ACEM 2008.2 PRIMARY VIVA EXAMINATION

SUBJECT: PHYSIOLOGY **Thursday am Q 1**

TOPIC: AP in Cardiac pacemaker cell + effect SNS/PNS NUMBER: _____

OPENING QUESTION	**Draw the action potential in a cardiac pacemaker cell, and explain the ionic fluxes.**	**COMMENTS**
POINTS REQUIRED	**1 Prepotential initially due to decrease in efflux K^+ , then completed by influx Ca^{2+} through T channels**	Suggested Pass/Fail Criteria in Bolded Type
	2 AP due to influx Ca^{2+} via L channels	
	3 Repolarisation due to efflux K, no plateau	All essential
PROMPTS	What electrolytes are responsible for each phase of the AP?	Stress cardiac pacemaker cell
SECOND QUESTION	**How do sympathetic and parasympathetic stimulation change the prepotential?**	
POINTS REQUIRED	1 Noradrenaline binds to Beta 1 receptor and raises cAMP, resulting in increased opening of L channels and Ca^{2+} influx. **Thus increased slope of prepotential and firing rate**	
	2 ACh binds to M2 receptor and decreases cAMP, resulting in both slowing of Ca channel opening and opening of special K channels (counters decay of K efflux) leading to greater fall in prepotential **Thus decreased slope of prepotential and firing rate**	
PROMPTS	What does noradrenaline do? What does vagal stimulation do?	

COMMENTS

ACEM 2008.2 PRIMARY VIVA EXAMINATION

SUBJECT: PHYSIOLOGY Thurs am Q 2

TOPIC: Distribution of Blood Flow in the Lung NUMBER:

OPENING QUESTION	**Describe the distribution of blood flow in the lung of an upright subject at rest.**	**COMMENTS**
POINTS REQUIRED	**1. Decreases linearly from base to apex**	Must identify 1, 2 ,3 and 5 to pass
	2. Due to hydrostatic pressure,	
	3. Under normal conditions, flow almost ceases at apex	
	4. Distribution more uniform with exercise	
	5. Explanation of West's zones 1 – 3 +/- zone 4 6. Zone 4 only at very low lung volumes	
PROMPTS	What are the zones of the lung described by West ?	
SECOND QUESTION (if needed)	**What are the main determinants of flow in these three zones ?**	
POINTS REQUIRED	**1. Zone 1 PA>Pa>Pv(not under normal conditions and is alv. dead space)** **2. Zone 2 Pa>PA>Pv (recruitment)** 3. **Zone 3 Pa>Pv>PA (distension + recruitment)**	Must identify 3 pressures and their relationship to pass
PROMPTS	What pressure gradients determine flow in zones 1-3	
THIRD QUESTION (if needed)	**How does the distribution of blood change when the subject becomes supine?**	
POINTS REQUIRED	1. Blood flow from base to apex is almost uniform but flow in posterior segments exceeds that in anterior segments	
PROMPTS		

COMMENTS

ACEM 2008.2 PRIMARY VIVA EXAMINATION

SUBJECT: PHYSIOLOGY Thurs am Q 3

TOPIC: Micturition NUMBER:

OPENING QUESTION	**Describe the physiologic process of Micturition**	**COMMENTS**
POINTS REQUIRED	1 **A spinal reflex inhibited and facilitated by higher centres** Intravesical pressure rises only after 400mls urine in bladder **Anatomy: Detrusor m, int and ext urethral sphincters** **During micturition Detrusor contracts, and perineal muscles and EUS relax.**	
	2.Nerve Supply **Parasympathetic (S 2,3,4,) via pelvic nn (afferent and efferents) to/from detrusor (efferent contraction) and pudendal nn to EUS (relaxation)** **Sympathetic** (L1,2,3) – Hypogastric nn via Inf Mesenteric Ganglion play no role in active micturition per se but role in prevention. (cause contraction of bladder muscle to prevent reflux of semen into bladder during ejaculation)	
	4. Initiation – remains unsettled, pelvic floor muscle relaxation initiates. Perineal muscles and EUS can be contracted voluntarily for prolonged periods. Bladder SM has intrinsic contractile activity Post urination, female urethra empties by gravity. Male expels by contraction of bulbocavernosus m	(optional/extra detail)
PROMPTS	What muscles and nerves are involved?	
SECOND QUESTION (if needed)	**List other factors that stimulate and inhibit micturition**	
POINTS REQUIRED	**1. Stimulants –** **a) Stretch/pressure** (intravesical volume > 400mls) **b) Higher centre input** **c) Parasympathetics** (eg organophosphates) **d) Sympathetic inhibiting drug**(eg a-blockers) **e)**Voluntary abdominal muscle contraction augments stream but does not initiate micturition per se	**(3 of 5 to pass)**
	2. Inhibitors **a) Parasympathetic inhibitors (atropine)** **b) Higher centres** **c) Sympathomimetics**	**(b) + one other**
PROMPTS	a) What is the effect of autonomic agents on micturition ? b) What non autonomic precipitants and inhibitors do you know?	(optional)

