ACEM 2008.1 PRIMARY VIVA EXAMINATION

SUBJECT: **ANATOMY**

TOPIC: Anatomy Thurs a.m Question 1 _____ **NUMBER:** _____

OPENING QUESTION	Identify the bones of the carpus	**COMMENTS**
POINTS REQUIRED	1 scaphoid, lunate, trapezium, trapezoid, capitate, hamate triquetrum, pisiform	7 out of 8 to pass
	2	
	3	
	4	
	5	
	6	
	7	
PROMPTS		
SECOND QUESTION	Describe the attachments of the flexor retinaculum (= Transverse palmar ligament)	
POINTS REQUIRED	1 Hook of hamate + pisiform medially (= ulnar)	2 to pass
	2 Trapezium (tubercle) + scaphoid (tubercle) laterally	
	3	
	4	
	5	
	6	
PROMPTS	What forms the carpal tunnel?	
THIRD QUESTION (if needed)	What nerves pass **superficial** to the carpal tunnel?	1 to pass
POINTS REQUIRED	1 Ulnar nerve	
	2 Palmar cutaneous branch of median nerve	
	3	
	4	
	5	
	6	

ACEM 2008.1 PRIMARY VIVA EXAMINATION

SUBJECT: **ANATOMY**

TOPIC: **MODEL ; Muscles of vocalisation / Nerve supply** **NUMBER:** **Thurs am/ Q2 _____**

OPENING QUESTION	On this model identify the structures of the larynx and upper airway.	**COMMENTS**
POINTS REQUIRED	1 Thyroid cartilage	**Identify 6 to pass**
	2 Cricoid cartilage	
	3 Cricothyroid membrane	
	4 Cricothyroid muscle	
	5 Epiglottis	
	6 Vocal cords; vocal folds	
	7 Aryepiglottic folds; vallecula	
PROMPTS		
SECOND QUESTION (if needed)	Name the muscles of vocalisation.	
POINTS REQUIRED	1 Cricothyroid ; Anterolat cricoid to inf margin and inf horn of thyroid. (Ext laryngeal nn)	**** to pass**
	2 Thyroarytenoid ; Lower ½ post angle of thyroid laminae and cricothyroid lig to anterolat arytenoid (Inferior laryngeal nn)	
	3 Post cricoarytenoid ; Post surface of cricoid lamina to vocal process of arytenoid. (inf lary nn)	
	4 Lat cricoarytenoid ; Arch of cricoid to vocal process of arytenoid. (inf lary nn)	
	5 Transverse and oblique arytenoids ; One arytenoid cart to contralat arytenoid (inf lary nn)	
	6 Vocalis ; Lat surface vocal process of arytenoid cart to ipsilat vocal lig	
PROMPTS		
THIRD QUESTION (if needed)	Please describe the nerve supply to the intrinsic laryngeal mm (mm of vocalisation)	
POINTS REQUIRED	1 All except cricothyroid supplied by recurrent laryngeal nn. **	
	2 Recurrent laryngeal nn is a branch of CN X	** main points
	3 Cricothyroid supplied by external laryngeal nn. **	
	4 External laryngeal nn is one of the 2 terminal branches of the superior laryngeal nn	
PROMPTS		

COMMENTS

ACEM 2008.1 PRIMARY VIVA EXAMINATION

SUBJECT: **ANATOMY**

TOPIC: Photo - femoral triangle boundaries and contents **NUMBER: Thurs am Q3 ___**

OPENING QUESTION	Identify the boundaries and contents of the femoral triangle in this photo	**COMMENTS**
POINTS REQUIRED	1 Ing lig, add long and Sartorius form triangle, pectineus (med) and iliopsoas (lat) form floor	4/5 to pass
	2 fem vein, fem art and fem nerve (med to lat)	3/3 to pass
	3	
	4	
	5	
	6	
	7	
PROMPTS		
SECOND QUESTION (if needed)	Describe the branches and course of the femoral artery.	
POINTS REQUIRED	1 4 **superficial branches** in fem triangle (superf epig, superf cx iliac, superf and deep pudendal)	The 3 general points to pass
	2 Profunda femoris ("**deep artery of thigh**"!) branches off post-lat in triangle to supply thigh, passes behind add longus. Gives med and lat cx fem arteries. Med cx fem supplies NOF	
	3 Fem artery continues down thigh deep to Sartorius and pass through adductor canal and becomes popliteal art at adductor hiatus	
PROMPTS		
THIRD QUESTION (if needed)		
POINTS REQUIRED	1	
	2	
	3	
PROMPTS		

COMMENTS

ACEM 2008.1 PRIMARY VIVA EXAMINATION

SUBJECT: **ANATOMY**

TOPIC:XR AP Pelvis **NUMBER:** Thur am qu4

OPENING QUESTION	**Describe the major bony features seen on this Xray**	**COMMENTS**
POINTS REQUIRED	1 Ilium – crest, ASIS, AIIS, acetabulum (pt), SI jt	15 features to pass
	2 Ischium – body, ramus, tuberosity, spine,	
	3 Pubis – symphisis, inf ramus, sup ramus, tubercle, pectineal line	
	4 Sacrum – vertebral foramina, L5-S1 jt	
	5 Coccyx	
	6 Femur – head, neck, gter trochanter, lesser trochanter	
	7 Acetabulum, obturator foramen,	
PROMPTS	Which bones can you see? Where do fractures usually occur?	
SECOND QUESTION (if needed)	**Demonstrate the bony attachments of the main muscles which flex the hip**	
POINTS REQUIRED	1 **Flexors** – Iliacus – iliac crest, fossa, ala sacrum, ant SI lig to psoas maj, Psoas maj – T12-L5 vert, discs, transv proc to lesser troch fem, Psoas min – T12 – L1 to pec line, iliopect eminence Rectus femoris – AIIS, ilium) Pectineus (superior ramus of pubis) Sartorius (ASIS)	Iliopsoas and one other
	,	
PROMPTS		

COMMENTS

ACEM 2008.1 PRIMARY VIVA EXAMINATION

SUBJECT: **ANATOMY**

TOPIC: Chest Wall ___ **NUMBER:** **Thurs AM Question 5**

OPENING QUESTION	Describe the intercostal muscles	**COMMENTS**
POINTS REQUIRED	1 **External intercostal** – from tubercles of ribs posteriorly to costochondral junction (thence external intercostal membrane) run infero-anteriorly and are most active during inspiration (to increase tonus of intercostal space) and during forced inspiration	External + internal + one other to pass
	2 **Internal intercostal** – deep to and at right angles to externals from sternum to angle of ribs posteriorly (continued posteriorly as internal intercostal membrane) run infero-posteriorly and are most active during expiration (to increase tonus of intercostal space). Interosseous portions act during forced expiration whilst interchondral portion act during active inspiration	
	3 **Innermost intercostals** – essentially the deeper parts of the internals separated from them by the intercostal nerves and vessels. Occur laterally	
	4 **Subcostals** – run in the same direction as the internals, but cross 2 or 3 spaces, lower spaces	
	5 T**ransverse thoracic** – run from sternum and xiphisternum transversely to lower ribs	
	6 **Levator costarum** – from transverse processes to ribs	
PROMPTS		
SECOND QUESTION (if needed)	Describe the pattern of distribution of neurovascular structures in the thoracic wall.	Order of neurovascular and relation to rib and groove
POINTS REQUIRED	1 Enter medial most part of posterior ICS	
	2 Run between parietal pleura and internal IC membrane in the middle of the space	
	3 Near angle of ribs pass between internal and innermost IC muscles	
	4 Here in costal grooves, with nerve inferior to artery inferior to vein	
	5 Collateral branches arise here and run along superior border of rib	
	6 Vessels also have anterior supply and drainage	
PROMPTS		
THIRD QUESTION (if needed)	Describe the arterial supply of the intercostal spaces	
POINTS REQUIRED	1 Posterior intercostal arteries (branches of the supreme intercostal from the subclavian [1 & 2] and of the thoracic aorta)	
	2 Anterior intercostal arteries (branches of the internal thoracic [previously: internal mammary] – spaces 1 to 6 & musculo-phrenic – spaces 7 to 9)	

PROMPTS		

COMMENTS

ACEM 2008.1 PRIMARY VIVA EXAMINATION

SUBJECT: **ANATOMY**

TOPIC: Anatomy _____ **NUMBER:** Thurs p.m Question 1

OPENING QUESTION	Identify the bones of the **tarsus**	**COMMENTS**
POINTS REQUIRED	1 Talus (head, neck, dome, groove for FHL post, groove for tibialis posterior on plantar surface, articular surfaces for calcaneum, navicular + ankle mortise)	6 out of 7 correct to pass
	2 Calcaneum (shelf= sustentaculum, groove for FHL, site of insertion of tendo achilles, insertion of long plantar ligament on plantar surface, articular surfaces for talus + cuboid)	(Extra marks for detail)
	3 Cuboid	
	4 Navicular	
	5 Medial, middle + lateral cuneiforms	
	6	
	7	
PROMPTS		
SECOND QUESTION (if needed)	Demonstrate the attachments of the medial collateral ligament (= ‘ deltoid ligament’)	2 of the 4 parts to pass
POINTS REQUIRED	1 Posterior tibio-talar (to medial tubercle of talus)	
	2 Tibio-calcaneal (to calcaneal shelf =sustentaculum tali)	
	3 Tibio-navicular (to tuberosity of navicular)	
	4 Anterior tibio-talar	
	5	
	6	
PROMPTS		
THIRD QUESTION (if needed)	Describe the structures running immediately posterior to the medial malleolus	2 to pass - correct order from superficial to deep needed
POINTS REQUIRED	1 Tibialis posterior tendon	
	2 Posterior tibial artery	
	3 Posterior tibial nerve (lying deep to the artery)	
	4	
	5	

	6	
PROMPTS		

COMMENTS

ACEM 2008.1 PRIMARY VIVA EXAMINATION

SUBJECT: **ANATOMY**

TOPIC: MODEL: Tongue / Muscles and nerve supply **NUMBER:** **Thurs pm Q2**_____

OPENING QUESTION	On this model, identify structures that make up the floor of the mouth.	**COMMENTS**
POINTS REQUIRED	Mandible; tongue; lip; teeth; geniohyoid muscle; hyoid bone; stylohyoid muscle; genioglossus muscle	Four structures to pass
SECOND QUESTION (if needed)	**Describe the innervation of the tongue**	
POINTS REQUIRED	1 Motor ; All mm except palatoglossus innervated by CN 12 (Hypoglossal nn) Palatoglossus is actually a palatine mm, therefore supplied by pharyngeal plexus	**To pass ;** **1) Hypoglossal main motor** **2) Lingual ant 2/3 sensation** **3) Chorda tympani ant 2/3 taste**
	2 General Sensation ; (touch and temperature) ; mucosa of anterior 2/3 supplied by lingual nn (branch of CN V 3 Mandibular). Taste for ant 2/3 (EXCEPT for vallate papillae) is via chorda tympani nn (branch of CN VII). The chorda tympani joins the lingual nn and runs anteriorly in it's sheath. Posterior 1/3 of tongue and vallate papillae, BOTH general sensory to mucous membrane and taste are supplied by the lingual branch of glossopharyngeal nn (CN IX)	
	3 Twigs of internal laryngeal nn (branch of vagus) supply mostly eneral but some special sensation to a small area of tongue just anterior to epiglottis. These mostly sensory fibres also carry parasympathetic secretomotor fibres to serous glands of tongue	
	4 parasympathetic fibres from chorda tympani travel with lingual nn to submandibular and sublingual salivary glands. These nn fibres synapse in the submandibular ganglion which hangs from the lingual nn.	
THIRD QUESTION (if needed)		
POINTS REQUIRED		
	5	
	6	
PROMPTS		

COMMENTS

ACEM 2008.1 PRIMARY VIVA EXAMINATION

SUBJECT: **ANATOMY**

TOPIC: Post. abdo wall - major structures and course of ureters **NUMBER: Thurs pm Q3**

OPENING QUESTION	This is a photo of the posterior abdominal wall. Please identify the major structures.	**COMMENTS**
POINTS REQUIRED	1 Psoas, iliacus	2/2
	2 Aorta, common, ext and int iliac arteries	3/4
	3 IVC and common and ext iliac veins	2/3
	4 Bladder, Ureters	3/3
	5 Gonadal vessels	+
	6 Genitofemoral nerve	+
	7	
PROMPTS		
SECOND QUESTION (if needed)	Describe the course of the ureter from the kidney to the bladder	
POINTS REQUIRED	1 Retroperitoneal, 25-30cm long	
	2 Arise from renal pelvis, ~L1 on left and L2 on right	
	3 Continue distally parallel to tp's of lumbar spine, adherent to parietal peritoneum	
	4 Pass over pelvic brim at bifurcation of common iliac art	
	5 run on lateral wall of pelvis then at level of ischial spine curve anteromedially to enter base of bladder	
		3/5 to pass
PROMPTS		
THIRD QUESTION (if needed)	What are the common sites of ureteric narrowing?	2/3
POINTS REQUIRED	1 Pelvoureteric junction	
	2 crossing pelvic brim	
	3 vessicoureteric junction	
	4	
PROMPTS		

COMMENTS

ACEM 2008.1 PRIMARY VIVA EXAMINATION

SUBJECT: ANATOMY

TOPIC: XR AP Pelvis **NUMBER:** Thur am qu4

OPENING QUESTION	**Describe the major bony features seen on this Xray**	**COMMENTS**
POINTS REQUIRED	1 Ilium – crest, ASIS, AIIS, acetabulum (pt), SI jt	15 features to pass
	2 Ischium – body, ramus, tuberosity, spine,	
	3 Pubis – symphisis, inf ramus, sup ramus, tubercle, pectineal line	
	4 Sacrum – vertebral foramina, L5-S1 jt	
	5 Coccyx	
	6 Femur – head, neck, gter trochanter, lesser trochanter	
	7 Acetabulum, obturator foramen,	
PROMPTS	Which bones can you see? Where do fractures usually occur?	
SECOND QUESTION (if needed)	**Demonstrate the important ligament attachments of the hip joint**	2 out of 4 to pass
POINTS REQUIRED	1 Iliofemoral lig – strong, ant sup ASIS, inf intertrochanteric line	
	2 Pubofemoral – med – obturator crest pubis inf-lat to merge with capsule deep to iliofemoral lig	
	3 Ischiofemoral – post, weakest of 3, from ischial pt of acetabular rim superolat'ly to femoral neck, med to base gter troch	
	4 Lig of head of femur – from acetabular notch to fovea for lig of head femur	
PROMPTS		
THIRD QUESTION (if needed)		
POINTS REQUIRED	1	
	2	
	3	
	4	
	5	
	6	
PROMPTS		

COMMENTS

ACEM 2008.1 PRIMARY VIVA EXAMINATION

SUBJECT: **ANATOMY**

TOPIC: Cubital Fossa _____ **NUMBER: Thurs PM Qu 5** _____

OPENING QUESTION	**Describe the boundaries of the cubital fossa**	**COMMENTS**
POINTS REQUIRED	1 Superiorly – imaginary line connecting the epicondyles	4 out of 5 to pass
	2 Medially – flexor muscles of forearm arising from common flexor attachment & medial epicondyle (pronator teres)	
	3 Laterally – extensor muscles of forearm arising from the lateral epicondyle & supraepicondylar ridge (brachioradialis)	
	4 Floor – brachialis and supinator muscles of the arm & forearm	
	5 Roof – deep fascia reinforced by bicipital aponeurosis, subcutaneous tissue and skin.	
PROMPTS	Look at your own cubital fossa	
SECOND QUESTION (if needed)	**Describe the contents of the cubital fossa**	First 4 to pass (and order correct)
POINTS REQUIRED	1 Brachial artery dividing into terminal branches; radial & ulnar arteries. It lies between	
	2 Biceps brachii tendon AND	
	3 Median nerve	
	4 Radial nerve – deep between brachioradialis and brachialis, dividing into deep and superficial branches	
	5 (Veins accompanying arteries)	
	6 Venepuncture veins (median cubital and medial and lateral antebrachial)	
PROMPTS		
THIRD QUESTION (if needed)		
POINTS REQUIRED	1	
	2	
PROMPTS		

COMMENTS

ACEM 2008.1 PRIMARY VIVA EXAMINATION

SUBJECT: ANATOMY

TOPIC: Mandible, TMJ, Muscles of Mastication_____ **NUMBER: 11/4 - 1**

OPENING QUESTION	**Demonstrate the features of the mandible?**	**COMMENTS**
POINTS REQUIRED	1 Condylar process (head & neck)	5 = pass
	2 Ramus, notch	10 = 10
	3 Coronoid process	
	4 Angle	
	5 Mental & Mandibular foramen	
	6 Mental tubercle and symphasis	
	7 Alveolar process	
PROMPTS	Indicate features and ask	
SECOND QUESTION (if needed)	**Describe the features of the Temperomandibular Joint?**	
POINTS REQUIRED	1 Bones – condyle of mandible, articular tubercle & Mandibular fossa	2 = pass
	2 Disc – separates superior synovial cavity (gliding in/out) and inferior synovial cavity (hinge up/down)	Superior & inferior cavities = pass
	3 Postglenoid tubercle and Temperomandibular (lateral) ligament prevent posterior dislocation	
	4 Stylomandibular and Sphenomandibular ligaments weak.	
PROMPTS	Questions – bones, synovial cavities, ligaments	
THIRD QUESTION (if needed)	**Describe the mandibular attachments of the muscles of mastication?**	Ask only if doing well and sufficient time
POINTS REQUIRED	1 Temporalis – temporal fossa to medial coronoid & anterior ramus	2 to pass
	2 Masseter – maxillary process & zygomatic arch to angle & lateral ramus	
	3 Lateral Pterygoid – greater wing sphenoid and lateral surface Lateral Pterygoid Plate to joint capsule, disc & Pterygoid fossa on neck of mandible	
	4 Medial Pterygoid – medial surface Lateral Pterygoid Plate & tuberosity of maxilla to medial ramus below foramen	
PROMPTS	Nil	

COMMENTS Must pass questions 1 & 2 to pass overall

ACEM 2008.1 PRIMARY VIVA EXAMINATION

SUBJECT: **ANATOMY**

TOPIC: Cardiac Chambers, Arterial Supply & Venous Drainage **NUMBER: 11/4 - 2 _**

OPENING QUESTION	**Demonstrate the chambers of the heart and their borders?**	**COMMENTS**
POINTS REQUIRED	1 Left ventricle	Need to identify chambers to pass
	2 Right ventricle – ant & post interventricular grooves	
	3 Right atrium & auricle	
	4 Left atrium & auricle – coronary groove	
	5 Crux of the heart	Additional information
PROMPTS	Ask candidate to demonstrate on model	
SECOND QUESTION (if needed)	**Demonstrate the arterial supply to the heart?**	
POINTS REQUIRED	1 RCA – SA nodal – R marginal – AV nodal – post interventricular (2/3) – interventricular septal	Underlined to pass
	2 LCA – circumflex – SA nodal (40%) – L marginal – post interventricular (15%)	
	3 LCA – LAD - ant 2/3 septum – lateral diagonal	
PROMPTS	Ask main branches of RCA & LCA	
THIRD QUESTION (if needed)	**Demonstrate the venous drainage of the heart?**	
POINTS REQUIRED	1 Coronary sinus	Coronary sinus to pass
	2 Great cardiac veins	
	3 Middle cardiac veins	
	4 Small cardiac veins	
	5 L post ventricular	
	6 L marginal	
	7 Anterior cardiac	
	8 Oblique veins on the L atrium	
PROMPTS	Ask to list veins	

COMMENTS Must pass Questions 1 & 2 to pass overall

ACEM 2008.1 PRIMARY VIVA EXAMINATION

SUBJECT: ANATOMY

TOPIC: Median nerve: Course and distal supply **NUMBER: 11/4 - 3**

OPENING QUESTION	**Identify on this specimen the median nerve.**	**COMMENTS**
POINTS REQUIRED	1 Identification of median nerve (numbered **16**)	Correct = pass
PROMPTS	Indicate features of a nerve versus tendon or vessel	Prompt = no pass
SECOND QUESTION	**Describe on the course of the median nerve in the forearm, using the photograph where able?**	At least all of 2, 4 and 5 to pass
POINTS REQUIRED	1 Emerges from **cubital fossa**	
	2 Passes between two heads of **Pronator teres (PT)**	
	3 Descends deep to **Flexor digitorum superficialis (FDS) (**numbered **12)** closely attached to fascial sheath	
	4 Continues distally between **FDS** and **Flexor digitorum profundus (FDP)**	
	5 Becomes superficial at wrist, passing between tendons of FDS and **Flexor carpi radialis (FCR)** (numbered **8**); deep to **Palmaris longus (PL)** if present	
	6 Passes deep to Flexor retinaculum	
PROMPTS	If state a structure, get candidate to identify it on the specimen.	Identify **FDS** and **FCR**
THIRD QUESTION (if needed)	**What are the structures supplied by the median nerve?**	8 structures to pass
POINTS REQUIRED	1 No branches in arm	
	2 **Articular branches** to elbow joint	
	3 **Muscular branches** to PT; FCR; PL; FDS	
	4 **Anterior interosseus nerve** to PQ; FPL; ½ FDP; articular branches to wrist joint	
	5 **Palmar cutaneous branch** to skin of lateral part of palm and adjacent thenar eminence	Numbered **18**
	6 **Recurrent branch** to thumb muscles (APB; OP; FPB)	Numbered **19**
	7 **Palmar digital branches** to lumbricals 1,2 and cutaneous supply	Numbered **17**
PROMPTS	What muscles are supplied by the median nerve? Identify the numbered branches – what do they supply?	

COMMENTS Must pass questions 1 & 2 to pass overall

ACEM 2008.1 PRIMARY VIVA EXAMINATION

SUBJECT: ANATOMY

TOPIC: Knee joint: ligaments; stability _____ **NUMBER: 11/4 - 4**

OPENING QUESTION	**Demonstrate the bony features on this x-ray.**	**COMMENTS**
POINTS REQUIRED	1 Bones – **femur; tibia; fibula**	8 = pass
	2 **Patella** (sesamoid)	
	3 Tibia – **intercondylar eminence (ICE)**; posterior intercondylar area; anterior intercondylar area	
	4 Tibia - **tuberosity**	
	5 Tibia –**condyles** (lateral; medial)	
	6 Femur – **condyles** (lateral; medial)	
	7 Femur – **epicondyles** (lateral; medial)	
	8 Fibula – **head of fibula**	
PROMPTS	Indicate features and ask	
SECOND QUESTION	**Using the x-ray as a guide, describe the cruciate ligaments.**	
POINTS REQUIRED	1 **Cruciates** – **anterior (ACL)** (anterior part ICE → postero-medial lat femoral condyle) and **posterior (PCL)** (stronger; posterior part ICE → ant-lat med femoral condyle)	Both correct to pass
	2 Ligaments of fibrous capsule: **ligamentum patellae** (continuation of Quadriceps Femoris tendon → tib tuberosity); **fibular collateral (lateral) ligament** (lat epicondyle of femur → head of fib); **tibial collateral (medial) ligament** (med epicondyle of femur → medial surface of tibia); **oblique popliteal ligament** (expansion of tendon of Semimebranosis; strengthens capsule posteriorly); **arcuate popliteal ligament** also strengthens capsule posteriorly; post aspect of head of fib → ICE and post aspect of lat epicondyle of femur)	Extra if doing well
	3 Others: menisci joined anteriorly by **transverse ligament**; medial cruciate joined to PCL by **posterior menisco-femoral ligament**	
THIRD QUESTION (if needed)	What are the factors that contribute to stability of the knee joint?	If doing well and sufficient time
POINTS REQUIRED	1 Strength of **surrounding muscles** (most important): particularly Quadriceps femoris (especially lower fibres of Vastus medialis and Vastus lateralis)	
	2 Strength of **surrounding ligaments**	
	3 **Bony** structures (minor)	

No prompts.
COMMENTS Must pass questions 1 & 2 to pass overall

ACEM 2008.1 PRIMARY VIVA EXAMINATION

SUBJECT: **ANATOMY**

TOPIC: Blood supply of the Gut **NUMBER: Friday am 5**

OPENING QUESTION	**Describe the branches of the abdominal aorta that supply the gut**	**COMMENTS**
POINTS REQUIRED	1. Coeliac trunk T12	All 3 to pass
	2. SMA L1	
	3. IMA L3	
PROMPTS		
SECOND QUESTION (if needed)	**Describe the arterial supply of the stomach.**	2 of 3 branches to pass
POINTS REQUIRED	1. lesser curvature - left gastric (from coeliac trunk)	
	2. lesser curvature - right gastric (from hepatic)	
	3. posterior gastric from splenic	
	4. short gastric arteries from distsl splenic	
	5. left gastro-omental (gastro-epiploic) from splenic	
	6. greater curvature - right gastro-omental (gastro-epiploic) from gastroduodenal (from hepatic)	
PROMPTS		
THIRD QUESTION (if needed)	**Describe the arterial supply of the colon.**	
POINTS REQUIRED	1. Superior mesenteric from aorta	
	a. Ileocolic, right colic, middle colic	
	b. Marginal artery	
	2. Inferior mesenteric artery	
	a. Left colic, sigmoid arteries,	
	b. Marginal artery	
	3. Anastamosis b/w Sup and Inf colic arts	

COMMENTS

ACEM 2007.1 PRIMARY VIVA EXAMINATION. PATHOLOGY 10 April 2008 am *Candidate Number……..*

	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Q1. Hypertrophy vs hyperplasia.	What are the differences between hyperplasia and hypertrophy?	1 Hyperplasia - increase in number of cells in organ/tissue - usually resulting in increase in volume - occurs if cellular population capable of synthesising DNA thus permitting mitotic division. 2 Hypertrophy - increase in size of cells - causes increase in size of organs. 3 Hypertrophy and hyperplasia often co-exist.	Prompt: What are the differences at a cellular level Pass criteria: 2/3 to pass
	Describe the different types of hyperplasia and give an example of each.	1 Physiologic: Hormonal, Compensatory. 2 Pathological - hormonal stimulation excessive e.g. oestrogen and effect on uterus, benign prostatic hypertrophy caused by androgens - growth factors e.g. proliferation of connective tissue cells and blood vessels in aiding wound repair.	Pass criteria: Need basic classification to pass
Q 2. Pathological calcification	Please describe the 2 different forms of pathological calcification and give an example of each.	1. Dystrophic calcification – normal serum calcium - in necrotic or dying tissue 2. Metastatic calcification - normal tissue - abnormal (raised) calcium	Prompt: "What is meant by dystrophic calcification / metastatic calcification" ?
	Prompt: Please give an example(s) of dystrophic calcification, and metastatic calcification.	1. Dystrophic calcification – atherosclerosis; calcific aortic stenosis; tuberculous node 2. Metastatic calcification – nephrocalcinosis; pulmonary calcinosis; gastric mucosal	Prompt: "What type of abnormal calcification is nephrocalcinosis" ?

	"Describe the different principal pathological causes of hypercalcaemia, with some clinical examples.	1. Increased PTH secretion + bone resorption - hyperparathyroidism 2. Destruction of bone tissue – skeletal metastases, myeloma, Paget's 3. Vit-D related disorders – sarcoidosis, hypervitaminosis D 4. Renal failure – secondary hyperparathyroidism + phosphate retention	Prompt: "Hyperparathryoidism from increased PTH secretion is one example. Can you give another" ? Pass criteria: 2/4
Q3. Hepatitis A	Describe the clinical course of Hepatitis A infection.	1) Oral faecal transmission. 2) Incubation period: 2-6 weeks. 3) No carrier state or chronic hep or cause hepatocellular Ca. 4) Rarely causes fulminant hepatitis, and so the fatality rate is about 0.1%.	Pass criteria: provide 3/4 Prompt: mode of transmission.
	How do the serological markers change with time in Hep A infection?	1) IgM anti HAV appears at the onset of symptoms. 2)Faecal shedding of the virus ends as IgM titre rises (2-12 weeks). 3) IgM Ab (months) 3) Replace by IgG anti HAV (years) . Encourage graph	Encourage graph.
Q 4. Nephrotic syndrome	What are the manifestations of the nephrotic Syndrome?	1. Massive proteinuria, with the daily loss of 3.5 gm or more of protein (less in children) 2. Hypoalbuminemia, with plasma albumin levels less than 30 gm/L 3. Generalized oedema 4. Hyperlipidemia and lipiduria	Pass criteria:3 out of 4
	What are the mechanisms of the oedema?	1. Loss of colloid osmotic pressure 2. Loss of serum albumin 3. Accumulation of water and sodium in tissues 4. Due to compensatory secretion of aldosterone Mediated by - Hypovolaemia - ↑ ADH - ↑ Sympathetic system	Pass criteria: 3/4

Q5. Sickle cell disease	What is sickle cell disease?	Hereditary haemoglobinopathy	An abnormal haemoglobin HbS is formed because of a point mutation in the beta globin chain
		(Generally heterozygous (about 40% HbS) is asymptomatic unless severe hypoxia. Homozygous most haemoglobin is HbS – leads to alteration of the Hb when deoxygenated – sickling (morphological alteration), as well as red cell membrane changes)	Pass criteria: Must state it is an abnormal haemoglobin.
	What are the major clinical features of sickle cell disease?	1.Haemolytic anaemia (anaemia, reticulocytosis, hyperbilirubinaemia) 2.Vaso- occlusive complications/crises 3. Splenomegaly/dysfunction Prone to infections esp pneumococcus/haemophilus	Pass criteria: 2 minimum
	What are the major precipitants for a sickle cell crisis in a prone individual?	1.hypoxia 2. dehydration 3. Drop in pH	2 of 3 Optional depending on time

ACEM 2007.1 PRIMARY VIVA EXAMINATION. PATHOLOGY 10 April 2008 pm *Candidate Number……..*

	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Q1. Apoptosis	What is apoptosis?	1.Pathway of cell death. 2.Induced by tightly regulated intracellular programme 3. Cells that are destined to die activate enzymes that degrade the cells' own nuclear DNA and nuclear/cytoplasmic proteins. 4. The cell's plasma membrane remains intact. 5. Apoptotic cell becomes target for phagocytosis. 6. Dead cell rapidly cleared before contents leak out so this does not elicit an inflammatory reaction in the host. 7. Cell shrinks	Prompt: What are the features at a cellular level? Pass criteria: 3/6 to pass Must get no.1 and cell contents don't leak out.
	Describe the physiologic situations where apoptosis occurs.	1.Programmed destruction of cells during embryogenesis. 2. Hormone dependent involution in adult such as endometrial breakdown. 3. Cell deletion in proliferating cell populations e.g. intestinal crypt cells. 4. Death of host cells that have served their purpose e.g. neutrophils in acute inflammation. 5. Elimination of potentially harmful self reactive lymphocytes. 6. Cell death induced by cytotoxic T cells.	Pass criteria: 2/6 required
Q2. Angiogenesis	Describe how angiogenesis occurs.	1)Mobilisation of Endothelial precursor cells (EPC) from the bone marrow & from pre-existing vessels. 2)EPC migrate to a site of injury or tumour growth. 3) EPC differentiate & form a mature network by linking with existing vessels. 4)Stabilisation: Endothelial cells from pre-existing vessels become motile & proliferate to form capillary sprouts. 5)Vessels mature involving pericytes & smooth muscle cells to form periendothelial layer.	Pass criteria: underlined

	Factors: VEFG Angioproteins 1 and 2 PDGF TGFB Receptors: $VEGFR^{-2}$ FGF^{-2} EC receptor Tie 2	1. Haemangioblast generates haemopoietic stem cells and angioblasts. Angioblasts like EPC are stored in adult bone marrow initiate antiogenesis. Participate in replacing lost endothelial cells, in vascular impant endothelization and in neovascularising ischaemic organs, cutaneous wounds and tumours. 2. Vasodilatation of pre-existing vessels, increased permeability, degradation of basement membrane, disruption of endothelial cell to cell contact, proliferation and migration towards angiogenic stimulus, and endothelial cell maturation/growth inhibition/remodelling capillary beds.	
Q3. Hepatitis C	Describe the potential outcomes of acute hepatitis C infections in adults.	1)Acute fulminant rare 2)15% resolve 3)85% chronic - >80% stable/20% cirrhosis (50% mortality) hepatocellular Ca	3 major points with most > chronic fulminant) and potential for cirrhosis/Ca
	How does the serology for Hepatitis C infection change in case of resolution?	1)Incubation period (2-26 weeks) 2)HCV-RNA (detectable for 1-3 weeks co-incident with transaminitis) 3)Anti HCV antibodies emerge. Only about 50% detectable during symptomatic acute infection. Remainder after 3-6 weeks. IgG/IgM. IgG persists.	All major points & 'window' when both virus & Ab may be – ve. Diagram encouraged.
10/4/2008 Q4	Describe the pathological features of gout	**Hyperuricaemia**	
		Acute arthritis Precipitation of urate crystals into the joint/s An event (sometimes minor trauma) releases crystals into synovial fluid Cascade occurs resulting in intense inflammatory reaction (complement activated, chemotaxis of neutrophils and macrophages with phagocytosis and activation of lysosymal enzymes, leukotrienes, prostaglandins and free radicals **Chronic arthritis and formation of tophi** which are urate deposits in synovium and periarticular areas **Nephropathy** – deposition of urate in kidney as well as formation of **uric acid stones**	Pass criteria: 3/4

	What are the causes of gout?	**Primary –** enzyme defect unknown (90%) (overproduction, underexcretion or increased excretion) - rare enzyme defect (HGPRT deficiency) **Secondary** (10%) Increased nucleic acid turnover e.g leukaemias (overproduction and excretion) Chronic renal disease (decreased excretion) Inborn error metabolism (complete HGPRT deficiency – Lesch-Nyhan syndrome) overproduction and excretion	Need primary & 1 secondary to pass.
Q5. Disseminated intravascular coagulation	What is Disseminated Intravascular Coagulation?	1 Intravascular activation of the coagulation sequence by a variety of processes and clinical conditions 2 resultant formation of micro-thrombi throughout the circulation, often uneven in distribution 3 consumption of platelets, fibrin & coagulation factors 4 coagulopathy secondary to loss of platelets, fibrin & coagulation factors 5 activation of fibrinolytic mechanisms aggravates haemorrhagic potential 6 clinical picture of tissue/organ hypoxia/infarction as well as haemorrhage 7 microangiopathic haemolytic anaemia (MAH) secondary to intravascular fibrin traumatising RBC	Pass criteria: 4 from 7 Prompt: In broad terms what occurs in DIC?

	List the major clinical disorders associated with DIC.	1.Obstetric: a. Abruption b. Retained dead fetus c. Septic abortion d. Amniotic fluid embolus e. Toxaemia 2.Infection/Sepsis a. Meningococcaemia b. Malaria c. Gram negative sepsis d. Aspergillosis e. Histoplasmosis 3.Neoplasm a. Ca pancreas, prostate, lung & stomach b. Acute promyelocytic leukaemia 4.Trauma a. Major diffuse b. Burns c. Extensive surgery c. Others a. Liver disease b. Heat stroke c. Shock d. Snakebite e. AAA	Pass criteria: suggest need two from at least 4 groups
	What are the major mechanisms which trigger Disseminated Intravascular Coagulation?	Pathological activation of the extrinsic and/or intrinsic coagulation pathways. OR impairment of clot-inhibition (RARE) 1. Release of tissue factor or thromboplastic substances into the circulation (placental origin in obstetric disorders; mucus from adenocarcinoma; endotoxins in gram negative sepsis) 2. Widespread / diffuse injury to endothelial cells (TNF is extremely important mediator), seen with heat stroke, burns, diffuse trauma, meningococcal & rickettsial infection	Underlined processes essential

ACEM 2007.1 PRIMARY VIVA EXAMINATION. PATHOLOGY 11 April 2008 am ***Candidate Number……..***

	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Q1.Scar formation	What are the phases involved in scar formation?	1. Fibroblast migration and proliferation 2. Extracellular matrix (ECM) deposition 3. Tissue remodelling	Prompt: "One phase is fibroblast migration and proliferation. Can you name another phase" ? Pass criteria 2/3
	What are the local triggers of fibroblast migration and proliferation (at the site of an injury)?	1. Growth Factors- TGF-β; PDGF; EGF; FGF Cytokines – IL-1; TNF	Prompt" Can you name a growth factor / cytokine involved" ? Pas criteria: 2 to pass
	What are the sources of these local triggers?	1. Platelets 2. Macrophages and other inflamm cells such as mast cells, eosinophils, lymphocytes 3. Endothelium	Prompt: "Which blood cells or constituents are involved. Platelets are one example. Can you give another" ? Pass criteria: 2 to pass.
Q2. Embolism	What clinical conditions may cause fat embolism?	1. (Microscopic) fat globules travelling in the circulation. 2. Long bone # 3. Soft tissue trauma/burns –rare 4. Very common with severe skeletal injury but rarely (<10%) of clinical significance	Pass criteria: 2 to pass.
	What is the pathogenesis of fat embolism syndrome?	1. Mechanical obstruction of microvasculature (lungs & brain): fat globules/aggregated platelet and RBCs. 2. Biochemical injury: FFAs from fat globules > endothelial injury, platelet activation & mediator release.	Main 2 points to pass
	What are the potential clinical sequelae of fat embolism?	1. Asymptomatic (Majority) 2. Neurological: altered LOC. 3. Pulmonary: Inc RR, SOB, hypoxia. 4. Haem: thrombocytopenia & anaemia.	2/4 to pass
Q3. Clostridial infections	Name some clostridial diseases and causative organisms.	1) Tetanus (lockjaw) – Clostridium tetani 2) Botulism (paralytic food poisoning) – Clostridium botulinum 3) Gas gangrene, necrotizing cellulitis – Clostridium perfringens, C. septicum 4) Pseudomembranous colitis – Clostridium difficile	Pass: Require 2 out of 4

	What is the pathogenesis of gas gangrene (C. perfringens, C. septicum)	Release enzymes – hyaluronidase; collagenase Virulence factors – TOXINS <u>α-toxin</u> - multiple actions - phospholipase C: degrades membranes; muscle; RBC - release phospholipid derivatives: ITP; prostaglandins - these cause derangement in cell metabolism and cell death	At least 2 & α-toxin
	Optional		
Q4. Peptic ulcer disease.			
	By what mechanisms may Helicobacter pylori cause peptic ulcers?	*1.H. pylori* secretes **<u>urease</u>**, which generates free ammonia; and a **<u>protease</u>** which breaks down glycoproteins in the gastric mucosa. 2. *H.* pylori makes **<u>phospholipases</u>** →damage surface epithelial cells glycoprotein complexes. *3.H.* pylori enhances gastric secretion and impairs duodenal bicarbonate secretion. This enhances metaplasia. 4. Several H. pylori proteins are immunogenic→evokes **<u>strong immune response</u>** in the mucosa. Activated T and B cells are both seen in chronic gastritis caused by *H. pylori.* 5. Thrombotic occlusion of surface capillaries is promoted by a **<u>bacterial platelet activating factor.</u>** 6. Other antigens (including **<u>lipopolysaccharide</u>**) recruit inflammatory cells to the mucosa. 7. Damage to the mucosa is thought to permit **<u>leakage of tissue nutrients</u>** into the surface microenvironment, thereby sustaining the bacillus.	**Prompt:** What does *H. pylori* produce which can help cause ulceration? Pass criteria: need to say it involves immunogenic response.
	What complications may arise from peptic ulcer disease?	1. **<u>Bleeding</u>** (15-20%of patients), →25% of ulcer deaths 2. **<u>Perforation</u>** ~ 5% of patients →2/3 of ulcer deaths 3. **<u>Obstruction</u>** from oedema and or scarring ~ 2% of patients Mostly due to pyloric channel ulcers Rarely causes complete obstruction with intractable vomiting & incapacitating, crampy abdominal pain	Pass criteria: 2/3
Q5. Pre-eclampsia	What are the proposed pathogenesis and consequences of pre-eclampia?	<u>Placental ischaemia</u> is the key feature leading to 1.Reduction in PGI2, PGE2, 2.Inc renin/angiotensin II, 3.Inc thromboxane and endothelial dysfunction, 4.Resulting in systemic hypertension & DIC.	Pass criteria: placental ischaemia & 1 other point.

	Describe the clinical course of pre-eclampsia.	Usually starts after 32 weeks gestation, charcaterised by 1.Hypertension, oedema and proteinuria. 2.Headache and visual disturbances are common. 3.Ecclampsia is characterised by convulsion and coma.	Need 2/3 to pass.
	Describe the morphological changes in the placenta.	1. Placental infarcts, 2. retroplacental haematoms, 3. villous ischaemia, 4. prominent syncytial knots, 5. thickened trophoblastic basement membrane, 6. villous hypovascularity, 7. fibrinoid necrosis , 8. intramural lipid deposition.	Needs to give 3 to pass.

ACEM PRIMARY 2008/2 ANATOMY VIVA Day 1 **Session (am/)** ***Candidate Number*……** **AGREED MARK…….……**

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1:	Demonstrate the bony features on these xrays	Elbow joint Distal Humerus: Med and lat epicondyles*, trochlea and capitulum*, olecranon fossa Prox. Radius: Radial head* and neck, prox. radio-ulnar joint, biceps tuberosity Prox. Ulna: Coronoid process, trochlear notch, olecranon*	At least 5 to pas
	Indicate the common extensor origin and name the muscles that arise from it.	Lat. Epicondyle*: Ext. carpi radialis brevis Ext. digitorum Ext. digiti minimi Ext carpi ulnaris Others that arise from lat epicondyle: anconeus and superficial head of supinator	* plus 2 of 4 muscles to pass
Question 2: Larynx	Using the model, demonstrate the main features of the larynx. Remove left side of mandible and tongue. What is the motor innervation of the muscles of the larynx	1) From epiglottis to inf border of cricoid 2) Cartilages(9). Thyroid: sup/inf horns/oblique line/laryngeal prominence/laminae/thyroid notch. Cricoid: cricothyroid joint>allows change in length of vocal cords. Epiglottis. Cunieform and Corniculate(paired). Arytenoid(paired) 3) Membranes: Thyrohyoid and median cricothyroid 4) Ligaments: Cricotracheal/med and lat cricothyroid. Aryepiglottic/thyroepiglottic. Med and lat thyrohyoid Extrinsic: Infrahyoid/suprahyoid/stylopharyngeus. CNX Recurrent laryngeal > sup and inf laryngeal Intrinsic: CNX > rec. laryngeal supplies all except cricothyroid supplied by ext branch of sup. Laryngeal. Principal adductors: lat. cricoarytenoid(plus trans and oblique arytenoids) Principal abductors: post cricoarytenoid	Need all to pass Thyroid cartilage Cricoid cartilage Epiglottis Cricothyroid membrane Location of the arytenoids To pass: Recurrent laryngeal nerve

		Tensor: cricothyroid Relaxer: Thyroarytenoid Sphincters: Action of most other than post cricothyroid	
Question 3: Bone: Tibia	Describe the features of the proximal end of this bone Prompt "Demonstrate the attachments of the menisci and cruciate ligaments."	Meniscal attachments Anterior and posterior cruciate attachments Capsular margin Tibial tuberosity Median and lateral condyles Tibiofibular joint	At least 4 bony features to pass
Question 4: Photo pelvis	Please demonstrate the major anatomical structures in the photo	Aorta Common iliac vessels Internal and external iliac veins and arteries Ureters Bladder Psoas muscle	At least 8 items to pass
	Please describe the innervation of the bladder	Presynaptic sympathetic fibers (T11-L2/3) via hypogastric plexus (Excite internal urethral sphincter) Presynaptic parasympathetic fibers (Motor to detrusor and inhibitarory to internal urethral sphincter) (S2-4) via Splanchnic nerve and inferior hypogastric nerve These synapse with post synaptic neurone on or near bladder wall Inferior to pelvic line (reflex and pain) -Visceral afferent follow P/S fibers retrograde to S2-4 spinal ganglia Superior to pelvic line (pain)- Follow sympathetic fibers retrograde to T11-L2/3 Somatic to external urethral sphincter, urethra via pudendal nerve (S2-4)	To pass: Describe the effects of sympathetic and parasympathetic stimulation on the bladder
	Please identify any nerves that innervate the bladder.	Inferior/superior hypogastric plexus, left and right hypogastric nerve Splanchnic nerve	Bonus question

Question 5: Discussion	Please draw the circle of Willis. What area of the brain does each branch supply? .	2 vertebrals (from subclavian) unite to form the basilar, which in turn divides into 2 post. Cerebrals. Basilar > brainstem, cerebellum and meninges Post. Cerebral > inferior aspect of cerebrum and occipital lobe L and R internal carotids(from common carotid). Each gives rise to an ant. and middle cerebral art. Ant cerebral > medial and sup surface and frontal poles. Middle cerebral(continuation of ICA after origin of ACA) > lat surface of brain and temporal lobe	Need Verts, ICAs and Anterior, middle and posterior cerebral (as well as areas of brain supplied by the latter 3 branches) to pass.

