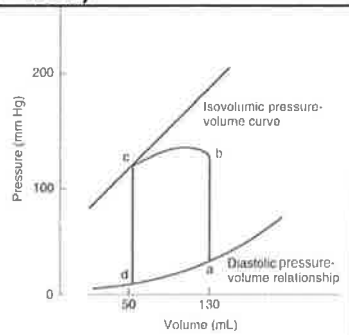
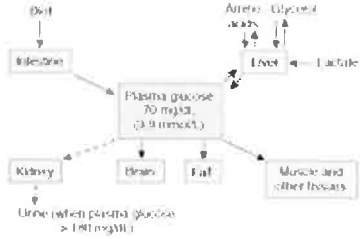
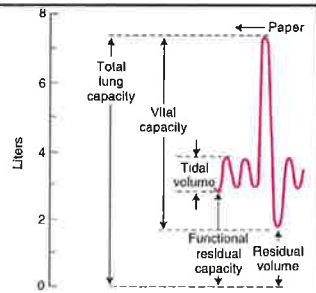
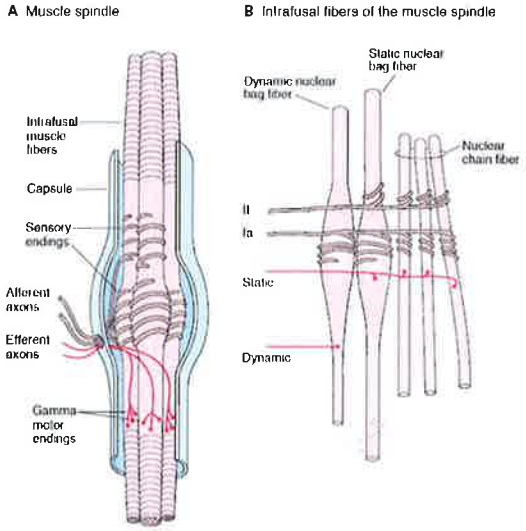
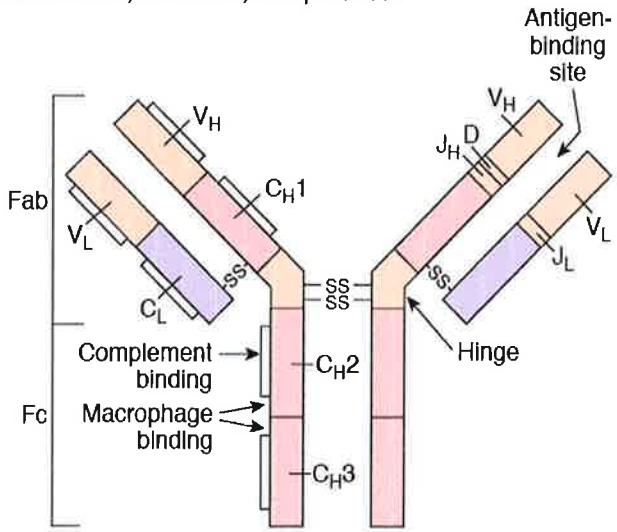
TOPIC	QUESTIONS	KNOWLEDGE (**essential in bold**)	NOTES
Question 1 LOA: 1	Please draw a pressure-volume loop for the left ventricle.	▪ a → b isovolumetric contraction ▪ b → c ventricular systole ▪ c → d isovolumetric relaxation ▪ d → a ventricular filling	**The candidate must be able to label the axes and draw a reasonable pressure-volume loop to pass this question.**
	Please relate the phases of the cardiac cycle to this pressure-volume loop.	▪ 75% along the line'd' to 'a' and closer to 'a' atrial systole (phase 1) occurs. ▪ The mitral valve closes at 'a' and the pressure rises sharply from 'a' to 'b' during isovolumetric ventricular contraction (phase 2) ▪ The aortic valve opens at 'b' and the pressure rises to a plateau and volume falls from 'b' to 'c' during ventricular ejection (phase 3) ▪ The aortic valve closes at 'c' and pressure falls from 'c' to 'd' during isovolumetric ventricular relaxation (phase 4) ▪ At 'd' the mitral valve opens and diastole commences (phase 5) from 'd' towards 'a'.	**The candidate must be able to relate three of the five phases of the cardiac cycle to the pressure-volume loop.**
Question 2 LOA: 1	1.What factors influence the rate of oxygen transfer from the alveolus into the pulmonary capillary?	Passive diffusion Determined by **Ficks law of diffusion** **Vgas α $\frac{A}{T}$. D. (P1-P2)** (Affected by surface area (A), membrane thickness(T), Difference in partial pressures gas between alveolus (P1) and Capillary(P2), and diffusion constant(D) D α $\frac{\text{gas solubility}}{\sqrt{\text{Molecular weight gas}}}$	Need to know the **basic Fick equation** to pass.