COMMENTS

ACEM 2008.2 PRIMARY VIVA EXAMINATION

SUBJECT: PHYSIOLOGY Thursday am Q 4

TOPIC: Transport Across Cell Membranes (inc Na-K Pump) NUMBER:

OPENING QUESTION	**Describe the structure and function of the sodium potassium ATP ase pump**	**COMMENTS**
POINTS REQUIRED	**1.** **Antiport: catalyses hydrolysis of ATP to ADP to move 3 Na out cell in exchange for 2 K in.** **Maintains electrochemical gradient ECF (Electrogenic pump 3+ out / 2+ in = net 1+ out)** and is large part of basal energy consumption - 33% energy use by cells (70% neurons) **Coupled to transport other substances** (secondary active transport) e.g.glucose in SI mucosa,	Need 3 / 5 (1 or 2, 3, 4 or 5)
	2. α and ß subunits which pass through cell membrane Both heterogeneous α subunit intracellular binding sites for Na & ATP α subunit extracellular binding sites for K & ouabain ß subunit has no binding sites Na / K Variable distribution of α 1 + 2 and ß 1+2 subunits	
	3. When Na binds to α subunit, ATP also binds. ATP is converted to ADP causing change in protein configuration extruding Na out of cell. **K then binds extracellularly dephosphorylating α subunit which returns to original configuration releasing K into cytoplasm**	
PROMPTS	**Describe the structure of the sodium potassium pump** **Describe how the sodium potassium pump works** **What are the effects of the sodium potassium pump ?**	

ACEM 2008.2 PRIMARY VIVA EXAMINATION

SUBJECT: **PHYSIOLOGY** **Thurs am Q 5**
TOPIC: **Vasopressin** _____ **NUMBER:**

OPENING QUESTION	**Describe the effects of Vasopressin**	**COMMENTS**
POINTS REQUIRED	**1 Retention of water (antidiuretic hormone) (V2 receptors)**	1 and 2 plus one other
	2 Vasoconstrictor effect (V1A receptors)	
	3 Central effect brain (area postrema) to decrease CO	
	4 Glycogenolysis in liver (V1A receptors)	
	5 Mediate increased ACTH secretion (V1B receptors)	
	6. Neurotransmitter in brain and spinal cord	
PROMPTS		
SECOND QUESTION	**How does vasopressin cause retention of water ?**	
POINTS REQUIRED	**Increases permeability of CD, acting on** V2 receptors **Insertion of protein water channels (aquaporin 2**) in luminal membranes. Water enters hypertonic interstitium Urine becomes concentrated and volume decreases Retention of water in excess of solute	Bolded
THIRD QUESTION	**What stimuli affect vasopressin secretion ?**	
POINTS REQUIRED	**1. Factors increasing vasopressin secretion** - **↑ effective osmotic pressure of plasma** - **↓ ECF volume** - Pain, emotion, "stress", exercise, standing - Nausea and vomiting - Clofibrate, carbamezepine, angiotensin 2	At least 2
	2. Factors decreasing vasopressin secretion - **↓ effective osmotic pressure of plasma** - **↑ ECF volume** - Alcohol	At least 2
PROMPTS		