ACEM PRIMARY 2008/2 ANATOMY VIVA Day 1 **Session (pm)** ***Candidate Number*……** **AGREED MARK……**

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: X-ray Abdo CT	1.Identify the structures present in this CT of the abdomen with contrast. 2. Which structures are retroperitoneal? 3. Demonstrate the potential spaces for fluid collection in the supine position.	Liver, spleen, kidneys, pancreas Pancreas, kidneys, aorta, IVC Hepatorenal space Splenorenal space	At least 6 to pass (1 point only for kidneys) At least 3 to pass Need both to pass
Question 2: (Day 1 pm session) Bone : Tibia and Fibula	1. Describe the proximal Tibiofibular joint (Tibia and fibula put together for candidates) 2. What structures can be damaged by direct trauma to the region of the proximal fibula? 3. Describe the consequences of injury to the Common peroneal nerve?	1. Identify proximal fibula and articular area of fibula and tibia Synovial joint, separate to knee joint, minimal movement possible 2 & 3 **Lateral collateral ligament** **Biceps femoris tendon** **Common peroneal nerve** Superficial fibular nerve – weakness of ankle eversion (and slight reduction in plantar flexion), sensory loss over lateral aspect of leg , reduced sensation over posterior aspect of leg and lateral aspect of foot ((Lateral) Sural nerve) Deep Fibular nerve (Anterior tibial) – weakness of ankle dorsiflexion (T.Anterior), sensory loss dorsum of foot and first interdigital cleft Injury to fibularis (peroneus) longus and brevis muscles– weakness of ankle eversion.	Pass 1 – Correctly identify joint surfaces, note that separate to knee joint and that no movement possible 2 To pass Common peroneal Nv and one other 3 Loss of dorsiflexion and eversion to pass
Question 3:	Identify the chambers of the heart (heart closed) Identify the valves (heart open) Demonstrate the structures of the conducting system of the heart	L+R atria*, plus auricles L+R ventricles* Aortic, pulmonary, mitral and tricuspid valves* SA node*: Ant-lat near the junction of the SVC and R atrium AV node*: Post-inf region of the inter-atrial septum, near the opening of the coronary sinus AV Bundle of His: Through the fibrous skeleton of the heart, along the membranous part of the inter ventricular septum. Divides into R +L bundles which pass on each side of the muscular IV septum	* essentials to pass *essentials to pass

Question 4 Photo neck	This is a lateral photo of the neck. Demonstrate the borders of the posterior triangle Demonstrate the borders of the anterior triangle What is this structure (12 – external carotid) - What are its branches?	 SCM, Trapezius, Clavicle Midline, SCM, Mandible Ascending pharyngeal Superior thyroid Lingual Facial Occipital Posterior auricular Superficial temporal Maxillary o If required, indicate not all are visible	 Need all 3 borders of each to pass Need all 3 borders to pass 2 branches to pass
Question 5 Discussion Dermatomes	What are the dermatomes of the upper limb? Prompt to demonstrate on own arm What is the peripheral nerve supply to the skin of the hand? (Forearm as bonus)	C 3,4: base of neck, lateral over shoulder C5: lateral arm C6: lateral forearm and thumb C7 middle and ring(or mid 3) fingers and middle post surface of limb C8: Little finger, medial hand and forearm T1: mid forearm to axilla T2: small part of arm and axilla Forearm: Post cut n of forearm, from radial > post forearm. Med cut n of forearm from medial cord of B plexus > ant and medial forearm. Lat cut n of forearm, from musculocut > lat forearm Hand: Radial > base of thumb and lateral dorsum of hand. Ulnar > ulnar 11/2 fingers and palm. Median > radial palm and 31/2 fingers inc post tips of these.	Need 5 correct to pass Need to name all 3 nerves to hand and reasonable distribution.

ACEM PRIMARY 2008/2 ANATOMY VIVA Day 2 **Session (am)** ***Candidate Number*……** **AGREED MARK……**

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: Xray Chest	Please demonstrate mediastinal borders on this X-ray	Aortic arch, SVC, Right atrium, Right ventricle, Pulmonary trunk, Left auricle, Left ventricle	Six to pass
	Please describe the surface anatomy of the heart.	Left 2nd costal cartilage, Right 3rd costal cartilage, Right 6th costal cartilage and 5th ICS in left Midclavicular line	Overall correct position to pass (allow 1 space difference for each location)
Question 2: Bone skull	Demonstrate the bones that make up the orbit.	Roof: Frontal Lateral: Frontal process of zygoma Medial: Ethmoid, Lacrimal Floor: Maxilla Posterior: Sphenoid, palatine	To pass – Frontal, zygoma, maxilla
	What are the names of these anatomical features?(pointing to sup and inf orbital fissures)	Superior and inferior orbital fissures	Both to pass
	What structures pass through the superior orbital fissure?	Sup: Ophthalmic N(V1), III, IV, VI, sympathetic fibres and ophthalmic veins.	III,IV, VI to pass
	Demonstrate the course of the infra orbital nerve and its' distribution.	Continuation of maxillary n(V2). Enters the orbit via infraorbital fissure > infraorbital groove. Exits through infraorbital foramen. Distribution to cheek, lower lid, lateral nose, upper lip and gums and antero/inf nasal septum	All of continuation of V2, exits infraorbital foramen, sensory to cheek and upper teeth to pass
Question 3: Model: Cubital fossa	Demonstrate the boundaries of the Cubital fossa	Lateral: medial border of brachioradialis Medial: Lat border of pronator teres Superior: Line between 2 epicondyles of humerus Floor: brachialis Roof: skin, deep fascia reinforced by bicipital apon.	To pass lateral, medial and floor
	Contents:	Med to lat. : median n, brachial art., biceps tendon, radial n deep to brachioradialis	To pass – median nerve, brachial artery and biceps tendon
	Describe the course of the brachial artery as it passes through the arm (pull off both upper forearm muscles from model)	From lower border of teres major(cont. of axillary art) to neck of radius. Medial to humerus > bicipital groove, post to biceps, anterior to triceps medial head. Comes to lie on brachialis as descends to cubital fossa. Median nerve crosses anteriorly. Ulnar nerve a post	To pass – continuation of axillary artery, Division over proximal radius into ulna and radial artery, median nerve crosses.

		relation in the upper arm	
Question 5:	1. What are the different parts of the diaphragm. 2. What are their attachments? 3 How is the nerve supply of the diaphragm? 4 How does contraction of the diaphragm result in ventilation of the lungs?	1. Costal muscular portion, Crural (lumbar) portion, Central tendinous portion 2. Costal portion attaches to lower 6 ribs and costal cartilages Crural portion attaches to L1-3 bodies, anterior longitudinal ligament and IV discs Central tendinous portion attaches to costal portion and inferior fibrous pericardium and falciform ligament 3. Phrenic nerves – only motor nerve to costal and crural portions, sensory to central tendon (and adjacent pleura, pericardium and peritoneum) Separate innervation of R+L sides Separate innervation of crural and costal portions Lower 6 intercostal nerves are sensory for costal portion. 4. Descent in inspiration causes increase in superior – inferior thoracic volume Diaphragmatic contractions responsible for 75% of inspiratory respiratory muscle action	To pass - name at least Tendinous and Costal portions 2 Identify costal portion attaches to lower ribs and tendinous portion. 3 Identify that phrenic nerves are only motor supply to diaphragm. (C3-5 +/-1) Bonus question
Question 5:	Describe the superficial boundaries of the popliteal fossa Using this photo demonstrate the contents? What is the distribution and supply of the common fibular nerve/	Superiorly: biceps femoris 1, semitendinosus 14 and semimembranosus 13 Inferiorly : lat 5 and med 6 heads of gastrocnemius Popliteal vessels 10&11 Small saphenous vein 15 Tibial 19 & common fibular 2 nerves Lymph nodes and lymphatics Superficial fibular nerve lateral compartment Antero lat leg and foot Deep fibular nerve ant comp and dorsum foot Skin b/w great &2nd toe	To pass – biceps, one of the semis, both heads of gastrocnemius To pass – Common fibular nerve, Tibial nerve, Popliteal artery and vein To pass – weakness of dorsiflexion and inversion

ACEM PRIMARY 2008/2 PATHOLOGY VIVA Thursday 18th September Session (am) ***Candidate Number*****…… AGREED MARK……**

TOPIC	QUESTION	KNOWLEDGE	PASS CRITERIA
1. Ischaemic Injury	1. What is the difference between ischaemic and hypoxic injury?	Ischaemic involves disruption or reduction in blood supply resulting in reduced oxygen delivery, reduced delivery of substrate and reduced removal of metabolic products Hypoxic involves reduced oxygen delivery only. I hypoxic, anaerobic (glycolytic metabolism can continue as new substrate is being delivered). As a result cellular, hence tissue injury is much more rapid in ischaemic injury.	Candidate to clearly differentiate the 2 processes
	2. Describe the morphologic intracellular changes that occur in ischaemic injury	**Reversible**; Cell swelling, ultrastructural changes including loss of microvilli and cell surface 'bleb' formation. Swelling of ER and mitochondria, Myelin figure formation, and clumping of nuclear chromatin **Irreversible**; severe mitochondrial swelling, plasma membrane damage, swelling of lysosomes	Mention of reversible & irreversible changes with examples from each
Question 2: Type 2 (Antibody mediated) hypersensitivity	1. Give some examples of Antibody-mediated (Type 2) hypersensitivity.	**1.Examples**: 1. *Transfusion reaction;2. Erythroblastosis fetalis;3. certain drug reactions; 4. Autoimmune haemolytic anaemia, thrombocytosis & agranulocytosis; 5. Myaesthenia gravis; 6. Grave's Disease; 7. Pemphigus vulgaris; 8. Glomerulonephritis (some forms); 9. vascular rejection in organ grafts*	3 to pass
	2. By what mechanisms is Type 2 hypersensitivity mediated? Prompt: More detail	**2a. Opsonisation & Complement- and Fc Receptor-mediated Phagocytosis:** Cells are coated (*opsonized*) with molecules attractive to phagocytes. *Complement* activation resulting in by-products (C3b and C4b). *Phagocytosis* results *Antibody-dependent cellular cytotoxicity (ADCC)*: no complement activation, leucocyte driven. **2b. Complement- and Fc Receptor-mediated inflammation**: Extracellular tissue inflammation - mainly antibody deposited activation of complement (by-products C5a; lesser C4a and C3a), which recruit neutrophils and monocytes. Fc receptors also bind the antibodies releasing enzymes and oxygen intermediates **2c. Antibody mediated cellular dysfunction**: antibodies against *cell-surface receptors* impair or dysregulate function ***without*** causing cell injury or inflammation	2 groups to pass
3. Tuberculosis	Describe the **pathogenesis** of tuberculosis in a previously **unexposed immunocompetent** person Prompt if doesn't mention airborne.	**Infection by M. tuberculosis airborne** • M. tuberculosis usually person to person airborne droplet spread **M tuberculosis enters alveolar macrophages and replicates** • Enters alveolar macrophages and replicates by blocking phagosome/lysosome fusion leading to bacteraemia (person generally asymptomatic or mild flu like illness **Immunity through T cell mediated delayed type hypersensitivity reaction that also causes hypersensitivity and tissue destruction- in particular granuloma formation and caseation** • About 3 weeks later T cell activation via MHC antigens on macrophages and IL-2 leading to macrophage becoming bactericidal (thru IFN-γ) This macrophage response also causes tuberculin positivity and formation of granuloma and caseation by recruiting monocytes ("epithelioid histiocytes") **Re- exposure or reactivation causes heightened immune reaction as well as tissue destruction** • Infection may be contained or may progress and may reactivate with immunosuppression from any cause	Highlighted

TOPIC	QUESTION	KNOWLEDGE	PASS CRITERIA
4. Calcific Aortic stenosis	1. What are the causes of Aortic valve stenosis?	Postinflammatory scarring (Rheumatic fever) Senile calcific Ao Stenosis Calcification of congenitally deformed valve	2/3 to pass
	2. What is calcific aortic stenosis?	Ao Stenosis most common valvular abnormality **Wear and tear** => calcification on normal or cong bicuspid valves Clinical attention in 6-7th decade in bicusid valves, 8-9th decade in prev. normal valves Heaped up **calcified masses within cusps**=> protrude through to outflow tracts. Functional valve area decreased.	Highlighted
	3. What are the consequences of calcific aortic stenosis?	**LV outflow obstruction**=> increased pressure gradient over valve. (severe when valve area 0.5-1cm^2) CO maintained by concentric **LVH**. Hypertrophied myocardium ischaemic. Impaired systolic and diastolic function. Decompensation => angina, CCF, syncope	Highlighted
5.Thyrotoxicosis	1.What is thyrotoxicosis?	Hypermetabolic state caused by elevated circulating levels of T_3 and T_4	Need to know
	2. What are the clinical features of thyrotoxicosis?	**Cardiac** – inc HR, dysrhythmias, CCF Neuromusc – tremor, prox myopathy **Ocular** – wide staring gaze, lid lag, proptosis CNS – anxiety, emotional lability, insomnia Skin – warm, flushed, inc sweating **Heat intolerance** **Thyroid storm** – fever, tachycardia, arrhyth., may be fatal if not treated promptly	Highlighted
	3. What are the main causes of thyrotoxicosis?	**Diffuse toxic hyperplasia (Graves disease)** Toxic multinodular goitre Toxic adenoma/carcinoma Neonatal from maternal Graves dis Non-hyperthyroidism – thyroiditis, etc	Highlighted + 1 other

ACEM PRIMARY 2008/2 PATHOLOGY VIVA Thursday 18th September Session (pm) ***Candidate Number*****…… AGREED MARK……**

TOPIC	QUESTION	KNOWLEDGE	PASS CRITERIA
1. Role of complement in inflammation	1. What is the complement system?	**Plasma protein system involved in immunity** against microbes. Complement proteins numbered C1-9 are present in plasma in inactive forms.	Highlighted
	2. Describe the main pathways by which complement activation occurs.	1. **Classical pathway:** involving an antigen-antibody complex 2. **Alternate pathway:** triggered by microbial surface molecules (e.g. endotoxin). No antibody involvement. 3. Lectin pathway: plasma mannose-binding lectin binds to carbohydrate on microbe All pathways result in cleavage and activation of C3 (most important and abundant complement component)	Highlighted & way activated
	3. How do activated complement products mediate acute inflammation?	1. **Vascular effects**: increased permeability; vasodilatation (via C3a, C5a mediated histamine release from mast cells) 2. Leucocyte adhesion, chemotaxis and activation: via C5a 3. Phagocytosis: C3b acts as opsonin on microbe and leads to phagocytosis 4. Cell lysis by the membrane attack complex (MAC) – composed of multiple C9 molecules	Vascular and one other
2. Local and Systemic influences on wound healing	1. Describe the factors that affect wound healing Prompt: Outline how they affect the healing process	**(Table 3-5) Local:** blood supply, denervation, local infection, FB, haematoma, mechanical stress, necrotic tissue, protection, surgical technique, tissue type **Systemic:** Age, anaemia, drugs, genetic disorders, hormones, diabetes, malignant disease, malnutrition, obesity, systemic infection, temperature, trauma, hypovolaemia, hypoxia, uraemia, vitamin deficiency (C), trace metal deficiency (Cu, Zn)	At least 3 local and 3 systemic. Must describe effect to pass.
	2. Describe the effect of an additional local/systemic factor.		If < 3 factors described in a group.
3. Malaria	1. What micro-organisms cause malaria?	Parasitic protozoa **Plasmodium falciparum**, vivax, ovale, malarie	Falciparum +1
	2. How does Plasmodium falciparum infection differ from other forms of malaria? Prompts: How does it compare clinically? By what mechanism?	All do: sporozoite→liver→merozoites formed → release & bind to RBC→ Hb hydrolysed → trophozoite→ schizont → merozoite/gametocyte *P. falciparum:* **infects RBCs of any age**, causing **clumping/rosetting** so ischemia, high cytokine production, **high level parasitemia**, severe anemia, cerebral symps, renal failure, pul oedema, death *Others*: infect only new or old RBCs, P vivax & ovale form latent hynpnozoites (relapses), low parasitemia, mild anemia, rarely splenic rupture, nephrotic synd	2/3 Highlighted and 1 clinical feature
	3. What factors can make people less susceptible to malaria?	**Inherited alterations in RBCs: HbS** trait, HbC, Duffy Ag neg Repeated exposure stimulates immune response: Ab and T lymphocytes (P falc avoids this), HLAB53	Highlighted

TOPIC	QUESTION	KNOWLEDGE	PASS CRITERIA
4. Pericarditis	1. What are the causes of acute pericarditis?	**Infectious**; **viral**, pyogenic bacteria Immune mediated(presumed); Rheumatic fever, SLE, Scleroderma, post cardiotomy. Post MI (Dressler's), Drug hypersensitivity reaction. Other; AMI, uraemia, post cardiac surgery, neoplastic, trauma, radiation	Need viral and three others
	2. What types of pericardial fluid exudate occur?	1. Serous; usually non-infectious inflammation, RF, SLE, uraemia, tumours 2. Fibrinous/serofibrinous; (most common) post MI, Dressler's, trauma, post surgery but also as in 1. 3. Purulent/suppurative; almost always bacterial invasion from local infection, lymphatic or blood seeding, or at operation 4. Haemorrhagic 5. Caseous	2/5 to pass
	3. Describe the clinical features of pericarditis	Pericardial rub (may be absent if large effusion). Pain, fever (chills and rigors if suppurative), signs of cardiac failure,	Rub, pain,fever required
Question 5: Pathogenesis of Type 1 Diabetes Mellitus	1. What is the pathogenesis of Type 1 Diabetes Mellitus	1. Genetic predisposition 2. Precipitating event 3. Autoimmune destruction of islet cells 4. Subclinical leading to overt DM	3 to pass
	2. What environmental factors may contribute to the development of Type I Diabetes Mellitus?	1. Infections (group B coxsackieviruses; mumps; measles; CMV; rubella; EBV): may induce tissue damage and inflammation, leading to the release of B-cell antigens. ***OR*** the viruses produce antigens which mimic self-antigens with the immune response cross-reacting with self-tissue.	
	3. How does genetic susceptibility contribute to the development of Type I DM?	2. Complex pattern of genetic associations: putative susceptibility genes mapped to at least 20 loci. Most important is ***class II MHC (HLA) locus → 50% of total*** genetic susceptibility: on chromosome 6p21 (HLA-D) 95% Caucasians with type I DM have HLA-DR3, DR4 or both. ***DQB1*0302 allele considered the primary determinant of genetic susceptibility***. **Non-MHC genes**: the first disease-associated non-MHC gene to be identified was *insulin*. Tandem repeats in the promoter region being associated with disease susceptibility. • **mechanism of association is unknown**: maybe the disease associated polymorphism makes the protein less functional or stable OR may influence the level of expression of insulin in the thymus, so altering negative selection of insulin-reactive T cells Another gene recently shown to be associated: encoding for the T-cell inhibitory receptor CTLA-4	

ACEM PRIMARY 2008/2 PATHOLOGY VIVA Friday 19th September Session (am) ***Candidate Number*****…… AGREED MARK……**

TOPIC	QUESTION	KNOWLEDGE	PASS CRITERIA
1: Cellular changes in inflammation	Describe the sequence of cellular events in acute inflammation Prompts: • What cells are involved in acute inflammation? • How do these cells get from the blood vessels to the inflammatory site?	**Leucocytes are the major cell type involved. In first 6-24 hours neutrophils, and monocytes/macrophages in 24-48 hours** • Leucocytes line endothelial wall – margination **First stasis of blood flow leading to increased leucocytes along endothelial wall** **Then leucocyte adhesion to endothelial wall and diapedesis or transmigration across into interstitium – extravasation** • Adhesion and transmigration and recruitment are mediated by various mediators such as histamine, PAF cytokines and various attraction molecules – variously called immunoglobulins, integrins, selectins, mucin-like glycoproteins **Then leucocytes migrate to site of injury- chemotaxis** • Chemotaxis and activation is mediated thru various bacterial products, cytokines, chemical factors, Ag-Ab complexes products of necrosis **Then leucocyte activation to enable phagocytosis and enzyme release** Phagocytosis and release of various enzymes from leucocytes	Highlighted
2. Type III immune mediated hypersensitivity	1. What is the pathogenesis of type III hypersensitivity? Prompt: Immune mediated	**Antibodies bind antigens & then induce inflammation** directly or by activating complement. The recruited leukocytes produce tissue damage by release of lysosomal enzymes and generation of toxic free radicals 3 phases (systemic diseases) a) Formation of antigen antibody complexes in circulation b) Deposition of **immune complexes** in various tissues c**) inflammatory reaction** at the site of deposition	Highlighted
	2. What are the common sites for immune complex deposition	Renal glomeruli, joints, skin, heart, serosal surfaces, small blood vessels	3 to pass
	3. Give some examples of diseases caused by Type 3 hypersensitivity	SLE, polyarteritis nodosa, post strep GN, Acute GN, reactive arthritis, serum sickness, arthus reaction	3 to pass

TOPIC	QUESTION	KNOWLEDGE	PASS CRITERIA
3. Candidiasis	1. What is the clinical spectrum of candida infection?	(Benign commensal) **Superficial mucosal infn** – mouth, vagina, oesophagus **Superficial cutaneous infn** – intertrigo, nappy rash, balanitis, folliculitis, paronychia, onychomycosis Chronic mucocutaneous (T-cell defects, endocrinopathy) **Invasive (disseminated)** – myocardial/ abscess/endocarditis, cerebral abscess/meningitis, renal/hepatic abscess, endophthalmitis, pneumonia	Highlighted – something from each category
	2. What mechanisms enable candida to cause disease? Prompt: What are the virulence factors?	1) **Phenotypic switching** to adapt rapidly to changes in host environment 2)**Adhesion to host cells** - imp. determ. of virulence –via adhesins (several types) 3) **Production of enzymes** (aspartyl proteases and catalases) degrade extracellular matrix proteins and may aid intracellular survival 4) secretion of adenosine – blocks neutrophil degranulation	1/3 Highlighted
4. Pathogenesis of atherosclerosis	1. Outline the steps involved in the pathogenesis of atherosclerosis.	Response to injury hypothesis: **1. Endothelial injury and dysfunction** 2. Lipoprotein (mainly LDL) accumulation and oxidation in vessel wall **3. Monocyte adhesion and migration into intima and transformation into foam cells and macrophages** 4. Platelet adhesion **5. Smooth muscle cell migration from media into intima** 6. Subsequent smooth muscle cell proliferation in intima **7. Enhanced lipid accumulation within intimal cells (macrophages and smooth muscle cells)**	Must have highlighted
	2. List the potential causes of endothelial injury?	**1. Hyperlipidaemia,** **2. Hypertension,** **3. Smoking** **4. Haemodynamic factors (disturbed flow patterns)** 5. Homocysteine, 6. Toxins, 7. Viruses, 8. Immune reactions	3 of highlighted and 1 other to pass
5. Pituitary Adenomas:	1. How are pituitary adenomas classified? Prompt: Name two cell types involved.	Classification based on **hormone cell-type**: prolactin cell, growth hormone cell (densely or sparsely granulated), thyroid stimulating cell, ACTH cell, gonadotroph cell (including silent and oncocytic), mixed GH-prolactin cell, Other plurihormonal cell, hormone negative.	Highlighted & 2 cell types to pass. If describe "functional" or "silent" adenomas – move to prompt
	2. What clinical syndromes may they produce?	Prolactinoma: amenorrhea, galactorrhea, loss of libido, and infertility Somatotroph (GH): gigantism or acromegaly ACTH: Cushing's syndrome Gonadotroph: local effects (headaches, visual impairment, diplopia, pituitary apoplexy), hypogonadism (lethargy, loss of libido, amenorrhoea)	

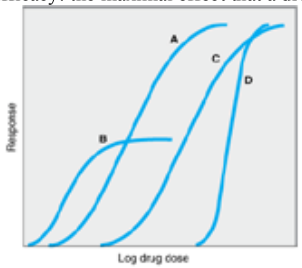
ACEM PRIMARY 2008/2 PHARMACOLOGY VIVA Day THURSDAY am *Candidate Number*…… AGREED MARK……

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: First Pass effect	1. What is first pass effect? Prompt "Can you define first pass effect?" 2. How can the first pass effect be reduced?	After absorption of an orally ingested drug, portal blood delivers drug to liver. *Metabolised in gut wall. *Metabolised in portal blood. *Metabolised by liver. *Excreted into bile Fraction of unchanged drug reaching systemic circulation may be reduced. ie. Reduces bio-availability of a drug Different route of administration IV; IM/SC; Sublingual; Transdermal; PR – Still may have some first pass metabolism, only 50% bypasses liver; Inhalational (may have first pass effect in the lung). Intrathecal	Pass: basic definition Mention 4 alternative routes
Question 2: Metronidazole	1. Describe the pharmacokinetics of metronidazole 2. What are the adverse effects of metronidazole?	(Class: Nitroimidazole antiprotozoal drug.) Pharmacokinetics: Well absorbed orally; Oral/IV/suppository (99% oral bio-availability); Metabolised in liver (can accumulate in hepatic insufficiency) and excreted in kidney; Low protein binding (10-20%); Dosage: 500mg tds or single dose of 2g for vaginitis; Half life 7.5 hours Nausea, diarrhoea, dry mouth, hairy black tongue Headache, paraesthesia, dizziness, insomnia Dysuria, dark urine, Disulfiram-like effect, hence avoid alcohol Potentiate the effect of coumarin anticoagulants, Lithium Teratogenic effect on mice, but not proven in human	Need 3 out of 6 PK Need 3 out of 6 categories
Question 3: Tricyclic antidepressants	1. What is the mechanism of action of the tricyclic antidepressants? Prompt: Name one amine? "Where does it happen? 2. Describe the toxic effects in overdose and how are they mediated?	Block amine (NAdr or Serotonin) reuptake pumps at presynaptic nerve endings prolongs duration of action of neurotransmitters at postsynaptic receptors. Most non selective Antimuscarinic. tachycardia, dry mouth, blurred vision, delirium, coma, Agitation; Urinary retention, reduced gastric motility, Respiratory depression; Neuromuscular irritability and seizures Sympathomimetic: tremor. Insomnia Sedation: additive effects alpha1-antiadrenergic – postural hypotension, Hypotension, dizziness fast sodium-channel blockade – reduced myocardial contractility, QT prolongation, cardiac arrhythmias;	Amine block, reuptake inhibitor some antimuscarinic cardiac (mix) Na channel block effects

Question 4: Sulfonylureas	1. What are the mechanisms of action of the Sulfonylureas? Prompt: How do sulphonylureas lower glucose? Describe another mechanism? 3. What are the adverse effects of sulfonylurea therapy?	Increased secretion of insulin - Bind to pancreatic B cell receptor causing increased release of Insulin -Reduced serum glucagon levels – with chronic use thought to be due to indirect inhib effects of insulin and somatostatin on a cells -Potentiation of insulin action on target tissues – increased binding of insulin to tissue receptors ?due to indirect effect of reduced glycemia or FFA levels Prolonged hypoglycemia; Alcohol intolerance – flushing; Dilutional hyponatremia (genetic predisposition) Jaundice, Leucopenia, thrombocytopenia (Chlorpropamide)	Bind to B cell; 1 of other 2 Hypoglycaemia

Question 5: Laxatives	1. Using examples, outline the mechanism of action of the various types of laxative? *Prompt: How does X work for example*	**Irritants or Stimulants** - (act early) castor oil -(act late)cascara, ***senna***, aloes (contain emodin alkaloids which are liberated after absorption from the intestine and excreted in the colon) -(prolonged action by enterohepatic circulation) phenolphthalein & biscodyl **Bulking agents** -hydrophyllic colloids, agar, psyllium seed, ***bran*** **Osmotic** -magnesium citrate and magnesium hydroxide, polyethylene glycol, sorbitol, ***lactulose*** **Stool softeners**: agents that emulsify with the stool and soften it (mineral oil, ***glycerine***, detergents such as docusate (dioctyl sodium sulphosuccinate)	3 out of the 4 mechanisms with at least 1 correct example NB –anything that distends intestine leads to peristaltic activity i.e. bulking and softening agents

ACEM PRIMARY 2008/2 PHARMACOLOGY VIVA Day THURDAY pm *Candidate Number*…… AGREED MARK……

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: Efficacy and Potency	1. What is the difference between Efficacy and Potency? Prompt: You can draw a diagram if you like?	Potency: the concentration (EC_{50}) or dose (ED_{50}) of a drug required to produce 50% of that drug's maximal effect. Efficacy: the maximal effect that a drug exerts.  	Definitions to pass **Examiner note: Drugs A and B are more potent than drugs C and D** because of the relative positions of their dose-response curves along the dose axis. **Drugs A, C, and D have equal maximal efficacy**, while all have greater maximal efficacy than drug B.
	2. What factors affect a drug's efficacy?	Affinity of receptor for drug; the drug-receptor interaction. . The route of administration, absorption, distribution through the body, and clearance from the blood or site of action	3 out of 6 to pass (NB not to do with potency)
Question 2: Cephalosporins	1. How are the cephalosporins classified and give examples? *Prompt:* *What are the different antimicrobial spectrums of the generations?*	1st-gen: (cephalexin, cephazolin, cephalothin) **very active against GPC** (pneumococci, strep, and staph). **GN org** (*E coli, K pneumoniae, & Proteus mirabilis*) **often sensitive**, but not against GN aerobes (*P aeruginosa,* indole-positive proteus, enterobacter, *Serratia marcescens,* citrobacter), & acinetobacter. 2nd-gen: (cefaclor, cefamandole, cefuroxime) **active against organisms inhibited by 1st-gen drugs,** but have **extended GN coverage.** Klebsiellae are usually sensitive. Some anaerobic 3rd-gen agents (cefotaxime, ceftazidime, ceftriaxone) have **expanded GN coverage & X BBB.** Less active against staphyl than earlier cephalosporins but are active against citrobacter, *S marcescens,* & providencia. Also **effective against β-lactamase-producing strains of haemophilus & neisseria.** Some anaerobic None above active against MRSA, enterococci or *P aeruginosa.* 4th see next column	**Know there are 4 generations, and understand principles of 3 of these** 4th-gen: (Cefepime) **extended spectrum of activity covering the majority of the enteric GNRs, including *Pseudomonas* and Enterobacter. Also active against *S aureus,* &*S pneumoniae***. More resistant to hydrolysis by chromosomal β-lactamases (eg, those produced by enterobacter).
	2. What are the adverse effects of the Cephalosporins?	**Hypersens reactions identical to penicillins**: **anaphylaxis, fever, skin rashes, nephritis, granulocytopenia, & hemolytic anemia**. Some individuals with a history of penicillin allergy may tolerate cephalosporins. **Frequency of cross-allergenicity** uncertain, probably around **5–10%.** Severe pain IMI. Thrombophlebitis IVI. **Renal toxicity: interstitial nephritis & ATN**. Cephalosporins with a methylthiotetrazole group (eg, cefamandole, cefotetan) may **cause: hypoprothrombinemia, bleeding** (preventable with Vit K_1 10 mg twice weekly) and **severe disulfiram-like reactions** with alcohol.	Essential penicillin cross reactivity + 2 others

Question 3: SSRIs	1 What is the mechanism of action of the SSRI drugs *Prompt selective serotonin reuptake inhibitors* *Prompt for delayed onset of action- possible mechanisms*) 2 What receptor/channel effects lead to the SSRI side effect profile *Prompt why are SSRIs safer than TCAs?*	i) **Amine hypothesis** – modulation of **NET + SERT** pathways by reuptake inhibition ? > serotonin response ii) Prolonged synaptic exposure to Serotonin leads to iii) prob **time frame 3-6 weeks** due to presynaptic/ post synaptic receptor / storage regulation iv) SSRIs **v HT specific** v TCA 300-7000:1 Very **specific for HT**(partic 1) receptors –therefore **serotonin syndrome/ restlessness. Minimal autonomic** NE activation + mild muscarinic / Na channel, **H1** block effects (**safety/ tolerance**). Possibly some α block (sexual dysfunction)	**General understanding** knowledge of amine hypothesis and b) delayed response c) prob alteration in pre/post synaptic **specific HT + 1 other**, Serotonin syndrome Minimal autonomic = good tolerance/ safety modulation receptors and storage
Question 4: Salbutamol	1). Describe the pharmacokinetics of salbutamol? 2). What are the pros and cons of the different routes of delivery *Prompt: MDI vs nebuliser*	1). Absorption –**complete all routes. Gut** fast, **resp tract slower**- depends on **mechanism delivery - gut 80% with Neb. 2).** Metab/elim- **50% 1st pass** (less if IV) (sulphated- inactive) liver, **rest renal/unchanged. 3). No resp metabolism. 4).** t1/2 3-6hr – prolonged if resp 1.Inhaled- Inhaler/ spacer: targeted/ low dose – minimal systemic ? local effects, co-ordination education; ii) Nebulised- less co-ord required> dose/systemic effects , noisy/frighten children- no benefit in co-ordinated patients 2.Oral- easier in v young/ disabled- longer t1/2, > SE profile, big doses, tachyphylaxis- possible increased deaths 3.IV/IM/SC – useful in asthma extremis or other indications, less 1st pass/. IV- pain/cost/staff use/high SE profile + high risk pts	**good fast absorption- all routes** **Metab 50% + renal.** **Grasp of 2 different routes** Inhaler/ spacer v Neb v IV minimum. Targetted proven effectiveness inhalers/pacers SE profile: < to > Inh v Neb v Oral v Systemic Co-ordination/delivery in extremis (age or severity) important

Question 5: Thioamides	1. How does carbimazole act in thyroid disease? 2. What are the major side effects of carbimazole? 3. How does carbimazole differ from propylthiouracil?	Metabolised to methimazole: **Major action block hormone synthesis** T3 and T4 **Inhibits thyroid peroxidase** – limits organification of iodine. Also blocks coupling of iodotyrosines Small action in blocking peripheral deiodination of T3 and T4. Slow onset as T4 may takes weeks to become depleted Rash maculopapular, pruritus – common; **B one marrow suppression**: neutropenia, agranulocytosis (reversible). Others – urticaria, arthralgia, lupus reaction, vasculitis, jaundice/hepatitis; nausea and GI, occur early **Carbimazole is a prodrug** - converted to methimazole in vivo. Methimazole is 10 times more potent **And one of the areas below** 1. PTU has greater action in inhibiting peripheral deiodination of T4 and T3 2. Propylthiouracil is strongly protein bound: preferred in pregnancy; not secreted in breast milk 3. PTU has shorter half life 1.5 vs 6 hours. PTU given qid, Carbimazole is daily 4. PTU bioavail 50-80%, vs Carb 100% Vd = TBW) 5. PTU excreted in urine as glucuronide metabolite <24 hours, carb in 48+ hours)	Bold to pass 1 side effect Bonus marks

CEM PRIMARY 2008/2 PHARMACOLOGY VIVA Day **FRIDAY am** ***Candidate Number*……** **AGREED MARK……**

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: P450 enzyme system	1. What is the role of the cytochrome P450 enzyme system? *Prompt: what does CP450 do?* 2. What is the mechanism of CP450 enzyme induction and give examples?	**Part of biotransformation system to detoxify drugs/substrates** **Acts by oxidation (phase 1 reaction)**: one molecule of oxygen is consumed per molecule of substrate Makes substrates more polar – easier to excrete or conjugate (phase2). Located on smooth endoplasmic reticulum Acts on a large number of lipophilic substrates, low specificity Relies on two enzymes: cytochrome P450, CP450 reductase (plus oxygen, NAPDH). **CP450 is a hemo-protein** – active in the oxidized -ferric state- Fe3+ Enhanced rate of synthesis - Reduced rate of degradation of CP450 enzyme Specific enzyme inducers eg: CYP/CP 450 2B1 - **barbiturates** CP 450 3A –steroids, macrolides, **anticonvulsants** CP 450 2E1 – isoniazid, chronic **ethanol** CP 450 1A1 – pollutants – aromatic hydrocarbons in tobacco smoke	Bold to pass 1 mechanism and 2 examples
Question 2: Gentamicin	1. Describe the mechanism of action of gentamicin? 2. What are the benefits of once daily dosing? *Prompt how does this improve clinical effectiveness?*	Irreversible inhibitor of protein synthesis. Passive diffusion via porin channels across outer memb, then active transport into cytoplasm by O2 dependant process; transmemebrane electrochem gradient supplies the E, transport coupled ti proton pump. Low ecf pH & anaerobic conditions inhibits transport as reduces gradient; transport enhanced by cell wall active drugs eg penicillin. Binds 30S ribosome & inhibits protein synthesis by simultaneously: 1). Inducing misreading of mRNA thus producing non toxic protein; 2) interfere with initiation complex of peptide formation; 3) cause break up of polysomes into non-functional monosomes Concentration dependant killing (at increased conc kill increased no of bacteria at a more rapid rate; post antibiotic effect (effect lasts longer than detectable serum levels); reduced toxicity (as toxicity is time & conc dependant –time above critical level will be longer with multi dose than single dose schedule); less nursing time; OPD therapy possible; convenience	Irreversible protein synth inhibitor A ribosome inhibitor Conc dependent kill + 1 other

Question 3: Phenytoin	1. Describe the pharmacokinetics of phenytoin?	Weak acid pKa 8.3; oral abs almost complete 90%, with peak serum conc 3-12hrs later. Slow release formulation also. IMI: incomplete abs with drug precipitation in the muscle, fosP OK **Highly plasma protein bound,** metabolised to inactive metabolities with urinary excretion, < 2% exc unchanged in urine. **Dose dependant kinetics**; Vd 45L/70kg. t1/2 av 24 hours (conc dependant). Therapeutic level 10-20mg/L. Drug interactions via plasma protein binding or via enz induction (CYP2C19 & CYP2C9). Alters TFT results; reduced CL neonates; foetal hydantoin syndrome	Pass: highly protein bound and dose dependant kinetics
	2. Describe the pharmacodynamics of phenytoin? *Prompt: what is the effect on action potentials?*	**Block sodium channels** & inhibits the generation of repetitive APs blocks sustained high frequency repetitive firing of APs). Preferential binding to & prolongation of the inactivated state of the Na channel (use dependant effect on Na conductance). **Other electrolyte effects** -alters K conductance; alters Ca conductance ad decreases Ca permeability, inhibits Ca influx therefore affecting neurotransmitter & hormone release; -interacts with membrane lipids ? stabilising membranes; -paradoxical excitation in some neurones; -alters membrane potentials and the conc of amino acids; affects neurotransmitters NA, Ach & GABA. High conc inhibits serotonin and NA release, promotes uptake of DA & inhibits MAO activity.	Pass: Na channel, And one other effect
Question 4: Metoclopramide	1. Describe the mechanism of action of metoclopramide? *Prompt: what receptor does it act on?* *What are the peripheral/central actions?*	**Dopamine antagonist** (D2 receptors) **Central** – via anti - nauseant and anti - emetic effect on the Chemoreceptor Trigger Zone (area postrema) **Peripheral** – blockade of GI dopamine receptors allowing cholinergic smooth muscle stimulation - increases oesophageal peristaltic amplitude - increases lower oesophageal sphincter pressure - enhances gastric emptying	Pass dopamine antagonist, peripheral & central action
	2.. List the adverse effects of metoclopramide?	Relate to central dopamine antagonist action - restlessness, drowsiness, insomnia, anxiety, agitation - **extrapyrimadal effects** – dystonias, akathisia, parkinsonian features. - risk of tardive dyskinesia with chronic use - hyperprolactinemia (galactorrhoea, gynecomastia, impotence, menstrual disorders)	Extrapyramidal + 1
Question 5: Flecainide	1. What is flecainide's mechanism of action?	**Na channel blockade** (class effect). Predominant action is to inhibit the fast, or sodium, channel which is largely responsible for the rapid upstroke of the myocardial action potential in cardiac conducting tissue **Class 1C action** - – minimal effect on the Action Potential Duration and dissociates from the Na channel with slow kinetics. (no effect on QT interval) Decrease the rate of rise (V_{max}, phase 0) of the action potential with little effect on duration.	Na channel block, class 1C
	2. Describe flecainide's pharmacokinetics. **Prompt** Usual oral dose Tambocar trade name	Well absorbed orally, half life ~ 20 hours, Peak plasma drug levels at ~ 3 hours (range 1-6 hrs), Vd ranges from 5 to 13.4 L/kg (mean 8.7 L/kg), 30% of a single oral dose (range 10 to 50%) is excreted in urine as unchanged drug – remainder by hepatic metabolism. Usual dose 100- 200 mg daily	2 things
	3. In which patients is it contraindicated?	Hypotension, LV dysfunction	Any answer

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		

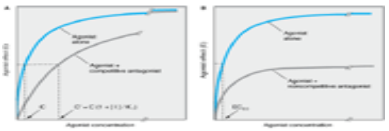
COMMENTS

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Second Messengers	In reference to drug action what is a second messenger? What steps are involved in the action of a drug via a second messenger ? (Prompt - Illustrate this with an example)	A chemical eg Ca++ or cAMP that converts receptor binding to end effect through the production of an active intracellular element. Extracellular ligand specifically detected by a cell-surface receptor. Receptor triggers the activation of a G protein located on the cytoplasmic face of the plasma membrane. Activated G protein changes the activity of an effector element (usually enzyme or ion channel) This element changes the concentration of the intracellular second messenger. Example cAMP - Gs stimulates adenylyl cyclase which converts intracellular ATP to cAMP which stimulates cAMP-dependent protein kinases. Ca, Phosphoinositides cGMP (Pass –understanding of the concept that there may be a secondary process producing drug effect and able to name at least 1 second messenger)	

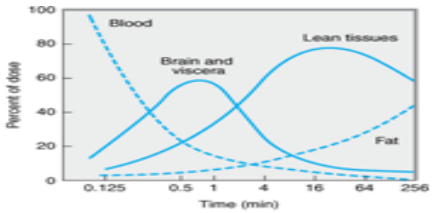
Angiotensin 2 Blockers	Describe the pharmacodynamics of therapeutic drugs that modulate the effect of angiotensin (Prompt to ACE & receptor blockers) What are the advantages of Angiotensin 2 receptor antagonists over ACE inhibitors ? (Specifically with respect to side effects)	ACE inhibitors – bind ACE reversibly preventing conversion of AI to AII. Inhibitory action on the renin-angiotensin system Stimulating action on the kallikrein-kinin system Angiotensin II inhibitors – competitive antagonists at A II receptor. As AII inhibitors do not result in production of bradykinins, there is a decreased incidence of cough and angioedema. Potentially greater effect as enzymes other than ACE can generate AII (Pass – able to describe actions and basic effects of ACE inhibitors and understanding that AII receptor antagonists and ACE inhibitors have different mechanisms.)	
LMWH	What are the pharmacodynamic differences between low molecular weight and unfractionated heparin? What are the advantages of low molecular weight heparin over unfractionated heparin?	Enoxaparin predominantly binds and inhibits factor Xa function, UFH binds to AT that inhibits factors II, IX, X Single daily or divided subcutaneous doses – facilitates patient mobility and OPD management. Routine monitoring not required (not mentioned in book) Reduced bleeding risk. Lower incidence of HITP. Improved efficacy over unfractionated heparin in ACS. Increased bioavailability (Pass – dosage differences and bleeding risk as well as factors II and IX less inhibited by LMWH (or at least that APTT is not accurate measurement of anticoagulation)	

Atropine	What is the mechanism of action of atropine ? What are the toxic effects of atropine? (Prompt - due to excessive use or abuse) What are the therapeutic uses for atropine ?	Antimuscarinic at cholinergic receptors Tachycardia, flushing, dry skin mucous, mydriasis membranes, ileus, urinary retention, acute angle glaucoma, central anticholinergic syndrome (delirium with visual hallucinations) Symptomatic bradycardias, especially when vagally mediated. OGP poisoning/ Inocybe Mushroom poisoning, drying of secretions. Adjunct to reversal of non depolarising muscle relaxants and suxamethonium administration in young infants. Antispasmodic, mydriatic. (Pass – antimuscarinic, at least 2 indications and 3 adverse effects involving 3 different body systems)	
Octreotide	Explain the rationale for the use of octreotide in upper gastrointestinal bleeding What are the pharmacokinetic differences between octreotide and somatostatin? (Supp Question – What other agents may be useful in the prevention and treatment of upper GI bleeding)	Octreotide reduces splanchnic blood flow, (? By glucagon release inhibition) therefore reduces portal venous pressure. This reduces blood loss from bleeding oesophageal varices and in some cases of severe duodenal ulcer related bleeding. Octreotide is a somatostatin analogue that has a longer half life than somatostatin (1.5hrs vs 3 min) so can be given as an IV infusion or subcutaneously. (Pass – reduces splanchnic blood flow)	

ACEM PRIMARY 1/2008 Pharmacology VIVA Afternoon Day 1 ***Candidate Number*****…… AGREED MARK……**

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Competitive vs Irreversible Antagonists	What is an antagonist ? Explain the difference between a competitive and irreversible antagonist (Illustrate this with an example)	Receptor antagonists bind to receptors but do not activate them. The primary action of antagonists is to prevent agonists from activating receptors In the presence of a fixed concentration of agonist, increasing concentrations of a reversible **competitive antagonist** progressively inhibit the agonist response; high antagonist concentrations prevent response completely. eg Propranolol and Noradrenaline **Irreversible** antagonists bind to the receptor either by forming a covalent bond with the receptor or by binding so tightly that the receptor is unavailable for binding of the agonist eg Phenoxybenzamine vs adrenaline  Changes in agonist concentration-effect curves produced by a competitive antagonist (Panel **A**) or by an irreversible antagonist (Panel **B**). In the presence of a competitive antagonist, higher concentrations of agonist are required to produce a given effect; thus the agonist concentration (C') required for a given effect in the presence of concentration [I] of an antagonist is shifted to the right, as shown. High agonist concentrations can overcome inhibition by a competitive antagonist. This is not the case with an irreversible (or noncompetitive) antagonist, which reduces the maximal effect the agonist can achieve, although it may not change its EC_{50}. Pass Be able to distinguish between competitive and irreversible antagonist	
Loop Diuretics	How does frusemide exert its action ? What are the adverse effects of frusemide? (Are any other organ systems effected ?)	Selective inhibition of NaCl reabsorption in the thick ascending loop of Henle Hypokalemic Metabolic Alkalosis Ototoxicity Hyperuricemia Hypomagnesemia . Allergy Skin rash Eosinophilia Interstitial nephritis Hyponatremia Pass – Na & loop of Henle, 4 adverse effects incl hypokalaemia & one non electrolyte	
Methylxanthine	What are the organ system effects of theophylline ? (Prompt both therapeutic and toxic)	CNS: Mild cortical arousal with increased alertness and deferral of fatigue. Bronchodilation. Nervousness and tremor. Overdose causes medullary stimulation, convulsions and death. CVS: Positive chronotropic and inotropic effects by inhibiting presynaptic adenosine receptors in sympathetic nerves and increasing catecholamine release at nerve endings. Produces tachycardia, increased cardiac output and BP. May cause arrhythmias. GIT: Stimulates gastric acid and digestive enzymes secretion. Kidney: Weak diuretic from increased glomerular filtration and reduced tubular sodium	

	How do these effects of theophylline correlate to its serum concentrations ?	reabsorption. Lung: Bronchodilation by relaxing airway smooth muscle and inhibits antigen-induced release of histamine from lung tissue. Theophylline has a narrow therapeutic window, and its therapeutic and toxic effects are related to its blood level: 5–20 mg/L: Improvement in pulmonary function. Anorexia, nausea, 15-20 mg/L: vomiting, abdominal discomfort, headache, and anxiety occur at concentrations of in some patients: >40 mg/L: Cause seizures or arrhythmias Pass –, CVS & Resp effects, narrow therapeutic window	

Thiopentone	Describe the pharmacokinetics of thiopentone What adverse effects does it cause when used as an anaesthetic induction agent ?	After IV bolus, rapidly crosses the blood-brain barrier. Plasma:brain equilibrium occurs < 1 min because of high lipid solubility. Rapidly diffuses out of the brain and highly vascular tissues, and redistributed to muscle and fat. Metabolized at rate of 12–16% per hour. <1% of the administered dose excreted unchanged by kidney.  Drops BP, SV, CO due to myocardial depressant effect and increased venous capacitance. Apnoea. Rarely precipitates porphyric crisis by inducing ALA synthase in liver Pass – 2 phase concept, hypotension	
Fluoroquinolones	What is the mechanism of action of fluoroquinolones ? What are the mechanisms of resistance to fluoroquinolones ? What are the clinical uses ciprofloxacin ?	DNA gyrase inhibitor/blocks protein production Resistance is due to one or more point mutations in the quinolone binding region of the target enzyme or to a change in the permeability of the organism. UTI Bacterial diarrhoea caused by Shigella, Salmonella, toxigenic *E coli*, Campylobacter Soft tissue, bone, joint, intra-abdominal and respiratory tract infections Treatment against multidrug-resistant organisms (pseudomonas and enterobacter) Prophylaxis and treatment against anthrax Gonococcal infection Chlamydial urethritis or cervicitis TB and atypical mycobacterial infections Eradication of meningococcal carrier state Prophylaxis in neutropenic patients Pass – DNA gyrase inhibition, 3 organ system uses	

ACEM PRIMARY 1/2008 Pharmacology VIVA Morning Day 2 ***Candidate Number*****……. AGREED MARK……**

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Bioavailability	Define the term bioavailability	Fraction of unchanged drug reaching the systemic circulation following administration by any route.	Need close approximation of defn
	What factors limit drug bioavailability following oral administration ?	(1) Extent of absorption (2) First-pass elimination (liver, gut)	Identify both factors (prompt if necessary)
	What methods of drug delivery are used to overcome bioavailability problems ?	Alternative route – sublingual, rectal, transdermal parenteral Administration pro-drug, increased dose	Give one example of an alternative route
Nitrates	What is the cellular mechanism of action of GTN ?	Denitration by glutathione S-transferase. Free nitrite ions released and form NO. NO activates guanylyl cyclise leading to increased cGMP and dephosphorylation of myosin and smooth muscle relaxation (precise mechanism unknown)	Production of NO leading to smooth muscle relaxation to pass
	How does GTN relieve angina pain ?	Venodilation leads to reduced venous return, reduce ventricular volume and reduced heart wall tension. This reduces myocardial O2 requirement.	Need to know that venodilation and reduced venous is major factor reducing myocardial o2 requirement.
	Outline the pharmacokinetics of sublingual GTN	Oral bioavailability is low due to extensive first pass hepatic metabolism by high capacity organic nitrate reductase. Rapid and efficient absorption by sublingual or intranasal routes but rapid elimination (t1/2 2-8 mins) and duration of action (15-30 mins) due to high capacity hepatic metabolism. Denitrited metabolites conjugated to to glucuronide and excreted in urine.	Poor oral bioavailability due extensive first pass metabolism and effective alternative routes of administration to pass
Beta Lactams	How does Penicillin exert its action ?	Inteferes in bacterial cell wall synthesis by binding to penicillin-binding-protein and preventing removal of terminal d-alanyl-d-alanine from peptides preventing crosslinking and formation of peptidoglycan.	Inhibits bacterial cell wall synthesis to pass.