	2. How do we measure diffusion capacity?	**Carbon monoxide** is used for measurement because **its uptake is diffusion limited**(not depend on amount blood available only on diffusion properties bld-gas barrier) (single breath method test can be used)	As bonus would need to explain why this is so – ie because the CO is so avidly taken up by Hb that the concentration gradient across the membrane never reduces, so membrane properties define flux
Question 3 RBF LOA: 1 RBF	1.What is normal renal blood flow (L/min)?	**1.2 – 1.3 L/min** (25% of C.O.) at rest	
	2. Describe the mechanisms which determine renal blood flow.	Perfusion pressure (**systemic MAP**); **renal arterial flow** (local constriction from NA & Ang II, dilatation from Ach, PGs, dopamine); **Renal nerves** (stim of sympath → NA → decreased RBF); **Autoregulation** (in part due to direct smooth muscle contractile response to stretch of the afferent arteriole; NO; Ang II has a role at low perfusion pressures); **Regional** differences in RBF (greatest at cortex, less in inner medulla)	Must say 3 of 5



Question 4 LOA: 1 Blood glucose control (Ganong 23) 22-23, 326-332	4.1 What factors determine blood glucose level? (Prompt: what are the broad principles [rather than specifics?]) 4.2 How does exercise affect glucose levels? **PROMPT**: By what mechanism?	4.1 Balance between glucose entering & leaving bloodstream • dietary intake • entry into muscle, adipose tissue, other organs • glucostatic activity of the liver (GNG, glycogenesis, glycogenolysis) 4.2 Increased entry of glucose into skeletal muscle • **insulin-independent incr in GLUT 4 transporters in muscle cell membranes** • persists for several hours • regular exercise can -> prolonged incr in insulin sens Exercise in T1DM can ppt hypo also cos abs of injected insulin more rapid during exercise	4.1 All three (**intake, uptake, hepatic**) Hepatic GNG acceptable if only mention 1 other mech ?  4.2 Bold
Question 5 Pain and its Modulation LOA: 2	5.1 Describe how pain is transmitted from the periphery to the brain 5.2 How can acute pain be modulated? 5.3 What sites do opioid peptides act on?	a. sense organ = **naked nerve endings** b. transmission via 2 fibre types - small, fast myelinated **A-delta fibres** - large slow unmyelinated **C fibres** c. spinal cord: both fibre groups end in **dorsal horn of spinal cord ("gate")** - A-delta fibres on neurons in laminas 1&4 -C fibres on laminas 1&2 d. from spinal cord to brain via **ventrolateral system – second order)** (including lateral spinothalamic tract) to **thalamus** and then third order neurons on to **cerebral cortex** a. "gate theory" : eg stimulation of large touch/pressure afferents causes inhibition of pain pathways in dorsal horn of spinal cord b. Stress-induced analgesia c. Drugs (eg opioids) d. Higher centre interpretation a. receptors in afferent nerve fibres b. dorsal horn region of spinal cord c. periaqueductal grey matter in brain	Must mention dorsal horn of spinal cord and at least 3 others of bold to pass Must get 'gate theory' + 1 other Supplementary Question if answers above