COMMENTS

ACEM 2008.2 PRIMARY VIVA EXAMINATION

SUBJECT: PHYSIOLOGY Thurs pm Q 1

TOPIC: Effects of hyper/hypokalaemia on ECG _____ NUMBER: _____

OPENING QUESTION	**Please draw a normal ECG tracing, showing the durations of the major intervals**	**COMMENTS**
POINTS REQUIRED	1. **correct shape**	All
	2. **times** PR 0.16 QRS 0.12 QT 0.4	
PROMPTS		
SECOND QUESTION (if needed)	**How does the ECG change with hyperkalaemia?**	
POINTS REQUIRED	1. progression	
	2 Initial **tall peaked T waves**. Intervals normal K 7.0	At least 3
	3. later **no atrial activity, QRS broad/slurred**	
	4 **ventricular arrhythmias** then fibres eventually unexcitable, sine wave appearance	
PROMPTS		
THIRD QUESTION (if needed)	**How does it change with hypokalaemia?**	
POINTS REQUIRED	1. **long PR**, ST depression, inverted T,	Both required
	2. **U wave**	
PROMPTS		

COMMENTS

ACEM 2008.2 PRIMARY VIVA EXAMINATION

SUBJECT: PHYSIOLOGY Thurs pm Q 2

TOPIC: Carbon Dioxide Transport _____ NUMBER:

OPENING QUESTION	**How is carbon dioxide transported from the tissues to the lungs?**	**COMMENTS**
POINTS REQUIRED	1. In plasma: • **Dissolved** • **Carbamino compounds with plasma protein.** • **Hydration - H^+ buffered - HCO_3^- in plasma.** 2. In red blood cells: • Dissolved. • Formation of carbamino–Hb. • Hydration - H^+ buffered - 70% of HCO_3^- enters plasma. 3. Of the approximately 49 mL of CO_2 in each decilitre of arterial blood, 2.6 mL (5%) is dissolved, 2.6 mL (5%) is in carbamino compounds, and 43.8 mL (90%) is in HCO_3- 4. In the tissues, 3.7 mL of CO_2 per decilitre of blood is added; 0.4 mL (10%) stays in solution, 0.8 mL (20%) forms carbamino compounds, and 2.5 mL (70%) forms HCO_3^-. The pH of the blood drops from 7.40 to 7.36.	Bolded
PROMPTS	Which is the most important? Anywhere else ? (other than plasma)	
SECOND QUESTION (if needed)	**What is meant by the term 'chloride shift'?**	
POINTS REQUIRED	1. **About 70% of the HCO_3^- formed in the red cells enters the plasma in exchange for Cl^-. The exchange is called the chloride shift.** 2. This process is mediated by Band 3, a major membrane protein and is essentially complete in 1 second. 3. Note that for each CO_2 molecule added to a red cell, there is an increase of one osmotically active particle–either an HCO_3^- or a Cl^-–in the red cell. Consequently, the red cells take up water and increase in size.	Bolded
PROMPTS		

COMMENTS

ACEM 2008.2 PRIMARY VIVA EXAMINATION

SUBJECT: PHYSIOLOGY Thurs pm Q 3

TOPIC: Renal regulation of K+ excretion _____ NUMBER:

OPENING QUESTION	**Describe how the nephron handles potassium.**	**COMMENTS**
POINTS REQUIRED	**1. K+ is freely filtered at the glomerulus (~600 mEq/day).** **2. Most is reabsorbed by active transport in the proximal tubule (~560 mEq/day).** 3. K+ is then secreted by passive diffusion into the tubular fluid in the distal tubule. 4. K+ is also generally passively secreted into the tubular fluid in the collecting ducts. 5. The total K+ excretion is approximately equal to K+ intake (~90 mEq/day) and K+ balance is maintained. 6. There is no direct exchange of K+ for Na+ in the tubular fluid of the distal nephron. However reabsorption of Na+ into the tubular cell tends to promote secretion of K+ (or H+) to maintain the potential difference across the apical membrane.	Bolded + at least one other
PROMPTS		
SECOND QUESTION (if needed)	**What factors influence this?**	
POINTS REQUIRED	**1. The rate of secretion of K+ is proportional to the rate of flow of tubular fluid through the distal nephron. With rapid flow the concentration of K+ in the fluid remains lower and secretion continues.** 2. Increased delivery of Na+ to the collecting ducts promotes increased secretion of K+ (e.g. thiazide diuretics). 3. Conversely decreased delivery of Na+ to the collecting ducts promotes decreased secretion of K+. 4. Inhibition of K+ absorption in the proximal nephron (e.g. osmotic or loop diuretics) promotes excretion of K+. **5. In the distal nephron K+ and H+ compete for secretion in association with reabsorption of Na+. Therefore in acidosis when H+ excretion is increased, K+ secretion is decreased.** **6. Aldosterone increases reabsorption of Na+ in the collecting ducts and thereby promotes K+ secretion.**	At least two of the three bolded
PROMPTS		