	What are the mechanisms of resistance to B Lactam antibiotics ?	(1) Inactivation by B-lactamase (2) Modification of target PBPs (3) Impaired penetration of durg to target PBPs (4) Presence of efflux pumps	Inactivation of B-lactamase and one other to pass
Antiemetics	What clarrrrsses of drug can be used as antiemetics ?	(1) Serotonin 5-HT3 antagonists: the "trons" (2) Phenothiazines: prochlorperazine, promethazine (3) Butyrophenones: haloperidol (4) Substituted benzamides: metoclopramide (5) H1 antihistamines: diphenhydramine (6) Anticholinergics: hyoscine (Benzos, Cannabinoids, Corticosteroids)	Name three groups to pass – if name agents, prompt for group or mechanism of action.
	List and explain the adverse effects of prochlorperazine ?	Acute dystonia (dopamine blockade) Sedation (antihistamine effects) Anticholinergic effects (antimuscarine effects) Allergy	Acute dystonia + one other to pass
Drugs Binding To Biogenic Amine Transporters	How do anti-depressants exert their action ?	Thought to enhance amine-dependent synaptic transmission (serotonin and noradrenalin) by: (1) Inhibition of metabolism within nerve terminal (MAOIs) (2) Inhibition of reuptake from synapse (TCAs, SSRIs) (3) Increased release due to antagonism of specific serotonin and alpha2 noradrenalin receptors (Mirtazapine)	2/3 mechanisms to pass
	What are the relative advantages of different classes of antidepressants ? (Direct to adverse effects if no response)	Adverse effect profile Cost Efficacy Risk of overdose Dosing schedule Drug interactions	Able to discuss pros and cons of at least two

Primary Physiology 2008.1 THURSDAY Morning Session **Candidate No.**

Topic	Questions	Core Knowledge	Score
1.1 VP inequality (West pp 67-72)	Describe the relationship between ventilation and perfusion of the lung in a person while standing? What are the effects of V/Q inequality on gas exchange? What effect does increasing ventilation to the lungs have on arterial PO2 and PCO2	• Max ventilation 3-4x greater at apex • PO_2 40mmHg higher at lung apex • Max perfusion basally Q nearly 20x greater at base • Prompt: are there regional variations in either • V/Q inequality impairs uptake or elimination of all gases • Majority of blood returns from lung bases where the oxygen saturation is low • Results in blood PO2 being lower than that of mixed alveolar PO2 • PCO2 reduces much more than PO2 increases	/2
1.2 Renal regulation K^+ (Ganong pp 724)	How does the kidney handle potassium? How do other ions affect potassium transport across the membranes in the nephron? Prompt: ***How is potassium transported into and out of the tubules?***	• K+ filtered ~600meq/24hrs • Active K+ reabsorption in prox tubules ~560meq/24hrs • K+ secretion ~502meq/24hrs at distal tubule – amount proportionate to flow rate through distal tubules • Secretion - Electrical coupling to Na+ reab, thus H+ also • Collecting tubules Na reab'd, K excreted, electrical coupling and passive K movement • Na reab'd in association with H secretion, K excretion decreased if Na low in distal tubule • Na/K 2Cl apical transporter/transport protein • 3Na/2K ATPase	/2
1.3 Pressure, flow & resistance (Guyton pp 164-170)	What are the basic factors which determine the rate of flow of blood through a blood vessel? What factors cause turbulent flow in a blood vessel?	Poiseulle's Law and formula describe these factors; **(Radius to 4th power + 2 others)** Where: F is the rate of flow; $P_A - P_B$ is the pressure differential; $\mathbf{F = \frac{P_A - P_B}{R}}$ R is the resistance: r is the radius of the tube; $\mathbf{R = \frac{8 \eta L}{\pi r^4}}$ η is the viscosity of the fluid L is the length of the tube $\mathbf{F = P_A - P_B \times \frac{\pi r^4}{8 \eta L}}$ Expressed by Reynold's number; **(3 out of 4)** Where: ρ is the fluid density; D is the diameter of the tube; $\mathbf{Re = \frac{\rho D V}{\eta}}$ V is the velocity of flow; η is the viscosity of the fluid. The higher the value of Reynold's number the greater the probability of turbulence' which usually occurs when Reynold's number is between 2000-3000.	/2

1.4 Thyroid hormones Ganong pp 319-328	What are the effects of thyroid hormones?	**(4 out of 7)** • Widespread actions • Metabolically active tissues • Heart - increased rate • Brain - development reticular Act. Sys. • Gut - increased carbohydrate absorbtion. • Muskuloskeletal growth • Adipose – lipolysis	
	What is the mechanism of action?	**(4 out of 8)** Intracellular--- • At the nuclear level • O2 consumption regulator. • T3 binds better than T4 to receptor • Hormone/receptor binds to DNA • Affects gene expression • Two genesites • Alpha Chromosome 17 • Beta Chromosome 3	/2
1.5 Vitamin D Ganong pp 387-388	What are the actions of vitamin D?	**(3 of 4)** • Increased absorption of calcium from the intestine by induction of calbindin-D proteins. • Increased resorption of calcium in the kidneys. • Increased osteoblast activity. • Aids calcification of bone matrix.	
	How is the synthesis of vitamin D regulated?	**(3 of 5)** • Not closely regulated. • Low calcium leads to increased PTH secretion and increased vitamin D is produced. • High calcium inhibits PTH and the kidneys produce inactive metabolites. • Low phosphate increases vitamin D production (and high phosphate inhibits it). • Vitamin D inhibits the enzyme involved in its synthesis.	/2
			Final Score

Topic	Questions	Core Knowledge	Score
2.1 Airway Resistance West pp 106-112	What factors impact on resistance in airways?	• Size of airway: R highest in medium sized bronchi, low in very small airways. • Lung volume: R decreases with expansion as airways pulled open • Bronchial smooth muscle tone: controlled by B sympathetics • Gas density: eg heliox -> low R • Forced expiration: intrathoracic pressure compresses airways = 'dynamic compression'	
	What factors cause turbulent flow in airways	Expressed by Reynold's number; **(3 out of 4)** Where: ρ is the fluid density; D is the diameter of the tube; V is the velocity of flow; η is the viscosity of the fluid. $\mathbf{Re} = \frac{\rho D V}{\eta}$ Laminar flow only in small airways, transitional most areas, turbulent in trachea (rapid breathing)	/2
2.2 Renal blood flow Ganong pp 702-705	What is a typical value for renal blood flow in an adult at rest?	**~25% of cardiac output or 1250 ml/min**	
	What factors regulate renal blood flow?	**Chemical**: Noradrenaline constricts interlobular and afferent arterioles. **(3 of 5)** Dopamine causes renal vasodilation. Angiotensin II constricts efferent arterioles to a greater extent than the afferent arterioles. Prostaglandins increase blood flow in the cortex and decrease blood flow in the medulla. Acetylcholine produces renal vasodilation. **Neural**: Strong stimulation of the sympathetic nervous system produces renal vasoconstriction. **Autoregulation**: Direct contractile response of smooth muscle of afferent arteriole to stretch. NO may be involved. **At low perfusion pressures angiotensin II plays a role in constricting efferent arterioles.**	/2
2.3 Factors controlling cardiac output & O_2 consumption Ganong pp 571-576	What factors control cardiac output?	**Cardiac Output = Heart Rate x Stroke Volume** **Heart rate** controlled by cardiac innervation – symp. / parasymp. **Stroke Volume:** • **Afterload** • **Preload** - Starling Curve (Fibre length-tension) **(2 out of 5):** Pericardial pressure Ventricular compliance Atrial filling Blood volume Intrathoracic pressure • **Contractile state (3 out of 7):** Cardiac innervations Hypoxia; hypercapnia; acidosis Drugs +ve & -ve inotropes Circulating catecholamines Loss of myocardium Intrinsic depression (Heart failure) Force-frequency relationship	
	What are the major factors which determine myocardial oxygen consumption?	**(2 out of 3)** Intramyocardial tension Contractile state of myocardium Heart rate (= Ventricular work/beat = SV x MAP)	/2

2.4 Glucocorticoids Ganong pp 372-380	What are the physiological effects of glucocorticoids?	• **Metabolic;** increased protein catabolism, increased hepatic glycogenesis and gluconeogenesis (raised plasma glucose). Raise peripheral tissue insulin resistance • Permissive effects on other reactions • Are required for catecholamines to produce **calorigenic and lipolytic** effects, **pressor responses** (vascular reactivity) and vasodilatation • Inhibit ACTH secretion (feedback) • Impair water excretion (mechanism unclear) • Reduce circulating basophils and eosinophils and increase other elements • Required for stress response • Affect EEG waveforms (mild personality changes in insufficiency)	
	How is glucocorticoid secretion regulated?	• Basal secretion and stress response both dependent on **ACTH** • (Other substances may stimulate adrenal directly but no evidence of role in physiologic regulation) • Free glucocorticoids produce negative feedback on ACTH secretion at both hypothalamic and pituitary levels. Effect mediated by action on DNA • Stress response ACTH secretion mediated almost exclusively via hypothalamic release of corticotrophin releasing hormone • **Circadian rhythm.** ACTH released in irregular bursts throughout day but much more common in early morning. 75% of cortisol secreted at this time	/2
2.5 Iron Ganong pp 474-478	Please describe how ingested iron is absorbed.	• Most ingested iron is ferric (3+) but the ferrous (2+) form is absorbed. • Minimal absorption in stomach but gastric secretions dissolve iron and aid conversion to the ferrous form. • Almost all absorption in duodenum. Iron is transported into enterocytes via DMT1. • Some stored as ferritin. • Remainder transported out via ferroportin 1 (basolateral transporter) in the presence of hephaestin. Then converted to ferric form and bound to transferrin. • Dietary heme is absorbed by an apical transporter andiron is removed from the porphyrin in cytoplasm.	
	What are the mechanisms that regulate iron absorption?	Precise mechanisms uncertain, probably related to: • Recent dietary intake of iron. • State of body iron stores. • State of erythropoiesis in bone marrow. • The regulatory mechanisms are unclear.	/2
			Final Score

Primary Physiology 2008. FRIDAY Morning Session **Candidate No.**

Topic	Questions	Core Knowledge	Score
3.1 Elastic Properties of the Lung West pp 96-106	Define lung compliance?	• Change in volume / change in pressure (Slope of pressure-volume curve) (Lung "stiffness")	
	What factors influence lung compliance?	**(3 out of 6)** • Fibrosis • Alveolar oedema • Elastic tissue • Emphysema / age • Volume / Size of lung • Surface tension in alveoli (Surfactant)	
	What else does surfactant do?	**(2 of 3)** • Reduces WOB Prevents collapse Keeps alveoli dry	/2
3.2 Loop of Henle, structure & function Ganong pp 700, 714-718	Please outline the structure of the Lof H	• Thin/descending, Thick/ascending. Situated mostly in the renal medulla • **Origin from PCT** • Short (cortical) and long (juxta med.)loops • **Macula densa at distal end, where joins DCT**	
	What happens to electrolytes in the loop	• **(Thin) Descending limb water permeable** • **Fluid becomes hypertonic as descends loop** • **(Thick) Asc limb impermeable to water, NaK Cl transported out, hypotonic at end, so K+ diffuses back** • **Active trans. ATPase**	
	Explain the counter-current concentrating mechanism	• Gradient • Exchange (vasa recta)	/2
3.3 Cerebral blood flow. Brain metabolism & O2 requirements Ganong pp 616-620	What factors determine cerebral blood flow?	**(4 of 5)** • Intracranial pressure • Local constriction/dilation of cerebral arterioles, autoregulation etc • MAP at brain level • Blood viscosity • Mean venous press at brain level	
	What substances are important for brain metabolism?	• Oxygen ~49ml/min = 20% body O2 consumption • Glucose (major energy source) ~77mg/min • Glutamate (converted to glutamine as detox mech NH3) ~5.6mg/min	/2

3.4 Insulin & Glucose Ganong pp 336-340	Describe the effects of insulin on various tissues What is the time frame for these effects	• **Adipose:** glucose in, fatty acid + glycerol synthesis, TG deposition, K in • **Muscle:** glucose in, glycogen synthesis, Aas in, protein synthesis, ketones in, K in • **Liver:** glycogen, protein + lipid synthesis, • General: cell growth • **Rapid:** glucose, AAs, K into sensitive cells • **Intermediate:** protein synthesis, glycolysis and synthesis, inhibition gluconeogenesis • **Delayed:** lipogenesis	/2

3.5
Regulation of calcium
Ganong pp 383-395
	What factors influence the level of free calcium in plasma?
	
	How does bone resorption occur?
		• Protein binding - depends on plasma protein level and pH.
		• Total body calcium
		  o bound in bone; bone calcium readily exchangeable or slowly exchangeable (resorption / deposition)
		  o Intake
		  o GI absorption under influence of vitamin D
		  o Renal excretion under vitamin D influence
		  o Parathyroid hormone
		  o Calcitonin
		
		• Osteoclasts are monocytes that develop from stromal cells under influence of RANKL.
		• Attach to bone via integrins in sealing zone of the membrane.
		• Hydrogen dependent proton pumps move into cell and acidify the area.
		• Acid dissolves hydroxyapatite and collagen.
		• Products move across osteoclast into interstitial fluid.
			/2

Final Score

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: X-ray: Lateral Ankle	Please identify the bones on this xray. What movements occur at **the ankle** joint?	Tibia, fibula, calcaneus, talus, navicular, cuboid Metatarsal and cuneiforms (grouped) Lateral and medial malleoli Dorsi and plantar flexion – some laxity in plantar flexion (<u>not</u> inversion/eversion)	These 6 to pass
Question 2: Bones: Humerus	Please identify this bone and its main features What factors stabilise the shoulder joint? Demonstrate the attachment of the rotator cuff muscles on the humerus	Proximal: Head, anatomical & surgical neck, greater and lesser tubercles, intertubercular groove, deltoid tuberosity, groove for radial nerve, Distal: condyles, epicondyles, trochlea, capitellum, coronoid and olecranon fossae Bones –unstable, glenoid labrum helps. Ligs: Intrinsic. glenhumeral ligs – ant, weak. Coracohum lig stronger, lies superiorly. Extrinsic support by coraco-acromial lig. Superiorly Muscles: rotator cuff muscles (SITS) stabilise superiorly. AP stability from TMaj, LD, Pec Maj	(6/8 to pass) (5 to pass) Name bone/muscle/lig concept ID bones as weakest part and muscles as most important Name and ID attachment 3 of 4 RC muscles
Question 3: Model: Orbit Extraocular muscles and eye movements	{using the model}Identify the muscles responsible for eye movement and describe their function Which nerves supply each of these muscles What are the effects of an oculomotor nerve palsy	sr- elevation, adduction , ir- depression, adduction, lr- abducts mr- adducts so- abduction depression, io- abduction, elevation iii, iv, vi Ptosis Dilated pupil fixed pupil "pupil down and out"	Also rotational element. All to prime mvts pass. All to pass 2/3 Addition; Explain why this happens

Question 4: Photo: Thoracic inlet Major vessels and relationships	Identify the vascular structures in this photo	Major - left common carotid 14, right brachocephalic trunk 4, right 19 common carotid, right subclavian art 21 , right 18 and left 13 brachiocephalic veins, right and left subclavian vv 24, LIJ 8 Minor – inferior thyroid vein, branches of thyrocervical trunk	5/8 major
	What are the branches of the subclavian artery? You will not be able to see all of them in the photo	Branches are vertebral artery, the internal thoracic artery 9, the thyrocervical trunk 32, the dorsal scapular artery.	2/4
	What are the posterior relations of the thyroid gland?	Cricothyroid membrane, trachea, right and left common carotid arteries laterally	Extra info
Question 5: Discussion: Anatomy of male urethra	Describe the parts of the male urethra. and the course of each	Internal urethral orifice (bladder) > Intramural > **prostatic urethra** > external urethral sphincter (= **membranous** (intermediate) urethra), thru perineal membrane to spongy urethra in bulb of penis > **spongy(penile)** in corpus spongiosum > external orifice.	Must name 3 parts
	Where is it narrowest?	Narrow:Membranous part and external orifice	1 of 2
	In a case of rupture of the spongy urethra, where does urine extravasate?	Around the penis/Scrotum/Anterior abdo wall/NOT into the thigh	Extra info

ACEM PRIMARY 2009/2 ANATOMY VIVA Day Thurs PM *Candidate Number*..... AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: X-ray: CT Head	Identify the anatomical features on this CT scan through base of the skull	I.D.: Bones: (occiput, temporal, sphenoid, nasal) Air spaces: (mastoid air cells, sphenoid sinus, ethmoid, nasal cavity) Intracranial: 4th ventricle, cerebellum, vermis, temp lobe, pons)	 2 of 4 to pass 2 of 4 to pass 3 of 5 to pass
Question 2: Bone: Ankle/foot	Identify the bones of the ankle and foot. What are the parts of the talus Demonstrate the attachments of the lateral ligament of the ankle	ID: lat/med malleoli, talus, calcaneus, navic, cub, cuneiforms, metat, phalanges (8 of 9) Trochlear (dome), head and neck PTFL, ATFL, CFL	8 of 9 2 of 3 ATF lig and 1 other to pass
Question 3: Model: Arm Extensor group of forearm muscles	{Using the model} Identify the extensor muscles of the forearm at the level of the wrist What is the nerve supply of this compartment? Describe how the action of these muscles produces thumb movement	1. apl 23, epb 22 2. ecrl 19, ecrb 18 3. epl 21 4. ed 27, ei 5. edm 6. ecu 16 Radial nerve and its deep branch becoming post interosseus nerve Apl abduction and extension at carpometacarpal jt Epl extension at ip joint Epb extension at mcp	7/9 pass Radial n. to pass All with prompts

Question 4: Photo: Aorta/IVC/kidneys Major vessels, branches and course of	Identify the structures visible in this photo.	Kidneys, ureters, psoas major, diaphragm, adrenals, IVC 7, L renal vein 12, R renal v 23, aorta 1, celiac trunk 2, sup mesenteric art 28,	8 to pass
	Name the branches of the abdominal aorta	Single - coeliac trunk, superior mesenteric artery, inferior mesenteric artery Paired –common iliacs, ovarian/testiculars, superior and inferior adrenals, right and left inferior phrenics, lumbar arteries	6 to pass
	Name the branches of the coeliac trunk and what do they supply	Arises at T12, supplies liver, stomach, spleen, oesophagus and superior part of duodenum and pancreas branches are L gastric, common hepatic and splenic	Extra info
Question 5: Discussion: LP Layers penetrated	Name the structures your needle would pass through when performing a lumbar puncture using a midline approach	Skin/ subcutaneous tissue/supra-spinous ligament/ inter-spinous ligament/ ligamentum flavum/epidural space + veins/ dura + attached arachnoid/ subarachnoid space (= CSF)	2/3 ligaments + know that dura is before SA space and CSF is in the subarachnoid space
	At what level would you do a LP in an adult and why?	Transcristal plane ~L4	Extra info

Thurs p.m.

ACEM PRIMARY 2009/2 ANATOMY VIVA Day Fri AM *Candidate Number*..... AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: Xray : AP/lateral Wrist	Please identify the bones on these xrays Prompt: identify the carpal on the lateral What movements occur at the wrist joint?	Ulna/ulnar styloid , radius/radial styloid . Scaphoid, lunate, triquetral, pisiform. Trapezium, trapezoid, capitate, hamate, metacarpals/phalanges Flexion >extension. Add>Abduction Circumduction	All carpal bones on AP and 2 others to pass, plus capitate/lunate/distal radius on lateral All to pass
Question 2: Bone: C2	Identify this bone and its anatomical features What are the major ligaments attaching to this bone and where do they attach	C2 (axis) – dens (must) - sup/inf articular facets, trans process, bifid s.p, lamina, pedicle, body (at least 5 to pass) General – tectorial mem (pll), ant a-a mem (all), lig flav (p a-a lig) Specific – cruciate (tranverse and long fibres) - alar	Dens and 5 others to pass 1 of 3 (alar & cruciate to pass)
Question 3: Model: Femoral triangle Muscles and contents	{Using the model} Demonstrate the boundaries of the femoral triangle Demonstrate the contents What does the femoral nerve supply?	Sup- ing lig; Med- add longus; Lat- Sartorius Floor- iliopsoas, Pectineus. Fem nerve, art, vein [Nodes and lymphatics] Musc- ant thigh esp quadriceps Artic- hip & knee. Sens- antmed thigh. Continues as saphenous nerve to supply antmed knee, leg, foot.	4/5 All Quads and skin over ant-med thigh to pass

Question 4: Photo: Pelvis Major vessels, course of ureter	Identify the ureters in this photo Describe the course of the ureter Where are its points of narrowing? What vascular structures you can identify in this photo?	- **exit the hilum at PUJ** - runs inf at level of trans proc along psoas - **cross the pelvic brim at the bifurcation of the common iliac arteries/SIJ** - along the lateral wall of the pelvis, towards ischial spines - turns medially to **enter the base of the bladder** Aorta, left and right iliac arteries, IVC, left and right iliac veins, femoral artery, femoral vein, inferior mesenteric artery, gonadal vessels	Must ID left and right Narrowings in bold Aorta, IVC, fem and iliacs to pass
Question 5: Discussion: Surface anatomy of the pleura	Describe the surface anatomy of the parietal pleura. How does surface anatomy of the lung compare to that of the pleura?	1)Sternoclavicular joint to midline at sternal angle 2) inferiorly to xiphoid at 6^{th} cc level on R, only to 4^{th} on L where passes laterally to margin of sternum then inf. to 6^{th} cc 3) reflection to 8th rib in MCL 4) $10^{th\ rib}$ in MAL 5) 12^{th} rib at its neck + PAL 6) parallel to vertebral column to T1 7) cervical cupola over the apex of the lung rising to 2-3 cm above the medial 1/3 of the clavicle at the neck of the 1^{st} rib. Lungs: 2 ribs higher at MAL and posteriorly (CD recess)	4 of 7 plus diversion on the left for the heart. Assumes quiet resps

Qn 1 ACEM PRIMARY 2009/1 PATHOLOGY VIVA Thursday 17 AM *Candidate Number*..... AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: Type I Hypersensitivity	What is a Type I hypersensitivity reaction?	**Rapid immunologic reaction (minutes)** **Antigen – antibody** **IgE** Mast cells Previously sensitised individual	Bold to pass
Question 2:	What are the Primary mediators within the mast cell granules and their actions.	1. **Biogenic amines/histamine**. The most important vasoactive amine is histamine. **Histamine** causes intense smooth muscle contraction, increased vascular permeability, and increased secretion by nasal, bronchial, and gastric glands. 2. **Enzymes. (named)** These are contained in the granule matrix and include neutral proteases (chymase, tryptase) and several acid hydrolases. The enzymes cause tissue damage and lead to the generation of kinins and activated components of complement (e.g. C3a) by acting on their precursor proteins. 3. **Proteoglycans**. These include heparin, a well-known anticoagulant, and chondroitin sulfate. The proteoglycans serve to package and store the other mediators in the granules.	Pass – 2 out of 3 groups must include biogenic amines and example of each
Question 3: **(if time)** Second, Late-phase Reaction	What characterizes the second, late-phase reaction?	The late phase reaction is characterized by infiltration of the tissues with eosinophils, neutrophils, basophils, monocytes, and CD4+ T cells as well as tissue destruction, typically in the form of mucosal epithelial cell damage. Time course 2-24 hours later without additional exposure – may last for days.	

Qn 2 **ACEM PRIMARY 2009/2 PATHOLOGY VIVA Thursday 17 am** *Candidate Number*..... **AGREED MARK**.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 2: Chronic inflammation	(a) What cell types are present in chronic inflammation?	**Macrophages** Lymphocytes Plasma cells Eosinophils Mast cells Neutrophils	Bold plus 2 others to pass
	(b) What processes mediate the persistent accumulation of macrophages seen in chronic inflammation?	1. **Continued recruitment** of monocytes (continued expression of adhesion molecules and chemotactic factors) 2. Local proliferation of macrophages 3. Immobilisation of macrophages	Bold to pass
	(c) What products are released by activated macrophages in chronic inflammation?	**Products associated with tissue injury:** • Toxic O_2 metabolites; Proteases (elastases, collagenases); Neutrophil chemotactic factors; Coagulation factors; AA metabolites; Nitric oxide **Products associated with fibrosis:** • Growth factors (PDGF, FGF, TGF); Fibrogenic cytokines; Angiogenesis factors (FGF); "Remodelling" collagenases	Processes in bold and an example of each Simple list (of 5 or more) passes. Better pass if organised into groups

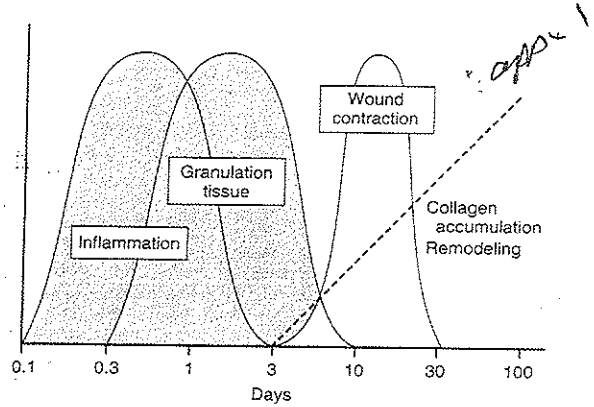
Qn 3 ACEM PRIMARY 2009/2 PATHOLOGY VIVA Thursday 17 am *Candidate Number*..... **AGREED MARK**.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1:	What is a paraneoplastic syndrome?	A complex of symptoms that cannot be readily explained by the local or distant spread of a tumour or by elaboration of hormones from the tissue in which the tumour arose.	Generally accurate description required to pass
Question 2:	What are the main types of paraneolastic syndromes?	**1. Endocrinopathies** - Cushing - Small Cell Ca lung (ACTH) - SIADH - Small Cell Ca lung, intracranial (ADH) - Hypercalcemia - Squamous Cell Ca lung, breast (parathyroid like hormones, TNF, TGF, IL-1) - Carcinoid – bronchial adenoma, ca pancreas and stomach – serotonin/bradykinin) - Polycythemia – Renal (EPO) **2. Nerve and Muscle Syndromes** - Myasthenia (bronchogenic Ca - ? immune mechanism) - CNS/neuro (breast) **3. Dermatological** - Acanthosis Nigricans (gastric, lung, uterine) - Dermatomyositis (bronchogenic. Breast) **4. HPOA** - bronchogenic	Endocrinopathies with at least 2 examples and at least one other to pass. Prompt: What syndromes or abnormal laboratory findings may be related to these syndromes? What are the mechanisms of these syndromes?
Question 3:	What is the cause of cachexia in cancer?	Not generally understood Anorexia Elevated BMR ? humoral factors – TNF, cytokines, Other tumour produced factors.	

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Qn 1	What are streptococci?	**Gram-positive cocci** growing in pairs or chains. Facultative or obligate anaerobes. Cause variety of suppurative infections and immunologically mediated post-streptococcal syndromes.	Bold to pass
Qn 2	Name some of the different types of streptococci and give examples of diseases they cause.	Alpha haemolytic - **S. pneumonia - pneumonia** - meningitis S viridans - endocarditis β Haemolytic - **Group A. (Pyogenes)** pharyngitis - scarlet fever - erysipelas - Impetigo - Rheumatic fever - Toxic Shock Syndrome - Glomerulonephritis Group B. (Agalactiae) - neonatal sepsis and meningitis - chorioamnionitis Strept. mutans - dental caries	3 major type/group + 6 diseases to pass
Qn 3	What factors in streptococci contribute to their virulence? .	Capsules pyogenes, pneumoniae M Protein prevents phagocytosis (anti M protein AL → Rh.F.) Complement C5a peptidase Pneumolysin lyses target cells (S pneumoniae) activates complement Pyrogenic exotoxin- rash and fever High MW glucans plaque formation aggregation of platelets Sucrose → lactic acid (S. mutans).	Any 3 to pass Capsule important.

Qn 5 ACEM PRIMARY 2009/2 PATHOLOGY VIVA Thursday am *Candidate Number*..... AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
DIC	What major clinical disorders are associated with DIC ? (same words as table)	**Most common are obstetric complications, malignancy, sepsis and major trauma** **Obstetric**: abruptio, retained dead fetus, amniotic fluid embolism, septic abortion. **Infections**: G-ve sepsis, meningococcus, malaria, rickettsia, histoplasmosis, aspergillosis **Neoplasia**: pancreas, prostate, lung, stomach. **Massive tissue injury**: trauma, burns, surgery. **Miscellaneous**: snakebite, shock, heat stroke, vasculitis, liver disease, leukaemia.	3 of 5 groups and an example of each.
	What is the pathogenesis of DIC?	2 major mechanisms - **release of tissue factor or thromboplastic substances** into the circulation, shift towards pro-coagulation, extrinsic pathway - **widespread injury to epithelial cells**, causing release of tissue factor, platelet aggregation, intrinsic coag pathway	Both mechanisms to pass
	What are the consequences of DIC?	- widespread deposition of fibrin leads to ischaemia and haemolytic anaemia - hemorrhagic diathesis (consumptive coagulopathy) from consumption platelets/clotting factors & activation plasminogen	

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1	Describe the process of skin wound healing by first intention. Prompt: Describe the timeline of these steps.  **FIGURE 3–20** Phases of wound healing. (Modified from Clark RAF: Wound repair. In Clark RAF (ed): The molecular and cellular biology of wound repair, 2nd ed, New York, Plenum Press, 1996, p. 3.)	• 24 hours: Scab; Neutrophils; Clot • 3 to 7 days: Mitoses; Granulation tissue; Macrophage; Fibroblast; New capillary • Weeks: Fibrous union *<24 hours*: neutrophils at the margins of the incision,. 24 to 48 hours: epithelial cells move from the wound edges and fuse in the midline beneath the surface scab, producing a continuous but thin epithelial layer that closes the wound. ***By day 3***, neutrophils replaced by macrophages. Granulation tissue progressively invades the incision space. Collagen fibres in the margins of incision. Epithelial cell proliferation thickens the epidermal layer. ***By day 5***, the incisional filled with granulation tissue. Neovascularization is maximal. Collagen bridges the incision. The epidermis recovers its normal thickness. ***During the second week***, continued accumulation of collagen and proliferation of fibroblasts. The leukocytic infiltrate, oedema, and increased vascularity have largely disappeared. ***By the end of the first month***, the scar is made up of a cellular connective tissue devoid of inflammatory infiltrate, covered now by intact epidermis.	Timeline + • Clot • Inflammation (neutrophils + macrophages) • Granulation • Remodelling

Qn 2 ACEM PRIMARY 2009/2 PATHOLOGY VIVA Thursday pm *Candidate Number*..... **AGREED MARK**.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 2: Normal Haemostasis	a) In the normal coagulation cascade, what happens after factor X is activated? Prompt: tell candidate factor X is where the intrinsic and extrinsic pathways join.	1. **Conversion of Prothrombin (II) to Thrombin (IIa)** requiring Calcium (Ca) and activated factor V (Va) as cofactors. Occurs on surface of damaged endothelium or activated platelets 2. **IIa catalyses fibrinogen (I) to fibrin (Ia)** in presence of Ca 3. IIa catalyses factor XIII to XIIIa in presence of Ca leading to **cross-linking of fibrin**	**Bold** essential to pass
	b) Describe the process of normal fibrinolysis.	1. **Plasmin is produced from circulating plasma protein plasminogen**, either by factor XIIa – dependent pathway, or by plasminogen activators. (PA, see 2. below) 2. **Plasmin breaks down fibrin** to FSPs, (eg D-dimer) and disrupts polymerisation 3. a) **t-PA from endothelial cells** most important PA, and most active when attached to fibrin b) Urokinase – like TPA (u-TPA) circulating protein 4. Free plasmin inactivated by alpha 2 plasmin inhibitor	**Bold** essential

Qn 3 ACEM PRIMARY 2009/2 PATHOLOGY VIVA Thursday PM *Candidate Number*..... AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: Diabetes Mellitus Type 1	What is the pathogenesis of diabetic ketoacidosis?	1. **Insulin deficiency** and glucagon excess →decreases peripheral utilization of glucose while increasing gluconeogenesis → severe **hyperglycaemia** 2. Hyperglycaemia causes **osmotic diuresis** and dehydration 3. Insulin deficiency increases **lipolysis and FFAs production.** FFAs are converted to **ketone bodies** by the liver. If rate of ketone bodies production exceeds rate of utilization by peripheral tissues→ketonaemia and ketonuria. Decreased urinary excretion of ketones leads to systemic metabolic ketoacidosis	1 from each of these groups to pass
Question 2:	What are the long-term complications of diabetes?	1. **Macrovascular**- coronary, peripheral vascular, cerebral and other large artery atherosclerosis, hypertension 2. **Microangiopathy**- nephropathy, cerebral microangiopathy, peripheral neuropathy, autonomic neuropathy 3. **Diabetic ocular complications**- retinopathy, cataracts, glaucoma	Macrovascular and microvascular with 2 examples of each to pass **or** Simple list of 6 to pass Higher score for organization in groups
Question 3:	Describe the stages in the development of Type 1 Diabetes?	1. Genetic predisposition 2. Precipitating event 3. Autoimmune destruction of islet cells 4. Subclinical leading to overt DM	Optional part of qn.

Qn 4 ACEM PRIMARY 2009/2 PATHOLOGY VIVA Thursday PM *Candidate Number*..... **AGREED MARK**.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 4: Ischemic bowel	1. What are the predisposing conditions for the development of ischemic bowel? Non-occlusive ischaemia • cardiac failure • shock • dehydration • vaso constrictive drugs Miscellaneous • radiation • volvulus • stricture • amyloid • diabetes • internal or external herniation	**Arterial thrombosis** • artherosclerosis. • vasculitis • aortic dissection • iatrogenic – angiography or aortic reconstruction • Hypercoagulable state. • Oral Contraceptive Pill **Arterial embolism.** • SBE • Angiography • Aortic atheroembolism **Venous Thombosis** • Hypercoagulation • OCP • AT III deficiency. • Intraperitoneal sepsis • Post-operative • Invasive neoplasms • cirrhosis • abdominal trauma	Simple list of 6 or more must contain examples of each of first 3 categories = straight pass headings + good examples of each = better pass.
	2. What are the clinical features of transmural infarction?	Pain Tenderness Nausea Vomiting Bloody diarrhoea, melanotic stool Shock Vascular collapse Absent bowel sounds Abdominal rigidity	Pain + any other 3 to pass

Qn 5 ACEM PRIMARY 2009/2 PATHOLOGY VIVA Thursday PM *Candidate Number*..... **AGREED MARK**.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 5 ARDS P715	What disorders can precipitate the Adult Respiratory Distress Syndrome, ARDS? *Prompt: What clinical conditions are associated with development of ARDS ?* *(same words as table)*	**Infection**: sepsis*, diffuse pulmonary infections*, gastric aspiration* **Trauma**: lung injury, head injury*, burns, radiation **Inhalation**: oxygen, smoke, irritants **Chemical injury**: heroin, salicylate, barbiturate, paraquat **Haematology**: transfusions, DIC **Other**: pancreatitis, uremia, CP bypass, hypersensitivity reactions (50% of ARDS cases associated with *)	4 groups, 1 example from each Need to include infection
	What is the pathogenesis of ARDS?	**Diffuse alveolar capillary damage**, variety of insults, initiated by different mechanisms. Capillary injury causes inc. vascular permeability, alveolar flooding & oedema, fibrin exudation, formation of hyaline membranes, **loss of diffusion capacity, abnormalities of surfactant.** Consequence of uncontrolled activation of acute inflammatory response; **most injury by neutrophils**. Macrophages alternative source of injury	3 out of 4 bold to pass
	What are the outcomes of ARDS?	Death, survival with organisation and **scarring.**	optional

Qn 1 ACEM PRIMARY 2009/2 PATHOLOGY VIVA FRIDAY 18 - AM *Candidate Number*..... **AGREED MARK**.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: Reversible Cell Injury	What are the morphological and chemical changes associated with early cell injury.	1. Decreased generation of ATP 2. Loss of cell membrane integrity 3. Defects in protein synthesis 4. Cytoskeletal damage 5. DNA damage	3 out of 5 to pass
Question 2:	What are the phenomena that characterize irreversible cell injury	The first is the inability to reverse **mitochondrial dysfunction** (lack of oxidative phosphorylation and ATP generation) even after resolution of the original injury. The second is the development of profound **disturbances in membrane** function.	Bold to pass
Question 3:	Can you give an example of a protein that leaks across degraded cell membranes? Prompt – "specific organs"	1. Cardiac muscle – contains a specific isoform of the enzyme creatine kinase and of the contractile protein troponin. 2. Liver (and specifically bile duct epithelium) – contains a temperature-resistent isoform of the enzyme alkaline phosphatase. 3. Hepatocytes – contain transaminases.	1 example to pass

Qn 2 ACEM PRIMARY 2009/2 PATHOLOGY VIVA Friday am *Candidate Number*..... **AGREED MARK**.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 2: Host Defences	(a) What are the normal barriers to infection by ingested pathogens in the gastrointestinal tract?	• Acid gastric secretions; • viscous mucosal layer; • lytic pancreatic enzymes; • bile detergents; • secreted IgA antibodies; • competition for nutrients with commensal bacteria; • clearance by defaecation	3/7 to pass
	(b) Describe the barriers to infection that exist within the respiratory tract.	• **Mucociliary blanket** within upper airways for trapping large microbes • **Coughing** (clears microbes from trachea) • **Ciliary action** within trachea and large airways (moves them up to be swallowed) • **Alveolar macrophages** or neutrophils attack and destroy microbes	2/4 to pass
	(c) What processes can disrupt the normal protective mucociliary action?	• Smoking; • cystic fibrosis (viscous secretions); • aspiration of stomach contents; • trauma of intubation; • viral infection; • bacterial infection	3/6 to pass

Qn 3 ACEM PRIMARY 2009/2 PATHOLOGY VIVA Friday AM *Candidate Number*..... AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: Embolism	What conditions predispose to the development of pulmonary thrombo-embolism?	**Hypercoagulable States:** 1. Primary- factor V Leiden, prohtrombin 20210 A, hyperhomocysteinaemia, antiphospholipid syndrome 2. Secondary – obesity, recent surgery, cancer, oral contraceptive pill, pregnancy **Other underlying medical conditions** – hip fracture, immobilization, cardiac disease, central venous lines	Simple list of 6 = straight pass Better pass with bold groups and examples of each
Question 2	What are the potential clinical sequelae of pulmonary thrombo-embolism?	Relates to size and number of emboli and overall status of cardiovascular system 1. Asymptomatic 2. Sudden death 3. Large PE –chest pain, dyspnoea, shock 4. Small PE-transient chest pain, cough and in predisposed individuals pulmonary infarct causing tachycardia, tachypnea, haemoptysis, fever,pleural rub. 5. Pulmonary hypertension	Any 3 to pass
Question 3:	What are the non-thrombotic types of pulmonary embolism?	1. Air 2. Bone marrow or Fat 3. Amniotic fluid 4. Tumour 5. Foreign bodies	3 to pass

Qn 4 ACEM PRIMARY 2009/1 PHYSIOLOGY VIVA Friday AM *Candidate Number*..... AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES

Question 4:	a. What are the causes of acute pancreatitis?	• Metabolic
		  ○ Includes alcohol
		• Mechanical
		  ○ gallstones
		  ○ trauma
		• Vascular
		• Infectious
		• Idiopathic (probably genetic basis)
			Identify alcohol and gallstones plus two others to pass.

	b. Describe the pathogenesis of acute pancreatitis	• Arises as a result of autodigestion by inappropriately activated pancreatic enzymes. • Trypsinogen is activated to trypsin. This in turn activates prophospholipase and proelastase, prekallikrein thus activating kinin system, and Hageman factor thus activated clotting and complement systems. Three potential pathways for initiation of pancreatic pathways: a. pancreatic duct obstruction b. primary acinar cell injury c. defective intracellular transport of proenzymes within acinar cells	Autodigestion and key role of activation of trypsinogen as triggering factor to pass.

