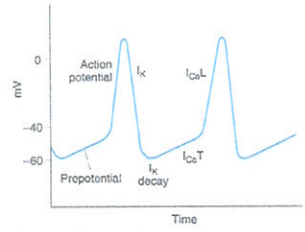
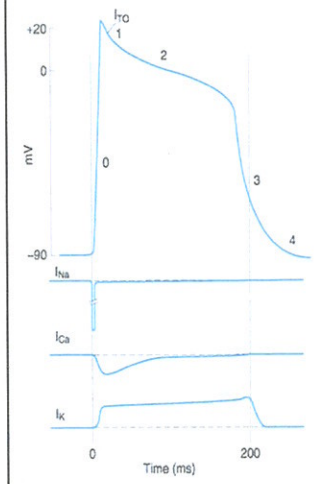
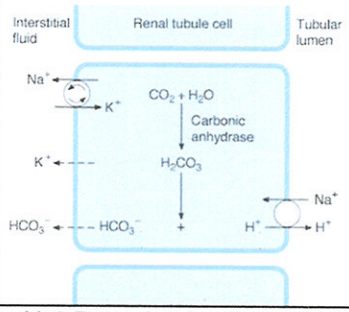
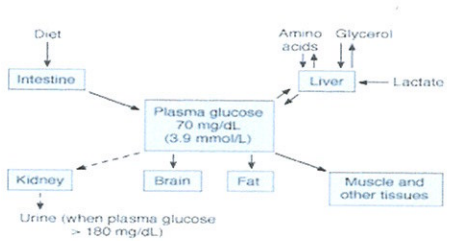
ACEM PRIMARY 2009/2 PHYSIOLOGY VIVA Day THURS **Session (am/pm)** *Candidate Number*.....

AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1a:	**Draw and explain the action potential in a cardiac pacemaker cell.**	 PROMPTS: What electrolytes are responsible for each phase of the AP?	**Pass-fail** **Must have shape to pass and know ion fluxes (↓effK + infCa T)- InfCa L – Eff K)** **1 Pre-potential initially due to decrease in efflux K^+, then completed by influx Ca^{2+} through T channels** **2 AP due to influx Ca^{2+} via L channels** **3 Repolarisation due to efflux K, no plateau**
Question 1b:	**Describe the major differences between a cardiac myocyte AP and the pacemaker**		1. Resting membrane potential, , –90mV rapid depolarisation voltage gated Na (overshoots) 2. Phase 1 rapid repolarisation = closure of Na channels.(inner v outer gates) 3. Plateau phase 2 voltage gated Ca2+ channels open (slower L type) 4. Phase 3 repolarisation Ca2+ ch close 5. Phase 4 due to various K+ efflux Differences- 1) Na fast v Ca dependent, 2) automaticity due to rising prepotential (K+/ Ca+), 3) plateau phase, 4) > resting potentials **Pass-Fail: Need correct shape + some knowledge of different channels (partic Na v Ca), no automaticity (no-prepotential, as no leaking K/ Ca) and plateau due to Ca ++ (>er inactive phase)**

Question 2:a) Score:	**What are the major factors that effect pulmonary vascular resistance in the normal lung?**	1) ↑Art or 2) Ven Pressure 3) Lung volume (U/J shaped curve) 4) Alveolar hypoxia > increased PVR via hypoxic vasoconstriction 5) Vascular Smooth Muscle Tone - response to endogenous/ exogenous factors 6)Area of lung (apex partic < base) 7) Position change 1	(A > V) (recruit – low P) (distension (high P) Low vol- collapsed ex-alveolar vessels Intermed Vol – vessels open High Vol – compressed alveol vessels (pulled open v normal elastic -cap 1st) (complex: Ipleural P < CO, alveolar P > capillary + caps squashed in alveoli) **Pass/Fail 3 of 6 , extra marks for detail in** eg nitrates, Ach, Isoprenaline, NO, decrease PVR; Increased sympathetic tone, serotonin, histamine and norepinephrine increase PVR, endothelin, thromboxane A2
b)	**Why is pulmonary flow so sensitive to pulmonary vascular pressures?**	) V low Pressure system – few resistance vessels 2) Easily distensible vessels 3) Recruitment 4) Only just enough P for normal gravity/ position to get apical flow 2/4 to pass	P just enough to reach only standing but (dependent lung may collapse) -due to < art pressure in low pressure system- partic if poor output V thin walls Vasc bed expands + geometry with alveolar expansion Surrounding IP/ alv P v significant effect on output Additional info 1/10th syst P (5-15 A-V diff) (low vol smooth muscle/high P and higher lung vol) (geometry-low P) (distension/ effects on cap) (due to v low P in system)