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Control of Blood Pressure LOA: 1	1.1 How is blood pressure maintained in the setting of acute blood loss?	1. <u>seconds/minutes</u> - **baroreceptors** (increased discharge with stretch, afferent nerve fibres pass to vasomotor area of medulla which in turn inhibits tonic discharge of vasoconstrictor nerves leading to drop in BP) - **chemoreceptors** (stimulation leads to peripheral vasoconstriction and rise in BP) - CNS ischaemic receptors 2. <u>minutes/hours</u> - **renin-anglotensin system** - blood volume changes - fluid shift through capillaries 3. <u>Longer term</u> - renal compensation via aldosterone - blood volume changes - salt intake	**Bold to pass + must understand baroreceptors**
	1.2 What other factors influence the vasomotor centre?	**Direct stimulation** - CO_2 , hypoxia **Excitatory inputs** - from cortex via hypothalamus - from pain pathways and muscles - chemoreceptors (carotid & aortic) **Inhibitory inputs** - from cortex via hypothalamus - from lungs - from baroreceptors	**Must get 2 of 3 bold**
Question 2 Lung Volumes LOA: 1	Please draw and label a diagram showing a spirometer tracing of static lung volumes.	▪ Tidal volume 500 mL ▪ Functional residual capacity 3L ▪ Residual volume 1.5-2.0 L ▪ Vital capacity 5.5-6L ▪ Total lung capacity 7-8 L 	**The candidate must be able to label the axes, draw a reasonable spirometer tracing and indicate three of the five major volumes.**
	What is residual volume and state a method or methods of measuring this volume?	▪ The residual volume is the volume of gas left in the lung after a maximal expiration. ▪ Residual volume may be measured by: o Helium dilution technique; o Body plethysmography; o Nitrogen washout and measurement. ▪ Helium dilution and nitrogen washout measure only the ventilated residual volume. The body plethysmograph measures the total volume of gas in the lung, including any that is trapped behind closed airways.	**The candidate must be able to provide a satisfactory definition.**

		▪ In young normal subjects, these volumes are virtually the same, but in patients with lung disease, the ventilated volume may be considerably less than the total volume because of gas trapped behind obstructed airways.	
Question 3 Renin secretion LOA: 1	1.What physiological factors are involved in regulating renin secretion? 2. What conditions increase renin secretion?	1. **Intrarenal baroreceptors**- An increase of afferent arteriolar pressure at the JG cells causes a decrease in rennin secretion (and vice versa) 2. **Amount of Na and Cl** entering the distal tubules in the macula densa cells(increase in NaCl causes a decrease in rennin secretion (? NO mediated)) 3. Plasma K level (probably thru NaCl effect) 4. Angiotensin II/Vasopressin (inhibitory) **5. Increase in sympathetic Nervous system** 6. Catecholamines and norepinephrine 7. Prostaglandins Sodium depletion Dehydration Diuretics Cardiac failure Hypotension Cirrhosis Haemorrhage Constriction renal Artery Upright position Constriction of aorta Various psychological stimuli	1-4 inhibit rennin secretion 5-7 stimulate renin secretion **3 conditions to pass**
Question 4 Stretch rflx LOA: 2	1. Describe or draw the components of a muscle spindle. 2. Describe the sequence of events involved in producing a stretch reflex.	**In parallel intrafusal muscle fibers** (3 types – dynamic nuclear bag, static nuclear bag and nuclear chain); **sensory nerve endings** (Group Ia afferent to all and efferent axons, Group II to nuclear chain and static nuclear bag); dynamic gamma **motor nerves** to dynamic bag fibers, static gamma motor nerves (to static nuclear bag and chain fibers).  Sequence: stimulus (muscle **stretch**); muscle; sensory organ (muscle **spindle**) within the muscle body; efferent **sensory** nerve; **synapse** in spinal cord to **motor** neuron supplying same muscle. Transmitter (glutamate).	**Bold to pass** **Must mention 3 of 5 bold**