COMMENTS

ACEM 2008.2 PRIMARY VIVA EXAMINATION

SUBJECT: PHYSIOLOGY Thurs pm Q 4

TOPIC: Skeletal muscle excitation /contraction / relaxation NUMBER:

FIRST QUESTION	**Describe the sequence of events in contraction and relaxation of skeletal muscle.**	
POINTS REQUIRED	**Steps in contraction** **(1) Discharge of motor neuron.** **(2) Release of transmitter (acetylcholine) at motor end-plate.** **(3) Binding of acetylcholine to nicotinic acetylcholine receptors.** (4) Increased Na+ and K+ conductance in end-plate membrane. **(5) Generation of end-plate potential.** **(6) Generation of action potential in muscle fibers.** (7) Inward spread of depolarization along T tubules. **(8) Release of Ca2+ from terminal cisterns of sarcoplasmic reticulum and diffusion to thick and thin filaments.** **(9) Binding of Ca2+ to troponin C, uncovering myosin-binding sites on actin.** **(10) Formation of cross-linkages between actin and myosin and sliding of thin on thick filaments, producing movement.**	Bolded at least?
	Steps in relaxation **(1) Ca2+ pumped back into sarcoplasmic reticulum.** (2) Release of Ca2+ from troponin. **(3) Cessation of interaction between actin and myosin.**	Bolded at least
PROMPTS		

SECOND QUESTION	**What is summation of contractions?**	COMMENTS
POINTS REQUIRED	1. The electrical response of a muscle fibre to repeated stimulation.	
	2. Contractile mechanism does not have a refractory period, so repeated stimulation before relaxation has occurred produces additional activation and a response added to the contraction already present.	
	3. With rapidly repeated stimulation, individual responses fuse into one continuous contraction (tetanus; tetanic contraction).	
	4. Complete tetanus: no relaxation between stimuli; tension developed ~ 4 times that of an individual twitch contraction	
	5. Incomplete tetanus: periods of incomplete relaxation between summated stimuli	
PROMPTS	Describe the response of a muscle fibre to repeated stimulation. What is a tetanic contraction?	

ACEM 2008.2 PRIMARY VIVA EXAMINATION

SUBJECT: PHYSIOLOGY Thurs pm Q 5

TOPIC: Physiology of Insulin **NUMBER:**

OPENING QUESTION	**What happens when insulin binds to an insulin receptor?**	**COMMENTS**
POINTS REQUIRED	1. Insulin receptor: tetramer - 2 α and 2 β glycosolated subunits α subunits extracellular + bind insulin; β subunits span membrane, intracellular parts have tyrosine kinase activity	
	2. Insulin binding triggers **tyrosine kinase** activity of β subunits → **autophosphorylation** of β subunits on tyrosine residues	2 of points 2 - 5
	3. → phosphorylation and de-phosphorylation of proteins	
	4. → **Effectors and secondary mediators** – Insulin receptor substrate (IRS-1);phosphoinositol 3-kinase (PI3K)	
	5. Once bound, insulin receptors aggregate in patches and are endocytosed → enter lysosomes → broken down or recycled;	
PROMPTS	What is the structure of an insulin receptor?	
SECOND QUESTION	**What are the principal actions of insulin?**	
POINTS REQUIRED	**Net effect: storage of CHO, protein and fat**	
	1. **Rapid (seconds):** ↑ transport of glucose, amino acids and K into insulin-sensitive cells	All 3
	2. **Intermediate (minutes):** stimulation of **protein synthesis** and inhibition of protein degradation; activation of **glycolytic** enzymes and glycogen synthase; **inhibition** of phosphorylase and **gluconeogenic** enzymes	
	3. **Delayed (hours):** ↑ mRNAs for **lipogenic**\other enzymes	
PROMPTS	What happens seconds, minutes and hours after insulin binds?	