Question 3a: Score:	**Discuss how and where H+ is secreted in the kidney?** **Prompt: how is Bicarbonate involved.**	Prox Na Bic co tranport Distal H+ ATPase H+/K+ (I cells) with large C Anh conc + Cl/ HCO3 BM exchanger  	Active secretion H+ (H+/Na+ co transport- 2ary active secretion), allows reuptake of HCO3- fromC anhydrase brush border_- H20/ CO2- Bic then into interstitium with Na via Na/K ATPase) Bic in cell transferred to Interstitium along gdt_ In DCT/ Coll ducts- Principle cells/ Aldo- have H+ ATPAse channels + H/K ATPAse linked to Bic/ Cl- exchanger in BM **PASS-FAIL must know 2 diff mechanisms, and mention bicarb**
3b:	**What is the limiting pH of urine and how is this limitation dealt with?**	pH 4.5 maximal acidity urine much > er acidity required excreted 3 major BUFFER systems H2CO3 (proximal), NH4+(throughout) and HPo4 (distal)	H+ load would be 100-100 x greater than max pH, Buffers all inc (partic H2CO3 and NH4 when acidotic)- NH4 via glutamate in interstitium, H2CO3 inc with H+ extra = >substrate + > C anh. HPO4 v concentrated in DCT
Question 4a: ***Score:***	**What are the major factors determining the plasma glucose level?** PROMPTS If discussing hormones XS- how does glucose enter and leave the plasma	1) Concept: Balance between glucose entering the bloodstream and glucose leaving the bloodstream. 2. Dietary intake 3. Cellular uptake (partic muscle/fat/ hepatic) 4. Hepatic glucostat / glycogenisis, glycogenolysis, gluconeogenisis 5. Renal freely filtered but PT reabsorbed to Tmax 6) Hormonal effects on these (partic 1, 3,4)	**COMMENTS** 3 for a pass + concept Complex hormonal effects not required 

4b :	**List the hormones which effect plasma glucose levels?** **Prompt- which way does gluc move**	↓BSL - Insulin (),Ins like GF 1and 2- (NSILA) ↑ BSL Catecholamines (Nor / Epi partic) (>),Glucagon (>), GH>, Cortisol>, Thyroid Pass requires 3 hormones + correct < or >	Insulin via glucose uptake (al tissues), glycogenogenesis, Liver - gluc to fat, - IGF- similar but much < Catechol –b receptor > cAMP- glycogenolysis/ gluconeogenesis Glucagon- cAMP direct- as catech TFTs- > absorption + ↑glycogenolysiss (liver partic) + ins bkdown↑ Cortisol- permissive to Glucagon/Catechols + some glucgenesis, prot to gluc liver- < uptake GH- > gluc liver, insulin block, <tissue upotake
Question 5:a	**Describe the typical serum / urine effects in hyperaldosteronism**	Na/ Cl mild ↑, fluid retention (follows Na), ↓K, alkalosis (alkalaemia only if K+ depletes) Urine K+/ H↑	**Na +/ Cl- mild rise in serum + fluid retention** **K+ <, mild alkalosis/ alkalaemia** **Why: Na + retained/ but drags fluid into ECFV01 (dilutes) + Na+ excretion >– escape phenomena** **C1 –retention with Na+.** **K+ depletion – K+ diuresis* (due to effect of aldosterone)** **H+ lost in urine - ↑ urinary acidity*, H+ loss in serum- only seen if K+ depletes and rely on H+ excretion**
5b	**How does aldosterone exert its effects in the kidney?**	Mineralocorticoid- Via Principal cells- collecting ducts, 2 effects 1) Genomic- Intracellular to nuc signalling > mRNA – a) Inc ENAC insertion/ activity (quick) b) > production (slow) 2) membrane bind IP3 mediated Na/K exchange > All = > Na reabsorb K/H loss to urine	**Is a medullary mineralo corticoid.** **Acts on P(rinciple cells ?) cells in collecting duct* (** **↑ reabsorption of Na+ and ct from urine in exchange for K+ and H+ causing ↑ pH and K+ diuresis.** **Action takes 10-30 minutes to develop and peaks later*** **Aldosterone – cytoplasmic receptor complex moves to nucleus where it alters transcription of mRNA.** **This now has 2 effects:** **1 Rapid - ↑ activity (+insertion*) of preformed/ active EpithNaChannels s, via activation of genes for SGK** **2 slower* - ↑ synthesis of ENaCs. There is a non genomic action. ↑ activitiy of the Na+ K+ exchangers via IP3 - ↑ intracellular Na+**