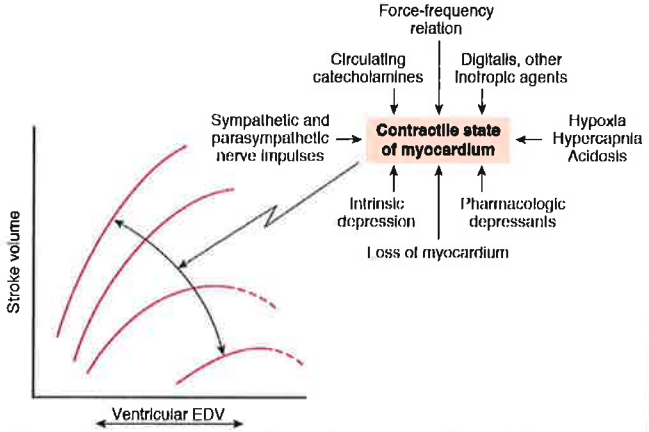
Question 5 Immunoglobulins LOA: 2	1. What are the types of immunoglobulins and what is the clinical significance of each?	1. Five Types A = Secretory D = Antigen recognition by B cells E = Anaphylaxis; release of histamine from basophils & mast cells G = Complement Activation; infections M = Complement Activation; infections, first produced	1. **3 of 5 to pass**
	2. Draw a typical Immunoglobulin Molecule and label the parts. *Prompt: Indicating the Variable region on their diagram; what is the significance of this region?*	 	2. **Bold to pass** **Light Chain** **Heavy Chain** Fab = Antigen Binding Fc = Effector Portion Hinge V = Variable Region

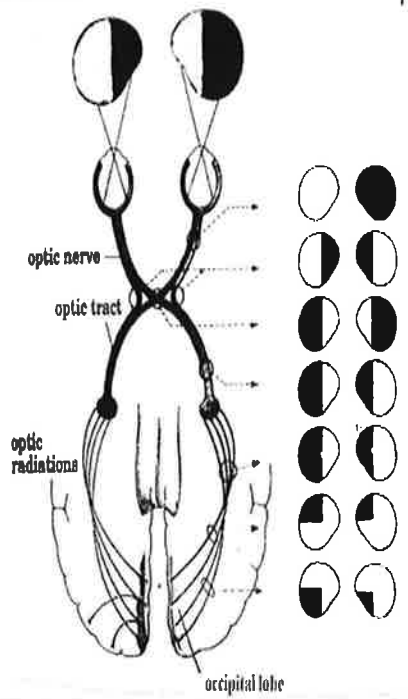
	BONUS
	3. What are the features of innate and acquired immunity?
		Innate immunity
		• triggered by cellular receptors (eg TLRs = "Toll-like Receptors")
		• bind molecular sequences common on MOs (not in eukaryotic cells)
		• activate defence mechanisms (interferons, phagocytosis, production of antibacterial peptides, complement activation, proteolytic cascades)
		• important in early response to infection
		Acquired immunity
		• T lymphocytes
		  ○ Cell-bound receptors related to antibody molecules
		  ○ APCs (Antigen Presenting Cells), MHC (Major Histocompatibility Complex) & HLAs (Human Leukocyte Antigens)
		  ○ encounter cognate antigen
		  ○ T cells proliferate & produce cytokines
		  ○ orchestrate immune response, including
		• B lymphocytes
		  ○ form clones to produce Abs
		• Memory cells
		  ○ small numbers of lymphocytes persist
		  ○ second exposure to same Ag provokes prompt & magnified immune attack	

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: LOA: 1	1.1 Please draw and label the intervals and segments of a normal ECG including times? 1.2 What electrophysiological event occurs during these periods?	**P wave, PR interval, QRS complex, ST segment, T wave (** U wave optional) and **QT segment** **PR interval: 0.12-0.2 sec. Atrial depolarisation and conduction through AV node** **QRS duration: 0.08 – 0.12 sec. Ventricular depolarisation** and atrial repolarisation. **QT interval: 0.40-0.43 sec. Ventricular depolarisation plus ventricular repolarisation** **ST interval** (QT minus QRS) **0.32 sec. Ventricular repolarisation**	Successfully draw an ECG tracing and label all of it + correctly identify the duration of 2 of the 4 intervals to pass 3 of 4 events
Question 2 [Pulmonary vascular resistance] LOA: 1	**2.1** What two mechanisms allow pulm vasc resistance to fall? (such as during exercise) **2.2** What other influences are there on pulm vasc resistance?	a. '**Recruitment**' of normally closed (non perfused) pulm capillaries b. '**Distension**' at higher vasc pressures, from near-flat to circular cross-section capillaries a. **Lung volume**: *when low*, pulm vasc resistance increased, due to smooth muscle and elastic tissue contraction: *when high*, again rises due to capil stretching and reduction in calibre b. **Hypoxia**: *increases* pulm vasc resistance from pulm vasoconstriction c. **Drugs**: *increased* by serotonin, histamine, norepi (contract vessel smooth muscle). : *decreased* by acetyl choline and isoprenaline (isoproterenolol)	Bold to pass Lung volume + one other.