COMMENTS

ACEM 2008.2 PRIMARY VIVA EXAMINATION

SUBJECT: PHYSIOLOGY **Friday AM Q1**

TOPIC: Systemic regulation of BP by Nervous system __ NUMBER:

OPENING QUESTION	**Where are Baroreceptors found in the body?**	**COMMENTS**
POINTS REQUIRED	1. **Stretch receptors in adventitia of vessel walls**, major ones found in **carotid sinus and aortic arch** to monitor arterial side of circulation.	
	2. Also "cardiopulmonary receptors" in right and left atria, and pulmonary circulation to monitor venous circulation	Both carotid sinus and aortic arch to pass
PROMPTS	Which blood vessels contain baroreceptors?	
SECOND QUESTION (if needed)	**What is the effect of vessel wall distension on a baroreceptor?**	
POINTS REQUIRED	**Stretch of vessel wall leads to increased baroreceptor discharge**, transmitted by afferents in glossopharyngeal and vagus nerves to **medulla. (vasomotor centre)** This results in release of inhibitory GABA which reduces sympathetic outflow, and excitatory effects on vagal motor neurones. Net effect is: **1. Inhibition of tonic discharge of vasoconstrictor nerves** **2. Excitation of cardiac vagal innervation** **Results in vasodilation, with decrease in BP, HR and CO.**	Bolded
PROMPTS	How does the baroreceptor respond to an increase in BP?	

ACEM 2008.2 PRIMARY VIVA EXAMINATION

SUBJECT: PHYSIOLOGY Friday AM Q 2

TOPIC: Control of Ventilation _____ NUMBER: _____

OPENING QUESTION	**What is the role of central chemoreceptors in control of ventilation?**	**COMMENTS**
POINTS REQUIRED	1. Located near ventral surface of medulla	
	2. Rise in blood CO2 increases CO2 in CSF	
	3. CSF has poor buffering capacity so pH changes rapidly	Bolded to pass
	4. Liberated H^+ ions stimulate chemoreceptors (increasing pH has reverse effect)	
	5. Efferents stimulate medullary respiratory centre to increase ventilation and return CO2 to normal.	
	6. Chronic CO2 elevation gives normal CSF pH and insensitivity	
PROMPTS	What happens in the brain when the blood CO2 level rises?	
SECOND QUESTION (if needed)	**What is the role of the peripheral chemoreceptors?**	
POINTS REQUIRED	1. Located in carotid and aortic bodies that have high blood flow	
	2. Respond mostly to decrease in O2 below 100mmHg	
	3. Impulses transmitted to respiratory centre to increase ventilation	3 of 5 to pass
	4. Responsible for all of the ventilatory response to hypoxaemia	
	5. Also responsible for small but rapid response to rise in CO2 and decrease in pH (carotid bodies)	
PROMPTS	How is hypoxaemia detected?	

COMMENTS

ACEM 2008.2 PRIMARY VIVA EXAMINATION

SUBJECT: PHYSIOLOGY Friday am Q 3

TOPIC: Renal Blood Flow _____ NUMBER: _____

OPENING QUESTION	**What is normal renal blood flow and how can it be measured?**	**COMMENTS**
POINTS REQUIRED	1. **Fick principle** (amount of a substance taken up per unit time divided by arterio-venous concentration difference)	
	2. PAH (excreted, not metabolised or stored, doesn't affect flow) is used to measure effective renal plasma flow (90% cleared) ERPF = Clearance of PAH = UV/P = 630 mL/min	2 of 3
	3. Actual renal plasma flow = ERPF/0.9 = 700 mL/min	
	4. Renal blood flow = RPF x 1/1-Hct (Hct = 0.45)	
	5. Renal blood flow = approx 1250 mL/min	
PROMPTS	What substance can be used to measure renal plasma flow?	
SECOND QUESTION (if needed)	**How do blood flow and oxygen extraction vary in different parts of the kidney?**	
POINTS REQUIRED	1. **Cortical flow is high** (5 mL/gm of tissue) and oxygen extraction is low	
	2. **Medullary blood flow is low** (2.5 mL/gm in outer cortex, 0.6 mL/gm in inner cortex) and oxygen extraction is higher (more metabolic work done)	2 of 3
	3. **Medulla is vulnerable to hypoxic damage** if flow is reduced (low flow, high oxygen usage)	
PROMPTS	How much blood flows to the renal medulla?	