Qn 5 ACEM PRIMARY 2009/2 PATHOLOGY VIVA Friday AM) *Candidate Number*..... **AGREED MARK**.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Friday 18th Morning Question 5: MS p1383	What are clinical features of Multiple Sclerosis	Distinct episodes of neurological **deficits separated by time.** Myriad of presentations as lesions separated by space. Unilateral visual impairment (optic neuritis) is common, brainstem, cord lesions	Bold to pass
	What is the pathogenesis of Multiple Sclerosis?	Exact etiology not established **Autoimmune, demyelinating** disorder, to white matter lesions separated in space. Genetic linkage, ?microbial / viral triggers. CD4+ Th1 T cells react against myelin antigens, release cytokines, activate macrophages. Inflammatory cells create **plaques**.	Need bold to pass
	What might be found in CSF of a patient with MS?	Mildly **elevated protein**; moderate pleocytosis; increased proportion of gamma globulin, oligoclonal bands – reflects B cells	Bold to pass

CEM PRIMARY 2009/1 PATHOLOGY VIVA Thursday am *Candidate Number*..... AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: **Vascular changes of inflammation**	**Describe the vascular changes that occur in acute inflammation**	**1.Vasodilation & increased blood flow mediated by histamine and NO, action on vascular smooth muscle** **2. Increased permeability** 3.Stasis - incr blood viscosity and concentration of RBCs 4 Accumulation of leukocytes on vascular endothelium	Need **1** + **2** and 1 other
	What are the causes of the increased vascular permeability?	**1. Gaps due to endothelial contraction via mediators** ("immediate transient response"): histamine (fast), bradykinin, sub P, leukotrienes, cytokines(longer). Venules. 2. Direct injury to vessel: ("immediate sustained") 3. "Delayed prolonged" 2-12 hrs burn, radiation, toxins 4.. Leukocyte mediated injury: venules, pulm caps, hours 5. Incr transcytosis: vesicles, vacuoles, incr channels VEGF 6. New vessel formation; new bvs leaky; VEGF, mediators	Need **1** and 2 others
Question 2: **Role of platelets in haemostasis**	**Describe the formation of a primary haemostatic plug after vascular injury**	1.Circulating platelets exposed to extracellular matrix (esp collagen) resulting in **adhesion** via vWf/Gp1b/V/IX. 2. **Activation** – a)Secretion of granule contents (esp Ca^{++} and ADP from dense granules) and b)expression phospholipids with platelet thromboxane A_2 leads to 3. **Aggregation** = primary haemostatic plug (reversible process)	Need **3/3 bold** Prompt: *What is the role of platelets at the site of injury?*
	How does this then become the secondary haemostatic plug?	1. **Thrombin binds** to platelet with ADP/TxA2 - increased aggregation 2. **Platelet contraction** occurs ("viscous metamorphosis") = secondary haemostatic plug 3. **Fibrin** formation locks platelets into clot (irreversible process)	Need **2/3 bold**
Question 3: **Tumour invasion and metastasis**	**Describe the steps involved in tumour cell invasion of the extracellular matrix**	1. **Detachment** ('loosening up') of the tumour cells from each other, with breaking of intercellular bonds 2. **Attachment** to extracellular matrix (ECM) components, via laminin and fibronectin receptors 3. **Degradation** of ECM, via type IV collagenase and plasminogen activator, creating passageways 4. **Migration** of tumour cells, which may then lead to vascular dissemination	Accept ≥ 3 of 4 **bolded words** (or a similar explanation) for a pass? Prompt: "*Detachment is the first step.*"
	Describe possible mechanisms that influence the distribution of metastases	1. **Tumour cell adhesion molecules** ligands *preferentially* expressed on target organ cells 2. **Chemokines** *for* target tissues 3. **Chemoattractants** *from* target organs	Prompt: "*Chemokines have an important role*"

Question 4: **Influenza**	**How does the Influenza virus cause pneumonia?**	1. **Attachment of virus to upper resp tract epithelium** 2. Necrosis of cells followed by inflammatory response 3. Interstitial inflammation with outpouring of fluid into alveoli 4. **Secondary infection by Staph / Strep**	Need bolded
	How does Influenza A cause epidemics and pandemics	Mutations of Influenza A haemagglutinin and neuraminidase allow virus to escape host antibodies (**antigenic drift**) and epidemics, whereas replacement of these with animal-derived RNA segments leads to new virus (**antigenic shift**) and pandemics	Needs bolded
Question 5: **Hypertrophic cardiomyopathy**	**What are the characteristics of hypertrophic cardiomyopathy?**	1. **Myocardial hypertrophy without ventricular dilatation** 2. **Asymmetrical septal thickening (septum >> free wall)** 3. Impaired diastolic filling and LV outflow obstruction in 25% of cases	Need bolded Prompt: What are the structural effects on the myocardium?
	What are the complications of HCM?	1. Heart Failure 2. Sudden death, ventricular arrhythmias 3. Atrial fibrillation, mural thrombus / embolisation 4. Stroke 5. Infective endocarditis mitral valve	Need 3/5

CEM PRIMARY 2009/1 PATHOLOGY VIVA Thursday pm *Candidate Number*..... AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: **Cellular changes following ischaemia**	**Describe the types of damage that occur inside a cell after severe ischaemia**	1. **ATP depletion** leading to NaK pump failure, anaerobic metabolism, Ca pump failure, reduced protein synthesis and protein misfolding 2. **Membrane damage** - mitochondria, lysosomes and plasma membrane 3. **Increased intracellular Ca^{++}** / loss **Ca^{++} homeostasis** 4. **Accumulation of reactive O_2 species** **5. Defects in membrane permeability**	Need 3/5 bold Prompt: What would happen to energy production in the cell?
Question 2: **Septic shock**	**What is an endotoxin?** **How does an endotoxin cause septic shock?**	**Bacterial cell wall Lipopolysaccharides** usually from Gram -bacilli. Consists of a generic fatty acid core and a complex polysaccharide coat unique for each species. **Dose dependent activation of neutrophils, macrophages and monocytes** → mediator release → local/systemic inflam. response. Activation via: LPS binding prot. + CD14 receptor via IC toll I receptor. Mediators: TNF, IL-1, 6, 8, chemokines → cytokine release Low dose: enhanced local inflammatory response and clearance of infection. Moderate dose: fever, procoagulant activity. **High dose: Syndrome of septic shock** • Systemic vasodilatation • Decreased myocardial contractility • Widespread endothelial injury → alveolar capillary damage (ARDS) • Activation coag system → DIC	**Bold** **3/4 needed**
Question 3: **Hepatitis C**	**What are the routes of transmission of Hepatitis C virus?** **What are the potential outcomes of infection?**	1. **Blood inoculation with IV drug use** 60%. 2. Unknown 3. Transmission via blood products pre1991 10% 4. Dialysis < 5% 5. Occupational exposure < 5% 6. Sexual transmission infrequent 7. Vertical transmission very low. 1. Acute Infection generally asymptomatic, rarely fulminant hepatitis 2. **85% progress to chronic persistent hepatitis** 3. 15% resolve completely 4. **20% of chronic infection progress to cirrhosis** 5. Some cirrhotics develop **Hepatocellular carcinoma**	Need bold and 2 other? Need bold

Question 4: **von Willebrand Disease**	**What are the haematological and clinical effects of von Willebrand disease?** **Describe the types of von Willebrand Disease**	Haem: Increased bleeding time with normal platelets Increased PT time (Types 1 & 3) Decreased Ristocetin cofactor activity Clin: Spontaneous bleeding from mucous membranes Increased bleeding from wounds Menorrhagia Bleeding into joints rare except in Type 3 **1. Type 1** and **Type 3** associated with decreased circulating vWF. Type 1 most common (70%), autosomal dominant and usually mild. Type 3 autosomal recessive and severe **2. Type 2** has defective vWF, autosomal dominant, mild severity and 25% of cases.	Need 3/4 Need 2/3
Question 5: **Malignant mesothelioma (pleural)**	**Describe the relationship between asbestos exposure and malignant mesothelioma** **Where can malignant mesothelioma arise?**	1. **Increased incidence among people with heavy exposure to asbestos.** Lifetime risk up to 7-10%. 2. **Asbestos bodies** found in increased numbers in lungs of patients with mesothelioma. 3. **Long latent period** for mesothelioma (25-45 yrs). 4. No increased risk in asbestos workers who smoke (in contrast to asbestos related lung carcinoma). Asbestos workers more at risk of dying from lung carcinoma (especially if they smoke). 1. **Pleura** 2. **Peritoneum** 3. pericardium 4. tunica vaginalis 5. genital tract	**2/4** **Bold**

ACEM PRIMARY 2009/1 PATHOLOGY VIVA Friday am *Candidate Number*…… **AGREED MARK**……

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: **Thrombosis**	**What factors lead to formation of a thrombus?**	1. **Endothelial injury: dominant** influence, by itself can lead to thrombosis, especially in **high flow** circulation (e.g. arterial circulation; cardiac chambers). Any alteration in **dynamic balance** of pro- and anti-thrombotic effects of endothelium can influence clotting 2. **Stasis or turbulence:** Turbulence contributes to thrombosis by causing endothelial injury or dysfunction, and local pockets of stasis. Disrupts laminar flow and bring platelets into contact with endothelium; prevents dilution of clotting factors by fresh flowing blood; retards inflow of clotting factor inhibitors. Stasis is a major factor in development of **venous** thrombi. 3. **Blood hypercoagulability:** Less frequent. Any alteration of the coagulation pathways that predisposes to thrombosis. **Primary**: Genetic mutations (e.g. Factor V gene; prothrombin gene); genetic deficiencies (e.g. antithrombin III; protein C; protein S) **Secondary (acquired):** High risk for thrombosis (prolonged bed rest; immobilisation; MI; AF; tissue damage; cancer; DIC; HITS; APLA); lower risk (cardiomyopathy; nephritic syndrome; hyperestrogenic states / pregnancy; OCP use; sickle cell anaemia; smoking)	**All three plus brief description to pass** Prompt: What is **Virchow's triad**?
Question 2: **Type 4** **Delayed type hypersensitivity**	**What are the cellular events in delayed type hypersensitivity** in a previously sensitised individual? **How does it differ in a naïve individual?**	1. Delayed type hypersensitivity T_H1cells are activated and secrete cytokines that are responsible for the delayed type reaction - IL-12, IFN-γ, TNF, lymphotoxin, chemokines. Accumulation of mononuclear cells around small veins and venules, p**erivascular cuffing,** increased **microvascular permeability, escape of plasma proteins** and **deposition of fibrin** in interstitium Typical example = tuberculin reaction 2. T cell mediated cytotoxicity Sensitised CD 8+ T cells (cytotoxic T lymphocytes) kill Ag bearing target cells In naïve individual, **CD4+T cells** differentiate into T_H 1cells after recognising antigen presented on APCs in association with class II MHC molecules. **T_H1cells** can enter the circulation and remain in the **memory** pool of T cells for long periods (years)	2/4 bold

Question 3: **Neisserial infections**	**What are the two clinically significant *Neisseria*?** **Describe the pathogenesis of a *N. meningitidis* infection**	1. *meningitidis* 2. *gonorrhoeae* 1. Respiratory spread 2. Common coloniser of the oropharynx 3. (10% of the population at any one time) 4. Colonisation lasts for months 5. Immune response leads to protection against that strain 6. Invasive disease crosses respiratory epithelium to enter blood 7. Capsule of *Neisseria* reduces opsonisation & protects against destruction by complement proteins 8. Outbreaks in young people living in crowded quarters who encounter new strains	Both Need 5/8 Prompt: How does it spread?
Question 4: **Vitamin K**	**What is the function of Vitamin K?** **What are the causes of Vitamin K deficiency?**	1. Required co-factor for a liver microsomal carboxylasewhich carboxylates a glutamate residue in Factors VII, IX, X & prothrombin (PLUS Proteins C & S and a few others) 2. Necessary for binding calcium and thus functional activity of the proteins 1. Fat malabsorption syndrome 2. Destruction of endogenous Vitamin K-synthesizing flora in the gut by broad spectrum antibiotics 3. Neonates (small liver reserves, no bacterial flora and low Vitamin K in breast milk) 4. Diffuse liver disease (hepatocyte dysfunction interferes with synthesis of Vitamin K dependent factors)	Need 3/4 Should know all the clotting factors and Protein C & S
Question 5: **Crohn disease**	**What are the pathological features of Crohn disease?** **What are the extraintestinal manifestations of Crohn disease?**	**1. Transmural inflammation of bowel with skip lesions** **2. Noncaseating granulomata** **3. Fissures and fistulae** Migrating polyarthritis, sacroiliitis, ank spondylitis, erythema nodosa, finger clubbing, sclerosing cholangitis (uncommon), Uveitis, mild hepatic pericholangitis, renal disorders due to trapping of the ureters (uncommon). Systemic amyloidosis (rare) GI tract cancer (less common than UC). May occur prior to intestinal symptoms.	2/3 Bold needed At least three systems Prompt: What other inflammatory conditions may be seen in Crohn disease?

ADDITIONAL QUESTIONS IF REQUIRED

ACEM PRIMARY 2009/1 PATHOLOGY VIVA Friday am ***Candidate Number*****…… AGREED MARK……**

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1:	**Assuming a patient survives the immediate effects, what is the fate of the thrombus itself?**	Some combination of the following four events: 1.**Propagation** (accumulates more platelets and fibrin, eventually leading to vessel occlusion); 2. **Embolisation** (dislodges and travels to other sites); 3. **Dissolution** (removal by fibrinolytic activity); and 4. **organisation** (inflammation leading to fibrosis) and **recanalisation** (vascular flow re-established or thrombus incorporated into a thickened vascular wall)	3 out of four to pass
Question 2:			
Question 3:	**What are the microbiological features of *Neisseria*?**	1. Aerobic 2. Gram negative diplococci 3. Coffee bean shaped 4. Require chocolate blood agar 5. 13 serotypes of *N. meningitidis*	Prompt: What are the staining characteristics of *Neisseria?*
Question 4:			
Question 5:			

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: Volume of Distribution	1. Define the "Volume of Distribution" of a drug	The apparent volume that a drug would occupy if it was evenly distributed according to its measured concentration in blood, plasma or water. Vd = Amount of drug in body / Concentration in plasma or blood	Pass: either definition or formula
	2. Fluoxetine has a volume of distribution of 2500L/70kg. What does this mean?	Has higher concentration in extravascular tissues than in the vascular compartment. high lipid solubility	
	3. Give an example of a drug with a low volume of distribution	aspirin, NSAIDS, warfarin, most antibiotics, tolbutamide	
Question 2: Adrenaline	1. What are the effects of adrenaline on the blood vessels in different tissue? 2. What receptors mediate these effects?	Vascular resistance Cutaneous α Mucous membranes α Skeletal muscle β_2, α Renal α, D Splanchnic α, β Venous tone α, β	Pass: 3 tissues + receptors
	3. Describe the effects of adrenaline on other organs besides the heart.	**Respiratory** Bronchodilation **Eyes** Pupillary dilation, Intraocular pressure – decreases, also decrease production of aqueous humor) Relaxation of **gastric** smooth muscle **Genitourinary** Uterine smooth muscle relaxation, Bladder relaxation, Bladder sphincter contraction, Ejaculation **Apocrine** sweat glands – palm of hands **Salivary** glands leading to dry mouth **Lipolysis** – increased fatty acids and glycerol in circulation **Liver** – enhanced glycogenolysis **Metabolic acidosis** Decreased extracellular **potassium** **Leucocytosis** **Insulin** inhibits or stimulates insulin secretion	3 organs
Question 3: Carbamazepine	1. Describe the mechanism of action of carbamazepine.	Anticonvulsant: a) blocks Na channels b) Inhibits high-frequency repetitive firing of neurones c) Presynaptic blocker of synaptic transmission d) similar action to phenytoin	Pass: (a)
	2. How is carbamazepine metabolised?	a) Metabolised by microsomal enzymes b) enzyme induction occurs c) active metabolites (clinical significance uncertain)	Pass: (a)
	3. What is its effect of the metabolism of other drugs?	If (b) is not volunteered above – Enzyme induction increases the rate of metabolism of other drugs eg primidone, phenytoin, valproate, clonazepam. Some of these drugs also can inhibit carbamazepine metabolism	Pass: Enzyme induction

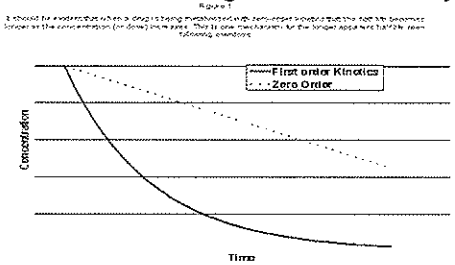
Question 4: Macrolides	1. Name some macrolide antibiotics?	Erythromycin, azithromycin ,clarithromycin, roxithromycin	Pass: 2 examples
	2. What is their mechanism of action?	Inhibits protein synthesis via binding to 50S ribosomal RNA and blocks aminoacyl translocation and the formation of initiation complexes	Pass: Protein synthesis and ribosomes
	3. What organisms are usually sensitive to macrolides?	Gram positive: eg pneumococci, staphylococcus Mycoplasma, legionella, chyamydia and some mycobacteria Gram negative : neisseria, bordatella pertussis, bartonella, campylobacter Treponema pallidum	At least 3
Question 5: Prescribing in the Elderly	1. In the elderly, what factors change with age and alter pharmacokinetics.	**Absorption**: No major change unless additional underlying associated condition with age **Distribution**: Dec lean body mass, Dec body water %, Inc fat body %, Dec serum albumin, Dec apparent Vd and sometimes increased Vd **Metabolism**: Liver metabolism does not decline for all drugs, Dec liver blood flow, Dec phase 1> phase 2 reactions, Liver slower to recover from injury **Elimination**: Dec renal function & Cr clearance, Half life inc of drugs variable, Dec excretion of volatile substances by the lung Associated age related illness affecting any of the above	Pass: renal function, 2 factors that may change Vd,
	2. Give some examples of drugs commonly used in the emergency department that must have their prescribing altered in the elderly?	Benzodiazepines – liver metabolism, renal function; PD sensitivity Opiods –PD sensitivity respiratory effects Antipsychotics –PD sensitivity; lean body mass NSAID – GI, renal Colchicine –renal, narrow therapeutic index Other drugs narrow therapeutic index Drugs primarily excreted renally –gentamicin, acyclovir Digoxin loading dose with dec Vd Amiodarone loading – Vd and PD sensitivity Many drugs as polypharmacy and must check for interactions i.e. Warfarin. So could argue extra precautions with all –polypharmacy, increase risk of error, compliance and administration issues Interactions with age related disease – IHD, COPD (B agonists or B Blockers) Sulphurs/Bactrim –adverse reactions Anticoagulants – falls Drugs which switch to zero order kinetics -phenytoin	Must get 4 relevant and plausible examples with correct associated mechanism & must include benzos and opiods. Prompts: • What about commonly used intravenous agents in the ED? • What about analgesic agents used in the ED? • What about sedative agents used in the ED? • Are there any drugs to be reduced with impaired renal function?

CEM PRIMARY 2009.1 PHARMACOLOGY VIVA Thursday pm *Candidate Number*..... AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: Drug Half-life	1. What is the definition of drug half-life?	1. time to change amount of drug in body by one half during elim (or infusion) OR t1/2 = (0.7 x Vd)/clearance	Either definition or formula
	2. What disease states can affect drug half life?	2. Factors affecting Vd: malnutrition, albumin levels, change in muscle mass or fat distribution, oedema, ascites, effusions Factors affecting CL: poor nutrition, renal disease, hepatic disease, heart disease(CO)	Need 2 Vd factors, and renal plus one other for CL
Question 2: Digoxin	1. What are the actions of digoxin on the heart at therapeutic levels?	Mechanical (Na-K ATPase) Electrical: Direct – alters action potential Indirect (autonomic) - parasympathetic effects predominate Sensitisation of baroreceptors Central vagal stimulation Facilitation of muscarinic transmission	Pass: Mechanical and one other.
	2. Are the parasympathetic effects uniform through-out the heart?	No Affect atrial and A – V nodal function more than Purkinje or ventricular function	
Question 3: Chlorpromazine	1. What are the clinical uses of chlorpromazine?	**Antipsychotic** especially for schizophrenia Sedative for agitation Antiemetic	Antipsychotic and one other
	2. What are the phamacodynamic properties responsible for these effects?	Antipsychotic D_2 blockade in mesolimbic & mesofrontal systems Antiemetic dopamine receptor blockade in medullary chemoreceptor trigger zone & peripherally on receptors on stomach Sedation 5HT blockade	Dopamine blockade
	3. What are its adverse effects?	Autonomic loss of accommodation, dry mouth, **urinary retention**, constipation **orthostatic hypotension**, sexual dysfunction CNS **Parkinsonism**, akathisia, dystonia, **Neuroleptic Malignant Syndrome** Tardive Dyskinesia Confusion Seizures **Sedation** Endocrine **Hyperprolactinaemia** – Amenorrhoea, galactorrhoea, infertility, impotence Ocular Corneal deposits	Any 3 adverse effectss

Question 4: Antibiotics for Staphylococcal infections	1. What classes of antibiotics are used in the treatment of **Staphylococcal** infections?	Beta-lactamase negative staph Penicillin 1st Generation Cephalosporins Beta-lactamase positive staph Beta-lactamase resistant penicillins – Methicillin / Naficillin, Isoxazolyl Penicillins (dicloxacillin, flucloxacillin etc) 1st Generation Cephalosporin Beta-lactamase inhibitor with penicillin combination – clavulinic acid, sulbactam, tazobactam Vancomycin Aminogycosides Macrolides	Pass: 3 classes
	2. What is the mechanism of **resistance** in Methicillin Resistant Staph Aureus?	Beta-lactam antibiotics normally bind to PBP's (Penicillin Binding Proteins) causing inhibition of transpeptidation, thus blocking cell wall synthesis and lead to cell wall death MRSA produce PBP's that have a low affinity for binding beta-lactam antibiotics and hence render then ineffective May be overcome if used in high enough concentrations, but not clinically achievable	Must demonstrate understanding of PBP's binding to pass
	3. What are the adverse effects of **Vancomycin**?	Local phlebitis Chills & fever Flushing due to histamine release (Red Man) Ototoxicity / nephrotoxicity if administered with aminoglycoside	Must get 1 to pass
Question 5: Prescribing in Pregnancy	1. List the factors affecting placental drug transfer?	Lipid solubility Molecular size Placental transporters Protein binding Placental and foetal drug metabolism	Pass: 2 of 5
	2. What is meant by foetal therapeutics?	Drug administration to the pregnant woman with the foetus as the target	
	3. Give examples of drugs administered for this purpose?	Corticosteroids (for lung maturation) Phenobarbitone (induce enzymes for glucuronidation of bilirubin) Antiretrovirals (decrease HIV transmission) Antiarrhythmics	

CEM PRIMARY 2009.1 Pharmacology VIVA Friday am *Candidate Number*..... AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1. Zero and First order kinetics	1. What is "First order elimination kinetics"?	First order: A constant fraction/percentage of the drug is eliminated per unit time. Rate of elimination is proportional to the amount of drug in the body. t½ constant. Most drugs eliminated this way (____) 	Definition to pass
	2. How is it different to zero order kinetics? (prompt – capacity-limited)	Zero order: a constant amount of drug is eliminated per unit time. Rate of elimination is constant and is independent of drug. There is capacity limited clearance or mechanisms have been saturated in overdose.	
	3. Give some examples of drugs with zero order kinetics?	Examples: Ethanol, phenytoin, salicylates, theophylline, and thiopentone (at high doses) (.....)	2 examples to pass
Question 2 Antihypertensives	1. What are the sites of action of antihypertensive drugs (with examples)?	Vasomotor centre – clonidine, methyldopa Sympathetic ganglia - trimethaphan Sympathetic nerve terminals – guanethidine, reserpine **β receptors of heart** – β blockers **Angiotensin receptors of bv** – AT II receptor blockers α receptors of bv - prazosin **Vascular smooth muscle** – hydrallazine, SNP, Ca blockers, GTN **Kidney tubules** - diuretics β cells juxtaglomerular cells – β-blockers **ACE**	Pass 4 of bold.
Question 3 Ondansetron	1. What is the mechanism of action of ondansetron?	selective 5-HT_3 receptor antagonists both peripheral in intestinal vagal afferents and central in chemoreceptor trigger zone and vomiting center in lateral medulla	Pass: serotonin
	2. What are the clinical uses of ondansetron?	a) Chemotherapy –induced nausea and vomiting eg 8 mg every 8 -12 hours b) Postoperative and post radiation nausea and vomiting. c) Other indications: acute or chronic medical conditions or gastroenteritis – not well evaluated	2 out of 3
	3. Name some side-effects of ondansetron?	Headache, dizziness and constipation. Small prolongation of QT interval	Pass: 1

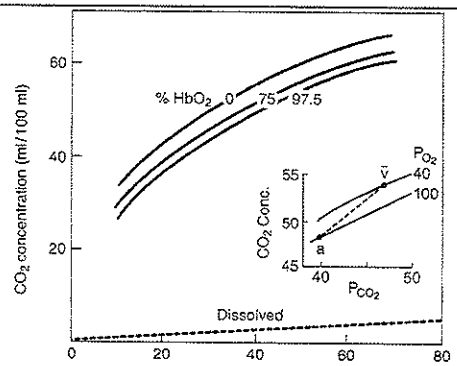
Question 4 Acyclovir	1. Describe the mechanism of action of acyclovir.	a. Converted to monophosphate by virus-specific thymidine kinase (infected cell specific) b. Converted to di- and tri- phosphates by host cell enzymes c. Inhibits viral DNA synthesis by irreversible binding to viral DNA polymerase, and chain termination	Pass: virus-infected cell specificity and inhibition of viral DNA synthesis (without detail)
	2. What are the indications for acyclovir?	Oral: initial or recurrent genital HSV2 infection Varicella-Zoster – within 24 h of varicella and 72 h or zoster (higher doses required) IV: HSV encephalitis, neonatal HSV, serious HSV or VZV	Pass: Use in HSV or VZV, plus encephalitis.
Question 5 Prescribing in children	1. In children, what factors change with age and alter pharmacokinetics?	Body Size and Composition – Growth of child – most doses calculated in mg/kg Adult is 50% water 20% extracellular Term neonate 70-75% water 40% extracellular Pre term neonate 85% water Influences drugs distributed in extra cellular space Fat 15% in adults 1% in pre term infants Plasma proteins Albumin – Decreased levels in neonate Potential for increased toxicity in neonates if drugs are highly protein bound Jaundiced neonates – if drug highly protein bound, will displace bilirubin and cause kernicterus Drug Metabolism Most drugs metabolised in liver Only 50-70% of adult values Slow clearance and prolonged elimination half lives Drug excretion GFR lower in newborns than older infants Neonate 30-40% adult values 3 weeks 50-60 % adult values 6-12 months Adult values	Pass: body size and composition, and drug metabolism and excretion.

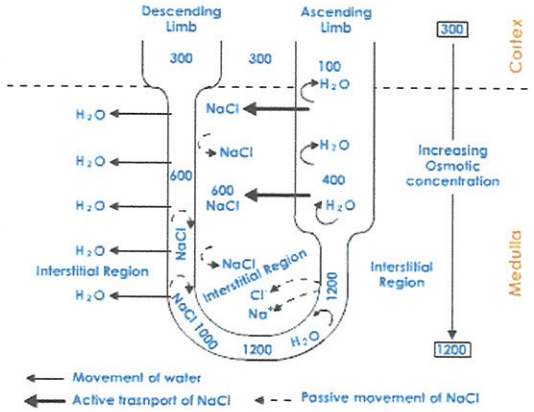
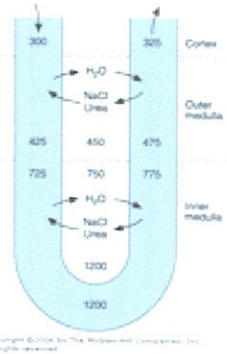
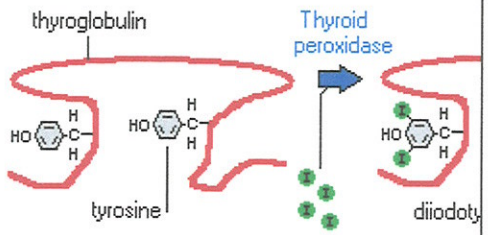
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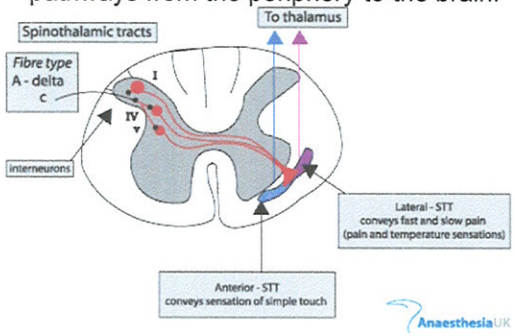
TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: Isovolumetric contraction and relaxation Ganong 565-8	In the heart: i) Describe the pressure and volume changes in the ventricles at the onset of systole. <u>Prompt</u>: describe the events that occur around the isovolumetric relaxation phase of systole. 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

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

ACEM PRIMARY 2009/1 PHYSIOLOGY VIVA 3rd April Fri Session (am) *Candidate Number*..... AGREED MARK.....

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.	

CEM PRIMARY 2009/1 ANATOMY VIVA Day Thursday am Question 1 *Candidate Number*..... AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1 (Photo)	Identify the bones visible on this x ray? (prompt to stay above C spine if needed) Prompt: what are the other facial bones that may not be visible	**Frontal**** **Nasal** **Maxilla **** **Zygoma**** **Sphenoid** **Mandible**** **Lacrimal** **Vomer** **Ethmoid**	Need 5 to pass frontal, mandible, maxilla, zygoma Plus one other
Question 2: (photo)	Identify the sinuses on this X-ray	• Frontal • Maxillary • Ethmoid • Mastoid ** (prompt if necessary)	3/4 to pass
Question 3: (photo)	Name this structure (point to infra orbital foramen). What passes through it, and what does it supply?	• **Infra orbital nerve** • **Mucosa of max sinuses** • **premolars, incisors, canines** • **skin of cheek** • **skin of lat nose** • **skin/conjunctiva of inf eyelid** • **ant/inf nasal septum** • **Skin of upper lip**	3 to pass

CEM PRIMARY 2009/1 ANATOMY VIVA Day Thursday am Question 2 *Candidate Number*..... **AGREED MARK**.....

rompt: Bone: Mid Cervical and Mid Lumbar Vertebrae, not articulated

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1:	a. Describe the features of this bone (Lumbar) b. What region of the spine is this from c. what is different about this bone (cervical)	Body: pedicles facet joints including articular facets, laminae Processes: transverse and posterior Spinal canal, vertebral artery (lateral foramina) for cervical only Key differences: Smaller body Longer thinner and downward sloping spinous process facet joints more horizontal allow greater range of movement foramen transversarium larger canal uncinate process anterior and posterior transverse process no accessory tubercle	4 Major features to pass. Correctly identifies cervical and lumbar describing key differences 4 to pass
Question 2:	What principle movements occur between this vertebra (cervical) and its neighbours Prompt if required: what are the movements at the facet joints	Cervical: relatively flat facet joints permitting free movement in all directions: rotation, flexion and lateral flexion.	Correct interpretation of facet joint orientation and functional result.

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Q1:	**Using this model, indicate the major ligaments of the knee and their attachments** **Prompt:** Extrinsic (5) & Intrinsic	**Patellar**: apex patella to tibial tuberosity . **Fibular collat**: lat femoral condyle to lat. fibula head **Tibial collat**: medial condyle to medial tibia & med. meniscus **Cruciates:** **Ant cruciate:** Ant intercondyle tibia to post part lateral fem condyle **Post cruciate:**Post intercond to anterolat medial condyle femur **Post Menisco-femoral**	Must know med, lat collats and cruciates and patella lig to pass
Q 2:	**What are the actions of these ligaments**	Patella part of ant joint capsule Fib and tibial collat: Taut in extension & relax in flexion to check rotation Cruciates: reduce medial rotation & allow lateral rotation until stopped by medial lig. Ant: prevent post displacement & hyperextension Post: prevent hyperflexion & stabilise knee in flexion	Ant and post cruciate to pass
Q 3:	**What are the attachments of the menisci**	Attached to intercondylar area tibia External parts attached to capsule 2 ligaments: coronary ligs from artic margins femur & tibia except under politeus tendon & transverse lig anteriorly to each other **Medial:** anterior horn to intercondylar are tibia in front of ACL & post horn in front of PCL **Lateral:**both horns attached intercondylar area tibia immediately in fron of & behind intercondylar spine	Predominate attachment is intercondylar ridge

CEM PRIMARY 2009/1 ANATOMY VIVA Thursday am Question 4 *Candidate Number*..... AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: (Photo)	Identify the major vascular structures in this picture. (Prompt if required)	**Superior vena cava (26)** **Left (13) and right (18) brachiocephalic vein (13)** **Left and right subclavian veins (24)** **Left internal jugular vein (8)** **Brachiocephalic trunk (4)** **Right common carotid artery (19)** **Right subclavian artery (21)** **Left common carotid artery (14)** **Thyrocervical trunk (32)** and its branches: inferior thyroid art. (6), ascending cervical art (3); internal thoracic art. (9)	Need 6/11 bold to pass
Question 2: (Not related to photo)	Describe the course of the right subclavian artery.	• **Arises** from **brachiocephalic trunk** • Runs **posterior to right sternoclavicular joint** as ascends through thoracic inlet • Arches superolaterally and **passes posterior to anterior scalene muscle** (relationship to this muscle defines its 3 parts) • Descends **posterior** to middle of **clavicle** and **crosses over 1st rib to become axillary artery**	3/4 Bold required to pass
Question 3: (Not related to photo)	Name the branches of the subclavian artery.	1st part: • **Vertebral artery** • **Internal thoracic artery** • **Thyrocervical trunk** 2nd part: • **Costocervical trunk** 3rd part: • **Dorsal scapular artery**	2/3 of part 1 bold to pass

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Q 1:	**Describe the palmar fascial spaces.** **Prompt:** **Direct to fascial spaces not compartments**	i) Midpalmar: Underlies central fascial compartment Related distally to synovial sheathes 3-5th digits Related proximally to common flexor sheath (as it emerges from carpal tunnel) ii) Thenar: Underlies thenar compartment Related distally synovial flexor tendon sheath of the index finger Related proximally to common flexor sheath distal to the carpal tunnel	3 of 6 to pass
Q 2:	**What are the contents of the central fascial compartment of the palm?**	Flexor tendons and sheaths Lumbricals Superficial palmar arterial arch Digital vessels Digital nerves	2 of 5 to pass
Q3:	**What are the boundaries of the central fascial compartment of the palm?**	Lateral – fibrous septum from palmar aponeurosis to 3rd metacarpal Medial – fibrous septum from palmar aponeurosis to 5th metacarpal Palmar aponeurosis superficial [covers it] Inferior mid palmar space	2 of 4

CEM PRIMARY 2009/1 ANATOMY VIVA Thursday pm Question 1 *Candidate Number*..... **AGREED MARK**.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1:	Name the intracranial structures that are visible on this non contrast CT Prompt 4th Ventricle	Cerebellum (right and left hemispheres united by central vermis). The pons. The 4th ventricle and pre-pontine cistern. The right & left temporal lobes in the middle cranial fossae. The mastoid, sphenoid and ethmoid sinuses.	Cerebellum, pons & 4th ventricle to pass.
Question 2:	Describe the posterior circulation of the brain *Prompt: What arteries contribute to the posterior circulation of the brain*	The vertebral As (originating from the subclavian As) give off the post and ant inf cerebellar As then unite (at the caudal border of the pons) to form the basilar A. The basilar A ascends to the superior border of the pons giving off the sup cerebellar A. It terminates by dividing into the 2 post cerebral As. The post communicating As join the post cerebral As to the middle cerebral A (& hence to the ant circulation).	Vertebral, basilar, post cerebral **and** post communicating As to pass.
Question 3:	What areas of the brain do the main arteries of the posterior circulation supply	Vertebral As -> cranial meninges & cerebellum. Basilar A -> brainstem, cerebellum & cerebrum. Post cerebral As -> inf aspect of cerebral hemispheres & occipital lobe. Post communicating As -> optic tract, cerebral peduncle, int capsule & thalamus.	Must correctly identify that the vertebral **and** basilar As -> cerebellum **and** post cerebral As -> inf of the cerebral hemispheres to pass.

CEM PRIMARY 2009/1 ANATOMY VIVA Thurs pm Question 2 *Candidate Number*..... AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1:	This is a right or left clavicle. Demonstrate the muscular attachments this bone	Deltoid – lateral 1/3 anterior Trapezius – lateral 1/3 posterior Pectoralis Major – medial 1/3 anterior - inferior Sternocleidomastoid – clavicular head, medial 1/3 ant - superior Subclavius – inferior, middle 1/3 (medial according to text)	Name all except subclavius **and** locate trapezius as posterior, SCM as anterior and deltoid as lateral attachments.
Question 2:	What are the anatomical relations of the medial third of the clavicle.	Medial: Sternoclavicular joint, manubrial notch Posterior: First rib, brachiocephalic vein (medial to scalenus anterior), internal jugular vein, subclavian vein (over scalenus anterior), subclavius, phrenic nerve (more posterior) Apical pleura, thoracic duct (left) Anterior/superior / inferior: Subcutaneous tissue, skin	Brachiocephalic **or** Subclavian vein **and** name 2 others to pass
Question 3:	Describe the course of the subclavian vein	Becomes subclavian vein from axillary vein medial to the outer border of the first rib. Courses medially posterior to clavicle, superior to flat section of first rib (groove). Lies immediately anterior to Scalenus anterior which separates it from the Subclavian artery. Becomes brachiocephalic vein at medial border of Scalenus Anterior when it joins the IJV.	Originates from Axillary vein **and** becomes Brachiocephalic vein **and** demonstrates course posterior to clavicle to pass.

CEM PRIMARY 2009/1 ANATOMY VIVA Thursday pm Question 3 *Candidate Number*..... AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1:	Name the bones of the foot Which of these constitute the medial longitudinal arch	• **Medial** – calcaneus, talus, navicular, 3 cunieforms, 3 metatarsals	Name all the bones with prompt if necessary Name major bone groups for med arch
Question 2:	What are the major factors contributing to the stability of the bony arches of the foot	**Passive** • **Bony** – shape of united bones Talus is the "keystone" • **Fibrous** (ligamentous) Plantar calcaneonavicular (Spring) lig Plantar calcaneocuboid (short plantar) lig Long plantar ligament Plantar aponeurosis **Dynamic** • Intrinsic muscles • **Long tendons** Flexor Hallucis & FDL – longitudinal arch Fibularis longus and Tib Post	Bold plus three ligs and three tendons to pass
Question 3:	What is the function of the longitudinal arches of the foot	• Shock absorbtion • Distribute bodyweight over the pedal platform • Act as springboards when walking, running and jumping	2/3 to pass

CEM PRIMARY 2009/1 ANATOMY VIVA Thursday pm Question 4 *Candidate Number*..... AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: (Photo)	What structures can you identify in this photograph?	**IVC** **Aorta** **Ureters** **Bladder** **Common iliacs** **Internal/ext iliacs** **Inguinal ligament** **Femoral vessels** **Testicular vessels** **Psoas** **If not already identified, point to ureter and ask, 'what is this structure?'**	Need 6 unprompted to pass
Question 2: Can demonstrate on photo	Describe the course of the ureters, and identify the 'narrow' points.	• **25 – 30 cm long** • **run from renal hila inferiorly** • **marked on x ray as running medial to tips of transverse processes** • **pass over pelvic brim at bifurcation of common iliacs** • **on lat wall of pelvis, inclining medially to insert post wall of bladder at VUJ** • **Narrow points are at..PUJ** • **...pelvic brim** • **....VUJ**	4/7 and 2 narrow points to pass
Question 3: (Not related to photo)	What is the arterial blood supply of the ureter?	Arterial Renal arteries in upper portion Gonadal vessels, sometimes in upper Mid portion from branches off abd aorta Inferiorly by branches of common iliacs Venous Renal and gonadal vessels.	Renal and gonadal to pass

CEM PRIMARY 2009/1 ANATOMY VIVA Thursday pm Question 5 *Candidate Number*..... AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1:	Describe the drainage of the superficial lymphatics of the upper limb?	Superficial lymphatics originate from lymphatic plexuses in the hand & ascend mostly with the superficial cephalic & basilar Vs. Some accompanying the basilar v enter the **cubital** LNs. Efferent vessels from here drain -> **humeral** (lat) axillary LNs (-> **central** axillary LNs -> **apical** axillary LNs). Most travel with cephalic v and enter the **apical** axillary LNs, but some enter the **deltopectoral** LNs earlier.	Travel with superficial veins **and** drain into axillary LNs to pass.
Question 2:	Describe the drainage of the deep lymphatics of the upper limb.	Deep lymphatics accompany the major deep vs in the UL & terminate in the **humeral** (lat) axillary LNs. These drain -> **central** axillary LNs - > **apical** axillary LNs -> supraclavicular LNs -> R & L subclavian lymphatic trunks.	Travel with deep veins **and** drain into axillary LNs to pass.
Question 3:	How do the right & left subclavian lymphatic trunks drain?	The R subclavian lymphatic trunk may be joined by R jugular & bronchomediastinal trunks to from the R lymphatic duct **or** it may enter the right venous angle (junction of int jug & subclavian vs) independantely. The L subclavian lymphatic trunk joins the thoracic duct.	R subclavian LT -> right venous angle **and** the L subclavian LT -> thoracic duct.

CEM PRIMARY 2009/1 ANATOMY VIVA Friday am Question 1 *Candidate Number*..... AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: (Photo)	Please indentify the intra-abdominal structures visible in this CT scan	**Liver** **Duodenum** **Small bowel** **Spleen** **L and R kidney** **Aorta** **Crura of diaphragm** **Pancreas** **Splenic vein** **IVC** **If not already identified as pancreas..'what is this structure?**	Need 6/10 to pass
Question 2: (Not related to photo)	What are the relations of the pancreas? You will not be able to see all of them.	• **Posteriorly…ivc, portal vein, r renal vein/artery, bile duct, sup mesenteric vessels, aorta, L2 verterbrae, L kidney and L adrenal** • **Lateral to right…duodenum 'C' shape around head** • **Lat to left…hilum of spleen** • **Anteriorly…stomach, peritoneum, lesser omentum, bowel, sup and inf panc-duod arteries**	6 to pass

CEM PRIMARY 2009/1 ANATOMY VIVA Friday am Question 2 *Candidate Number*..... **AGREED MARK**.....

rompt: Bone: Mid Thoracic Vertebra, not articulated

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1:	Describe the features of this bone If required prompt: What region of the spine does this bone come from. Begin with the features.	Body: location of invertebral disc attachments pedicles, facet joints including articular facets, laminae, articular facets for ribs on body and transverse process Processes: transverse and posterior Spinal canal	4 Major features to pass. Identify thoracic vertebra
Question 2:	How does this differ from vertebrae in other regions	Cervical: smaller body, larger canal, very small and often bifid spinous process, canal for vertebral artery, facet joints flatter, no ribs. Lumbar: larger body, smaller canal, spinous process square and more directly posterior, no articulations for ribs, more prominent transverse processes. Sacral: only mention to dismiss	Minimum 3 differences for each. Reasonable description of differences.