Question 3 LOA: 1	1.1 What is the normal Glomerular Filtration Rate? 1.2 What factors affect GFR? Prompt: what agents affect GFR and how?	Rate: ~125mL/min normal adult Factors: **Size** and **permeability** of capillary bed Primarily by mesangial cell contraction / relaxation [and loss of renal tissue] Agents: Increased – ANP, Dopamine, PGE2, cAMP Decreased – Endothelins, AG II, Vasopressin, Norepinephrie, PAF, Platelet-derived growth factor, TxA2, PGF2, Leukotrienes C4 & D4, histamine. **Hydrostatic and oncotic** pressure gradients. Renal blood flow, Systemic BP (esp below auto-reg range), afferent and efferent arteriolar constriction Ureteral obstruction, oedema of kidney, changes in plasma proteins (dehydration hypoproteinaemia), changes in capillary permeability	100-150 3 of 4 Bold

Question 4 Withdrawal LOA: 2	Describe the withdrawal reflex. (Prompt – what are the components?)	A **polysynaptic** reflex occurring in response to a painful stimulus to skin/subcut tissue and muscle. **Survival/protective** basis. A pre-potent reflex (takes priority of all other concurrent reflex activity) The "**crossed**" response is flexor muscle contraction and extensor muscle inhibition, so the body part is flexed and withdrawn from stimulus. ALSO extension of opposite limb. 'Irradiation of stimulus' up and down spinal cord results →recruitment of motor units' Reflex is enhanced by abolition of brain modulation.	Need the bold concepts to pass
Question 5 LOA: 2	Describe the metabolism and excretion of bilirubin ? What are the causes of jaundice ?	**1)** a) Bilirubin **ex breakdown of Hb**. Bound to **albumin** in circulation. b) Most dissociates in liver, **enters liver cells** as free bilirubin via organic anion transporting polypeptide (OATP), bound to cytoplasmic proteins; c) **conjugated to glucuronic acid via glucuronyl transferase** in smooth ER to form water soluble bilirubin diglucuronide; d) transported against conc gradient into bile canaliculi; **excreted in bile into intestine** Small amount of bilirubin diglucuronide escapes into blood where loosely bound to albumin and excreted in urine. Total plasma bilirubin includes free bili plus small amount conjugated bili. e) **Intestinal bacteria – convert conj bili to urobilinogen** which can be absorbed by the intestinal mucosa , reabsorbed into portal circulation; some re-excreted into bile, some enters general circulation and excreted in urine **2)** a) excess bilirubin production – **haemolysis** b) **decreased uptake bilirubin into cells** c) **disturbed intracellular protein binding or conjugation** d) **disturbed secretion of conjugated bilirubin into the bile cannaliculi** e) intra- or extra-hepatic **bile duct obstruction**. (First 3 liberate free bilirubin, latter 2 cause elevated conjugated bilirubin in plasma)	1) Conjugation + 3 more bolded processes to pass 2. Haemolysis, obstruction + 1 more

ACEM PRIMARY 2012/2 Physiology VIVA **Afternoon Session 4** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: LOA: 1	Draw or describe the Frank-Starling law as it applies to human cardiac muscle? What factors influence the FS curve?	Curve of SV against Ventricular EDV  Circulating catecholamines; inotropes (inc dig); hypoxia, hypercarbia, acidosis – (negative); pharmacological depressants; loss of myocardium (-ve); intrinsic depression; sympathetic and parasympathetic input	Draw or describe a curve and + explain 2 +ve, 2 -ve