COMMENTS

ACEM 2008.2 PRIMARY VIVA EXAMINATION

SUBJECT: **PHYSIOLOGY** **Friday pm Q 4**

TOPIC: **Synthesis and fate of catecholamines at synaptic junction** **NUMBER:** _____

OPENING QUESTION	**Describe the biosynthesis and storage of norepinephrine at the synaptic junction.**	**COMMENTS**
POINTS REQUIRED	1. dietary **tyrosine** mostly (some formed from phenylalanine)	
	2. tyrosine transported into catecholamine-secreting neurones by concentrating mechanism	
	3. tyrosine → **dopa** by tyrosine hydroxylase [this is the rate-limiting step & is subject to feedback inhibition by dopamine and norepinephrine] → **dopamine** by dopa decarboxylase in cytoplasm	At least 4 in correct order
	4. dopamine enters granulated vesicles → **norepinephrine** by dopamine β-hydroxylase (DBH)	
	5. norepinephrine stored bound to ATP, with protein **chromogranin A**	
PROMPTS		
SECOND QUESTION (if needed)	**How is Norepinephrine removed from the synaptic junction?**	
POINTS REQUIRED	**1. norepinephrine is removed from the synaptic junction by:** **i. binding to postsynaptic receptors** **ii. binding to presynaptic receptors** **iii. reuptake into presynaptic neurons** iv. **catabolism (MAO)**	Bolded to pass
	2. catabolism at noradrenergic nerve endings is catalysed by MAO (monoamine oxidase) and COMT (COMT mainly in liver, also at postsynaptic noradrenergic nerve endings)	
	3. norepinephrine → DOMA (3,4-dihydroxymandelic acid) & DHPG (3,4-dihydroxymandelic aldehyde) → VMA (vanillylmandelic acid) & MHPG (3-methoxy-4-hydroxyphenylglycol) by systemic COMT. These deaminated derivatives are physiologically inactive.	
PROMPTS		

COMMENTS

ACEM 2008.2 PRIMARY VIVA EXAMINATION

SUBJECT: PHYSIOLOGY Friday pm Q 5

TOPIC: Aldosterone synthesis/effects/feedback loop____ NUMBER: _____

OPENING QUESTION	**Describe the actions of Aldosterone.**	**COMMENTS**
POINTS REQUIRED	**1. increase reabsorption of Na^+ from urine** **Acts on principal cells (P cells) of collecting ducts, leading to increased amounts of Na^+ exchanged for K^+ and H^+ in renal tubules, producing a K^+ diuresis and fall in urine pH.**	Aldosterone cause retention of Na^+ in ECF leading to ECF volume expansion
	2. increase reabsorption of Na^+ from sweat, saliva and colon	
PROMPTS		
SECOND QUESTION (if needed)	**List the stimuli that increase aldosterone secretion**	
POINTS REQUIRED	**1. ACTH from pituitary**	1, 2 and two others at least
	2. renin from kidney via angiotensin II	
	3. direct stimulatory effect of rise in plasma K^+ concentration on adrenal cortex	
	4. Clinical causes: Surgery Anxiety Physical trauma Haemorrhage High K intake Low Na intake Standing Constriction of IVC in thorax 2^0 hyperaldosteronism (eg CCF, cirrhosis, nephrosis)	
PROMPTS		
THIRD QUESTION (if needed)	**Describe the feedback regulation of aldosterone secretion.**	via **renin-angiotensin** system feedback loop.
POINTS REQUIRED	1. Fall in ECF / blood volume → reflex increase in **renal nerve discharge** & **decrease in renal artery pressure**	Bolded
	2. → increase in **renin** secretion → increase in **angiotensin II** → increase in aldosterone secretion	
	3. → Na^+ & water retention → expanded ECF volume → decrease in stimulus that initiated renin secretion	
PROMPTS		

COMMENTS