CEM PRIMARY 2009/1 ANATOMY VIVA Friday am Question 3 *Candidate Number*..... AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1:	a. Identify and describe the fibularis muscles	**Fibularis (Peroneus) Longus (lat comp) (18)** Head and upper shaft of fibula - passes deep to sup fibular retinaculum, post to lat malleolus then through separate compartment deep to inferior fibular retinaculum. Then passes inf to the fibular trochlea of the calcaneus, through groove on ant inf aspect of cuboid – crosses sole of the foot to insert on the base of the 1st MT and medial cuneiform **Fibularis (Peroneus) Brevis (lat comp) (17)** Distal shaft of fibula deep to PT. Post to lat malleolus deep to FL tendon. Passes sup to fibular trochlea of calcaneum. Inserts on base of 5th MT Fibularus (Peroneus) Tertius (ant compartment) (14) Slip of muscle from EDL Passes ant to lat malleolus inserts on base of 5th MT	'reasonable' description of FL and FB to pass
	b.What nerves supply these muscles	**Nerve supply** **FL and FB supplied by superficial Fibular (peroneal) nerve** FT supplied by Deep Fibular (Peroneal) nerve	Bold to pass
Question 2:	What are the actions of the fibular muscles	**Evertors of foot** **Stabilise foot** in 'toe-off' phase of walking / running **FL &FB – weak plantar flexors** because they run post to t/v axis of ankle joint FL – contributes dynamic stability tot the transverse arch of the foot FT – weak dorsiflexor	2/3 Bold to pass
Question 3:	What joins are involved in inversion and eversion of the foot	• **Subtalar (talocalcaneal) joint** • Transverse tarsal joint (**calcaneocuboid** and **talonvavicular joint**)	2/3 to pass

CEM PRIMARY 2009/1 ANATOMY VIVA Friday am Question 4 *Candidate Number*..... AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: (Photo)	On this photo, identify the structures bound by the extensor retinaculum	**From radial to ulnar, grouped by common synovial sheaths** **APL (2)/EPB (11)** **ECRL (6)/ECRB (5)** **EPL (12)** **Ext indicus (10)/Ext digitorum (9)** **Ext digiti minimi (8)** **Ext carpi ulnaris (7)**	Need 6/9 to pass
Question 2: (Not related to photo)	What is the motor supply of these muscles?.	• All radial/post interosseus nerve (C7, C8, except ECRL which is C6 too)	
Question 3: (Not related to photo)	Describe the sensory supply of the dorsum of the hand.	• Ulnar…medial 1 ½ digits via palmar cut branch • Radial….sup branch..lat half of dorsum of hand excluding distal digits. • Median…dorsum of radial 3 ½ digits, via dorsal branches of palmar dig nerves	

CEM PRIMARY 2009/1 ANATOMY VIVA Friday am Question 5 *Candidate Number*..... AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: (Discussion)	Define the boundaries of the anterior triangle of the neck.	• **Anterior border of sternocleidomastoid** • **Midline of neck** • **Inferior border of the mandible**	All 3 to pass
Question 2:	(a) Describe the surface markings of the carotid sheath in the neck. (b) What are the contents of the carotid sheath?	(a) Carotid sheath **runs along a line** joining the **sternoclavicular joint** to a **point midway** between the **mastoid process** and the **angle of the mandible.** (b) **Common carotid artery, internal jugular vein, vagus nerve**	All to pass
Question 3:	Describe the location of the thyroid gland in the neck.	• Located anteriorly in the neck at level of C5-T1 • Lies **deep to sternothyroid and sternohyoid muscles** • **Right and left lobes sit anterolateral to the larynx and trachea** • A thin **isthmus** unites the two lobes **across the trachea (usually at 2nd and 3rd tracheal rings)**	2/3 bold to pass

ACEM PRIMARY 2009.2 PHARMACOLOGY VIVA Thursday 17 Sept Session (am) *Candidate Number*..... AGREED MARK.....

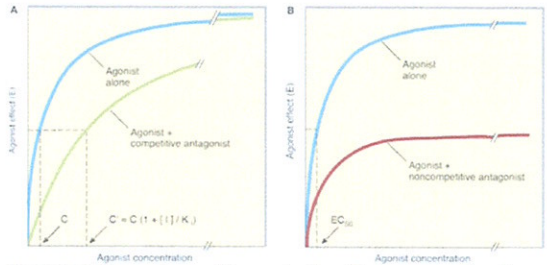
TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: **Drug Biotransformation**	(a) What are the sites of drug biotransformation? (Prompt – Which is the major?)	**Liver** - GIT - lung - skin - kidneys	Must get Liver and two others
	(b) What is a **Phase 1 biotransformation** reaction?	Conversion of a parent drug to a **more polar / water soluble** form by the adding or unmasking of a **functional group**, most commonly by **oxidation** but also by reduction or hydrolysis. The hepatic CYP (P450) enzymes are responsible for the majority of these reactions.	Must mention **more polar** or **water soluble** & **oxidation**
	(c) What is meant by **enzyme induction,** in liver biotransformation?	**Repeated administration** of a substrate brings about either **enhanced enzyme synthesis** or **reduced enzyme degradation** causing **increased metabolism** of the substrate	Must mention **enzyme more active**, therefore **increased metabolism and reduced drug action** (2 of 3 bolds to pass)
Question 2: **Classification of drugs in acute asthma**	(a) Outline the groups of drugs that might be used in asthma and give an example of each?	• **sympathomimetics** • **corticosteroids** • **muscarinic** antagonists • other bronchodilators -magnesium • antihistamines (allergic basis) • methylxanthines • cromolyns • leukotriene inhibitors [antagonists] – montelukast, zafirlukast,zileuton • heliox –changing airflow dynamics • ?other smooth muscle dilators – ketamine; calcium channel blockers • Experimental -; IgE monocloncal antibodies - omalizumab	Must get 3 bolded groups and one other with one correct drug example per group to pass.
	(b) Outline the mechanism of action of corticosteroids in asthma	Corticosteroids do the following: • Reduce bronchial reactivity • **Inhibition of** (lymphocytic and eosinophilic) **airway mucosal inflammation** • Increase airway calibre	Must get bolded point to pass
Question 3: **Propofol**	(a) Describe the pharmacokinetics of propofol?	Intravenous administration **Distribution $t_{1/2}$ 2-8 min**, redistribution $t_{1/2}$ 30-60 min **Metabolism- rapidly in liver**; total body clearance is greater than hepatic blood flow, suggesting extrahepatic mechanisms **Excretion- urine** as glucuronides and sulphates- <1% unchanged	Required for Pass: a) bold

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
	(b) What are the side effects of propofol?	Respiratory- dose-related **depression of central ventilatory drive**, apnoea, Cardiac- **Marked decrease in blood pressure** through decreased peripheral arterial resistance and venodilatation, and direct negative inotropic effect. Soy/egg allergy, Pain on injection	b) Knowledge of respiratory and cardiac effects of propofol
Question 4: **Warfarin**	(a) What is the mechanism of action of warfarin?	• Blocks synthesis of Clotting Factors II, VII, IX, X and Anticoagulant proteins C and S • Coupled to Deactivation of Vitamin K	Blocks factors II, VII, IX, X

	(b) What drug interactions with warfarin prolong the INR (prompt for mechanism)?	• Pharmacokinetic: (↑ INR)
		  • Inhibit transformation of Warfarin: S-Metronidazole, Fluconazole, Bactrim; R & S-Amiodarone, Disulfiram, Cimetidine
		  • Displace albumin bound warfarin: phenylbutazone, sulphinpyrazone
		• Pharmacodynamic: (↑ INR)
		  • Aspirin – affects platelet function
		  • 3rd generation Cephalosporins – reduce gut flora producing Vit K
		  • Heparin – directly prolongs INR
			2 examples

	(c) How is the action of Warfarin reversed?	• Vitamin K: FFP:Prothrombin complex – Prothrombin X: Recombinant Factor VIIa	2 of 4
Question 5: **Activated Charcoal**	(a) In a poisoned patient what modalities are available for decontamination?	Skin – remove clothes, wash contaminated skin GIT – emesis, gastric lavage, activated charcoal & cathartics / whole-bowel irrigation	3 of 5 to pass
	(b) How does activated charcoal work?	Adsorption due to its large surface area	
	(c) Name some drugs or agents that activated charcoal is NOT effective in adsorbing?	Ions: Fe, Li, K Alcohols, cyanide Corrosives (acids and alkalis)	2 examples
	(d) Name a drug where repeated doses of activated charcoal may assist in elimination of the drug	Carbamazepine, dapsone, theophylline	One drug

ACEM PRIMARY 2009.2 PHARMACOLOGY VIVA Thursday Session (pm) *Candidate Number*..... AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: **Dose Response**	(a) Draw and explain a Dose-Response curve for an agonist		Must demonstrate relationship of concentration to effect
	(b) Show how this curve is altered in the presence of an irreversible (non-competitive) antagonist	 **Figure 2-3.** Changes in agonist concentration-effect curves produced by a competitive antagonist (**A**) or by an irreversible antagonist (**B**). In the presence of a competitive antagonist, higher concentrations of agonist are required to produce a given effect; thus the agonist concentration (C') required for a given effect in the presence of concentration [I] of an antagonist is shifted to the right, as shown. High agonist concentrations can overcome inhibition by a competitive antagonist. This is not the case with an irreversible (or noncompetitive) antagonist, which reduces the maximal effect the agonist can achieve, although it may not change its EC_{50}.	Pass - Non-competitive antagonist has lower maximal effect
	(c) How does this differ from a competitive antagonist?		Pass – Higher conc. of agonist to produce similar effect
Question 2: **Classification of drugs used in diabetes mellitus**	(a) Outline the groups of drugs that are used to treat hyperglycaemia in diabetes mellitus.	• **Insulin** • **Sulfonylureas -** • **Biguanides** • Meglitinides • D- phenylalanine derivatives • Thiazolidinediones • Alpha-glucosidase inhibitors	Must get 3 bolded groups to pass.
	(b) Contrast the mechanism of action of sulfonylureas and biguanides.	Sulfonylurea: • **Increase insulin release from pancreas** • Reduction of serum glucagon levels • Closure of potassium channels in extrapancreatic tissues Biguanide: • **Action does not depend on functioning pancreatic B cells** • May directly stimulate glycolysis in tissues with increased glucose removal from blood; • May reduce hepatic gluconeogenesis; • May slow of absorption of glucose from the GI tract; • May reduce glucagon levels	Bold to pass

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 3: **Ketamine**	(a) How does ketamine affect the cardiovascular system?	HR, BP and cardiac output increase Stimulate central SNS, and inhibits re-uptake of noradrenaline at sympathetic nerve terminals	(a) Demonstrated understanding of CV effects of ketamine
	(b) What are the side effects of ketamine?	Sialorrhoea **Decreased RR** Postoperative disorientation Sensory and perceptual illusions **Emergence phenomenon** Vomiting **Raised ICP**- increases cerebral blood flow, oxygen consumption and ICP Rash	2 bold + 1 other
Question 4: **TPA**	(a) How does TPA work?	• Fibrinolytic. Binds to fibrin in a thrombus and converts entrapped inactive plasminogen to active plasmin to initiate local fibrinolysis	Definition
	(b) What are the indications for TPA use?	• STEMI • PE with haemodynamic instability • Acute Ischaemic Stroke: • Severe DVT	AMI, stroke and 1 other
Question 5: Amphetamines	(a) What is the mechanism of action of amphetamines?	**- Indirectly cause increased release of catecholamines at synapses** - Competitively inhibits dopamine transport in pre-synaptic neurone (DAT), inhibits VMAT causing non-vesicular release of dopamine into synapse (& similarly for other catecholamines)	First point to pass
	(b) Describe the effects of amphetamines?	1. Catecholamines; (increased arousal & decreased sleep) elevated HR (dysrhythmias) and BP (CVA) 2. Dopamine release; euphoria, potentially abnormal movements & psychosis 3. Serotonin; Appetite suppression, hallucinogenic & hyperthermia	CNS stimulation and cardiovascular effects to pass

ACEM PRIMARY 2009.2 PHARMACOLOGY VIVA Friday Session (am) *Candidate Number*..... AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: **Drug Clearance**	(a) What formula describes **Drug Clearance**?	Ratio of rate of elimination of a drug to its concentration in blood / plasma or $CL = \frac{\text{Rate of elimination}}{\text{Conc}}$	Must get formula to pass
	(b) What is **Flow Dependent Elimination**? (prompt if needed – High extraction)	For drugs that are readily cleared by their organ of elimination (high extraction ratio), **the rate of elimination is dependent on rate of drug delivery to the organ – determined by blood flow** and plasma protein binding. (Systemic $CL = CL_{renal} + CL_{liver} + CL_{other}$)	Must mention **drug delivery / blood flow** to pass.
	(c) Can you name any drugs that have Flow dependent elimination	Hepatic –lignocaine; propanolol; verapamil' morphine; pethidine	One example to pass
Question 2: **Side effects of NSAIDs**	(a) What are the side effects of the non-steroidal anti-inflammatory agents?	**Allergy;** rash; pruritis Nausea, abdominal pain, diarrhoea **GI irritation / ulcers** **Bleeding secondary to inhibition of platelet aggregation** **Nephrotoxicity** Peripheral oedema; fluid retention Headache	3 bold to pass
	(b) What specific side effects occur with aspirin?	Salicylism – vomiting, tinnitus, hearing loss and vertigo Exacerbation of asthma Histamine induced flushing Irreversible platelet inhibition Raised LFTs	Any 2 to pass
Question 3: **Suxamethonium**	(a) Describe the mechanism of action of suxamethonium	Phase I (**depolarising**)- reacts with Nicotinic receptor, opens the channel, causing depolarisation of the motor end plate, not metabolised at the synapse, and so membranes remain unresponsive to subsequent impulses- lack of "repriming" leads to **flaccid paralysis.** Phase II (desensitising) Unclear, but channel block may be more important than agonist action. Action is terminated by diffusion away from the end plate into the extracellular fluid, where it is metabolised by plasma cholinesterase.	Demonstrated understanding of mechanism of action
	(b) What are the side effects of suxamethonium?	b) **Bradycardia**- negative inotropic and chronotropic effects (inc. second dose bradycardia)	3 bold to pass

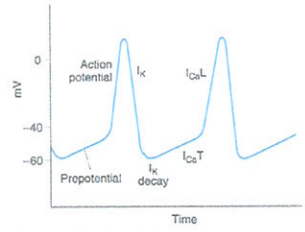
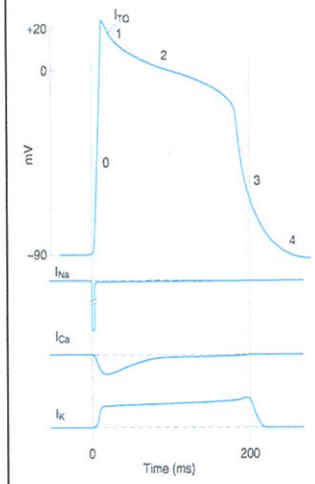
TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
		Hyperkalaemia (esp burns, nerve damage, NM disease, closed head injury) **Increased intra-ocular pressure** **Increased intragastric pressure** (inc. aspiration) **Muscle pain** (in up to 20%) **Malignant hyperthermia** (when combined with volatiles) ***Sux apnoea*** in susceptible patients	

Question 4:
 	
Calcium Channel Blockers
	• What is the mechanism of action of CCBs?	• CCBs bind to receptors on alpha1,2, gamma and delta subunits of L-type Ca channel→↓ frequency of opening of Ca channels in response to depolarisation→↓ transmembrane Ca current→ ↓ Ca influx→
		  ◦ vascular smooth muscle relaxation
		  ◦ ↓ contractility in cardiac muscle
		  ◦ ↓ SA node pacemaker rate
		  ◦ ↓ AV node conduction velocity
			Need anti-arrhythmic and smooth muscle effects

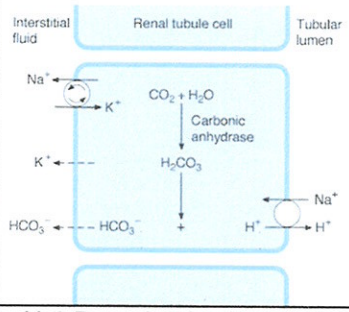
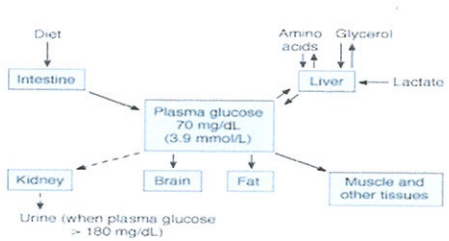
	• What are the toxic effects of CCB's	• Cardiovascular: cardiac arrest; bradycardia; AV block; heart failure, hypotension • Minor: flushing, dizziness, nausea, constipation, peripheral oedema	2 cardiovascular
Question 5: Topical Anaesthetics	(a) What is the mechanism of action of local anaesthetics?	**- blockade of voltage-gated Na channels in neurones** - increasing doses lead to higher excitation threshold, slower impulse conduction, lower AP **- blocks conduction if 2-3 nodes of Ranvier in a myelinated nerve affected**	Blockage of Na channels and blocked conduction to pass.
	(b) Which local anaesthetics are used topically?	Lignocaine – oral spray for procedures, viscous for pharynx, with prilocaine in EMLA, other mixtures for wound and ENT care, eye drops EMLA (Eutectic Mixture of Local Anaesthetics – mixture of lignocaine and prilocaine) – skin anaesthesia for cannula insertion, etc. Cocaine – ENT procedures (combines vasoconstriction) Proxymetacaine, amethocaine, oxybuprocaine – eye drops Benzalkonium – oral gels	2 agents

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

AGREED MARK.....

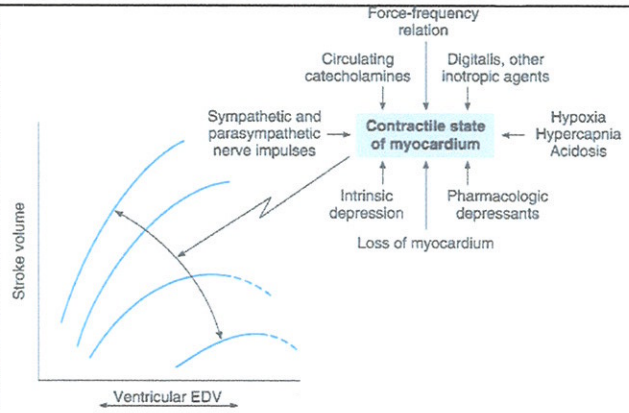
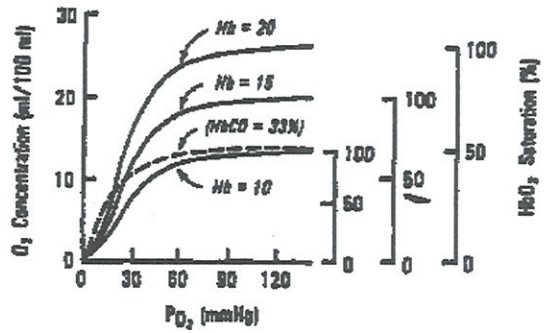
TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1a:	**Draw and explain the action potential in a cardiac pacemaker cell.**	 Source: Ganong WF: *Review of Medical Physiology*, 22nd Edition: http://www.accessmedicine.com Copyright © The McGraw-Hill Companies, Inc. All rights reserved. PROMPTS: What electrolytes 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**	 Source: Ganong WF: *Review of Medical Physiology*, 22nd Edition: http://www.accessmedicine.com Copyright © The McGraw-Hill Companies, Inc. All rights reserved.	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+**

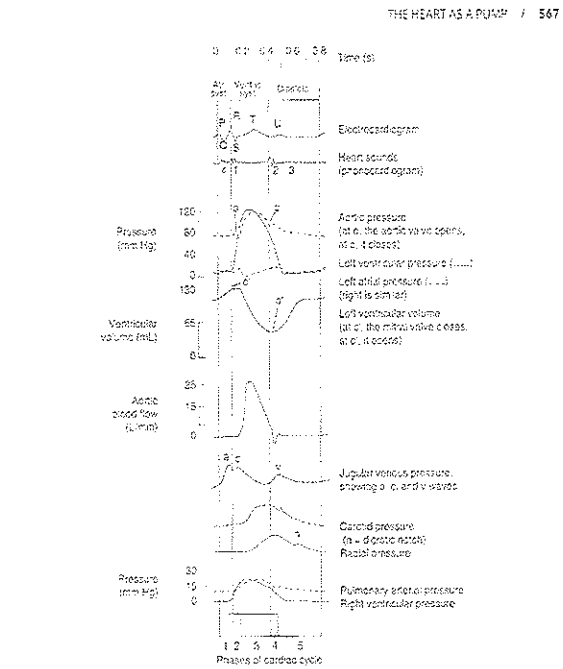
CEM PRIMARY 2009/2 PHYSIOLOGY VIVA Day Thurs Session (pm) *Candidate Number*..... AGREED
MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: a) b)	**Draw Frank Starling curve.** **List the factors that alter contractility**	 Source: Ganong WF: *Review of Medical Physiology*, 22nd Edition: http://www.accessmedicine.com Copyright © The McGraw-Hill Companies, Inc. All rights reserved.	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**

ACEM PRIMARY 2010/2 PATHOLOGY VIVA Thursday (September 16) Session: Morning *Candidate Number*…… AGREED MARK……

TOPIC	QUESTION	ESSENTIAL (BOLD) KNOWLEDGE (UNBOLD)	NOTES
Question 1.1 Vascular Changes of Inflammation	1. Describe the vascular changes in acute inflammation 2. What are the mechanisms of increased vascular permeability?	1.1. **Vasodilatation:** opening of arterioles and capillary beds mediated by histamine and Nitric Oxide leading to increased blood flow 1.2. **Increased vascular permeability** 1.3. **Stasis:** due to PP permeability and increased viscosity 2. 2.1. Endothelial contraction / retraction: gaps in venules due to histamine and leukotrienes < 30mins, immediate transient response eg.ultraviolet radiation and kinins and leukotrienes 2-12hrs, delayed prolonged leakage eg. late appearing sunburn 2.2. Direct vascular endothelial injury eg. in severe burns, microbial toxin injury, amplified by neutrophil activation, rapid onset but may last days 2.3. Leukocyte mediated leakage, in venules and pulm capillaries, long lasting for hours 2.4. Trancytosis increased Tx of fluid and protein thru endothelial cell, VEGF	1. All 3 2. 2 out of 4
Question 1.2 Antibody Mediated Hypersensitivity	1. What is antibody - mediated hypersensitivity? 2. Describe the mechanisms which mediate the hypersensitivity response 3. List an example or examples for each mechanism	1. **Caused by antibodies that react with antigens present on cell surfaces or in the extracellular matrix** Antigens can be intrinsic to the membrane or matrix or extrinsic eg. Drug metabolite 2. Mechanism of hypersensitivity response 2.1. **Opsonisation and phagocytosis: IgG antibodies opsonise cells plus complement activation** generates C3b and C4b recognized by phagocyte Fc and protein receptors resulting in **phagocytosis and destruction of opsonised cells** **Examples:** transfusion reaction, erythroblastosis fetalis, autoimmune haemolytic anaemia, agranulocytosis, thrombocytopaenia, drug reactions when a drug acts as a hapten 2.2. **Complement and Fc receptor mediated inflammation: antibodies bind to fixed tissue** such as basement membranes, extracellular matrix … **activates complement** … generate by-products particularly chemotactic agent C5a … direct PMN migration and C3a and C5a **= increase vascular permeability. PMNs activated** by C3a and Fc receptors… release of pro- inflammatory substances like prostaglandins, production of lysosomal enzymes, reactive O2 species **Examples:** glomerulonephritis, vascular rejection in organ grafts, vasculitis caused by ANCA, Goodpastures 2.3. **Antibody mediated cellular dysfunction:** antibodies directed against cell surface **receptors impair or dysregulate function without causing cell injury or inflammation** **Examples:** myasthenia gravis, Graves's disease, insulin resistant diabetes, pemphigus vulgaris **2.4. Antibody dependant cellular cytotoxicity** **Examples:** IgG coats cells, effector cells such as monocytes, neutrophils, eosinophils and NK cells then bind and lyse cells without phagocytosis, role in specific diseases uncertain.	1. Bold 2. Bold 2/4 3. 2/4
Question 1.3 Hepatitis C Infection	1. What causes Hepatitis C infection? 2. Describe the clinical course of Hepatitis C infection 3. What are the risk factors for acquiring Hepatitis C?	**1.1. Flaviviridae family RNA Virus** 2.1 Incubation period 2-26 wks (mean 6-12 wks) 2.2 Acute infection usually mild or asymptomatic (1-3 weeks) 2.3 Persistent and Chronic hepatitis with exacerbations in 80% 2.4 Cirrhosis in 20-30% 2.5 Fulminant hepatic failure rare **3.1 IVDU (54%)** **3.2 Multiple sex partners (36%)** 3.3 Needle stick (10%) (risk of HCV is 1.8% v 0.3% for HIV) 3.4 HCW (1.5%) 3.5 Blood Transfusion (in the 1980's), 3.6 Vertical,	1. Bold 2. 3/5 3. 3/7

		3.7 Unknown (32%)	
	Additional question for good candidates. After completion of 5 questions		4. 2/4
	4. What features of the Hepatitis C virus make vaccine development difficult?	4.1 Highly stable core, **extremely variable envelope (E protein)** 4.2 RNA polymerase inherently unstable; frequent mutations, multiple *quasispecies* found in any one pt 4.3 Genomic and Antigenic variability 4.4 Actively inhibits interferon mediated cellular response at many levels	
Question 1.4 Disseminated Intravascular Coagulation	1. Describe the pathophysiology of "disseminated intravascular coagulation"? ("Trigger" can be a prompt)	1. 2 major mechanisms **trigger** DIC: 1.1 release of tissue factor into circulation 1.2 widespread injury to the endothelial cells 1.3 Acute, subacute or chronic thrombo-haemorrhagic disorder characterized by 1.3.1 excessive activation of coagulation leading to 1.3.2 formation of **thrombi in the microvascular** circulation 1.3 .3 secondary activation of **fibrinolysis causing bleeding** 1.3.**4** **consumption of platelet**s, fibrin and coagulation factors	1. 1 trigger and 2/3 bolds
	2. What are some of the important causes and triggers of severe DIC?	2.1 **Obstetric complications** (eg amniotic fluid embolism, FDIU) responsible for approx 50% cases 2.2 Malignant neoplasms (33% cases) **2.3 Sepsis** **2.4 Major trauma**, severe burns, extensive surgery 2.5 Transfusion reaction 2.6 Most mild cases probably due to sepsis, esp in elderly, but not usually diagnosed – low plts	3. 3/6
Question 1.5 Post Streptococcal GN	1. Describe the aetiology and pathogenesis of post streptococcal glomerulonephritis.	1.1 G**roup A β-hemolytic streptococci** (eg: 90% types 12, 4, and 1) 1.2 Typically post pharyngeal/skin infections (impetigo) - sometimes epidemic, partic in overcrowded insanitary conditions 1.3 An **immunologically mediated disease** ? Type 2/ or 3 type e.g. ? Circulating or antigen deposit disease. 1.4 Granular **immune deposits in the glomeruli** (IgG & C3) - partic GBM- leading to leaking glomeruli. 1.5 Streptococcal antigen found in the glomeruli. 1.6 Complement activation – low serum complement **1.7** Elevated titres of anti streptococcal Ab 1.8 Nephritis associated streptococcal plasmin receptor NAPlr, Strep pyogenic exotoxin B (SpeB), zSPeb	1. 2 x Bold + 1 others
	2. Describe the clinical features of post Streptococcal GN.	1. 1 to 4 weeks after a streptococcal infection of the pharynx or skin (impetigo). 1.1. Malaise, fever, nausea, oliguria, and **haematuria** 1.2. Red cell casts, mild **proteinuria** (usually < 1 gm/day), periorbital and other **oedema**, mild to moderate **hypertension** 1.3. 95% will recover quickly in 1-3 weeks, 4 % chronic, 1% severe acute renal failure. Adult onset has worst prognosis 1.4. Depleted C3 and almost always Strep Ags.	2. 2 x Bold + 2 others

ACEM PRIMARY 2010/2 PATHOLOGY VIVA Thursday (September 16) Session: Afternoon *Candidate Number*…… AGREED MARK……

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 2.1 Hypertrophy	1. What is tissue hypertrophy? 2. What are examples of hypertrophy (Prompt: How is it classified??) 3. How is hyperplasia different form hypertrophy?	1.1. **Increase in cellular size not number** leading to overall organ/tissue size increase 1.2. Cell size increased by **more structural components and** increased synthesis of cellular proteins 1.3. Triggered by increased functional demand or stimulation by hormones or growth factors 1.4. Can be selective hypertrophy of specific sub-organelles 2. Examples 2.1. **Physiological** skeletal muscle enhancement through training or uterus under influence of hormones such as oestrogen 2.2. **Pathological** such as cardiomegaly in hypertension and CCF (has an **upper limit** after which regression occurs -> cell injury -> apoptosis/necrosis) 3. Hyperplasia involves an **increase in the number** of cells.	1. Bold 2. Bold (+ 1 example of each) 3. Bold
Question 2.2 Role of Platelets in Haemostasis	1. What are the 2 main roles of platelets in haemostasis? 2. How is the primary haemostatic plug formed?	**1.1. Primary Haemostatic Plug** **1.2.** Provides surface to **recruit and concentrate activated coagulation factors** **2.** After vascular injury, platelets contact exposed ECM eg. collagen, adhesive glycoprotein, vWF 2.1. **Adhesion** – via glycoprotein 1b (GpIb) receptor to vWF forming bridge between plat and ECM collagen 2.1.1. necessary to overcome high shear force of blood flow, deficient in vW disease or Bernard-Soulier syndrome 2.2. Activation resulting in shape change and s**ecretion** – **granule release** (**ADP**, **TxA2**). 2.3. **Aggregation** – ADP potent activator of platelet aggregation and +ve feedback for more ADP release. Agonist binding causes intracellular protein phosphorylation cascade => degranulation, including dense body content release of **Ca^{++}**, required for coagulation cascade. Platelet activation causes appearance of negatively charged phospholipids on surface => bind Ca, critical nucleation sites for assembly of coagulation factor complexes. 2.4. TxA2 amplifies platelet aggregation **=>** leads to formation of primary haemostatic plug. 2.5. Aggregation reversible at this stage but not after next stage of stabilization via coagulation cascade with formation of thrombin.	1. **Bold** to pass 2. **4/7 Bold** to pass
Question 2.3 Ulcerative Colitis	**1.** What are the pathological features of Ulcerative Colitis? **2.** What extra-intestinal manifestations occur in ulcerative colitis?	**1.1.** One of two disorders that compromise **inflammatory bowel disease (IBD)** **1.2.** Severe ulcerating inflammatory disease **1.3.** Limited to **colon and rectum.** 1.3.1. Continuous distribution (Starts in colon and extends continuously – No skip lesions) 1.3.2. Extends only into mucosa and submucosa (ie not trans mural) 1.3.3. Pancolitis if entire colon affected, limited distal disease eg ulcerative proctitis 1.4. Superficial broad based ulcers 1.5. Pseudopolyps 1.6. Malignant potential 1.7. Toxic megacolon 2. Extra-intestinal Manifestations 2.1. Polyarthritis, 2.2. sacroiliitis, ankylosing spondylitis 2.3. Uveitis 2.4. Skin lesions 2.5. Pericholangitis 2.6. Primary sclerosing cholangitis	**1.** Bold (+ 2) 2. 4 for pass

Question 2.4 Cholera	1. What is the causative organism of cholera? 2. Describe the pathogenesis of cholera (Describe how the enterotoxin causes diarrhoea).	1. **Vibrio cholera** = **gram neg bacteria** (comma shaped/flagellate) 2. Pathogenesis 2.1. **Non invasive** 2.2. **Flagella proteins** for attachment & colonization **2.3. Preformed enterotoxin** 2.3.1. Cholera enterotoxin • 5 B subunits • 1 A subunit 2.3.2. B subunit **binds** to intestinal (mainly duodenum/jejunum) – epithelial cells • Retrograde transport in ER 2.3.3. A subunit Tx to cytoplasm • A subunit activates G protein • Stimulates adenyl cyclase→ c-amp • Opens cystic fibrosis transmembrane conductance regulator (CFTR) • **Releases Cl^- into lumen** o **secretion of HCO3, Na and H_20** o **massive diarrhoea which overwhelms colonic resorption**	1. Bold 2. Need 4 bold to pass
Question 2.5 Parkinsonism	1. Describe the clinical features of Parkinsonism. (Prompt: How do Parkinsonian patients look?) 2. What are the causes of Parkinsonism? (Prompt: what part of the brain is affected?) 3. Outline the possible pathogenesis of Parkinson's Disease.	1. Diminished facial expression, stooped posture, slowness of voluntary movement, festinating gait (progressively shortened, accelerated steps), rigidity and a "pill-rolling" tremor. **2. Conditions that cause damage to the <u>nigrostriatal dopaminergic system</u>** 2.1. Parkinson disease 2.2. Post-encephalitic 2.3. Familial forms (rare – auto dominant & recessive) 2.4. trauma/ injuries 2.5. Drugs – dopamine antagonists/toxins/pesticides 2.6. Multiple system atrophy, progressive supranuclear palsy 3. Possible pathogenesis – no unifying pathogenic mechanism identified 3.1. Misfolded protein/stress response triggered by α-synuclein aggregation 3.2. Defective proteosomal function due to the loss of the E3 ubiquitin ligase parkin 3.3. Altered mitochondrial function caused by the loss of DJ-1 and PINK1 3.4. Genetic variants with gene defects 3.5. Possible damage to dopaminergic cells from toxins drugs/AI conditions	1. 3 of 6 2. Bold + 2

ACEM PRIMARY 2010/2 PATHOLOGY VIVA Friday (September 17) Session: Morning *Candidate Number*…… AGREED MARK……

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 3.1 Type 1 (Immediate) Hypersensitivity	1. What are the features of Type 1 hypersensitivity? 2. What are the actions of mast cell mediators in Type I Hyeprsensitivity (and give examples) 3. What is the late phase reaction	**1.1. Immediate reaction, , previously sensitised individuals, IgE mediated** 1.2. **Mast cell** and or basophils involved 1.3. **Mediators** involved include Histamine, other amines, enzymes proteases, proteoglycans, heparin, leukotrienes, C4, PAF, Prostaglandins, Cytokines **2.1 Cellular infiltration** – leukotrienes, chemotaxis, PAF, Cytokines 2.2 **Vasoactive effects –** Hist, PAF, Leukotrienes, PG D4 **2.3 Smooth muscle spasm –** leukotrienes, histamine, PG, PAF **3 Ongoing inflammatory reaction without additional exposure to triggering ag**	1. 3/5 bold 2. Histamine + 2 others + reasonable actions 3. Ongoing
Question 3.2 Angiogenesis	1. What is angiogenesis? 2. Please give some examples? 3. What steps are involved in angiogenesis from pre existing vessels?	1. **The process of blood vessel formation in the adult.** 2 methods 1.1. Branching and extension of existing vessels 1.2. Recruitment of endothelial progenitor cells (EPCs) 2. Wound healing, chronic inflammation, proliferating endometrium, tumours, etc 3. Steps in angiogenesis 3.1. Vasodilation 3.2. Proteolytic degradation of basement membrane 3.3. Endothelial cells migrate to angiogenic stimuli 3.4. Maturation 3.5. Capillary formation 3.6. Recruitment of periendothelial cells for support structure formation **4.** Inhibitors such as endostatin are released by proteinases (This is a small fragment of collagen that inhibits endothelial proliferation and also angiogenesis)	1. **Bold** and one other 2. **Any 2** 3. **Any 3**

Question 3.3 Pseudo-membranous Colitis	1. Describe the pathogenesis of pseudomembranous colitis. 2. What are the clinical features of pseudomembranous colitis? 3. What is the pseudomembrane?	**1.1. Disruption of normal bowel flora (ab's – esp. 3rd gen ceph) allowing overgrowth of *C. difficile*** **1.2. *C. difficile* elaborates toxins that cause:** 1.2.1. Ribosylation of small GTPases 1.2.2. Disruption of epithelial cytoskeleton 1.2.3. Tight junction barrier loss 1.2.4. Cytokine release 1.2.5. Apoptosis 1.3. Denuded surface epithelium 1.4. Superficial lamina propria contains dense infiltrate of neutrophils & occasional fibrin thrombi in capillaries 1.5. Damaged crypts are distended by mucopurulent exudates that erupt "volcanically" 1.6. Coalesce to form the pseudomembrane 2. Causes fever, (leukocytosis), profuse watery diarrhoea, abdo pain **3. Pseudomembrane is an adherent layer of inflammatory cells and debris at sites of colonic mucosal injury**	1. Toxin + one otherbold + 1 other (1.3 to 1.7) 2. 2/3 3. bold
Question 3.4 Cholecystitis	1. Describe the pathogenesis of acute calculous cholecystitis 2. How does acalculous cholecystitis differ from this? 3. Describe the clinical features of acute cholecystitis.	1. Acute Calculous (90% of all) 1.1. **Obstruction by stones,** stasis- activates hydrolases 1.2. Lecithins -> (mucosal Phospholipases) **-> lysolecithins** 1.3. Disrupts glycoprotein mucous -> **epithelium exposed to bile salts** 1.4. Prostaglandin release -> **inflammation,** mucosal and mural 1.5. **Dysmotility** & **raised intraluminal pressure** **1.6. Bacterial infection secondary to stasis** **2. Acalculous (10%) – rarer, in predisposed individuals, slower often masked** **2.1. Ischaemia, end arteries (cystic)** **2.2. Other promoting features – sludging micro-crystals, stasis, local inflammation, distension** 2.3. Sepsis with hypotension, immunosuppression, major trauma and burns, diabetes, infection, severe atherosclerosis (drugs/ABs- ? vasculitic). 3. Right upper quadrant or epigastric pain, 3.1. Mild fever, anorexia, tachycardia, sweating, nausea, and vomiting, tender RUQ (Murphy's)	1. 3/6 2. 3/6 3. 4/7
Question 3.5 Osteoarthritis	1. What factors lead to osteoarthritis 2. Describe the pathological changes that occur in an affected joint 3. Describe the major clinical features of osteoarthritis	1.1. Genetic & environmental (mechanical) 1.2. Age – virtually ubiquitous (80-90%) after 65 1.3. Other exacerbating diseases e.g. Obesity, diabetes, injury, abnormal joints, 2.**Chondrocyte injury** 1.3.1. Early OA: **chondrocytes proliferate (cloning) and secrete inflammatory mediator**s, collagens, proteoglycans, and proteases which initiates secondary inflammatory changes. 1.3.2. Later OA: repetitive injury and chronic inflammation lead to chondrocyte drop out, marked loss of cartilage, and extensive **subchondral bone changes** 3.Mostly **asymptomatic** <50y.o. 1.4. Deep, achy **pain** worse with use, **morning stiffness**, **crepitus,** and **limited ROM** 1.5. **Oligoarthritis 95%** (occas generalized/early) 1.6. Impingement on spinal foramina by osteophytes results in cervical and lumbar nerve root compression and **radicular pain**, muscle spasms, **muscle atrophy**, and **neurologic deficits**. 1.7. Common: hips, knees, lower lumbar and cervical vertebrae, PIP, DIP of the fingers, 1st carpoMC joints, and 1st TarsoMT joints. Not wrists, elbows, shoulders	1. 2/4 answers 2. 2/3 bold, 3. 2/4

ACEM PRIMARY 2010/2 PATHOLOGY VIVA Friday (September 17) Session: Afternoon *Candidate Number*…… AGREED MARK……

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 4.1 Cellular Events of Inflammation	1. How do leucocytes get to an area of acute inflammation? 2. What is the role of leukocytes in acute inflammation?	1.1 **Margination** of WCC in vessels, rolling and adhesion to endothelium (pavementing) (Selectins) 1.2 **Migration** and **diapedesis** across endothelium (PECAM1, CD31, Integrins) 1.3 **Migration towards chemotactic** stimulus in tissue (bacterial products, cytokines, IL8, C5A) 2 2.1 **Recognition and attachment** to materials (opsonins) mediated by receptors 2.2 Killing of microbes: **phagocytosis /engulfment /killing and degradation** (H2O2-MPO-Halide) 2.3 **Release of products** – Amplify the inflammatory reaction (lysosomal enzymes, reactive oxygen/nitrogen)	1. All Bold 2. 3/5 Bold
Question 4.2 Acute Pancreatitis	1. What is the aetiology of acute pancreatitis? 2. What is the suggested pathogenesis of acute pancreatitis? 3. What are the laboratory findings of acute pancreatitis?	1.1 Metabolic – **Alcohol** 5% (UK), 65% (US), M:F = 6:1, drugs eg. azothioprine, hyperlipoproteinemia, hypercalcaemia, 1.2 Genetic – trypsinogen and trypsin genes 1.3 Mechanical – **Gallstones** 35-60%, M:F = 1:3, trauma, iatrogenic/intraoperative/ERCP 1.4 Vascular – shock, atherosclerosis, vasculitis 1.5 Infectious – mumps 2. 2.1 **Autodigestion of pancreatic substance by inappropriately activated pancreatic enzymes** 2.2 3 mechanisms 2.2.1 **Pancreatic duct obstruction** eg. by impacted gallstone => accumulation of lipase in interstitium => local fat necrosis => release of proinflammatory cytokines => leaky vessels + oedema => vascular insufficiency and ischaemic damage to acinar cells 2.2.2 **Primary acinar cell injury** eg. alcohol, mumps, trauma, drugs, organ insufficiency aftershock/ischaemia 2.2.3 **Defective intracellular transport of proenzymes within acinar cells** – digestive enzymes and lysosomal hydrolases intermingled causing release of activated enzymes. Human mechanism not clear. 3 **3.1 Marked elevation of serum amylase** in first 24 hours **3.2 Rising serum lipase** within 72-96 hours **3.3** Glycosuria – 10% cases **3.4** Hypocalcaemia – poor prognostic sign if persistent **3.5** Leukocytosis **3.6** Acute renal failure	1. **Bold** + 2 of the other causes from different groups **2.1** **Bold** to pass **2.2** **2 of 3** bold **3.** **Bold** + 2 others to pass
Question 4.3 Abdominal Aortic Aneurysm	1. Describe the pathogenesis of an aneurysm 2. What are the clinical consequences of an AAA? 3. What is the risk of rupture of an AAA?	1. **Structure or function of the vascular wall connective tissue is compromised** 1.1. **Poor intrinsic quality of the vascular wall connective tissue eg Marfan syndrome**, Ehlers-Danlos 1.2. **Collagen degradation vs synthesis by local inflammation (** proteolytic enzymes**) eg atherosclerotic plaque, vasculitis,** 1.3. **Loss of vascular smooth muscle cells** or the inappropriate synthesis of noncollagenous or nonelastic ECM (cystic medial degeneration) 2. 2.1. **Rupture** into the peritoneal cavity or retroperitoneal tissues with massive, potentially fatal haemorrhage 2.2. **Obstruction of a branch vessel** resulting in ischemic injury, eg. iliac, renal, mesenteric, or vertebral arteries 2.3. **Embolism** from atheroma or mural thrombus 2.4. **Impingement** on an adjacent structure, e.g. ureter, vertebrae 2.5. Nothing (if < 4cm and no embolic complic's) 3. Related to size - 3.1 4 cm or less in diameter nil 3.2 between 4 and 5 cm 1% per year 3.3 between 5 and 6 cm 11% per year 3.4 greater than 6 cm in diameter 25% per year	1. 2/3 **bold**, 2 examples 2. 3 out of 5 3. Low < 5cm, much higher > 5cm

Question 4.4 Iron Deficiency Anaemia	**1.** What is the aetiology of Fe deficiency anaemia?	**1.1. Chronic blood loss – GIT, menorrhagia** 1.2. Increased requirement – pregnancy 1.3. Dietary deficiency – vegetarians 1.4. Impaired absorption – celiac	1. Bold + 1
	2. What are the laboratory findings in Fe deficiency anaemia?	2. 2.1. **Microcytic hypochromic anaemia** (low Hb) 2.2. Low S. Fe levels 2.3. Low S. Ferritin levels (correlates well with body iron stores) 2.4. High TIBC (high transferrin levels) 2.5. Low Transferrin saturation levels	2. Bold +3
	3. What are the clinical features of Fe deficiency anaemia?	3. 3.1. **General** - pallor, weakness, lethargy, fatigue, SOBOE, angina 3.2. Features of blood loss – GI, menorrhagia 3.3. **Specific** features – koilonychia, alopecia, glossitis, pica	3. At least 5 from 2 groups
Question 4.5 E. coli Gastroenteritis	List the types of E. Coli enteritis and describe their features	**1.1 Enterotoxic E coli** (ETEC) **1.1.1** Food and water, traveller's **1.1.2** **LT heat labile toxin**, adenyl cyclise **-> inc cAMP** -> **inc Cl- secretion** and **decr absorption (cholera like)** **1.1.3** ST **heat stable toxin**, guanylate cyclase -> **incr cGMP** **1.2 Enterohaemorrhagic E coli** (EHEC) **1.2.1** **Beef esp. ground**, milk vegetable **1.2.2** **O157:H7** and non O157:H7 **1.2.3** **Shigella like toxin** **1.2.4** Large outbreaks, **bloody diarrhoea**, **haemolytic uraemic syndrome** **1.2.5** Thrombotic Thrombocytopenic purpure (2%) **1.3 Enteroinvasive E. Coli** (EIEC) **1.3.1** Food, water, person to person **1.3.2** No toxins, invades mucosa, colitis **1.4 Enteroaggregative E. coli** (EAEC) **1.4.1** Adheres via **adherence fimbriae**. **1.4.2** Dispersin (removes –ve charge/ protection) **1.4.3** Shigella like toxin and ETEC ST toxin **1.4.4** Non bloody diarrhoea, prolonged in AIDS	2 of 4 groups to pass 1 feature of any two