CEM PRIMARY 2009/2 PHYSIOLOGY VIVA Day Thurs Session (pm) *Candidate Number*..... AGREED
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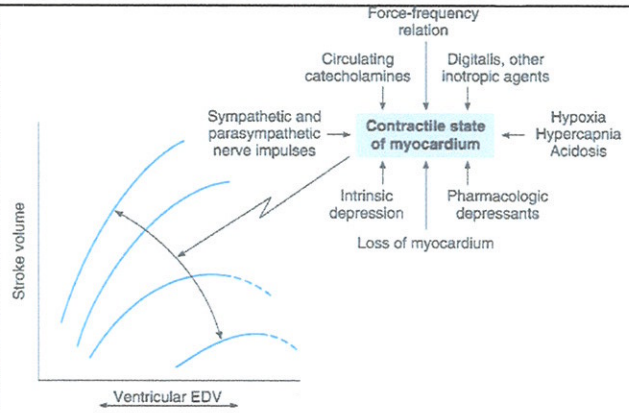
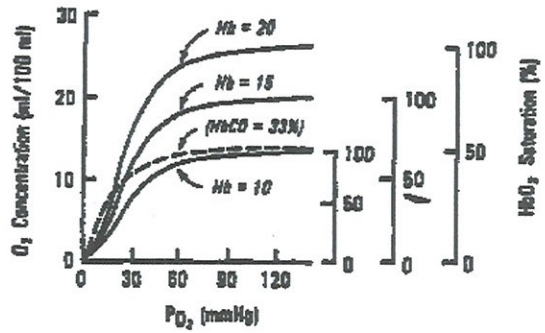
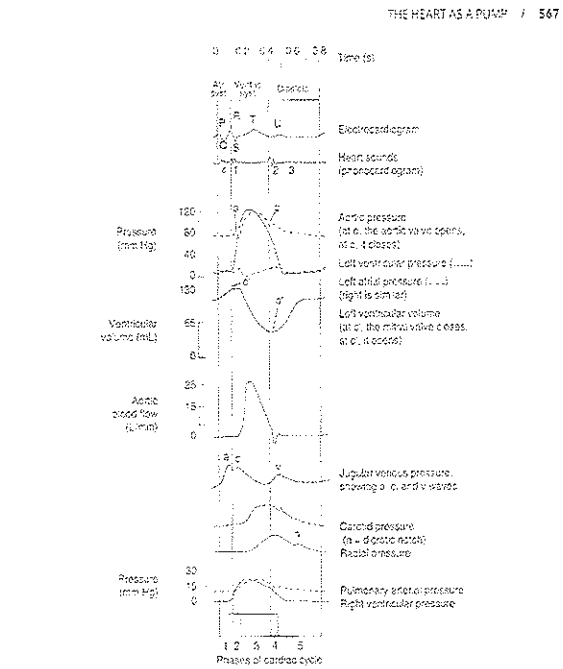
TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: a) b)	**Draw Frank Starling curve.** **List the factors that alter contractility**	 	1. Sympathetic nerves moves up & left, 2. Parasympathetic nerves move down and right; 3. Force frequency relationships, postextrasystolic potentiation (Ca2+mediated 4. Catechols (via beta 1 and cAMP), digitalis (via Na/K ATPase block) and inotropes increase 5. Hypoxia, hypercarbia, acidosis, quinidine, procainamide, barbs etc depress MC 6. Intrinsic depression with CHF, AMI **Must label FS curve, and 2up /2 down**
Question 2:	**Please draw the curve demonstrating the relationship between O_2 concentration v pO2** **How does this change in anaemic and polycythaemic individual?** **What is the effect of carbon monoxide on these curves?**	 1) decrease in effective Hb per percentage 2) COHb shift to Left	Prompt if draws saturation dissociation curve