Question 2 LOA: 1	a) What happens to normal ventilation, perfusion and the ventilation-perfusion ratio (V/Q) from top to bottom of the upright lung? b) Explain the reasons for the alveolar-arterial O_2 difference ?	a) **Both ventilation and perfusion increase** with **blood flow (perfusion) (Q) increasing more than ventilation (V)** and this results in **V/Q ratio DECREASING down the lung.** b) Normally 4 mmHg 1)Even though P Alv O_2 at apex 40 mm Hg above base, **most of blood flow (Q) comes from base where P Alv O_2 is low** → decrease in P Art O_2 2)Also **non-linear shape of O_2 dissociation curve means that addition of small amount of shunted blood with low O_2 concentration greatly decreases P O_2 of arterial blood** and units with high P O_2 have little effect on O_2concentration because curve is flat at high O_2 concentration	a) 3 of 3 bold to pass (know it all) b) 1 of 2 bold to pass OK Need to discuss both mechanisms

Question 3 [Renal compensation acidaemia] LOA: 1	**3.1** Describe how the renal tubule cells respond to metabolic acidaemia. **3.2** In metabolic acidosis, describe which buffer systems in the urine are involved that allow excretion of large amounts of H+? **3.2b** What happens to glutamine synthesis in the liver in chronic metabolic acidosis?	a. Acidaemia: renal tubule cells **secrete H+** into tubular fluid, in **exchange for Na** Secreted H+ reacts with buffers: a. **HCO3-** to form CO2 and H2O with bicarbonate absorption b. **HPO4 2-** to form H2PO4- c. **NH3** to form NH4+ a. Glutamine synthesis increased in liver, to provide kidney with additional source NH4+, as well as NH3 secretion increasing over days	Bold to pass Need two out of three bold Need to mention that **glutamine synthesis increased**
Question 4 LOA: 2	4.1 Describe the neural connections of the visual pathways? 4.2 Describe the visual field defects of nerve sectioning at optic chiasm and optic tract on the right.	1.Retina – **optic n – optic chiasm –optic tract** - lateral geniculate body (thalamus) – geniculocalcarine tract – **primary visual cortex (occipital lobe,** Brodmann 17) **(Bold to pass)** Other connections a) lat geniculate nucleus to pretectal midbrain and sup colliculus (papillary refexes, eye movement) b) to frontal cortex (refined eye movement-vergence, near point response c) optic chiasm to thalamic suprachiasmatic nucleus (endocrine and circadian responses to day/night cycle) 2. See diagram. **Both to pass**	Visual Pathway Diaphragm – looking from above, R side lesions 

Question 5 LOA: 2	5.1 Describe the ABO blood types and their inheritance.	Inheritance – Mendelian **co-dominance of A and B antigens**. Complex oligosaccharides differing in terminal sugar. A and B phenotypes may be homozygous (AA, BB) or heterozygous (AO, BO) genotypes. **O – no antigens (universal donor), anti-A and anti-B antibodies** **A – anti-B** **B – anti-A** **AB - both antigens, no antibodies (universal recipient)** Most individuals have H antigen (terminal fucose coded by H gene) A – N-acetylgalactosamine added on the H antigen B – terminal glactose added. Similar antigens common in intestinal bacteria and possibly foods so rapidly develop antibodies to those not represent in own cells.	Understand co-dominance + Bold
	5.2 Why is Group O blood is used as a 'universal donor'? Additional if quick: i)How does the Rh system differ.	**O – no antigens (universal donor), anti-A and anti-B antibodies** Rh (first described Rhesus monkeys) C, D, E and others but only D clinically important (most antigenic). Rh only present on red cells so need exposure to Rh ve blood to develop antibodies. Occurs during transfusion and mixing at child birth or other bleeding (50% sensitized by transfusion) Antibodies take time to develop so first baby OK, second Rh +ve preg carries risk of HDNB (17% of 2nd Rh+ve preg if not treated). Rh antibodies IgG cross placenta. ABO IgM and don't.	Bold - absence of antibodies in O minimizing transfusion reactions.