ACEM PRIMARY 2010/1 ANATOMY VIVA **Day: Thursday** **Session: am** *Candidate Number*..... AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: Xray:Facial	(a) Demonstrate the walls of the orbit on this xray and name the bones that form them. Prompt: What bone forms each wall? (b) Demonstrate the position of the infra-orbital nerve on this xray. (c) What structures does the infra-orbital nerve innervate?	Roof – orbital part of frontal b and posteriorly the lesser wing of the sphenoid. Medial- ethmoid with contributions from frontal process of maxilla, lacrimal and sphenoids Lateral – frontal process of zygomatic b and the greater wing of sphenoid Floor – Maxilla and partly by zygomatic and palatine bone Mucosa of max sinuses **Upper medial teeth** (Premolars, canines, incisors) **Skin of cheek** Skin of lateral nose Skin/conjunctiva of inferior eyelid Anteroinferior nasal septum **Skin and oral mucosa of upper lip**	**All 4 walls and** **Frontal** **Maxilla** **Ethmoid** **Zygomatic** Need Bold to pass Demonstrate region of infra-orbital foramen/notch Bold to pass

Question 2:
 	
Bone: Pelvis
	a. What bones make up this structure, and what are their major features?	a. Pelvis
		• **Acetabulum** (with acetabular notch)
		• **Obturator foramen** (with obturator groove)
		• **Ilium**
		  ○ Ala
		  ○ **Iliac crest**
		  ○ Inferior, anterior and posterior gluteal lines
		  ○ **Anterior superior**, anterior inferior, posterior superior and posterior inferior iliac spines
		  ○ Greater sciatic notch
		• **Ischium**
		  ○ **Ischial spine**
		  ○ Lesser sciatic notch
		  ○ Ischial tuberosity
		  ○ Ischiopubic ramus
		• **Pubis**
		  ○ **Superior pubic ramus**
		  ○ Pubic symphysis
			a. Need all bold to pass, and able to demonstrate aspects of bony parts

	b. What are the lateral rotators of the femur, and where do they originate?	b. Lateral rotators of hip
		• **Piriformis**
		  ○ anterior sacrum and sacrotuberous ligament
		• **Obturator internus / (externus)**
		  ○ Pelvic/ext surface of obturator membrane and surrounding bones
		• **Superior gemellus**
		  ○ Ischial spine
		• **Inferior gemellus**
		  ○ Ischial tuberosity
		• **Quadratus femoris**
		  ○ lateral border of ischial tuberosity
		• **Gluteus maximus** (minor)
		  ○ ilium posterior to posterior gluteal line, dorsal surface of sacrum and coccyx, sacrotuberous ligament
			Need 2 to pass

Question 3: Model: Heart (Heart model assembled at start of question, remove aorto-pulmonary root as question asked)	a) Using this model, describe the arterial supply of the heart	Main coronary vessels arise from **corresponding aortic sinus** above aortic valve. **R coronary** courses inf in av groove Gives off branches to SA node **Marginal** **Post interventric** AV nodal\ **L coronary** bifurcates into **Circumflex** and **LAD (anterior I – V art)** Circ gives off **Marginal** branch, and LAD gives off **diagonals.**	5/9 bold to pass
	b) What does the R coronary artery supply?	**R atrium** **Most of RV** **Diaphragmatic surface LV** **Post 1/3 septum** **60% SA** **80% AV**	3/6 bolded to pass
	c) Demonstrate the venous drainage of the heart (BONUS MARKS)	Major drainage is via the **Coronary sinus** 3 main tributaries are **great**(accompanies LAD, then Circ), **middle** (accompanies PIV) and	3/6 to pass

		small cardiac veins (accompanies R marginal). Oblique vn L atrium marks start of sinus. **Ant cardiac vn's** start ant surface RV, drain straight into r atrium **Smallest cardiac vn's** (venae cordis minimae) drain direct into chambers	
Question 4: Photo: Brachial Plexus	a) Identify the components of the brachial plexus as shown in this photo. The vessels have been removed. PROMPT: Direct candidates to start at superior aspect. b) Identify the muscles visible in this photo	**Posterior Cord** **Radial Nerve (Terminal br)** Axillary Nerve (Terminal br) Thoracodorsal n Upper and Lower Subscapular nn **Lateral Cord** Musculocutaneous n (terminal br) Lat root of median n (terminal br) **Medial Cord** **Ulnar nerve (Terminal Br)** Med root of median nerve (terminal br) Medial cut nn of arm and forearm **Median nerve** Surrounding muscles: Deltoid, biceps, coracobrachialis, pec minor, triceps, lat dorsi, subscapularis, teres major	Identify – 3 cords, 3 bolded branches and 2 others to pass Identify 3 muscles to pass
Question 5: Discussion	Describe the superficial venous drainage of the lower limb	Dorsal v arch of foot drain to GSV, ascends ant to MM, then behind med fem condyle (hand breadth post to patella), then up med thigh through fascia lata in saph opening into fem V. Numerous valves, perforators to deep system and anastomoses with SSV. Laterally, Small SV arises from dorsal venous arch, ascends behind LM, lateral to Achilles, penetrates fascia at mid-line, between heads of gastroc to join popliteal vein	1.Identify MM, fem condyle and saph opening landmarks of GSV 2.Name SSV and general location 3.Identify connection with deep system via perforators

ACEM PRIMARY 2010/1 ANATOMY VIVA Day: Thursday Session: pm *Candidate Number*..... AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: Xray : lateral C spine	(a) Identify the major bony features of the cervical spine on this xray.	**Atlas** - Anterior and posterior arches **Axis –Dens,** spinous process **C3-7: Body**, pedicle, lamina, superior and inferior articular process, **spinous process** **Zygapophysial (facet) joint** Intervertebral disc space	Bold to pass
	(b) Describe the ligaments which maintain alignment of the cervical spine. Prompt: Which ligaments support the dens?	**Anterior Longitudinal ligament** / Anterior atlanto-occipital membrane **Posterior longitudinal ligament** / Tectorial membrane Ligamentum flavum / Posterior atlanto-occipital membrane Interspinous ligaments Supraspinous/nuchal ligaments Intertransverse ligament **Transverse ligament of the atlas** Cruciate ligament Alar ligament	Bold plus 2 others
Question 2: Bone: First rib	a. What bone is this? PROMPT: What side is it from?	a. **First rib** **Appropriate side**	a. Need all bold to pass

	b. What are the bony landmarks?	b. Landmarks
		• **Head** (with single facet for T1)
		• Neck
		• **Tubercle** for transverse process of T1
		• Superior surface, medial to lateral:
		  ○ **Groove for subclavian artery**
		  ○ **Scalene tubercle and ridge**
		  ○ **Groove for subclavian vein**
		• Costal groove
			b. Need all bold to pass

	PROMPT: What vessels are related to this bone? c. What muscles attach to this bone?	c. Muscles • **Anterior scalene** (tubercle) • **Middle scalene** (medial to groove for artery) • Longissimus portion of erector spinae attaches between tubercle and angle • **Intercostals** • Subclavius (at costochondral jn)	c. Need all bold to pass

		• Serratus anterior (lateral)	
Question 3: Model: Elbow	a. Demonstrate the bony features of the elbow joint. b. Demonstrate the capsular attachments of the elbow. c. Describe the collateral ligaments of the elbow. d. Describe the movements of the elbow joint	Humerus, Olecranon of ulna, and Radial head. Other features are coronoid of Ulna, Trochlea, Capitulum, lateral and medial epicondyles, olecranon fossa, coronoid fossa Fibrous layer: surrounds whole joint, proximally above the coronoid and olecranon fossae, distally across neck of radius and adjacent ulna. Synovial layer, lines the internal surface of fibrous layer and non articular parts of the humerus….is continuous distally with synovial membrane of prox radioulnar joint **Radial-** lat epicondyle of humerus and blends distally with annular ligt of radius. Ulnar- medial epicondyle of humerus to 1) coronoid tubercle (**anterior band**)..strongest 2) **Posterior fan like band** weakest and 3) slender **oblique band.** Flexion Extension	7/10 to pass Need either good description of upper or lower margin to pass 2/4 bold to pass Both to pass (Pronation/supination is critical error)
Question 4: Photo: Female Pelvis	a. This is a midline sagittal section of a pelvis. Name the major anatomical structures. PROMPT: this is a female pelvis b. Describe the boundaries and relations of the Pouch of Douglas	Major: **Pubic symphysis, Bladder, Vagina, Uterus, Rectum, Sacrum, Blue marker through cervix., External anal sphincter.** Minor: Ovary, Tube, suspensory ligament (difficult), L5/S1 disc, Sigmoid, Ureter (difficult) "Recto-uterine pouch". Inferior most extension of the peritoneal cavity, between anterior rectum and posterior uterus. Close to cervix and posterior fornix of vagina. Open above to peritoneum	5 of bold to pass Rectum, uterus, open above.
Question 5: Discussion: Common fibularis (common fibular n/common peroneal n)	a. Outline the course of the common fibular nerve and its main branches. b. What does it supply? (Motor and sensory)	Origin: from **sciatic n** as it bifurcates in **apex pop fossa** Passes over **post head of fib** and then winds around neck of fib Divides into **sup and deep fib n,** also br to knee jt. Common- supplies **skin posterolat leg** Superficial br –**motor supply to lateral compartment**, and sensory supply **distal 1/3 ant leg and foot** Deep branch motor to **ant mm of leg and dorsum of foot**, and Sensory to **1st web space foot.**	For a pass: Origin, Fib head, Main branches, Motor and sens supply

ACEM PRIMARY 2010/1 ANATOMY VIVA Day Friday Session: am *Candidate Number*..... AGREED MARK.....

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: Xray: CT abdomen	a. Identify the intra-abdominal structures visible on this CT scan	**Liver**/ porta hepatis /duodenum/IVC / **pancreas** /splenic vein/**kidneys/spleen/ aorta**/ coeliac trunk/ crus of diaphragm/ small bowel	Bold + 2 to pass
	b. Describe the relations of the right kidney (see diagrams Moore page 291-293,324)	Surrounded by peri-nephric fat Superiorly- R adrenal + liver+ portal vein Supero-laterally- Right lobe of liver Medially-Psoas + vertebrae Posteriorly- 12th Rib + abdo muscles(TA, IO, EO),deep back muscles (erector spinae/quadratus lumborum) Anteriorly- gall bladder + duodenum + ascending colon Antero-medially-R renal vein + IVC, pancreas more anteriorly	Correct structures related in 3 directions to pass

Question 2:
 	
Bone: Femur
	a. Identify this bone, and identify the significant bony landmarks of its proximal portion
	
	PROMPT: Ask for side if needed
		a. Femur, appropriate side (head superomedially, distal intercondylar fossa posterior)
		Landmarks:
		• **Head**
		  ○ fovea for ligament of the head
		• **Neck**
		• **Lesser Trochanter**
		• **Greater trochanter**
		• Intertrochanteric line (anteriorly)- continues as spiral line
		• **Intertrochanteric crest** (posteriorly)
		  ○ Quadrate tubercle
			a. Need all bold

	b. What is the blood supply of the neck and head of the femur?
	
	PROMPT: Which is the main supply?
		b. Blood supply to neck and head
		  ○ Medial and lateral circumflex femoral aa
		  ○ Usually branches of deep artery of thigh (profunda femoris)
		  ○ Branch to form retinacular aa (from medial>lateral), feed under the posterior unattached capsule (med) or through the iliofemoral ligament (lat)
		• Artery to the head of the femur (less)
		  ○ Branch of obturator a
		  ○ travels in the ligament of the head
			b. Need to demonstrate understanding of dual supply, and relative contributions (circumflex aa>a of head of femur)

	c. How does the capsule of the hip joint attach on this bone? (BONUS)	c. Capsule of hip joint on femur • Most fibres spiral around the joint to attach at the intertrochanteric line and root of greater trochanter • Posteriorly, fibrous layer crosses the neck proximal to the intertrochanteric crest, but is not attached • deep fibres pass circularly around the neck to form orbicular zone • The capsule thickens into three ligaments: o Iliofemoral- intertrochanteric line o Pubofemoral- merges with fibrous capsule o Ischiofemoral- into femoral neck, medial to the base of the greater trochanter	c. Be able to demonstrate understanding of capsular attachment
Question 3: Model: Larynx	a. Identify the structures that make up the larynx PROMPT: Identify the structures that make up the larynx b. Describe the nerve supply of the larynx	Single Cartilages: **thyroid, cricoid, epiglottis** Paired Cartilages: arytenoids, corniculates and cuneiforms Ligaments/Membranes: *thyrohyoid memb; cricotracheal, hyoepiglottic and thyroepiglottic ligaments* Intrinsic: quadrangular memb, **cricothyroid membrane,** includes cricothyroid ligs , vestibular fold/ligaments, **vocal cords** (are the upper part of the cricovocal memb), **ari-epiglottic fold/ligament** Muscles: Extrinsic and Intrinsic – eg cricothyroid and arytenoid muscles Recesses: vallecula, piriform fossa Nerve supply: Motor: (Cranial X- vagus N) **Infr laryngeal nerve** (continuation of Recurrent laryngeal nerve) supplies all muscles except cricothyroid, and sensory below the cords (**Ext laryngeal N** supplies Cricothyroid M – tenses the cords) Sensory: Internal Laryngeal N above the cords (sensory and autonomic) and NB Int and Ext Laryng N are terminal brs of Supr Laryng N	**Bold to pass**
Question 4: Photo: Upper Limb	Describe the superficial venous drainage of the upper limb. Use this photograph if you need to. PROMPT: identify the veins first. Once identified, track distally and describe proximal path	Hand: dorsal and palmar networks drain to **cephalic** (anterolateral) and **basilic** (posteromedial, around medial epicondyle). Connected in cubital fossa by **median cubital.** Basilic goes deep distal/middle 1/3s of arm with axillary artery to become axillary vein. Cephalic in deltopectoral groove, deep through DP fascia,	Bold to pass

		inferior to clavicle.	
Question 5: Discussion	a. Draw or describe the circle of Willis	**IC x 2 → MCAx2 and ACAx2** **VA x2 → Basilar x1→PCAx2** ACA linked by ant communicating artery PCA each join IC by post comm. Art	a. Identify 3 paired arteries and their origin
	b. Which part of the brain is supplied by each of the major arteries?	ACA – medial/sup surface of cerebrum except occ MCA – lateral surface and temporal lobes PCA – occipital lobe, inferior cerebrum	b. Name supplied area of at least one major artery

ACEM PRIMARY 2010/2 ANATOMY VIVA 16th Sept MORNING ***Candidate Number……..…*** **AGREED MARK……**

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: **Soft tissues on CXR**	Demonstrate the borders of the mediastinum on this Xray	SVC RA RV (Apex) (L ventricle) L Auricular appendage Pulmonary trunk Aorta R brachiocephalic v.	At least 6 correct to pass
Structures transected at the sternal angle	In the supine position, which mediastinal structures are located at the same level as the sternal angle. (Prompt: What mediastinal structures would you see if you looked at a transverse slice though the chest at the level of T4-5?)	**Mediastinal structures** Carina (bifurcation) Division of pulmonary trunk Reflection of the pericardium SVC (enters R atrium) Hila of the lungs Transverse fissure of R lung Ascending aorta becomes arch Arch becomes descending Aorta Phrenic nerve Vagus nerve L recurrent laryngeal nerve origin Azygos vein Thoracic duct (crosses from R to L) Pleura approaches the midline anteriorly	At least 6 correct to pass
Question 2: **BONE: Sacrum**	a) Identify the features of this bone?	**Sacrum** consists of 5 fused bones and the coccyx **4 pairs of sacral foramina** – S1-S4 anterior larger than posterior **Ala** **Sacroiliac joint** **Superior Articular facets** **Lumbrosacral joint** **5 Vertical lines** – median, intermediate and lateral	**Any 4** to pass

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 3: **Model: Hand**	Could you please identify the muscular structures visible in the hand of this model? (**Prompt** away from thenar/hypothenar muscles)	Potentially 10 ..3 thenar (op, apb, fpb) 3 hypothenar (adm, fdmb, odm), Add Poll, Lumbrical, Dorsal and palm int 6/10	5 to pass
	Could you demonstrate the actions produced by the lumbricals and the interossei and describe their innervation?	**Lumbs** do Z, and PAD/ DAB for **interossei**, along with extension.. actions are **combined** to produce Z All deep br ulnar, except for lat 2 lumbs..median nerve..,	All to pass
	Could you demonstrate the origins and insertions of the short muscles of the hand?	**Lumb**..orgn1,2 lat side of lat 2 tendons of fdp, 3, 4 bipennate from med 3 tend fdp, **dors int** orgn bipenn from adjacent mc's, insert base prox ph, ext expans, **palm int** palm surface 2,4,5 mc, ins as for dors, 2,4,5	**Bonus**
Question 4 **Photo: Gluteal Area**	a) This is a photograph of the gluteal region. Identify the structures. Prompt if needed – what is this (Sciatic Nerve)	15-**Piriformis** **Sciatic N**: 23-Tibialpart; 1-Common Fibular (Peroneal) part 2-**Gluteus maximus**; 16-Post Fem Cutaneous N 13-Obturator Externus 18-Quadratus femoris 7-Inferior gluteal art. 17-Pudental N; 9-Internal Pudental art; 11-N to Obturator Internus 20-Superior Gamellus; 14-Obturator Internus 6-Inferior Gamellus 21-; 22-; 8-Inferior gluteal N 3-Gluteus medius; 4-Gluteus minimus 5-Greater Trochanter Femur 19-Sacrotuberous Ligt 10-Ischael Tuberosity	**2 Bold** plus 2 others to pass

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 4 cont'd **Photo: Gluteal Area**	b) Describe the course of the Sciatic Nerve, and the muscles it supplies.	Enters gluteal region via greater sciatic foramen **inferior to piriformis** and **deep to gluteus maximus;** decends in midline posterior thigh deep to biceps femoris; **bifurcates** into **tibial and common fibula** (perioneal) nerves at **apex of popliteal fossa** No supply in gluteal region. Supplies all **muscles of posterior compartment of thigh** (common fibula short head biceps, tibial division all the rest)	**Bold** to pass
Question 5: **Discussion: Facial Nerve**	What is the motor supply of the muscles of facial expression? Describe its course. Discuss the non motor component of the facial nerve.	7th cranial nerve Temporal bone Stylomastoid foramen -posterior auricular Parotid gland - temporal - zygomatic - buccal - marginal mandibular - cervical Intermediate nerve Taste join lingual nerve ant 2/3 tongue Parasympathetic Somatic sensory	Facial n and 3 terminal branches Bonus question.

ACEM PRIMARY 2010/2 ANATOMY VIVA 16th Sept afternoon ***Candidate Number*……** **AGREED MARK……**

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: **CT Head**	Identify the non – bony features on this CT scan.	Orbits Temporal lobes in middle cranial fossa Pons 4th ventricle Cerebellum and vermis	Need temporal lobe, pons and cerebellum
	Which bony sinuses are shown?	Ethmoid, sphenoid, mastoid	2/3
	What is the blood supply of the cerebellum?	Vertebral arteries – basilar artery – post cerebral Ant & post inferior cerebellar art Superior cerebellar art	Need posterior circulation
Question 2 **Bone: Femur**	a) Identify the landmarks of this bone	**Head**, **fovea**, neck **Greater trochanter, lesser trochanter**, Trochanteric fossa intertrochanteric line intertrochanteric crest pectineal line **shaft and/or linea aspera** medial / lateral supracondylar lines adductor tubercle medial / lateral epicondyles **medial / lateral condyles** intercondylar fossa	**Bold** plus 3 others to pass
	b) Demonstrate the attachments of the adductor muscles of the hip.	Adductor longus - Middle 1/3 linea aspera Adductor brevis - Pectineal line and proximal linea aspera Adductor magnus - Adductor part – linea aspera, medial supracondylar line Hamstring part (not strictly in this Q) adductor tubercle [Gracilis] Not femur (tibia)] Pectineus Pectineal line inferior to lesser trochanter Obturator Externus Trochanteric fossa	3 to pass

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 3: **Model Thumb**	a. Could you please identify the muscles of the thenar eminence, and demonstrate their origins and insertions? b. Please demonstrate the movements produced by the thenar muscles. What nerves innervate these muscles?	APB, FPB, OP (all originate fl ret and scaphoid/trap tubercles) apb inserts lat side base prox phal, op inserts lat 1st mc, and both heads fpb insert lat prox phal. base Op opposes (mc to middle palm, rotates), abd abducts, helps opposition, fl flexes..all recurrent br. Med n, except dp hd fpb…deep br ulnar	3/6 pass
Question 4: **Photo: Anterior Triangle neck**	a. Define the boundaries of the anterior triangle of the neck. b. The internal jugular vein has been removed. Name some structures in the anterior triangle c. Name the branches of the external carotid	SCM, midline, mandible Muscle – SCM, strap muscles Lymph nodes – jugulo-digastric Artery – common carotid, int and ext carotid, sup thyroid, lingual, facial Vein – Branchiocephalic, subclavian Nerve – recurrent laryngeal Ant. Asc. pharyngeal, superior thyroid, lingual, facial Post. Occipital, post auricular, superificial temporal, maxillary	(need all 3) 3 to pass 2 to pass
Question 5: **Surface Anatomy of the Pleura**	Describe the surface anatomy of the parietal pleura. What is the clinical significance of the attachment of the pleura? (Prompt – Are there any parts of the pleura that are more likely to be injured?)	Sternoclavicular joint to midline at SM joint Passes inferiorly parasternally to 6th ICC on R and 4th ICC on left where it deviates to the left. 8th rib in MCL 10th rib in MAL 12th rib in PAL Small section medial to this is inferior to the 12th rib Posteriorly - parallel to vertebral column to T1 Cupola rises 2-3 cm above medial 1/3 of clavicle at the neck of the 1st rib Cervical pleura may be injured Deviation of pleura to the left provides a window for pericardiocentesis without traversing the pleura. The attachment of the pleura at a lower level than the lungs (posterolaterally) favours collection of pleural fluid in this area (drainage, clinical findings). Penetrating injuries to the upper lumbar region	At least 5 correct to pass At least 2 to pass

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1 **XR: Pelvis**	Identify the bony features of this x-ray	**Iliac crest,** Ala of ilium **Sacro iliac joint, Sacrum** Lumber vertebrae Pelvic brim Anterior superior iliac spin Anterior inferior iliac spine Ischial spine Ischial turberosity **Obturator foramen** Acetabular fossa **Superior rami** **Inferior rami** Symphysis pubis	(a) **6 Bold** + 3 others to pass
	Describe the anatomy of the iliopsoas muscle.	**Iliopsoas** – consists of **Iliacus** & **Psoas major** **Psoas major:** Superior attachment (a) Transverse process of lumbar vertebrae (b) Sides of vertebral bodies T12-L5 (c) Intervertebral discs T12- L5 Inferior attachment (a) **Single tendon to lesser trochanter of femur** Innervation (a) Anterior rami of L1, L2, L3 **Iliacus** Superior attachment (a) Superior 2/3 of iliac fossa (b) Ala of sacrum (c) Anterior sacro-iliac ligaments Inferior attachment (a) **Lesser trochanter of femur and shaft inferior to it** (b) Psoas major tendon Innervation (a) **Femoral nerve** L2- L4	Name two muscles and origin and insertion

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 2: **Bone: SKULL**	Show me which bones make up the orbital rim?	Orbital rim: Frontal, Zygomatic, Maxilla	**Pass Criteria:** Need 2/3
	Describe the course of the infra-orbital nerve?	Entrance into the orbit via the inferior orbital fissure Traverses infra-orbital groove and canal in orbital floor. Emerges via infraorbital foramen	Need Inferior orbital foramen
	What does the infra-orbital nerve supply?	Mucosa of maxillary sinus; premolar, canine, and incisor maxillary teeth; skin and conjunctiva of inferior eyelid; skin of cheek, lateral nose, and anteroinferior nasal septum; skin and oral mucosa of superior lip.	Need cheek, superior lip, upper teeth
Question 3 **Model: Ankle**	a) Identify the ankle dorsiflexors on this model?	1) tibialis anterior 2) Extensor digitorum longus 3) extensor hallucis longus 4) fibularis (peroneus) tertius	**3/4 to pass**
	b) What is their nerve supply? c) Identify the insertions?	All supplied by deep fibular (peroneal) nerve (L4) 1) TA medial cuneiform and base 1st MT 2) EDL middle and distal phalanges lateral 4 digits 3) EHL base distal phalanx hallux 4) FT 5th MT	**know nerve and 3/4 to pass**
Question 4: **L wrist & hand photo**	a) Identify the median nerve in this photo and adjacent structures.	16. Median n (15. Flexor retinaculum (anterior) – divided) 12. Flexor digitorum superficialis (posterior) 14. Flexor pollicis longus (lateral) 11. Flexor digitorum profundus (deep posterior) 18. palmar cutaenous branch of median n	Median n and 2 other structures to pass
	b) Demonstrate where sensation changes may occur if the median nerve is injured in the forearm.	Palmar 3 ½ digits, adjacent palm and dorsal distal fingers	Finger distribution to pass

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Q 4, cont.	c) Demonstrate these changes if the median nerve is compressed in carpal tunnel syndrome	Same, except palmar area is preserved (palmar cutaneous branch arises proximal to flexor retinaculum	Bonus question
Question 5 **Discussion: Abdomen surface anatomy + Transpyloric plane**	(a) Describe the transpyloric plane.	Transpyloric line (halfway between manubrium and symphysis pubis- typically at L1) Or half-way between the xiphisternum and umbilicus	
	(b) What are the anatomical structures transected at the transpyloric plane?	Pylorus Fundus of gall bladder Neck of pancreas SMA origin Hepatic portal vein Root of transverse mesocolon Duodenojejunal junction Hila of kidneys (L above R below) Hepatic and splenic flexures of the colon Conus medullaris	4 to pass

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: **PEG XR**	Demonstrate the bony features of the upper cervical vertebrae on this x-ray? What ligaments stabilise these bones?	Lateral mass of Atlas (C1), Body of Axis (C2), Dens of Axis (C2), Lateral atlanto-axial joint, Spinous process of Axis (C2) Cruciate ligament Alar ligament Anterior longitudinal ligament = anterior atlanto-axial membrane = anterior atlano-occipital membrane Posterior longitudinal ligament = tectorial membrane Ligamentum flavum = posterior atlanto-axial membrane = posterior atlano-occipital membrane Nuchal ligament Interspinous ligament	**Pass Criteria** Need lateral mass, dens, body, axis Need cruciate ligament, alar ligament + 1 other
Question 2: **Bone: Ankle Joint**	a) identify the bony landmarks of the ankle ▪ prompt if not provided – what are the features of this bone (point at talus or name if already named) b) Name the structures passing behind the medial malleolus	▪ lat malleolus ▪ medial malleolus ▪ talus ▪ trochlea talus ▪ head talus ▪ neck talus ▪ body talus ▪ lateral tubercle talus ▪ medial tubercle talus ▪ groove for flexor hallucis longus ▪ Tibialis posterior ▪ Flexor digitorum longus ▪ Posterior tibial artery ▪ Tibial nerve ▪ Flexor hallucis	**5/10 to pass** **3/5 to pass**
Question 3: **Model of Heart**	Identify the chambers of the heart on this model Demonstrate where the major components of the conducting system would be found on this model	**RA, LA, RV, LV,** SAN – junction of SVC and RA AV-node – postero-inferior interatrial septum near coronary sinus AV bundle Right and left bundles	All 4 correctly identified to pass. Name major parts and generally accurate location

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 4: **Photo: Cubital fossa**	1 Identify the contents of the cubital fossa shown in this photograph.	(Boundaries - PT, BR, line b'n epicondyles.) Contents - **Median n, Brachial a, Biceps tendon,** Radial n, veins with Brachial a	Bold = required for pass
	2. Demonstrate the course and branches of the Radial nerve, and name the structures they supply	2. Radial n under BR muscle laterally**, divides into superficial and deep, former through Supinator to post. compt. muscles/jt., lateral to ant. compartment forearm sensory only to dorsum hand**	Bold to pass
	3. Which veins in the cubital fossa are usually accessed during venepuncture? What are the commonly observed variations to these vessels?	3. Median cubital, **Basilic and Cephalic v's** Median basilic and median cephalic in 20%	(Optional)
Question 5 **Discussion: Abdominal Aorta – Branches p313**	(a) Name the branches of the abdominal aorta	Anterior midline branches -**Celiac** -**Superior mesenteric** -**Inferior mesenteric** Lateral branches -Supra-renal -**Renal** -Gonadal Posterolateral -Subcostal -Inferior phrenic -Lumbar	4 **Bold** to + 3 others to pass
	(b) Describe the anatomy of the superior mesenteric artery.	Origin L1 level, midgut vessel, pancreas above, duodenum below, L renal v below	

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TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: Reperfusion Injury	(a) What is reperfusion injury?	Further **cell death** in ischaemic tissues following **restoration of blood flow**	(a) Highlighted
	(b) What are the proposed mechanisms of reperfusion injury?	1. **Generation of oxygen free radicals** – formed from incomplete reduction of in-coming O2 by damaged mitochondria in affected tissue and action of oxidases (generated from ischaemic cells and leucocytes) 2. **Associated inflammation** - cytokines, adhesion molecules generated by hypoxic cells; they recruit neutrophils etc in re-perfused tissue; ensuing inflammation causes additional injury 3. **Activation of complement system** – IgM Ab deposit in ischaemic tissue; restored blood flow brings complement proteins that bind to Ab and are activated; causing further cell injury and inflammation 4. Mitochondrial permeability transition – via reactive O2 species – effects mitochondrial function - precludes recovery of ATP / energy supplies for the cell	(b) 2 for pass
Question 2: Apoptosis	a) What is apoptosis ?	**Programmed cell death** / " suicide programme"	
	Prompt *Describe features and purpose of apoptosis*	Remove degraded- un needed cells, Stop excess growth, Tightly controlled Activates degradation enzymes, Intact membrane packaging(es), Phagocytosis encouraged = end point **Non inflammatory**,	Physiological or pathological initiators/ Capsases/ Intrinsic/ extrinsic paths Mitochondrial v death receptor
	b) List some important stimuli for apoptosis ?	a) loss of growth/ stimulating hormones (e.g. GH, nerve growth, loss of sex hormones) b) excessive DNA damage (via p53 build up) c) unfolded protein build up d) developmental atrophy, (embryogenesis) e) proliferative tissues- homeostasis – non useful cells/ excess to function- loss of contact inhibition f) loss of useful cells after finished purpose (e.g. neutrophils/ lymph post infl) g) cells with harmful characteristics (e.g. autoimmune antigens /xs mutations) h) infections (viral leading to cell death) i) parenchymal damage after duct obstruction	3 concepts
Question 3: Emphysema	1. What is the pathological definition of emphysema?	"Emphysema is a condition of the lung characterised by abnormal permanent **enlargement of the airspaces** distal to the terminal bronchiole, accompanied by **destruction of their walls**, & without obvious fibrosis"	**Bold to pass** Permanent Enlargement + destruction
	2. Describe the pathogenesis of emphysema	**Protease – antiprotease theory** ***Alveolar wall destruction results from an imbalance between proteases (mainly elastase) and antiproteases*** **Elastases** from **Neutrophils**, also Macrophages, Mast cells, pancreas, bacteria **Anti-elastases**: **α_1AT**, secretory leukoprotease inhibitor, serum α_1 macroglobulin **α_1AT** inhibits neutrophil proteases. PiZZ variant predisposes to emphysema Neutrophils normally sequestered in lung (L > U) and a few gain access to the alveolar space. - Any stimulus that ↑ number of leukocytes (neutrophils / macrophages) in lung or release of their elastase containing granules ↑ elastocytic activity. - Stimulated neutrophils also release **oxygen free radicals** which inhibit α1-AT activity meaning process of elastic tissue destruction is unchecked	Know that key is imbalance between proteases (mainly elastase) and antiproteases
	3. What is the role of cigarette smoke?	• Smokers have ↑ **neutrophils & macrophages in alveoli**, - smoking stimulates neutrophil chemotactic factor (e.g. IL-8), nicotine chemotactic, smoke activates alternative complement pathway • **Smoking stimulates release of neutrophil elastase,** proteinase 3, Catepsin G • Smoking ↑ elastase activity in macrophages (not inhib by α1-AT) • Reactive oxygen species in cigarette smoke deplete glutathione and superoxide dismutase Note centri-acinar distribution due to impaction of smoke particles in small bronchi / bronchioles with neutrophil influx. Differs to pan-acinar emphysema associated with α1-AT deficiency and chronic low level proteolysis from neutrophils in transit through the lung circulation.	2 effects

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Question 4: Aortic dissection	a) Describe the pathogenesis of an aortic dissection.	a)Medial weakness (commonly from **hypertension**), medial hypertrophy vasa vasorum, **intimal tear**, blood flow dissects the media -> medial haematoma. Cystic medial degeneration Risk factors - HT, CT disease eg Marfan's, Ehlers-Danlos, iatrogenic, pregnancy,	**Bold to pass**
	b) How are aortic dissections classified?	**By site of involvement, proximal (A) and distal (B),** DeBakey I, II, III I – ascending and descending II – ascending only III – descending only (better prognosis)	**bold**
	c) What are the potential consequences of the disease?	Rupture back into intima or out through adventitia Most common cause of death is rupture into pericardial, pleural or peritoneal cavities Other outcomes include cardiac tamponade, aortic insufficiency, MI, extension into any of the branches of the aorta causing obstruction +/- ischaemia, transverse myelitis	At least 3
Question 5: Meningitis	a) Classify meningitis with examples of important causes.	- Acute pyogenic: bacterial - Aseptic: viral, chemical - Chronic: infection: TB, infiltration: carcinomatous	Must have bacterial and viral and at least one other
	b) What are the likely organisms causing acute bacterial meningitis in different age groups?	◦ E coli/Group B strep: neonates ◦ Pneumococci: infants/older(all ages beyond neonates really) ◦ Meningococci: All ages beyond neonates esp. young adults ◦ Haemophilus: Children but decreased incidence with Immunisation ◦ Listeria. extremes of age ◦ Unusual orgs e.g staph aureus post N/surg. Immuno compromised eg gram negatives.	3 of 6
	c) What are the typical CSF findings in acute bacterial meningitis?	◦ Raised pressure ◦ Turbid ◦ Raised protein ◦ Lower glucose ◦ *Raised neutrophils ◦ *+ve bacteria on gram stain or culture	* and one other

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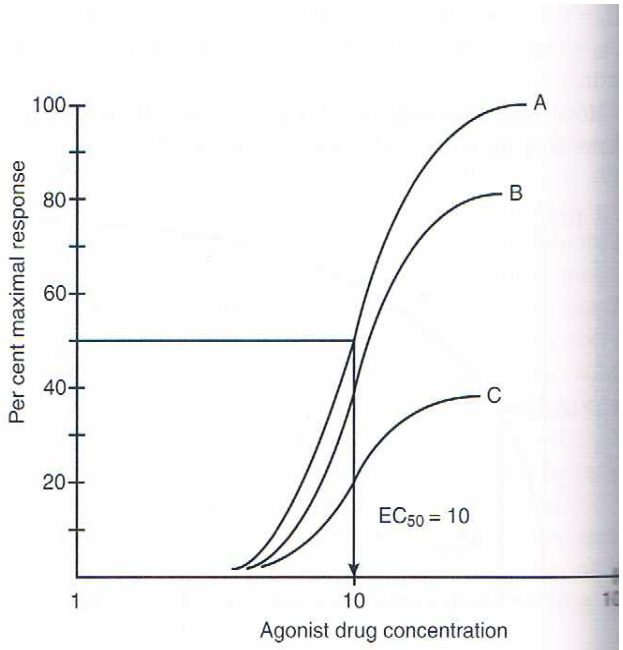
TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1 Metaplasia	a) What is metaplasia and give some examples?	◦ **Reversible change** (Among differentiated cells such as epithelial or mesenchymal) ◦ Where **one cell type is replaced by another** by reprogramming of precursor stem cells or undifferentiated mesenchymal cells Examples: ◦ Respiratory tract: trachea and bronchi in respiratory tract – due to chronic irritation such as smoking; ciliated columnar to stratified squamous ◦ GIT: oesophagus – due to chronic gastric acid reflux; squamous to intestinal-like columnar "Barrett's oesophagus"	a) Highlighted and 1/2 examples
	(b) How may metaplasia progress? (Prompt: What is the potential undesirable outcome of metaplasia?)	Cells lose normal protective function Persistence of influence that initiated the metaplasia **initiates malignant transformation** (e.g. squamous cell lung ca; adenocarcinoma oesophagus) Reverses	(b) Highlighted
Question 2 Wound Healing	a) What systemic factors affect wound healing? (50%)	1)Nutrition- (protein/ Vit C/ zinc/debilitation) 2) Metabolic- partic diabetes/ hypermetabolic/ unwell/ sepsis 3) Hormonal (steroids? effect of other hormone deficiencies/ XS catechols) 4) Circulatory status (e.g shocked/ hypotensive PVD/ venous ob, lymphatic obstruction)/ 5) age 6) drugs	Need **3 factors + 3 examples**
	b) What local factors impede wound healing (50%)	a) **Infection** b) Type/ / size of wound/not opposed c) Position- eg vasc/mvt/ pressure d) foreign bodies e) Wound vascularity/ local pressure excess f) Movement- excess g) Genetic features h) Excessive granulation " proud wounds" i) Neuropathic wounds	**Bold plus** **At least 3 local factors**- some
Question 3: Coagulation Cascade	1. What is the coagulation cascade?	"The coagulation cascade is essentially a **series of conversions of inactive pro-enzymes to activated enzymes**, culminating in the formation of thrombin which then converts the soluble plasma protein fibrinogen into the **insoluble fibrillar protein fibrin**"	**Series of reactions** **Fibrin formed**
	2. What mechanisms restrict the activity of the coagulation cascade. *Prompts:* *How is fibrin broken down?*	A. **Restriction of factor activation** to sites of exposed phospholipids B. **Three types of natural anticoagulants** 1. **Antithrombins** (e.g. AT3) - Inhibit the activity of thrombin & other serine proteases (IXa, Xa, XIa, XIIa) - AT3 activated by binding to heparin like molecules on endothelium → utility heparin in thrombosis 2.**Proteins C & S** - Vit K dependant proteins characterised by ability to inactivate factors Va and VIIIa. 3. **Plasmin (fibrinolytic system)** Plasminogen to plasmin by factor XII dependant pathway or 2 groups of plasminogen activators (PA) u-PA or t-PA Breaks down fibrin & interferes with polymerisation - Resulting fibrin split products (fibrin degradation products) also act as weak anticoagulants Endothelial cells modulate the coagulation / anticoagulation cascade balance by releasing PAI - block fibrinolysis by inhibiting t-PA binding to fibrin **4. Tissue factor Pathway Inhibitor (TFPI)**	**Plasmin + 1 other** **Description of plasmin action**

Question 4: Portal Hypertension	1. Classify portal hypertension giving examples for each. Prompt for most important hepatic cause.	**Increased resistance to portal blood flow classified as:** - **Pre hepatic**: portal vein thrombosis or narrowing - ***Hepatic: cirrhosis,** granulomatous disease, massive fatty change, schisto, nodular regenerative hyperplasia - **Post hepatic**, R heart failure, constrictive pericarditis, hepatic vein occlusion	3 groups including hepatic. Cirrhosis and one other cause
	2. What are the major clinical consequences of portal hypertension due to cirrhosis?	- Ascites: with potential for infection - Porto systemic venous shunts: varices > upper GI bleed. Other sites e.g caput, h'roids, retroperit. - Splenomegaly: thrombocytopenia - Hepatic encephalopathy > coma	At least 3 consequences
	3. What mechanism are involved in the formation of ascites?	- **Starlings forces**: increased pressure, decreased albumin - Increased formation of hepatic lymph overwhelms thoracic duct drainage > percolation into peritoneum - Intestinal fluid leak: ^pressure in intestinal capillaries and osmotic effect of protein rich ascitic fluid - Renal retention of Na and H2O due to 2ndary ^aldosterone.	Starlings forces and one other
Question 5: Influenza	i) Describe the structure and classification of influenza viruses	**ssRNA, bound by nucleoprotein that determines type (A, B or C) and a lipid bilayer that contains both haemagglutinin and neuraminidase** (determining subtype eg H1N1)	Need RNA and major types
	ii) What is the difference between antigenic drift and shift?	Only in influenza type A **Drift – mutation** of the haemaglutinin and neuraminidase antigens allowing escape from most host antibodies (epidemic) **Shift – antigens replaced via recombination of RNA** segments with those of animal viruses (pandemic) Types B and C do not show drift or shift, mostly infect children, who develop antibodies preventing re-infection	Bold
	iii) How does the human body clear a primary influenza virus infection?	**2 mechanisms – cytotoxic T cells and macrophages** cytotoxic T cells kill virus infected cells, an intracellular antiinfluenza protein (Mx1)is induced in macrophages by cytokines IFN-a and IFN-b. Future infection is prevented (haemagglutinin Ab) and ameliorated (neuraminidase Ab)	Bold to pass

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TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: Hypertrophy	(a) What is hypertrophy?	**Increase in the size of cells** – due to the **synthesis of more structural components** – resulting in an increase in the size of the organ; caused by increased functional demand or by hormonal stimulation. – pathological or physiological.	a) Highlighted
	(b) Give examples of physiological and pathological hypertrophy	Physiological – Skeletal muscle (gym etc – workload); Uterus in pregnancy (hormonal) Pathological – Myocardium (due to hypertension, aortic stenosis – workload); BPH	b) One example of each
Question 2: Oedema	a) What factors govern the movement of fluid between the vascular and interstitial spaces? (30%)	Hydrostatic Pressure – Osmotic Pressure- protein/ Na Normal capillary walls- most protein retained Small fluid out art end Most back venous end Small amount back via lymphatics	3 concepts mentioned A > c >V May know some Pressures, may mention gravity/ leg v head. Capillaries are fluid leak vessels. Normal tissue flow important. Thoracic duct return of lymphatics
	b) What are the major mechanisms of oedema formation (with examples)? 70%	>**Increased Hydrostatic Pressure** (local- DVT/ systemic- CCF)/venous obstruction < **Oncotic P** (mainly prot loss e.g. Nephrotic syndrome or poor production eg cirrhosis/ malnutrition or loss via gut) **Capillary leak-** (inflammatory injury/ systemic / infection) **Obstructive lymphatics-** e.g. lymphodema/ tumour/ op etc **Na retention with H2O** (renal insuff/ renin angio)- mainly dilutional	3 key features + a couple of examples
Question 3: Type 2 Hypersensitivity – Antibody mediated	1. What is type 2 hypersensitivity?	"Type 2 hypersensitivity is **mediated by antibodies** directed toward antigens present on the surface of cells or other tissue components"	Antibody mediated One of cell surface & extracellular matrix
	2. Describe the different types of type 2 hypersensitivity reactions and give examples of each.	**Three types** **(A) Opsonisation, Complement & Fc Receptor Mediated Phagocytosis** • Ig G,M activate complement, C3b & C4b recognised by phagocytes • activates complement system & membrane attack complex causing lysis of cells • Ig G recognised by phagocytes • Ab dependent cellular cytotoxicity (ADCC) Mono, Macro, Neut, Eosin, NK - transfusion reactions - erythroblastosis foetalis - auto immune haemolytic anaemia; agranulocytosis; thrombocytopaenia - certain drug reactions **(B) Complement and Fc receptor Mediated Inflammation** *C5A (+C4A & C3A) stimulate Neutrophil and Monocyte attack via Fc receptors releasing enzymes and Oxygen free radicals* *e.g: glomerulonephritis* *Vascular Organ graft rejection* *Goodpastures* **(C) Antibody Mediated Cellular Dysfunction** *Antireceptor antibodies disturb the normal function of receptors without causing cell injury.* e.g. myasthenia gravis (ACh receptor antibodies) Graves Disease - pemphigus vulgaris	2 of 3 types with one example Able to describe complement dependant reaction plus one other with examples

Question 4: Hepatitis D	i) Describe how the Hepatitis D virus infects the human body	RNA virus **Must always be in conjunction with Hep B** 1) **acute infection** – indistinguishable from classical acute Hep B.) Exposure to serum containing both Hep B and D. HBV must establish first to provide HBsAg necessary for development of complete HDV viridons 2) **superinfection.** -chronic HBV carrier exposed to new inoculum of HDV. Disease develops 30-40 days later 3) helper-independent latent infection- in liver transplantation patients	Bold to pass
	ii) Prompt: *Superinfection is one of the ways that Hepatitis D can infect the human host.* How does superinfection with HDV manifest?	1) severe acute hepatitis in previously unrecognised HBV carrier 2) exacerbation of preexisting mild chronic hepatitis B 3) 80-90% chronic progressive disease and cirrhosis	Need one
	iii) How is Hepatitis D infection diagnosed?	**IgM anti-HDV** – most reliable marker of recent HDV exposure but late and short lived HBV an HDV coinfection – best with IgM against both HDAg and HBcAg *2 phases –* *acute phase – active HDV replication, suppression of HBV, high ALT levels* *chronic phase – HDV replication decreases, HBV replication increases, ALT levels fluctuate, progression to cirrhosis and hepatocellular cancer* *HDV RNA detectable in blood and liver just prior and in early days of acute symptomatic disease* *In chronic delta hepatitis, HBsAg is present and IgM and IgG anti-HDV antibodies persist for months*	At least one
Question 5: Haemolytic anaemia	1. Classify haemolytic anaemias	- Intravascular/extravascular Or - extrinsic/intrinsic to the RBC. Or - hereditary/acquired	One classification,
	2. Describe the common features of haemolytic anaemias.	Features: - *Decreased RBC life span(< 120/7) due to premature destruction - ^ erythropoietin and erythropoiesis - Accumulation of products of Hb catabolism - reticulocytosis	• premature RBC destruction and one other feature
	3. Give some important causes of intravascular haemolysis. Prompt for examples	Intravascular - Mechanical injury: cardiac valves, microangiopathic. repetitive physical trauma - Complement fixation: ABO incompatible blood transfusion - Intracellular parasites: malaria - Exogenous toxins: clostridia	• 2 of 4
	If required **4. Apart from anaemia what are the results/manifestations of intravascular haemolysis?**	**- *Haemoglobinaemia** **- Haemoglobinuria** **- *Unconjugated hyperbilirubinaemia(jaundice) from catabolism of haem groups in mononuclear phagocyte system** **- Haemosiderinuria and renal haemosiderosis** **- Decreased serum haptoglobin due binding with free Hb and then cleared by monophag system.** **- Free Hb oxidized to metHb** **- Reticulocytosis**	* **Hbaemia and hyperbilirubinaemia to pass and one other** **OR 3 of 7**

QUESTION	KNOWLEDGE	PASS CRITERIA
1. a. With regard to drugs, what is "potency".	Potency refers to the affinity or attraction between an agonist and its receptor. A good measure of drug potency is the EC_{50} – the concentration that produces 50% of the maximal response.	Demonstrate understanding of efficacy and potency.
b. How is this different to Efficacy?	Efficacy is the maximal response that a drug (agonist) can produce (E_{max}) when all receptors are occupied, irrespective of the concentration required to produce that response.	
c. Draw a concentration-response curve showing 2 drugs with the same potency but different efficacy.		