Question 3: a) b)	**Describe how sodium is handled in the glomerulus and the PCT** **List the mechanisms that effect Na reabsorption**	**Most Filtered** out with solutes/ AAs (90%) **Most (60%)** Na-H counter-transport, **Bicarbonate** is main anion reabsorbed with Na Absolutely depends on Na K ATP ase (Basement M)/ C Anhydrase-tub cell to generate H+/ Bic Small co-transport with nutrients /anions/ Cl latter part Approx 60% 1)Tubulo-glom - Macula Densa,↑Na↑adenos/ Ca, aff vasocon 2)Glomer/tub balance- > filtered = > resorbed (good capacity)- mainly oncotic p in eff capillaries 3) Humeral Aldosterone- distal CT / ENaC, K+/H+ PGE2 – pron Na K ATP ase block/ Ca ++ > Ouabain endog- ATP ase block effect Endothelin and IL-1 cause natriuresis (prob > PGE2) ANP-↑ cGMP – less ENaC Angio 2- renal ACE ↑circ Ang 1 + renal -↑ PCT > reabs	NB – good candidates will volunteer Na resorbtion through out except TALH, 60/30/7/3 % - all Na excretion last 3% **Reqd :1 humeral / 1other**
Question 4:	**What are the sequence of events in contraction and relaxation of a skeletal muscle?** **Prompt: what about relaxation**	1)Motor neurone d/c + Ach presyn release 2)AcH to post syn- Nicotinic receptors ↑ Na/K in end plate generates AP along muscle fibre 3) T tubules spread depolarisation releases Ca++ from sarcoplasmic reticulum (terminal cisterns) 4) ↑ Ca around myosin/actin filaments, to TropC uncovers myosin binding sites on Actin 5) 5) X-links form thin/ thick – shorten as slide **Relaxation** Ca pumped out, trop C reactivated and blocks actin/myosin bind.	Pass /FailShould have 3 /5 steps mentioned with some detail and know active Ca ++ reverses for relation

Question 5:	**What are the actions of the parathyroid hormone on Calcium?** **What are the other effects of PTH?**	PTH- 1. ↑ plasma Ca^{++} by: ↑ Ca^{++} mobilization ↑ bone reabsorption, ↑ Ca^{++} reabsorption in distal tubule and (3) Ca reabsorption 2. ↓ plasma phosphate: ↓ PO_4 reabsorption in proximal tubules 3. ↑ 1,25 dihydrocholecalciferol: renal (> Ca absorption) 4. Over a longer time: ↑ osteoblastic *and* osteoclastic stimulation- prob anabolic	Pass: Ca ++ ↑ PO4 ↓ + some idea of how these achieved OR additional other mechs Parathyroid related hormone- (prob fetal/ cartilage growth + teeth/ breast- skin) ? **PO4 < +1 other in either section**

CEM PRIMARY 2009/2 PHYSIOLOGY VIVA Day Fri Session (am/) *Candidate Number*.....
AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1:	**On this sheet of paper, please draw an ECG trace and, below this, identify the 5 phases of the cardiac (contractile) cycle**	1. Atrial systole 2. Isovolumetric ventricular contraction 3. Ventricular ejection 4. Isovolumetric ventricular relaxation 5. Ventricular filling	4/5
	On this sheet of paper, please draw an ECG trace and, below this, demonstrate the left ventricular volume trace. Please give approximate volume values on the y-axis.	 *Figure 29–3.* Events of the cardiac cycle at a heart rate of 75 beats/min. The phases of the cardiac cycle identified by the numbers at the bottom are as follows: 1, atrial systole; 2, isovolumetric ventricular contraction; 3, ventricular ejection; 4, isovolumetric ventricular relaxation; 5, ventricular filling. Note that late in systole, aortic pressure actually exceeds left ventricular pressure. However, the momentum of the blood keeps it flowing out of the ventricle for a short period. The events in the lower figure for the right ventricle and pulmonary artery are similar. Abbreviations: AV, atrioventricular; SL, semilunar.	1. The end-diastolic ventricular volume is approx. 130ml 2. The end-systolic ventricular volume is approx. 50ml [thus about 80ml is ejected by each ventricle per contraction, at rest and the ejection fraction (the percent of the EDV that is ejected with each contraction) is about 65%.

