times due to interneurones. o Irradiation of the impulse up and down the spinal cord. o Recruitment of motor units. o Reverberation of the circuit as some interneurons turn back on themselves. • Above effects result in after-discharge due to continued bombardment of motor neurones by impulses arriving by complicated and circuitous paths.	**To pass: Magnified response**
	iii) What is meant by the term prepotency of the withdrawal reflex?	• The reflex pre-empts spinal pathways from any other reflex activity occurring at the same time.	