2. a. Describe the mechanism of action of glyceryl trinitrate. b. What are the clinical effects of nitrates	• Taken up by **vascular smooth muscle** • Interacts with tissue sulfhydryl groups • Releases free radical **nitric oxide** • Activates cGMP • Dephosphorylates myosin light chains • R**educes intracellular Ca levels** • Smooth muscle relaxation & **vasodilation** • Low doses – venodilation ⇒ ↓ preload & stroke volume • Higher doses – arterial dilation ⇒ **↓ blood pressure** ⇒ ↓ cardiac output & **↓ myocardial oxygen demand** + dilation of coronary arteries/redistribution of perfusion ⇒ **i**mproved oxygen delivery to myocardium & resolution of ischaemic pain **[Prompt if needed "What other clinical effects may be seen?"]** • Adverse effects: postural hypotension, tachycardia, dizziness, headache, flushing, blurred vision, dry mouth, rash	Must state • **vascular smooth muscle** • **nitric oxide** • **vasodilation** Must state • **↓ BP** • **↓ myocardial oxygen demand** • 2 listed other effects
3. a. What is pancuronium?	**Non-depolarising NM blocker** Quaternary ammonium compound Potent competitive antagonist of ACh at nicotinic receptors skeletal muscle motor end-plate Interruption of transmission requires > 70% occupancy; blockade requires > 95% occupancy	Nondepolarising NM blocker

b. Describe the pharmacokinetics of pancuronium?	Poorly absorbed after oral admin Rapidly and widely distributed after iv Rapid elimination ($T1_{/2}$ 30min) by urinary excretion unchanged drug (highly water soluble), and hepatic metabolism with biliary excretion **[Prompt: Describe its distribution and elimination]**	Rapid distribution Rapid elimination
c. What are the adverse effects of pancuronium?	Uncommon Minor tachycardia, hypertension, sl ↑ CO can occur Life-threatening anaphylaxis < 1:10,000	A cardiac and allergy effect
4. a. Describe the pharmacokinetics of lithium	Rapidly absorbed (except SR preparations) with peak plasma concs in 1-3hrs. High bioavailability. Not metabolised Renally excreted unchanged with partial reabsorption from PT. Long T ½ of 24hrs in adults Steady state plasma concs not reached for 5-7 days **(PROMPT – How long does it take to reach steady state plasma conc?)**	Long T ½ so steady state plasma concs not reached for days. Renally excreted unchanged.
b. What are the adverse effects of Lithium at therapeutic levels?	Tremor, nausea, polydypsia /polyuria, diarrhoea, weight gain. Long-term: Acne / psoriasis, hypothyroidism, nephrogenic diabetes insipidus (inhibits the effect of ADH on the DT cells -> polyuria).	Polyuria & Polydipsia OR NDI.
c. What are the signs/symptoms of lithium toxicity?	GIT: Vomiting. Neuro: Tremors, confusion, slurred speech, ataxia, drowsiness, blurred vision, seizures.	CNS effects with at least 3 symptoms

5. a. List the advantages of eye ointments over eye drops.	More stable Less absorption into lacrimal ducts Longer retention time on conjunctival surface Safer with potent drugs Ointment bases provide protection and comfort at night	2 to pass
b. List by action the types of drugs used topically in the eye	Mydriatics Miotics Cycloplegics Decongestants Antibiotics Antivirals Antiseptics Corticosteroids Local anaesthetics Stains eg. Fluoroscein	4 to pass
c. List the ideal properties of an ocular local anaesthetic	Quick onset of action (10-20 secs.) Useful duration of action (10-20 mins) No obvious effects on function or healing No interactions with drugs used concurrently	Quick onset and useful duration of action

QUESTION	KNOWLEDGE	PASS CRITERIA
1. a. What routes of drug administration are there?	Enteral: Sublingual, buccal, oral, rectal Parenteral: SC, IM, IV, intrathecal, epidural Inhalational Topical	**Enteral/oral** + 3 non-enteral
b. What factors affect the rate of drug absorption from the small intestine?	Ionisation status of drug: alkaline Intestinal pH (7-8) favours absorption of un-ionised basic drugs Intestinal motility; increased motility lead to reduced transit time and drug absorption Gut surface area, blood flow, solubility of drug, formulation of drug **PROMPT: What is a specific drug factor**	Must mention drug factors and gut factors
c. What are potential disadvantages of rectal drug administration?	Erratic absorption because of rectal contents Local drug irritation Uncertainty of drug retention	1/3

2.
a. Describe the mechanism of action of ACE inhibitors
	• competitive **block conversion of angiotensin I to II** ⇒
	  ○ **decreased vascular tone** from prevention of vasoconstrictor effects of Ang II (main effect)
	  ○ **inhibition of aldosterone secretion** caused by Ang II leading to reduced Na & H_2O resorption ⇒ decreased BP
		3 in **bold** to pass

b. What are the adverse effects of ACE inhibitors	• **dizziness**, **hypotension** • headaches, weakness • loss of taste, nausea, diarrhoea • rash, fever, joint pain • **cough** • mild hyperkalaemia due to decrease in aldosterone secretion • acute renal failure	• hypotension or dizziness • cough • plus 2 others

c. What are some drug interactions that occur with ACE inhibitors	• Diuretics ⇒ hypotension • General anaesthetics ⇒ hypotension • Lithium ⇒ lithium toxicity • NSAIDS ⇒ hyperkalaemia & reduced effects of ACE inhibitor • Potassium sparing diuretics / potassium supplements ⇒ hyperkalaemia	2 to pass
3. a. What is the mechanism of action of erythromycin?	**Inhibits** RNA-dependent **protein synthesis** by binding to the 50S ribosomal subunit. **Bacteriostatic** (at high conc with selected organisms can be bactericidal)	Protein synthesis inhibitor Bacteriostatic
b. What is the mechanism for the drug interactions associated with erythromycin & give some examples?	**Inhibits hepatic CYP3A4.** Usually inhibits metabolism of other drugs metabolism causing increased activity. Examples: benzodiazepines, carbamazepine, cisapride (cardiotoxicity), digoxin, warfarin, theophylline, cyclosporine, tacrolimus	Inhibit hepatic metabolism One example
c. What are the adverse effects of erythromycin?	Common: **GIT**: abdo cramp, diarrhoea, N&V, candida (oral,vag) Rare: hypersensitivity, hearing loss, pancreatitis, hepatotoxicity Rapid iv may cause ventric arrhythmias.	GIT plus another
4. a. Describe the pharmacokinetics of phenytoin.	Oral absorption slow and variable: Time to peak levels 1.5-3hrs. Saturable hepatic metabolism leading to non-linear PK and variable T ½ of 7-42hrs. Metabolites excreted in the bile & urine.	Saturable metabolism/non-linear pharmacokinetics

b. What are the adverse effects of phenytoin?	Idiosyncratic: hirsuitism. gingival hyperplasia & overgrowth with bleeding, acne & facial coarsening. Dose related neurotoxic effects: drowsiness , dizziness, blurred vision, hallucinations, slurred speech, clumsiness, dizziness and confusion. Rapid IV administration associated with CV collapse. **PROMPT: Are there any specific problems with IV administration.**	Dose-related CNS effects Cardiac with IV administration & 1 other.
5. a. What are the indications for benzodiazepine use?	Anxiety Disorders Preoperative Medication Insomnia Sleep Disturbances Seizure Disorders Panic Disorder Alcohol Withdrawal Muscle Spasm Induce amnesia during cardioversion/endoscopic procedures	Seizures and 2 others
b. Explain the rationale for use of benzodiazepines in alcohol withdrawal	Down-regulation of neuro-inhibitory GABA receptors in alcohol dependent individual leads to symptoms of GABA deficiency in withdrawal. BZD act at a modulatory site on the the $GABA_A$ receptor to facilitate GABA binding to the $GABA_A$ receptors, enhance chloride channel opening, and overcome neuroexcitatory symptoms of GABA deficiency.	Facilitate GABA binding to the $GABA_A$ receptors Control neuroexcitatory symptoms of alcohol withdrawal.

QUESTION	KNOWLEDGE	PASS CRITERIA
1. a. What is meant by Total Body Clearance" of a drug	Describes the **ability of the body to eliminate a drug**. It refers to the theoretical volume of plasma emptied of drug per unit time (usually L/h). Total body clearance reflects the sum of all clearance process including **renal**, **hepatic** and **other**.	Definition
b. Name 2 drugs that have a high hepatic clearance and explain why this is important.	Lignocaine, Morphine, Propranolol, Pethidine. Drugs with high hepatic elimination may only be suitable for parenteral administration or have significant dosing variations depending on the route of administration. **PROMPT: How might it impact on route of administration**	2 drugs Demonstrate understanding
c. What factors determine drug half-life	Volume of Distribution and Clearance ($t_{1/2}$ = 0.693 x Vd/ Cl) Vd and clearance change with disease states - cardiac, hepatic and renal failure	Vd and clearance
2. a. What are the pharmacokinetic properties of frusemide?	• Rapid absorption after oral admin • Oral bioavailability 50% (range 10 –100%) • Highly protein-bound (>95%) • 50% conjugated in kidney & 50% excreted in urine unchanged (tubular secretion) • Elimination $t^1/_2$ 1.5 – 2 hours • Peak effect 30 minutes IV / 1 hour oral	Must list 3 properties
b. What are the site and mechanism of action of frusemide ?	• Actively secreted into lumen of nephron from proximal tubule cells via organic-base pump • Inhibits Na^+-K^+-$2Cl^-$ transporter in **thick ascending limb of loop of Henle** thus **preventing resorption of Na^+ & Cl^-** • Abolishes counter-current concentrating mechanism leading to a dilute urine	Must mention thick ascending limb of loop of Henle and reduced resorption of Na and Cl.

C. What are the adverse effects of the frusemide?	• Electrolyte disturbances – **hypokalemia**, **hyponatraemia**, hypomagnesaemia, hyperuricaemia • Postural **hypotension** & dizziness • Increased LDL & triglycerides, decreased HDL • Ototoxicity (high dose IV) • Drug interactions	Must list • Hypokalemia • Hyponatremia • Hypotension or dizziness • 1 other
3. a. What is the mechanism of action of cephalosporins	I**nhibit bacterial cell wall synthesis**, cell division and growth (similar to penicillins) **Bactericidal** Most effective in rapidly dividing cells.	Bolded material
How does the spectrum of microbiological activity for the 4th generation cephalosporins compare to that of earlier generations?	**Gram negative** as for 3rd generation e.g. E Coli, H Influenza, Klebsiella S**ome gm positive** (S Pneumonia) but less than 1st generation M**ore resistant to B Lactamases** than earlier generations	Bolded material
What is the relationship between penicillin allergy and cephalosporin allergy.	5-15% possibility of cross-reaction with penicillin allergy.	Aware of cross-reactivity
4. a. Describe the general pharmacokinetic characteristics of antipsychotic drugs	Most are readily but incompletely absorbed. Many undergo significant first pass metabolism Most are lipid soluble (lipophilic) Most have high PPB (92-99%) Most are completely metabolised by hepatic enzymes (oxidation; demethylation) These are catalysed by liver enzymes. **PROMPT: Use chlorpromazine as an example**	Lipid soluble. Hepatic metabolism + 1 other

Define the term “atypical” antipsychotic and provide an example.	Newer antipsychotic agents with less propensity to cause extra-pyramidal side-effects. Better at treating negative features of schizophrenia. They share a greater ability to alter $5HT_{2A}$ receptor activity than to interfere with D_2-receptor action. Examples: olanzapine; clozapine; quetiapine; risperidone; loxapine	Less EPS One example
c. Describe the adverse drug reactions to olanzapine.	Weight gain Sedation (but less than typical antipsychotics) Minor orthostatic hypotension Minor anticholinergic effects (dry mouth, urine retention etc) (Extrapyramidal effects less prominent)	2 effects
5. a. What is the mechanism of action of flumazenil?	Antagonist at the BZD binding site on the $GABA_A$ receptor (ligand-gated chloride channel). Decreases the binding of GABA. Blocks GABA-induced increase in Cl^- permeability and influx of Cl^- into the cell causing hyperpolarisation and decreased excitability of the neuron.	Specific BZD receptor antagonist at GABA receptor
b. What are the indications for flumazenil use	Avoid intubation or ICU admission in BZD overdose. Reverse BZD sedation after procedures Diagnostic role	Reverse the sedative effects of BZD
c. What potential problems should be anticipated when using flumazenil?	Precipitate seizures in mixed overdoses with BZD and proconvulsants Precipitate seizures in pts taking BZD to control epilepsy Precipitate withdrawal symptoms and seizures in BDZ-dependent Duration of action is only 1-3hrs thus repeated administration may be necessary Reversal of BZD-induced respiratory depression has not been demonstrated, so resp and cardiovasc support may be required Adverse Effects: headache, visual disturbance, increased anxiety, nausea, light-headedness	Precipitate fits Need for repeated doses

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TOPIC	QUESTION	NOTES	ESSENTIAL KNOWLEDGE
Question 1: Spare receptors & their significance P13-4	1. Define the term "spare receptor"	**Receptors "spare" if maximal biologic response possible at an agonist concentration that does not result in all available receptors being occupied.** Describes concept of receptors "spare in number". Can also have spareness "temporally" if effects produced by binding last much longer than the time the agonist occupied the receptor	Highlighted section concept
	2. What is the significance of spare receptors? *How is it related to the maximal response of a drug?* *What do the terms spare in number and temporal spareness mean?*	Increasing the number of receptors coupled to an effector can allow lower concentrations of agonist to still produce a given proportion of maximal response - tissue thus more sensitive	concept
Question 2: Antibiotics in urinary sepsis P 732, 755-61, 765-6	1. Describe the mechanism of action of gentamicin ?	Irreversible inhibitor of protein synthesis-possible mechanism: 1. Passive diffusion via porin channels across outer memb, then active transport into cytoplasm by an O2 dependant process. . 2. Binds 30S ribosome & inhibits protein synthesis by 1) Inducing misreading of mRNA thus producing toxic or nonfunctional protein; 2) interfere with initiation complex of peptide formation; 3) cause break up of polysomes into non-functional monosomes.	Irreversible protein synth inhibitor. Binds ribosomes
	2. What are the benefits of once daily dosing ? *Prompt how does this improve clinical effectiveness*	1. *Concentration- dependant killing* (at increased conc kill increased no of bacteria at a more rapid rate; 2. *Postantiobiotic effect*- activity lasts longer than detectable serum levels; 3. *Reduced toxicity* – time above critical level will be longer with multi dose than single dose schedule); 4. Less nursing time; OPD therapy possible; 5. Drug level not required unless >3 day therapy.	Conc dependant kill + 2 others.
	3. How do penicillins enhance the efficacy of gentamicin? *Optional question*	Low ECF pH & anaerobic conditions inhibits transport Transport enhanced by cell wall active drugs eg. penicillin	
Question 3: Aspirin P575-8	1. Describe the pharmacokinetics of Aspirin *What's the significance of it being a weak acid?*	Aspirin has pKa 3.5; Rapidly absorbed from stomach and upper small intestine→peak plasma level in1-2 hrs. Half life: 15 min. Rapidly hydrolysed→Acetic Acid+Salicylate by esterases in tissue and blood. Salicylate non-linearly bound to albumin. Alkalinisation of urine increases rate of excretion of free salicylates and its water soluble conjugates. Small Vd, capacity limited metabolism	Rapid abs, small Vd, renal excretion

	2. What are the adverse effects of therapeautic doses of Aspirin? *What are the respiratory effects of aspirin?* *Are there any other systems affected?*	CNS: Headache, tinnitus, dizziness CVS: Fluid retention, H/T, oedema GIT: Abdo pain, N,V, Ulcers, Bleeding Haem: Thrombocytopenia, neutropenia, Aplastic a Hepatic: Abn LFTs, liver failure Pulmon: Asthma Skin: All types of rashes, pruritis Renal: Impairment and failure, hyperK, proteinuria	GIT + allergy + bronchospasm
Question 4: Amiodarone P228-9	1. What are the cardiac effects of amiodarone at a cellular level?	Prolongs AP duration (by blocking K+ channels) Blocks inactivated Na channels. The AP prolonging action reinforces this effect. Blocks depolarized cells > normal cells. Mild antisympathetic. noncompetitive inhibitor of beta receptors; Weak adrenergic blocker - slows HR and A-V node conduction. Weak Ca channel blocker. Inhibits abnormal automaticity; slows sinus rate; increases PR interval	K block and 1 other
	2. What are the mechanisms of pharmacokinetic drug interaction with Amiodarone and give two examples.	Inhibits liver cytochrome metabolising enzymes Digoxin, Warfarin levels increase. Cimetidine increases amiod toxicity by decreasing hepatic clearance. Interacts with statins (artorvastatin and simvastatin; instead use pravastatin as not P450). Concentration and effects of Phenytoin, anaesthetics, cyclosporins, theophylline, procainamide, flecainide, quinidine are increased by amiodarone	Enzyme induction/inhibition + 1example of either
Question 5: Passive immunisation in ED p1073-8	1. What is passive immunisation?	Giving preformed antibodies to a recipient. Source may be human, animal	Concept
	2. What is passive immunisation useful for?	1. prevention of disease when time does not allow immunisation 2. treatment of disease normally prevented by immunisation 3. for patients unable to form antibodies 4. for treatment of conditions for which active immunisation is unavailable or not possible eg snakebite	2 uses
	3. What passive immunisations might we consider in ED?	tetanus, botulism. measles, rubella, vaccinia, varicella Hep B, Hep A; diphtheria, rabies. antivenoms – spiders, snakes; Rhesus incompatibility	Tetanus + 1 other

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TOPIC	QUESTION	NOTES	ESSENTIAL KNOWLEDGE
Question 1: Antagonist / agonists P14-16	1. Describe the difference between a Competitive and an Irreversible antagonist	Competitive - in fixed conc. of agonist, increasing conc. of antagonist will lead to progressively inhibited response, but an increasing agonist conc. can overcome to still evoke maximal response (agonist conc / effect curve shift to right) High comp. antagonist conc. prevent response completely if agonist conc. fixed Irreversible (Noncompetitive) - bind so tightly or covalently as to make receptor unavailable to agonist. Number of remaining receptors may then be too low to allow maximal response to occur regardless of agonist conc. (unless spare receptors) Length of effect of irrev. antagonist will reflect turnover of receptors involved rather than rate of elimination of antagonist	Description visual or verbal
	2. Give an example of an antagonist?	Competitive: naloxone, flumazenil, Propranolol, isoprenaline, naltrexone, nalmefene Irreversible: phenoxyenzamine, MAOI	1 example
Question 2: Antibiotics in CNS infections P737-40, 751-2, 790-5, 835	1. How are cephalosporins classified? *What are the differences between the classes?*	1- GPs; 2- + haemophilus & kleb; 3-GP + GN; 4- pseudomonas	1-4 with increasing GN spectrum activity; less GP activity
	2. Why are 3rd generation cephalosporins used in CNS infection?	Expanded GN activity & cross the BBB; penetrate body fluids well; good toxicity profile	Spectrum activity & penetration CNS
	3. Are there any bacteria responsible for CNS infection that cephalosporins do not cover?	Listeria Resistant pneumococci may need vancomycin Resistant E Coli; use with aminoglycoside to cover Pseudom	1 example
Question 3: Paracetamol Toxicity P 591-2, 56-7	1. Describe the mechanism of Paracetamol hepatotoxicity	In normal doses, Paracetamol undergoes glucuronidation and sulphation to the corresponding conjugates, making up 95% of total excreted metabolites. The alternative P450 dependant pathway accounts for 5%. When intake far exceeds therapeutic intake, glucuronidation and sulphation pathways are saturated, so P450 dependent pathway becomes impt. So long as there is hepatic GSH available for conjugation, no hepatotoxicity occurs. Once hepatic GSH is depleted faster than its regeneration, a reactive toxic metabolite-N-acetylbenzoiminoquinone is produced. This reacts with the nucleophilic groups of cellular proteins to produce hepatotoxicity.	concept of 2 paths with saturation Glutathione key word
	2. What is the antidote and how does it work?	NAC glutathione substitute, binding to the toxic metabolite Anti oxidant	NAC + donor/substitute (GSH)

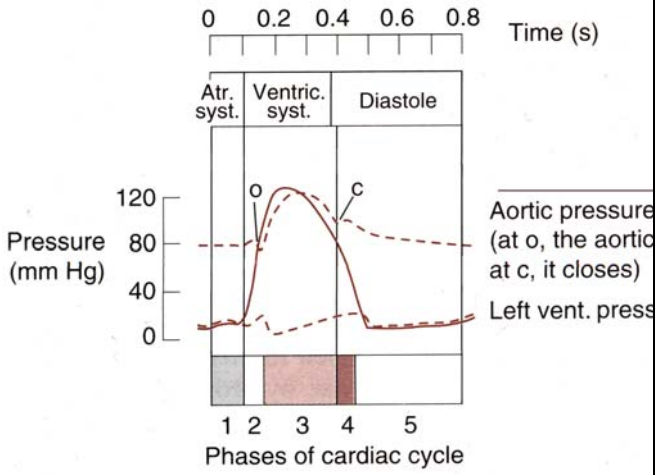
Question 4: Metoprolol P147-55, 169	1. Describe the pharmacokinetics of metoprolol *What's the bioavailability?* *Why is this so?* 2. How does metoprolol differ from propranolol in its action at beta receptors? 3. How do B Blockers control hypertension?	Oral or IV, Well absorbed Bioavailability 50% due to first-pass effect Large volume of distribution Half-life, 3-4 hours Metabolised in the liver B1 equipotent B2 50-100 fold less potent Not fully understood Negative inotropic and chronotropic effects Slow a-v node conduction Antagonises release of renin caused by sympathetic nervous system	Large Vd + first pass B1 selective Negative inotrope/chronotrope
Question 5: Evaluation of drugs and clinical trials Katzung 68-73	1. During clinical drug trials, what factors might confound the results? *What are some of the host factors?* *What are some of the observer factors?* *Why do you blind trials?* 2. What can be done to minimise the confounders?	1. variable natural history of most diseases 2. presence of other diseases and risk factors 3. subject and observer bias 1. large populations over sufficient time; cross-over trials 2. exclusion criteria; randomisation; cross-overs 3. placebo controls; blinding; cross-overs	Bias Bonus points for comment

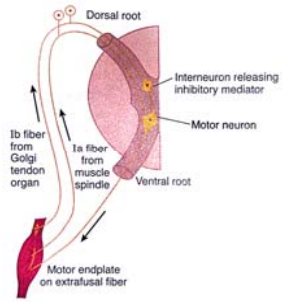
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TOPIC	QUESTION	NOTES	ESSENTIAL KNOWLEDGE
Question 1: Second messengers P21-26	1. What are the steps in activation of a second messenger?	Method of transmembrane signalling Drug binds to a receptor on extracellular side plasma membrane Triggers activation of G protein on cytoplasmic side Activated G protein changes an enzyme or ion channel This changes concentration of intracellular second messenger which mediates a response	Binding Transmembrane signal G protein Effector
	2. Give an example of a second messenger and the type of response it produces? *What about cAMP?*	cAMP via adenylate cyclase Mobilization of fat and carbohydrates Conservation of water by kidney Increase rate and contractility of heart Ca++ regulation Adrenal hormone regulation, relaxation of smooth muscle Ca++ and Phosphoinositides cGMP via transmembrane guanylyl cyclase (atrial naturetic peptide) or nitric oxide which binds to a cytoplasmic guanylyl cyclase GTN, Na nitroprusside Inhibition of phosphodiesterase – increased cGMP eg sildenafil	name 1 and some knowledge of a response
Question 2: Drugs in status epilepticus P374-92	1. Describe how phenytoin is administered in status epilepticus? *What's the mg/kg dose?*	IV load 13-20mg/kg,, given diluted in saline (precipitates in glucose at max rate in adults of 50mg/min Continued 100mg Q6-8hrly	Dose mg/kg, iv route safe rate
	2. Describe the adverse effects of phenytoin ? *What about short term vs long term effects?* *What about in iv administration?*	Dose related nystagmus, ataxia, diplopia long term: gingival hypertrophy, hirsuitism mild facial coarsening & peripheral neuropathy abnormal Vit D levels (osteomalacia) low folate levels; megaloblastic anaemia; Foetal hydantoin syndrome. Idiosyncratic: skin rash; SJ syndrome; Lymphadenopathy; agranulocytosis. Rapid iv may cause hypotension/arrhythmia Drug interactions; reduced CL & binding in neonates	CNS + skin + CVS in iv admin

Question 3: Morphine P489-98	1. Describe the effect of morphine on the different opiod receptors?	Morphine is a full agonist in the μ (mu) receptor→ analgesia, sedation, ↓respirations, ↓ GIT transit, modulation of hormone and neurotransmitter release ; also affects σ (delta) → analgesia, modulation of hormone and neurotransmitter release and Ќ (kappa)→analgesia, psychomimetic effects,↓ GIT transit	Agonist mu receptor + 1 receptor
	2. Describe the effects of morphine on different organ systems?	CNS: Analgesia, euphoria, sedation, respiratory depression; miosis, hyperthermia, -stimulates release of ADH, prolactin and somatotrophin, -truncal rigidity, Resp: depression, Cough suppression, CVS: bradycardia GIT: constipation, contracting biliary smooth muscle, N&V, Renal: Depressed renal function Gynae: Decreases uterine tone Skin: Pruritis, urticaria	CNS + resp + 2 others
Question 4: Atropine P108-9, 114-6	1. Describe the pharmacokinetics of atropine	Oral or IV (usually), neb, topical; Well absorbed orally Widely distributed (including CNS) Half-life 2 hours Elimination: 60% excreted renally unchanged 40% phase I and phase II metabolism and renally excreted	Wide distribution + short t1/2
	2. At which receptors does atropine act?	Muscarinic (equipotent at M1, M2 and M3) Nicotinic (minimal potency)	Predominant Muscarinic
	3. What are the effects of atropine on heart rate?	Lower doses often an initial bradycardia, (Blocks prejunctional M1 receptors); Tachycardia	Dose dependant
Question 5: Therapeutic drug monitoring p46-49	1. What pharmacokinetic variables affect drug levels? *Patient factors?* *Specific drug examples?*	absorption – eg small bowel abnormalities clearance – eg impaired renal, liver, cardiac function volume of distribution – changes in either tissue or plasma binding impact drug availability; eg decreased muscle mass in elderly, hypoalbuminaemia, drug interaction.	2 variables
	2. What pharmacodynamic variables affect drug dosing?	maximum effect (Emax) – vs toxicity by increasing dosing beyond maximum effect sensitivity (EC50) – eg hyperkalemia decreases sensitivity to and effect of digoxin	

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TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: Mechanical events/phases of cardiac cycle.	List in order, the mechanical phases of the cardiac cycle Please draw the pressure changes in the ventricle that occur during the cardiac cycle	• Atrial systole. • Isovolumetric ventricular contraction. • Ventricular ejection. • Isovolumetric ventricular relaxation. • Ventricular filling.  	Three out of five phases to pass this subsection. Basic diagram and correct pressure .
Question 2	1) What is normal GFR 2) what factors affect GFR	1. @ 125ml/min in 70kg male, 10% less in female 2.a) Net filtration pressure= hydrostatic pressure in capillaries and bowmans capsule AND colloid osmotic pressure -afferent – efferent pressure- under control of – autoregulation, sympathetic, AgtII, dobutamine, PGs, serum Na - renin Pressure in bowmans capsule – renal obstruction 2. b)capillary filtration coefficient Relates to surface area and permeability of capillaries Surface area controlled by mesagial cells under control	1) reasonable value 2) 3 to pass 3) 1 of 2

	3)As well as filtration , by what other means does the kidney regulate the composition of urine –	of AgtII, ADH, NA, PGs (constrict) ANP, dopamine, cAMP, PGE2(relax) 3.secretion and resorption	
Question 3 Vasopressin	(a) What stimuli influence vasopressin (ADH) secretion? (b) How does vasopressin exert its anti-diuretic effect?	Increased by • decreased ECF volume • increased effective plasma osmotic pressure • angiotensin II • nausea & vomiting; stress, exercise, • clofibrate, carbamazepine; Decreased by • increased ECF volume • decreased effective plasma osmotic pressure • alcohol (b) *It increases permeability of the collecting ducts to water resulting in renal retention of water*. activation of V2 receptors, causing insertion of proteins called water channels (aquaporins) into apical (luminal) membranes of the principal cells of the collecting ducts.	Need – volume, osmolarilty and aquaporin
Question 4 Monosynaptic reflex	**Please describe a monosynaptic stretch reflex**	 Muscle spindle and its reflex connections are involved in proprioception	Essential to pass Monosynaptic, sensory organ, effector

Question 5	a.Please explain the concept of compliance as it relates to the lung b.What factors affect compliance	a. Volume change per unit pressure change (slope of pressure volume curve) =~200ml/cm H2O Depends on lung volumes and demonstrates hysteresis , may draw compliance curve, point out that inc compliance at low volumes Depends on structural proteins and surface tension b. Decrease – fibrosis, pulmonary oedema, not ventilated, inc pulmonary venous pressure Increase- ,emphysema, asthma surfactant	Basic concept 1 of each

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TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: Myocardial contractility	Please draw the starling curve What factors influence myocardial contractility?	• Positively Inotropic: o Sympathetic stimulation via nerves or circulating catecholamines; o Post-extrasystolic potentiation; o Increased heart rate (small effect); o Drugs such as xanthines, glucagon, cardiac glycosides, adrenergic agents; o Increased myocardial mass (chronic). • Negatively Inotropic: o Parasympathetic stimulation (small) o Hypercapnoea, hypoxia, acidosis; o Drugs such as calcium channel blockers, beta-blockers, quinidine, barbiturates; o Cardiac failure (intrinsic myocardial depression; o Cardiomyopathy or infarction.	Five of the factors listed with at least two each positively or negatively inotropic to pass this subsection.
Question 2: Thyroid hormones	1) Outline the physiological effects of thyroid hormones 2) Describe the mechanism regulating thyroid hormone	(a) Heart: chronotropic; inotropic (↑ number of B-adrenergic receptors; ↑response to catecholamines; ↑ proportion of a-myosin heavy chain); Adipose tissue: catabolic (stimulated lipolysis); Muscle: catabolic (↑protein breakdown); Bone: developmental (promote normal growth(Cretin) and skeletal development); Nervous system: promote normal brain development; Gut: metabolic (↑ CHO absorption); Lipoprotein: metabolic (formation of LDL receptors); other – calorigenic (↑ metabolic rate, ↑ stimulation O_2 consumption) (b) Negative feedback effect of T4 and T3 on hypothalamus and pituitary to inhibit TRH and TSH secretion respectively. Cold stimulates	3 to pass incl cardiac

		thyroid hormone secretion, stress and glucocorticoids inhibit.	
Question 3: Renal blood flow	1) what is the normal RBF 2) How is renal blood flow regulated	1) RBF @ 1250ml/min, 25% CO 2) a)neuroendocrine; NA constrictor, DA dilator, Angio2 constrictor, PG's (incr cortical decrease medulla), ACH dilates b) autoregulation, probably vessel wall stretch reflex as occurs in denervated isolated vessels c) renal nerves.	1) approximate value 2) Must have 2 of DA, NA, AgtII, autoreg
Question 4 Glycogen	1) What are the physiologic actions of glucagon? 2)What factors affect glucagon secretion?	• Glycogenolysis in liver – not muscle • Gluconeogenesis from aa's only at very high levels • Lipolysis – • Ketogenesis • +ve inotropic effect on heart • Inc blood flow to kidneys • • Stimulates secretion of GH, insulin and somatostatin 2)Stimulators beta adrenergic stimulants Cortisol, protein meal,vagal stimulation, starvation,stress, exercise CCK gastrin theophylline <u>Inhibitors</u> Glucose ----most important,nsulin Somatostatin FFA Ketones α Adrenergic stimulators. GABA Phenytoin	3 bulleted – essential to pass. Rest additional –3 to pass from each Rest additional

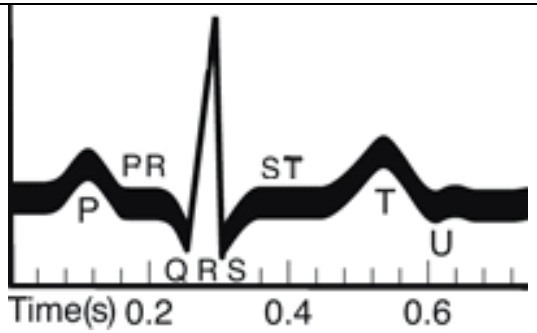
Question 5	What are the metabolic functions of the lung? (Prompt – What substances are metabolised in the lung?)	Metabolism of vasoactive amines a. Activation of Angiotensin 1 → AT 2 (ACE in capillary endothelium) b. Inactivation of bradykinin (ACE); PGs E/F c. Uptake & storage of Serotonin d. Arachadonic acid metabolites → leukotrienes / SRS-A & Prostaglandins Synthesis of a. Surfactant b. IgA c. Phospholipids d. Proteases (collagen/elastin breakdown)	2 of each

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<table>
<tr><th>TOPIC</th><th>QUESTION</th><th>ESSENTIAL KNOWLEDGE</th><th>NOTES</th></tr>
<tr><td>Question 1:

Normal ECG and cardiac membrane polarisation changes/ECG findings in MI</td><td>Please draw a normal ECG tracing

Describe the cardiac events that relate to each of the intervals

What is the electrophysiological basis for ST elevation in acute MI?</td><td> 

The ST segment represents the plateau (Phase 2) of the AP and the T wave is repolarisation (Phase 3).

ST segment elevation concave upwards.

<table>
<tr><th>Polarisation anomalies in infarcted Cells</th><th>Current flow</th><th>ECG Change in leads over the Infarct</th></tr>
<tr><td>Rapid repolarisation</td><td>Out of infarct</td><td>ST segment elevation</td></tr>
<tr><td>Decreased resting membrane potential</td><td>Into infarct</td><td>TQ segment depression (manifested as ST segment elevation)</td></tr>
<tr><td>Delayed depolarisation</td><td>Out of infarct</td><td>ST segment elevation</td></tr>
</table></td><td>Recognisable shape with QRS

Name intervals.

Describe the ST segment representing plateau (Phase 2) to pass this subsection.

One of three</td></tr>
<tr><td>Question 2:
Insulin</td><td>(a) What metabolic effects does insulin have on the liver?

B, What are the effects of insulin deficiency on the body over</td><td>(a) ↓ketogenesis; ↑protein synthesis; ↑lipid synthesis; ↓glucose output due to ↓gluconeogenesis, ↑glycogen synthesis, ↑glycolysis

(b) 1. Decreased cellular glucose uptake –
; total body dehydration and acidosis.</td><td>2 to pass</td></tr>
</table>

Question 3	Describe the Physiological process of Micturition Prompts: What muscles and nerves are involved?	• Spinal reflex facilitated and inhibited by higher centres • First urge to void at 150ml • Marked fullness at 400ml • During micturition, the Detruser muscle contracts and perineal muscles/external urethral sphincter relax • Parasympathetic(S2,3,4) afferents respond to stretch receptors in bladder wall to initiate reflex contraction via parasympathetic efferents. • Pudendal nerve to External Urethral Sphicter causes relaxation. • Spinal reflex integrated in sacral portion of spinal cord • Sympathetic (L1,2,3) play no role in micturition but only in prevention. EUS and perineal muscles can be controlled voluntary for a period of time but eventually void reflex overcomes voluntary control.	To Pass Spinal Reflex Parasympathetic control Voluntary Control
Question 4 thermoregulation	What factors are responsible for heat production and heat loss? Describe the body's adaptive response to a cold environment	**Heat Production** Basic metabolic process Specific dynamic action of food Muscular activity **Heat is lost by** Radiation and conduction Vaporisation of sweat Respiration Urination and defaecation **Mechanisms activated by cold** Shivering	To Pass 2 of each 3 to pass

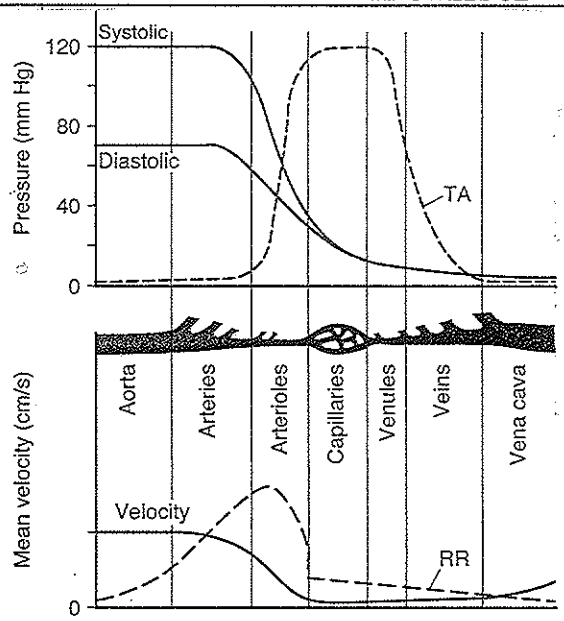
		Hunger ↑ Voluntary activity ↑ Secretion of adrenaline/nor adrenaline ↓ Heat loss Cutaneous vaso constriction Curling up Horripilatian (Controlled from posterior hypothalamus)	
Question 5	a. What factors influence the rate of transfer of oxygen from the alveolus into a pulmonary capillary b. Could you give some clinical examples of when these may be affected	A)Process is passive diffusion (ficks law of diffusion) Affected by – surface area, membrane thickness, gradient of p O2 (o2 in alveolus and O2 binding capacity of Hb) Also – constant –solubility and MW ($V = A/T \times D \times (P_1-P_2)$ $D = Sol/MW^{1/2}$ b) exercise alveolar hypoxia and thickening of blood gas barrier	2 to pass

ACEM PRIMARY 2010/2 PHYSIOLOGY VIVA Day Friday Session (pm) pm *Candidate Number*…… AGREED MARK……

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: Cardiac output and its measurement.	What are the parameters that define cardiac output? What are the factors that influence stroke volume? How can cardiac output be measured?	Cardiac output (CO) = heart rate (HR) X stroke volume (SV) • Preload • Afterload • Myocardial contractility The Fick principle states that the amount of a substance taken up by an organ (or by the whole body) per unit of time is equal to the arterial level of the substance minus the venous level (A-V difference) times the blood flow. The principle can be used to determine cardiac output by measuring the amount of O_2 consumed by the body in a given period and dividing this value by the A-V difference across the lungs. $$\text{Output of left ventricle} = \frac{O_2 \text{ consumption (mL/min)}}{[A_{O_2}] - [V_{O_2}]}$$ Whole body O_2 consumption is calculated by collecting expired gas in a spirometer and determining its O_2 content, which is then subtracted from the calculated O_2 content of inspired gas. The arterial O_2 content can be measured in an arterial sample and the mixed venous blood O_2 content is obtained from a pulmonary artery catheter. In the indicator dilution method, a known amount of a substance is injected into a vein and the concentration of the indicator in serial samples of arterial blood is determined. The output of the heart is equal to the amount of indicator injected divided by its average concentration in arterial blood after a single circulation through the heart. The cardiac output for that period is	Full equation to pass this subsection. 3 to pass this subsection. Basic explanation of either principle.

		calculated and then converted to output per minute. $\text{Flow} = \frac{\text{amount of indicator injected}}{\text{instantaneous concentration of indicator in arterial blood}}$ The indicator must, of course, be a substance that stays in the bloodstream during the test and has no harmful or haemodynamic effects. A popular indicator dilution technique is thermodilution, in which the indicator used is cold saline.	
Question 2	Can you draw a nephron and describe the functions of each part	• Glomerulus - filtration • Afferent arteriole(contain juxtaglomerular cells – secrete renin) then capillary tuft then efferent arteriole encapsulate in bowmans capsule • PCT- resorption of most solute – Na, glucose, aa, reclaim HCO3 • Desc limb of LOH – thin, water permeable • Thick Asc LOH – site of Na K 2 Cl – generates concentration gradient • DCT – site of Na K Cl pump • Proximal part is the macula densa forms juxtaglomerular apparatus - • CD- p cells - under control of ADH and aldosterone(water and Na resorption) • I cells – involved in H+ excretion	To Pass: Draw basic shape and label 1 function of each part
Question 3 Mineralocorticoids	(a) What is the physiological role of aldosterone (b) What conditions increase aldosterone secretion?	(a) Aldosterone causes Na+ and water retention, expanded ECF volume and shutting off the stimulus to increased renin secretion. (b) Primary: – stress hormone, low pressure/volume states secondary hyperaldosteronism: (eg. CCF, cirrhosis & nephrosis). Drugs:	Bold to pass To Pass: Primary and secondary

Question 4 Regulation of serum calcium level	a. What hormones are involved in serum calcium regulation Outline the effects of PTH (Parathyroid Hormone)	PTH, Calcitonin, 1,25 DHCC ↑ Parathyroid hormone secretion A Kidneys - ↑Calcium reabsorption ↑ 1,25- (OH)2D formation ↓ Urinary excretion of Calcium ↑ Plasma 1,25- leads to (OH)2D levels cause – B Intestine - ↑ Calcium absorption C Bone – ↑ Resorption ↑ Release of Ca 2+ into plasma	To Pass 2 of 3 <u>Additional</u> To Pass: 1 of each
Question 5	a. Please draw a diagram showing static lung volumes b. How does physiological dead space differ from anatomical dead space?	a. TV 500ml b. DS 150 ml c. TLC 7L d. FRC 2L e. VC6L a. Anatomical dead space, conducting zones of lung 150 mls b. Physiological dead space- Parts of lung with ventilation but no perfusion	To Pass: Draw diagram Label diagram Prompt for both physiological and anatomical dead space Normally very nearly the same but physiological dead space much greater in disease because of V/Q mismatch, esp with increased airway pressures

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>
</table>
</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

ACEM PRIMARY 2011/1 PHYSIOLOGY VIVA Day Thursday AM Session ***Candidate Number*****…… AGREED MARK……**