Question 2:	**What are the physiological changes that allow survival at high altitude ?**	1) Hyperventilation > decreases CO_2, > O2 2) Increased Hb (> EPO), 3) Alkalosis moderated by movement of bicarbonate from CNS (1-2/7) and renal excretion 4) Increased 2,3,DPG - R shift, 5) Pulm hypertension (due to alveolar hypoxia inducing pulm vasoconstriction) - 6) RV hypertrophy – not really an "adaptation" 7) Decreased work of breathing	All hypoxia driven, > viscosity helpful as pick up more difFic (3 of 7 to pass)
Question 3:	**Describe how anti-diuretic hormone/ Vasopressin acts on the kidney.**	ADH binds to G-receptor, V2 activates adenylate cyclase. ↑ IC c-AMP ▶migration of IC endosomes. **H20 channels (aquaporin2) inserted into luminal membrane ↑water permeability, with ↑water reabsorption**	
	What factors influence ADH secretion	**Table 39-1.** Summary of stimuli affecting vasopressin secretion. **Vasopressin Secretion** **Increased** / **Decreased** osmotic P of plasma↑ 285mmol / osmotic P of plasma ↓ Decreased ECF / Increased ECF volume Pain, emotion, "stress," exercise / Alcohol Nausea and vomiting Standing Clofibrate, carbamazepine Angiotensin II	Osmolality mediated via osmoreceptors OVLT **ECF via Baroeceptors** **NB low P baro rec (pulm/atria > arterial) via NTS/ CVLM).** **If bled/ hypotens reinforces via Angio 2 > ADH and allows lower osmolality to trigger ADH** **Pass /Fail** **must know ECF/ Osmolality + 1 other or some sensor/ effector pathway**
Question 4:	**What are the principal functions of the Liver.**	**1) Bile formation (500 mls a day) - Excretion, elimination,** digestion **2) Synthesis- protein/** coag/ biding prot/alb **3) Inactivation/ detox –**drugs/toxins/ active circ substances **4) Nutrient vitamin absorption, metabolism/ control (e.g. glucostat)** AAs, lipids, fat sol vits etc **5) Immunity (partic gut orgs)**- Kupffer/ Macrophages in sinusoid endothelium	3/5 named functions (or part of function eg some idea)

b)	**Describe bilirubins path from production to excretion?**	1. **Most formed by breakdown of Heme /Hb.** 2. **Bilirubin bound to albumin *** 3. **In liver actively transported (OATP) as dissociates – binds to cytoplasmic proteins.** 4. **Conjugated by gluc-transferase (*in ER) with glucoronic acid to H20 sol bil-digluc** 5. **Bil di gluc active transport (MDRP2) against gdt to bile canaliculi – to gut. (<5% bil/bdg reflux to blood)** 6. **Intestinal mucosae relatively impermeable** 7. **Gut bacteria act / convert most to urobilinogens*** 8. **Some bile pigments/ urobilinogens/unconj bil reabsorbed in portal circn –most resecreted– entero hepatic circulation.** 9. **Small amounts urobil in blood excreted in urine – urobilinogen and faeces – stercobil.**	Pass Fail 4 elements in proper order/ prompt if stuck on excessive detail e.g. just a general overview of production to excretion. Pass does not require this detail!
Question5:	**What are the effects of thyroid hormones on nervous and vascular systems** **What other physiological effects does thyroid hormone have on the body?**	CNS- 1)**Development CNS** -cerebral cortex, basal ganglia cochlea 2)↑ **activity, mentation speed/ agitation** (catechol / dop+ direct brain effects) 3) ↑ **refexes** CVS- 1)**vasodil** (2ary heat)- 2) > **circ vol/ HR/ CO** - **3)Ht- > myosin heavy chain (+ isoforms)**/ faster twitch genes (+ Ca ++, Na K ATPase etc↑) + down reg others, **> contraction/ HR**/ speed of contraction - **4) > sens to Catechols** (synergistic effects + up regulated ß receptors and effector systems) HR, contract more Lipolysis - adipose tissue Formation of LDL receptors on lipoprotein Protein breakdown in muscle Skeletal development promoted Increased carbohydrate absorption from the gut Stimulates O_2 consumption by metabolically active tissues Increased BSL/ insulin resistance	Impt issues highlighted 3/6 for a pass. **Pass: 3-4 overall at least 1 in each** **Prompt- what features of thyrotoxicosis** **2 required**