Question 1

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES & MARKS
Question 1	1.1 Describe the normal sequence of electrical excitation of the cardiac conduction system and cardiac muscle?	Normal sequence of depolarisation: **SA node** **Atria** (pathways) **AV node** Bundle of His **Major bundles** (Right & Left) Purkinje fibres **Ventricular muscle** Left side of IV septum first Spread down septum to apex Then up to AV grooves Spread from endocardial to epicardial surfaces	All of bold to pass
	1.2 What are the common mechanisms which cause abnormalities of cardiac conduction?	**Abnormal pacemakers** **Re-entry circuits** **Conduction defects** Prolonged repolarisation Accessory pathways	2/3 bold to pass
	1.3 What are the possible clinical consequences of these conduction abnormalities? (Flexibility between 1.2 & 1.3)	Abnormal pacemakers • **ectopic beats** • pacemaker failure (sinus arrest) • **fibrillation (atrial or ventricular)** Re-entry circuits • leading to tachyarrhythmias Conduction delays • **heart block** • bundle branch blocks Prolonged repolarisation • Long QTc Accessory pathways • WPW or LGL	2 bold + 2 others to pass

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Question 2

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES & MARKS
Question 2 Pulmonary blood flow	2.1 Describe the normal distribution of pulmonary blood flow.	2.1 Influenced by gravity-3 main zones Zone 1(apex) PA>Pa>Pv – least blood flow Zone 2(mid) Pa>PA>Pv Zone 3(base) Pa >PA>Pv – Most blood flow	Posture/Hydrostatic pressure and describe 3 zones to pass
	2.2 How is the distribution of pulmonary blood flow actively controlled?	2.2 Hypoxic pulmonary vasoconstriction-alveolar hypoxia constricts pulmonary arteries, directs blood away from poorly ventilated diseased lung areas. Mechanism-NO, endothelin-1, TXA2, low pH, autonomic system	Hypoxic pulmonary vasoconstriction to pass
	2.3 Please explain how cardiogenic pulmonary oedema occurs.	2.3 Starling's Law- differences in capillary and interstitial hydrostatic and colloid osmotic pressures. Significant increases in net outward pressure of Starling equation results in interstitial oedema especially at perivascular and peribronchial spaces. Further increases of outward pressure results in fluid entering alveolar spaces.	Basic description of Starling forces

Question 3

Question 3:	3.1 What are the principal buffering systems in the body? PROMPT: How about in other fluid compartments?	Blood: Bicarbonate, Protein and Haemoglobin Interstitium: Bicarbonate Intracellular: Protein, Phosphate Urine: also uses ammonia	3 buffering systems to pass 2 fluids to pass
	3.2 Outline how the body responds to a metabolic acid load.	a) Buffering in blood, interstitial and intracellular spaces b) Respiratory response: H_2CO_3 converted to H_2O and CO_2, CO_2 expired via lungs through increased minute ventilation. c) Renal: • Renal mechanisms operate to compensate for metabolic acidosis and return the serum pH towards normal • Anions that replace HCO_3^- 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 further H^+ secretion) • Buffering systems include: Bicarbonate, Phosphate, Ammonia	Buffering in blood CO_2 expiration via lungs Acid secretion in kidney + buffering in urine All 3 to pass

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Question 4

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES & MARKS
Question 4 Thermoregulation	4.1 What mechanisms does the body use to regulate temperature? PROMPT: What mechanisms are activated by cold? PROMPT: Are any voluntary?	a) **Activated by cold:** Shivering, Hunger, Increased voluntary activity, adrenaline and noradrenaline secretion, decreased heat loss, cutaneous vasoconstriction, curling up, horripilation	4 to pass
		b) **Activated by heat:** Increased heat loss, cutaneous vasodilation, sweating, increased respiration, decreased metabolic heat production, anorexia, apathy & inertia	4 to pass
	4.2 How are these temperature regulating mechanisms controlled?	Reflex responses activated by cold controlled from posterior **hypothalamus** Those activated by warmth are controlled primarily from the anterior **hypothalamus**	Bold to pass

Question 5

Question 5.1	Please outline the different ways in which a substance can cross a cell membrane	Passive • Diffusion • Facilitated diffusion Active • Endo/exocytosis • Ion channels –ligand, voltage, mechanical gated • Active transport Primary and secondary	3/5 methods to pass
Question 5.2	Can you please explain the process of secondary active transport? PROMPT: Give a clinical example	The movement of an ion down its electrochemical gradient provides energy to transport another substance against its electrochemical gradient. Example – Na/glucose, Na/ aminoacids	Basic concept or clinical example to pass

ACEM PRIMARY 2011/1 PATHOLOGY VIVA Thursday (April 7) Afternoon Session ***Candidate Number*****…… AGREED MARK……**

TOPIC	*QUESTION*	*ESSENTIAL KNOWLEDGE*	*PASS CRITERIA / COMMENTS*
Question 1. **Atrophy**	1**. What is atrophy?**	**Shrinkage in the size of an organ or tissue** due to decrease in cell size and number.	Must know
	2. **What are the causes of atrophy?**	• Disuse • Denervation • Diminished blood supply • Inadequate nutrition • Loss of endocrine stimulation • Pressure	At least 4
	3. Give **some examples of atrophy**	• Fracture disuse • damage to nerves causing muscle atrophy • breast/reproductive organs from oestrogen lack	At least 2
Question 2. **Normal Haemostasis**	1. List the **sequence of events in normal haemostasis after vascular injury**	1. **Transient vasoconstriction** [Neurogenic & humoral factors (include endothelin – endothelium derived vasoconstrictor)] 2. **Primary haemostatic plug - platelet.** 3. **Secondary haemostatic plug**: coagulation cascade activated by tissue factor and platelet phospholipids, fibrin polymerization "cementing" platelets 4. **Limit spread**: tissue plasminogen activator & thrombomodulin	3 of 4 bold
	2. Describe the **creation of the Primary Haemostatic Plug?**	**Platelets** bind via 1. glycoprotein Ib (GpIb) receptors to 2. von Willebrand factor (vWF) on 3. exposed extracellular matrix (ECM) are 4. activated undergo 5. shape change and 6. granule release: adenosine diphosphate (ADP) and thromboxane A_2 (TxA_2) 7. additional platelet aggregation through platelet GpIIb-IIIa receptor binding to fibrinogen	3 of 7 (plus must say **platelets**)

Question 3. Tuberculosis	**1. What is secondary tuberculosis?**	Pattern of disease that arises in a previously sensitised host	**previously sensitised host**
	2. How may infection occur in secondary tuberculosis?	1. May follow shortly after primary infection (<5%) **2. Reactivation of latent organisms** • Typically in areas of low disease prevalence **3. Reinfection** • Typical in regions of high prevalence	**Items 2 and 3**
	3. Describe the pathological features in the lung of secondary infection with TB.	• Locale - **apical UL in secondary** • **Area of inflammation / granuloma** / multinucleate giant cells • Central **caseous necrosis** • **cavitation** • **Healing with fibrosis and calcification** • +/- Complications include tissue destruction, **erosion of blood vessels, miliary spread, pleural effusion, empyema, fibrous pleuritis**	**Need 3 of:** • Apical site • Inflammation / granuloma • Caseous necrosis • Cavitation • Fibrosis / calcification
Question 4. **Chronic gastritis**	1. What are the **causes of chronic gastritis?**	• **H Pylori** • Chronic bile reflux • NSAIDS • Autoimmune • Allergic response • Infections • Radiation • Mechanical • Psychological stress • Chronic irritants (coffee, alcohol, caffeine) • Systemic disease • (Crohns, amyloid, graft vs host)	**H pylori +** 2 others
	2. Describe the features of **H pylori induced chronic gastritis**	• Most **common cause** • predominantly **antral** • **High acid** production • **Hypogastrinaemia** • Generates **ammonia** (specific test) • **Disruption normal mucosal defence** mechanisms	2/5
	3. What are the **complications of gastric ulcer?**	• Bleeding (15-20%) o Accounts for 25% of ulcer deaths • Perforation • Obstruction • Gastric adenocarcinoma (complication of chronic H. Pylori pangastritis)	2/3

Question 5. **Subarachnoid Haemorrhage**	1. What is the **most frequent cause of subarachnoid haemorrhage?**	• **Rupture of an aneurysm** • (less common causes include ext of traumatic haem, H/T intracerebral bleed into ventricular system, AVM, bleeding disorders, tumour)	Rupture of aneurysm to pass
	2. **Where are saccular aneurysms commonly located?**	• **Most** near major arterial branch points along the circle of Willis or a major vessel just beyond (= **anterior cerebral circulation**) • 40% ant comm art • 34% middle cerebral art • 20% int carotid/PICA • 4% Basilar/Posterior Cerebral	At least anterior circulation and 1 other to pass

	3. What are the genetic risk factors for saccular aneurysms?	• Generally unknown, not 'congenital'
		• Some genetic risk
		  o Polycystic kidney
		  o Ehlers Danlos type 4
		  o Neurofibromatosis type 1
		  o Marfan's)
		  o Fibromuscular dysplasia
		  o Aortic coarctation
			2/6

	4. What are the pathological **consequences of subarachnoid haemorrhage?** Prompt for "Late"	• **Early** - vasospasm and additional ischemic injury - increased intracranial pressure • **Late** - meningeal fibrosis & scarring - CSF obstruction	Need 2

Comments: _____

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ACEM PRIMARY 2011/1 PATHOLOGY VIVA Friday (April 8) Morning Session *Candidate Number*…… AGREED MARK……

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	PASS CRITERIA / COMMENTS
Question 1. **Cell Death / Necrosis**	**1. Describe the cellular changes in necrosis** PROMPT Start with the cellular features.	• Usually irreversible injury • Often adjacent inflammation • **Swollen cells** • Increased eosinophilia • Myelin figures (whorls of cell membrane bits) • Nucleus fades (karyolysis), may shrink (pyknosis) and then fragments (karyorrhexis) • Organelle disruption → amorphous mass • **Cell membrane disrupted**, contents released	• Swelling • Disruption of cell integrity.
	2. What are the patterns of tissue necrosis? PROMPT What are the different macroscopic appearances of necrotic tissues?	• **Coagulative** (architecture preserved) • **Liquefactive** (digestion → liquid viscous mass) • Caseous (friable white) • *Gangrenous (usually applied to limb. Typically coagulative. Superimposed liquefaction from infection → 'wet gangrene') • *Fat necrosis (focal areas of fat destruction) • Fibrinoid (microscopic feature of Ag-Ab complexes in vessel walls from immune mediated)	• Coagulative • Liquefactive Prompt with names needs to describe difference *these terms clinical not true pathology terms

Question 2.
 	
Cell derived mediators of inflammation
	1. Which mediators of inflammation are derived from cells?	• Preformed
		  ○ **Vasoactive amines**
		  ▪ **Histamines**
		  ▪ Serotonin
		• Newly synthesized
		  ○ **Arachidonic metabolites**
		  ▪ Prostaglandins
		  ▪ Leukotrienes
		  ▪ Lipoxins
		  ○ Reactive Oxygen Species
		  ○ Platelet activating factors
		  ○ Nitric Oxide
		  ○ Cytokines (TNF, IL1)& Chemokines
			Pass = bold + 1 other

	2. Which cells release histamine?	Widely distributed in tissues, richest sources: • **Mast cells** • **Basophils** • **Platelets**	**Pass =/> 2**
	3. What **are the effects of histamines in an inflammatory response?**	• **Dilation of the arterioles** • **Increased vascular permeability of the venules** • Can cause constriction of large arteries	**Pass = bold (2)**

Question 3. **Measles**	1. **Describe the pathogenesis of measles** PROMPTS: What type of virus is measles? What is the mode of transmission?	1. **Paramyxovirus (single stranded RNA)** 2. **Respiratory droplet spread** 3. Multiplies in upper respiratory tract epithelial cells 4. >lymphoid tissue where it replicates in mononuclear cells 5. haematogenous spread 6. Preventable by vaccination as only single strain. 7. Epidemics amongst un-vaccinated individuals	• **Virus** • **Respiratory droplet spread** • **+ 1 other**
	2. What type of immune responses occur in measles?	• **T cell mediated immunity** controls infection + causes rash • **Antibody mediated** protects against re-infection • epidemics in unvaccinated hosts	• cell mediated • antibody mediated
	3. **Describe some of the systemic features of measles virus infection.** Prompt: What are some complications of measles infection?	• **Rash**–blotchy, red/brown. Skin hypersensitivity reaction • Oral mucosal ulceration – **Koplik's spots** • **Croup** • **Interstitial pneumonia** • **Conjunctivitis, Keratitis**, with scarring and visual loss • **Encephalitis**; - plus SSPE, measles inclusion-body encephalitis • **Diarrhoea** with protein losing enteropathy • **Immunosuppression** • **Secondary bacterial infection**	• Rash • + 3 others
Question 4. **Ischaemic bowel disease**	**1. What conditions can lead to infarction of bowel?** **PROMPT; by what mechanisms do these conditions cause injury**	**Acute vascular obstruction** -atherosclerosis (esp. origin major vessels) -aortic aneurysm -hypercoagulable states -OC use -embolism **Intestinal hypoperfusion** -cardiac failure -shock -dehydration -vasoconstrictive drugs Systemic vasculitis -Henoch-Scholein purpura -Wegener's granulomatosis Mesenteric venous thrombosis -hypercoagulable states -invasive neoplasms -cirrhosis -trauma -abdominal masses	Bolded headings with 4 clinical examples to pass
	2. **Describe the intestinal response to an acute ischaemic insult.** Prompt: what is the mechanism by which ischaemic bowel injury occurs?	• Initial hypoxic injury • **Secondary reperfusion injury** - major injury in this phase - free radical production, neutrophil infiltration, inflammatory mediator release • Magnitude of response determined by - vessels affected - timeframe over which ischaemia develops	Must know that it is predominantly a **reperfusion type injury**
	3. **Which parts of the bowel are most susceptible** to acute ischaemic injury and **why**?	**Watershed zones** -splenic flexure, sigmoid colon and rectum -located at end of arterial supply Surface epithelium: Villi more at risk than crypts -intestinal caps run from crypts up villi to surface	Must be able to explain why **watershed zones are most susceptible** to injury.

Question 5. **Hepatic Failure**	1 What are the **causes of acute liver failure?**	• **Drugs and toxins:** Paracetamol, halothane, rifampicin, mushrooms, CCL4 • **Infections**: hepatitis A, B and (rarely) C. Mechanism: direct toxic eg paracetamol,mushrooms Or toxicity and/or immune mediated eg Hepatitis virus	**3 causes -** at least 1 drug and 1 infection
	2. What are the **clinical features of liver failure?**	• Jaundice • Ascites • Hypoalbuminaemia • Hyperammonemia→ encephalopathy • Coagulopathy • Portal hypertension • Foetor hepaticus • Spider naevi • Palmar erythema • Hypogonadadism + gynaecomastia	At least **5 features**
	OPTIONAL (Good candidates) What **do you understand by hepato-renal syndrome?**	• Renal failure in pt with severe chronic liver disease with no obvious cause for the renal failure. Features include: • Na retention • Impaired free water excretion • Decreased renal perfusion and GFR	Any features

Comments: _____

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ACEM PRIMARY 2012/1 Anatomy VIVA March 29 Thursday Morning Session 1 *Candidate Number……..* AGREED MARK……

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: CXR Borders of heart, lung anatomy LOA: 2	a) Identify the structures that make up the mediastinal contours on this CXR b) Describe the lobes of the lungs and their fissures. (note: these may not be actually visible on the CXR we have, but candidates can show where they would be..)	**Right: R Brachiocephalic v, SVC, R pulmonary trunk , R atrium** **Left: Aorta, Pulm trunk, L atrium, L Ventricle** **Both lungs: upper and lower lobes are separated by the oblique fissure** (from T2 posteriorly to 6th costal cart anteriorly). **On the right the upper and middle are separated by the transverse fissure** (at level of R lung hilum along line of 4th rib) Left lung **–** prominent cardiac notch in lower lobe.	Pass criteria: At least 6 of **bolded** to pass? All **bold**
Question 2 Bone: Ankle joint LOA: 1	(a) Demonstrate the bony features of the ankle joint (b) Demonstrate the ligaments that stabilise the ankle joint (name and describe / show attachments)	• Articular surface of **distal tibia** including **medial malleolus**. • **Lateral malleolus** of distal fibula. • Articular surface of **talus** • **Lateral ligament:** From **lateral malleolus**. **Ant. talofibular**(weakest), **Post talofibular**(strong), **Calcaneofibular** • **Medial ligament** (deltoid): Fans out from **medial malleolus to attach to talus, calcaneus and navicular** (4 parts: tibionavicular /tibiocalcaneal /ant. and post tibiotalar) • Ant. and post tibiofibular ligaments also shown on model	All bold All 3 bolded for lat, and medial (at least two attachments)
Question 3 Lateral compartment of leg (Model lower limb) LOA:1	a) Identify the muscles of the lateral compartment of the leg and describe their origins and insertions b) What is their nerve supply? c) What are their actions?	1. **Origins & Insertions** a. F. longus i. Origin: **Head** + **prox** 2/3 lat surface of **fibula** ii. Insertion: **Base of 1st MT** + medial cuneiform **b.** F. brevis **i.** Origin: **inferior 2/3** of lat **fibula** ii. Insertion: Dorsal tuberosity **base of Vth MT** 2. **Superficial fibular** (peroneal) nerve: L5 S1 S2 **Everts** foot / weakly plantarflexes ankle	Fibularis tertius is in the anterior compartment F. longus passes behind the lateral malleolus and crosses the plantar aspect of the foot to insert medially

Question 4 Photo: upper limb, nerves of hand-motor and sensory Pg 163 McMinn's LOA: 1	a) Identify the ulnar nerve in this photo and adjacent structures b) Demonstrate where sensation changes may occur if the ulnar nerve is injured in the forearm	**25. ulnar n** 23. ulnar artery 9. flexor carpi ulnaris 26. deep branch of ulnar nerve 11. flexor digitorum profundus **16. median n** **Palmar and dorsal aspects of 1 and a half ulnar fingers**, adjacent palmar and dorsal aspects of hand and ulnar aspect of wrist	Ulnar n and Median n and 2 other structures to pass Finger distribution to pass
Question 5 Discussion: Blood supply of the myocardium LOA: 2	a)Describe the arterial blood supply of the myocardium. Prompt: Tell me about the coronary arteries. b)What is the blood supply of the conducting system? c*)Describe the venous drainage of the heart.*	LCA/RCA from aorta. LCA branches into 1. **LAD** (or AI)– IV groove to apex, anast with PDA in IV groove. Anterior surface both **ventricles +** ant 2/3 **IV septum** 2. **Circumflex** – Coronary groove to posterior surface heart. Supplies **lat LV**. Anast with RCA. PDA in 1/3. (L dominant) **RCA** coronary groove. **RV**, posterior 1/3 IV sept, post. surface, PDA in 2/3 (R dominant) **SA node: RCA** in 60%. LCA in 40%. **AV node: RCA** in 80%. LCA in 20%. AV Bundles: LAD in most. *Coronary sinus into RA receives from* 1. *great cardiac vein: ant IV groove → coronary groove → coronary sinus* 2. *middle cardiac vein: Post IV groove → coronary sinus* 3. *small cardiac vein: inferior surface → coronary groove → coronary sinus* *Some ant cardiac veins into RA.*	Must describe 3 vessels in bold and some description of what they supply to pass. SA/AV node: usually by RCA + AV bundles by LCA to pass *Bonus details*

ACEM PRIMARY 2012/1 Anatomy VIVA March 29 Thursday Afternoon Session 2 *Candidate Number……..* AGREED MARK……

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: CT Brain LOA: 2	a) Identify anatomical features of the brain shown in this CT scan b) Describe the territories that the cerebral arteries supply.	F**rontal, temporal or parietal (or both) and occipital lobes**, including gyri and sulci. Thalamus, internal capsule(ant/post limbs)**,** caudate nucleus **Lateral ventricles** (ant/post horns), choroid plexus posteriorly **Falx cerebri** Branches of Circle of Willis**:** **Anterior cerebral a – Frontal lobe,** medial and superior surface **Middle cerebral a - Temporal lobe and lateral surface** **Posterior cerebral a - Occipital lobe,** inferior surface	B**old to pass** All **bold**
Question 2 Bone: Hip joint LOA: 1	(a) Demonstrate the bony features of the hip joint (b) Describe the ligaments that stabilise the hip joint and demonstrate their attachments.	• **Acetabulum**: Formed by the **ilium, ischium and pubis.** Lunate surface of acetabulum. Acetabular notch. • **Femoral head** • **Iliofemoral: AIIS and acetabular rim(very strong) to intertrochanteric line** • **Pubofemoral:** obturator crest of pubis to blend with medial aspect of iliofemoral lig**.** • **Ischiofemoral:** posterior acetabular rim (weakest), spirals supero-laterally to base of greater trochanter • Transverse acetabular: bridges acetabular notch • Ligament of head (minimal role in stability), acetabular notch to fovea of head	Bold Iliofemoral and one other

Question 3 Posterior compartment of leg (Model leg) LOA: 1	a) On this model demonstrate the muscles of the **posterior** compartment of the **leg**. b) Demonstrate the origins and insertions of the superficial group c) What is their Nerve Supply? d) Describe their action	**1. Superficial**: **Gastrocnemius /soleus/**plantaris a. Gastroc **i. Lat head** from lat aspect **lat femoral condyle** ii. **Medial head from popliteal surface of femur** above medial femoral condyle. iii. **Insertion**-Into posterior surface of **calcaneum** via **calcaneal (Achilles) tendon** (**along with soleus** + plantaris) iv. **Soleus** Origin from prox ¼ fibula + soleal line & middle 1/3 tibia 2. Nerve supply - All **tibial nerve S1 S2** 3. Action: All **plantarflex** ankle. Gastrocnemius flexes leg at knee	Superficial + deep groups divided by transverse intermuscular septum. Nerves and blood vessels run in deep sub-compartment Bolded
Question 4 Photo upper limb: Rotator cuff muscles- actions and nerve supply LOA: 1	a) Identify the rotator cuff muscles in this image b) What are the actions of the rotator cuff muscles? c) What are their innervations?	**Supraspinatus Infraspinatus Teres Minor Subscapularis** They form a musculotendinous structure around the shallow glenohumeral jt, protecting the jt and gives it **stability**. **Supraspinatus – initiates shoulder abduction** **Infraspinatus and teres minor-lateral arm rotation** **Subscapularis- medial arm rotation** Supraspinatus- **Suprascapular n** (C4,**C5,**C6), Infraspinatus- **Suprascapular n** (**C5**,C6), Teres minor- **Axillary n** (**C5**,C6) Subscapularis- **Upper and lower subscapular n**(C5,**C6**,C7)	All bold Joint stability plus one bold 2/4 required
Question 5 Portal Systemic Anastamoses LOA: 2	a)Describe the portal-systemic anastamoses. b) When do these become clinically significant?	1. **Oesophageal veins** draining into azygos (systemic) or left gastric vein (portal) 2. Rectal: inf & middle rectal veins into IVC (systemic) and sup rectal vein into inf mesenteric (portal) 3. Umbilical: Paraumbilical (portal) and epigastric veins ant abdominal wall (systemic) 4. Retroperitoneal: visceral (portal) veins on bare areas of organs (liver/ colon/ spleen) and veins of post abd wall (systemic) 1. Obstruction to portal flow from liver disease/ other obstruction (portal hypertension) 2. Large volume portal-systemic shunting (no valves) with dilation 3. Risk of **major haemorrhage (Oesophageal varices)**	Oesophageal + 1 other to pass Bold

ACEM PRIMARY 2012/1 Anatomy VIVA March 30 Friday Morning Session 3 *Candidate Number……..* AGREED MARK……

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: Ureter in the pelvis LOA: 2	a) Describe the path of the ureter in this Xray b) Where does the ureter narrow in the pelvis anatomically c) Give a clinical example of why this may be important	Descends on Psoas m just medial to the **tips of the vertebral T/V processes** to **cross the pelvic brim at the** bifurcation of the common iliac arteries/**SIJ** Bends laterally along the **lateral wall of the pelvis**, towards ischial spines, then turns medially to **enter the base of the bladder** 1.**Pelvic brim** 2. **VUJ** at bladder base Site of ureteric calculus obstruction	B**old to pass** One of two **bold**
Question 2 LOA: 1 Bones : Elbow Joint	(a) Demonstrate the bony features that form the elbow joint articulation (b) Describe the ligaments of the elbow joint and demonstrate their attachments	• Humero-ulnar articulation – between **trochlea** of humerus & **trochlear notch** of ulna • Humero-radial articulation – between **capitulum** of humerus & **head of radius** • **Radial collateral ligament** (lateral) – from **lateral epicondyle** of humerus to **annular ligament** of radius • **Ulnar collateral ligament** (medial) – from **medial epicondyle** of humerus to **coronoid process / olecranon** of ulna - 3 components : *anterior* (strongest), *posterior*, *oblique* bands	Bold to pass
Question 3 Quadriceps muscles LOA: 1 Introduce: "We'll remove the Sartorius muscle and TFL, can you please…"	1. Demonstrate the quadriceps muscles on this model Prompt: what are the origins and insertions?	1. Quadriceps a. *Rectus femoris*: i. Origin: **Anterior inferior iliac spine** + ilium superior to acetabulum **ii.** Insertion: Via **quadriceps tendon** into **tibial tuberosity** *b.* *Vastus medialis* i. Origin: Inter-trochanteric line and medial lip of linea aspera ii. Insertion: **Quadriceps tendon** + **medial patella** /	*Minimum: Correctly identify all four and name origin of Rectus femoris and insertion of all.*

	2. What are their actions? 3. What is their nerve supply?	patellar retinacula c. *Vastus lateralis* i. Origin: Greater trochanter + lateral lip of linea aspera ii. Insertion: **Quadriceps tendon** + patellar retinacula d. *Vastus intermedius* i. Origin: Ant + lat shaft of femur **ii.** Insertion: **Quadriceps tendon** **2. Extend the knee.** **Rectus femoris also assists in hip flexion** **3. Femoral nerve** L2,3,4	
Question 4 Cubital fossa LOA: 1 Introduce: "This image is the LEFT elbow, and this is the LATERAL side"	a) Describe the boundaries of the cubital fossa b) Identify its contents in this photo	**Superiorly – imaginary line connecting the epicondyles** **Medially – Pronator teres** (flexors of forearm from CFO) **Laterally – Brachioradialis** (extensors from lat epicondyle) **Floor – Brachialis and Supinator muscles** **Roof – deep fascia/bicip. aponeurosis**, subcut fat, **skin** **Brachial a**(3) dividing into radial(13) & ulnar (15) arteries. **Biceps brachii tendon/aponeurosis**(1+2) **Median n**(9) **Radial n** (14)– deep between Brachioradialis and Brachialis Posterior interosseous n(11)	**Bold** required
Question 5 Nerve supply to face LOA: 2	a) What is the sensory supply of the face? (Prompt: what nerves supply skin sensation on the face?) b) What is the motor supply to facial muscles ?(Prompt: muscles of facial expression)	**Trigeminal nerve branches:** **Ophthalmic**; supratrochlear, supraorbital, infratrochlear, ext nasal, lacrimal..line from angle of eye, dorsum nose **Maxillary;** Zygomatic (temporal, facial), infraorbital, lat. nose **Mandibular;** auric temporal, buccal, mental Small supply to angle of jaw from great auric **Facial nerve, motor root:** Emerges from stylo mastoid foramen, and engulfed by parotid 5 motor branches: **Temporal** (above eyes) **Zygomatic** (below eyes) **Buccal** (upper lip) **Marginal mandibular** (lower lip) **Cervical** (platysma, neck)	**Bold** required 3 of 5 branches required

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Haemostasis LOA: 1	**In hemostasis, describe the sequence of events at the site of vascular injury**	• Transient **vasoconstriction** by neurogenic and via local secretion of factors eg endothelin • Endothelial damage exposes ECM, leads to • **Platelet adherence, secretion & activation** leading to the primary haemostatic plug • Tissue factor is exposed, resulting in activation of **coagulation cascade** and **thrombin** generation, converting **fibrinogen to fibrin** leading to secondary haemostasis consolidating the initial platelet plug • **Polymerised fibrin and platelet aggregates** to form permanent plug • Counter regulatory mechanisms limit plug to site of injury	**Must state** • Vasoconstriction • Platelets • Coagulation cascade • Fibrin
	What factors restrict clotting to the site of vascular injury? **Prompt:** What prevents runaway clotting of the vascular tree?	• **Endogenous anticoagulants** o **Antithrombins** eg AT III, inhibit thrombin and IXa, Xa, Xia, XIIa o **Proteins C and S -** inactivate Va, VIIIa o **TFPI** (Tissue factor pathway inhibitor) • **Fibrinolytic cascade activation** o **Plasmin** from plasminogen (via factor XII or plasminogen activators) to break down fibrin & interfere with its polymerisation o **tPA** = the most important plasminogen activator	**Must include** concepts of : • Endogenous anticoagulants • Activation fibrinolysis
Question 2 Fracture healing LOA: 1	**How do fractures heal?** Prompt: What are the timeframes of these stages? **What factors impair fracture healing?**	1 Haematoma formation/fibrin mesh - hrs 2 Inflammatory cell influx - days 3 Fibroblast/ Osteoprogenitor cells-procallus 4 Organised haematoma - 1wk, 5 Woven bone , bony callus - 2-3 wks 6 Callus maturation remodelling - 6 wks Inadequate immobilisation, severe displacement, vascular compromise, infection /FBs, poor nutrition, systemic illnesses	Must have reasonable sequence and approximate times, at least 4 components to sequence **At least 3**

Question 3 Subarachnoid haemorrhage	**Where in the cerebral circulation are saccular (berry) aneurysms commonly located?** ***Prompt:*** At what part of these vessels are they most likely to arise? **What factors increase the likelihood of rupture of these aneurysms?** **What are the pathological sequelae of subarachnoid haemorrhage?**	90% near major arterial **branch points – Anterior** Cerebral A / ACoA (40%); MCA / AChoroidalA (34%); ICA / PCoA (20%); Basilar A / PCoA. Multiple in 20% – 30% cases at autopsy. Increased likelihood with **size** (> 10mm) – 50% risk of rupture per year. May occur at anytime but in about 1/3 associated with **acute increases in ICP** (e.g. straining at stool; orgasm). Acute events (hours to days) – **ischaemic injury (stroke)** from **vasospasm** (especially basal SAH). Late events (healing process) – meningeal fibrosis and **scarring**; may lead to obstruction to **CSF** flow and /or to CSF absorption. Death	Mention of branch points and anterior circulation to pass. **Bold** to pass. **Two of bold** to pass.
Question 4 Endocarditis LOA: 1	**What factors predispose patients to infective endocarditis?** **Which organisms commonly cause infective endocarditis?** **What are the complications of infective endocarditis?** (Prompt to get each group)	**Cardiac factors** – Myxomatous mitral valve, calcific aortic stenosis, bicuspid aortic valve, prosthetic valves, rheumatic heart disease **Host factors** – neutropaenia, immunodeficiency, malignancy, therapeutic immunosuppression, diabetes, alcohol, intravenous drug use, bacteraemia. **Streptococcus viridans**; **Staph aureus**; Staph epidermidis; enterococci; HACEK (Haemophilus, Actinobacillus, Cardiobacterium, Kingella); fungi **Local – erosion / destruction** of underlying cardiac tissue (valve, myocardium); abscess formation. **Systemic – systemic emboli** – infarcts / septic infarcts – brain, kidneys, lung, subcutaneous tissues, retina. Other - glomerulonephritis (immunologically medicated)	Need 4 (2 from each group) **Bold plus one other** to pass **1 local and 1 systemic**
Question 5 ALTE/SIDS LOA: 2	**What is Sudden Infant Death Syndrome?** **What risk factors have been identified?**	**The sudden death of an infant under 1 year of age which remains unexplained after thorough investigation and autopsy.** Parental risks- young mum <20, maternal smoking or drug use, low SES, deficient pre-natal care Infant risks- premature, low BW, male, SIDS in sibling, brainstem anomalies. Environment- prone sleeping, soft bedding and co-sleeping, hyperthermia	Accurate definition (age & unexplained nature) **At least 3** risk factors

ACEM PRIMARY 2012/1 Pathology VIVA March 29 Thursday Afternoon Session 2 ***Candidate Number……..*** **AGREED MARK……**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Thrombosis LOA: 1	**What factors predispose to thrombus formation?** (Prompt: Give an example of a clinical situation where each factor occurs) **Expanding on hypercoagulable states, what are the broad categories and give examples of each type?**	Virchow's triad - • **Endothelial injury** • **Alteration in blood flow** • **Hypercoagulability** • **Primary (Genetic)** Mutations- Factor V Leiden, Prothrombin Increased - factors VIII, IX, XI, or fibrinogen Deficiencies- AT3, Protein C, S • **Secondary (Acquired)** Prolonged bed rest, immobilisation, MI, AF, Tissue injury, prosthetic valves, cancer, DIC, HITS, Anti phospholipid Antibody Cardiomyopathy, nephrotic syndrome, pregnancy, post partum, OCP, sickle, smoking Note often multifactorial	**Bold 3** Plus **1 example for each** **Bold + 2 examples** **Bold + 3 examples**
Question 2 Septic shock LOA: 1	**How do microbes initiate septic shock?** **What are the effects of the mediators on the coagulation pathway?** **What are the consequent effects on tissues?**	1. Interaction with innate cells of immune system- neutrophils. macrophages and monocytes 2. Humoral interaction to activate complement and coagulation path 3. Direct endothelial action 4. End result is mediator release TNF,IL 6,8,10, NO,PAF, PAI-1 Microvascular thrombosis, decreased fibrinolysis, DIC Tissue ischaemia, multi organ failure	**at least 3 to pass** **2/3 to pass** **Either**
Question 3 Jaundice LOA: 1	**Outline the normal metabolism and elimination of bilirubin?**	Bilirubin production from heme (breakdown of senescent **erythrocytes**) 2. Binds to serum **albumin** and delivered to liver. 3. **Hepatocellular uptake**. 4. **Glucuronidation** – bilirubin glucuronides excreted into bile. 5. Gut **deconjugation** – colourless urobilinogens. These and pigment residues excreted in faeces. ~20% urobilinogens **reabsorbed** in ileum and colon and returned to liver. Small amount of reabsorbed urobilinogen excreted in urine	**three of bold** to pass

	What are the common causes of jaundice? (Prompt for bold)	Disorders that affect the production and metabolism of bilirubin: 1. Predominantly unconjugated: ↑**production** (haemolyisis; resorption of blood from internal haemorrhage; ineffective erythropoiesis); ↓hepatocyte uptake (drug interference with membrane carrier systems; Gilbert syndrome – some cases); impaired bilirubin conjugation (physiological jaundice of newborn - ↓UGTA1 activity; breast milk jaundice - β-glucuronidases; genetic deficiency of UGTA1 (Crigler-Najjar); Gilbert syndrome (autosomal recessive ↓UGTA1 activity); **hepatitis** (diffuse hepatocellular disease eg viral; drugs; cirrhosis). 2. Predominantly conjugated: **impaired bile flow;** deficiency of canalicular membrane transporters (Dubin-Johnson syndrome; Rotor syndrome)	**Bold to pass**
Question 4 ARDS LOA: 2	**Describe the pathogenesis of ARDS** **What conditions are associated with the development of ARDS?**	Initial **injury to alveolar capillary membrane** (endothelium); **acute inflammatory response** (neutrophil mediated); results in **increased vascular permeability** and alveolar flooding; fibrin deposition; formation of hyaline membranes; and widespread **surfactant abnormalities** (damage to Type II pneumocytes); eventually – organisation with scarring **Infection** (sepsis, diffuse pulmonary infection, gastric aspiration) **Physical / Injury** (trauma – head, pulmonary, fractures, near drowning, burns, radiation) **Inhaled irritants** (O2 toxicity, smoke, irritant gases and chemicals) **Chemical injury** (Heroin, barbituate, acetylsalicylic acid, paraquat) **Haematological conditions** (multiple transfusions, DIC) **Other** (pancreatitis, uraemia, cardiopulmonary bypass, hypersensitivity – organic solvents, drugs)	**3 of 4 bold** **Need 3 groups** (with **example** from each); **must include infection**
Question 5 Anaemia LOA: 2	**What are the causes of intravascular haemolysis?** **What are the manifestations of intravascular haemolysis?** (Prompt: In the blood? In the urine?)	-mechanical injury to cells (valves, microthrombi, other physical trauma) - complement fixation (eg transfusion reaction) -toxic injury (eg clostridia), - parasites (eg malaria) Anaemia, haemoglobinuria, haemoglobinaemia, jaundice, haemosiderinuria	**3 causes** **3 manifestations**

ACEM PRIMARY 2012/1 Pathology VIVA March 30 Friday Morning Session 3 *Candidate Number……..* AGREED MARK……

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Embolism LOA: 1	**What is an embolus?** **What types of emboli do you know of?** **What are the features of fat embolism syndrome?** **Prompt** – What systems may be affected in fat embolism syndrome?	A **detached intravascular** solid/liquid/gas **mass** that is carried by the blood stream from its site of origin to a **distant site**. • Pulmonary • Arterial thromboemboli • Fat emboli • Air emboli • Amniotic fluid • **Associated with long bone fractures,** rarely soft tissue injury/burns • Only 10% symptomatic • **Pulmonary insufficiency**- SOB, ↑RR,↑HR • **Neurologic symptoms**- irritability, restlessness, delirium, coma • **Anaemia**- due to RBC aggregation/haemolysis • **Thrombocytopaenia**- platelet adhesion/aggregation, leads to petechial rash	**Bold to pass** **3 examples to pass** **3/5 bold to pass**
Question 2 Wound Healing LOA: 1	**Describe the process of healing of an incised skin wound?** (Prompt: include the timing of these processes.) **What factors influence wound healing?**	**a) Formation of a blood clot – immediate** **b) Neutrophil migration at wound margins – within 24 hours** **c) Formation of granulation tissue (fibroblasts and vascular endothelial tissue). Blood vessels are leaky and proteins and fluid pass into the extravascular space leading to oedema– 24-72 hours** d) Cell proliferation and Collagen deposition – neutrophils are replaced by macrophages between 48 and 96 hours e) Scar formation – leucocytic infiltrate, oedema and increased vascularity disappear; increased accumulation of collagen – second week f) Wound Contraction – formation of myofibroblasts at the wound edges that contract. g) Connective tissue remodelling h) Recovery of Tensile strength – 10% at 1 week to a peak of 70-80% at 3 months a) Local (infection / mechanical eg motion of wound / FB / size, location, type eg incised vs blunt trauma) b) Systemic (nutrition / metabolic status / circulatory status / hormones)	**Bold 3 and 2 others = 5** **To pass: 2 local & 2 systemic**
Question 3 Cor	**What is cor pulmonale?**	Right sided heart failure that is **not secondary to left sided heart failure (pure RHF).** It can be acute (eg massive PE) or chronic (eg chronic lung disease).	**Bold** to pass

pulmonale			
	What are the common causes of cor pulmonale?	Diseases of pulmonary parenchyma (COPD; fibrosis; bronchiectasis). Diseases of pulmonary vessels (Primary pulmonary hypertension; recurrent PE; extensive pulmonary arteritis eg Wegener's granulomatosis). Disorders affecting chest movement (marked obesity; kyphoscoliosis; neuromuscular). Disorders causing pulmonary arterial constriction (hypoxaemia; metabolic acidosis; chronic sleep apnoea; altitude sickness). Common feature of all these is **pulmonary hypertension.**	**Bold** plus 3 other to pass
	What are the major morphological features of cor pulmonale? (Prompt: what are the organ features?)	Pulmonary congestion is minimal whereas engorgement of the systemic & portal venous systems may be pronounced. Heart: right ventricular hypertrophy and dilatation; leftward bulging of septum. Liver / portal system: congestive hepatomegaly; centrilobular necrosis; congestive splenomegaly Pleura, pericardial and peritoneal spaces: effusions; ascites. Subcutaneous tissues: oedema (dependent and peripheral portions of body; anasarca)	At least **three** to pass.
Question 4 UTI	**What organisms cause acute pyelonephritis?** Prompt: what are the most common?	**G-ve bacilli** (>85%), endogenous organisms E Coli, proteus, klebsiella, enterobacter, strep faecalis Other: staph, fungi, (viruses in immunocompromised and renal transplant patients)	**G-ve & 3 organisms pass**
	What steps are involved in ascending infection?	**5 steps:** 1. colonisation distal urethra 2. entry into bladder 3 . urinary tract obstruction / stasis of urine 4. vesicoureteric reflux 5. intrarenal reflux	Need to explain the steps clearly
	What are the features of chronic pyelonephritis?	**Chronic** = chronic reflux or obstruction causes pelvocalyceal damage. Recurrent infections lead to recurrent bouts of renal inflammation and **scarring**	**Bold** & concept
Question 5 Chronic Pancreatitis	**What are the morphological features of chronic pancreatitis?**	Parenchymal fibrosis, reduced number and size of acini with relative sparing of islets of Langerhans. Variable dilation +- blockage of pancreatic ducts. Destruction of exocrine parenchyma and in later stages destruction of endocrine parenchyma. Calcification.	**Any 3.**
	What are the clinical consequences?	Irreversible impairment of pancreatic function including: Diabetes; Steatorrhea; Malabsorption chronic attack not immediately life threatening but long term outlook poor(50% 20-25 mortality) Disease may be silent. Amylase, lipase may not raise in chronic attack	**Any 3**

		Pseudocyst	

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 LOA: 1 **HALF LIFE**	Define drug elimination half life Is there a formula you can use? *Prompt: What factors affect half-life?* *Prompt: Can you explain what that means?* How does knowledge of a drug's half life help us clinically?	**Time required to change the amount of drug in the body by ½ during elimination** T1/2 = 0.7 x Vd/clearance (0.7 approx log 2) **Indicates time to steady state after dose change.** 50% after 1, >90% after 4	Concept required Both bold to pass
Question 2 LOA: 2 **PENICILLINS**	Describe the mechanism of action of penicillins How does resistance to penicillins occur? In general, what is the anti-microbial spectrum of penicillin G? *Prompt: Could you be specific*	**Inhibition of cell wall synthesis**. Interfere with transpeptidation. Covalently binding to PBP. Important in the cross linkage. Bacteriocidal,. Only kills growing cells. a. Inactivation by beta lactamases b. Modification of target PBPs (Pneumo/entrococci) c. Impaired penetration of drug to PBP; impact on porin channels. Gram negatives d. Efflux pump (gram neg) Streptococci, meningococci, enterococci, some pneumococci, treponema pallidum, clostridia, non-betalactamase producing staphylococci	**At least 2 including beta-lactamases** **At least 3 bacteria**
Question 3 LOA: 1 **LITHIUM**	Describe the pharmacokinetics of Lithium What are some of the drug interactions with lithium What are the some side effects of lithium *Prompt: What other organ systems effects are there?*	**Absorption; rapid and near complete**. peak levels in 30-120min **Distribution; total body water** Vol.D 0.5 to 0.9L/kg Slow distribution Metabolism; none T ½; @20 hours. **Elimination; renal excretion** **Thiazide diuretics**- 25% reduction in lithium clearance Newer NSAID's – similar reductions in clearance Neuroleptics (except clozapine) and antipsychotics- enhancement of extrapyramidal syndromes **Neurological;** tremor, confusion, ataxia, dysarthria, new psychiatric symptoms Reduced thyroid function Nephrogenic diabetes insipidis – loss of responsiveness to ADH. Oedema Skin reactions; acneiform eruptions	2 neurologic symptoms

Question 4 LOA: 1 **ANTIEMETICS**	Name some antiemetics used in the Emergency Department. Compare the mechanisms of action of ondansetron and metoclopramide Describe the potential adverse effects of metoclopramide.	**Ondansetron (or Granisetron or Tropisetron)** **Metoclopramide** **Prochlorperazine** Diphenhydramine (or other antihistamines). Meclizine. Hyoscine. Benzodiazepines. Chlorpromazine. Droperidol Act at different receptors: Ondansetron: **Peripheral 5HT3 blockade** (vagal and spinal afferents, Reduces sensory visceral output) + Central 5HT3 blockade (vomiting centre and CTZ) Metoclopramide: **D2 blockade** (CTZ). Increases oesophageal motility. Increases LOS pressure. Increase gastric emptying CNS: Restlessness, drowsiness, insomnia, anxiety, agitation – common (20%), esp. elderly Extrapyramidal effects: **acute dystonia**, akathisia, parkinsonian effects, more likely with higher doses Tardive dyskinesia with chronic dosing	Bold to pass Bold to pass Must mention acute dystonia + one other CNS effect
QUESTION 5 LOA: 1 **DRUGS IN AGITATED PATIENTS**	List the drug classes which are used in management of acute agitation in the ED *Prompt: Can you give some specific examples?* What is the predominant mechanism of action of the atypical antipsychotics. Describe adverse effects of the atypical antipsychotics	**Benzodiazepenes** **Antipsychotics – Phenothiazines** eg chlorpromazine **Butyrophenones** eg haloperiodol **Atypicals** eg olanzapine , risperadone Barbiturates – phenobarbital **Serotonin** ($5HT_{2A}$**) receptor antagonism** Dopamine (D2) receptor antagonism (weaker effect) **Extrapyramidal reactions** - – less common than with older typical antipsychotics Tardive dyskinesia **Antimuscarinic effects** – dry mouth, urinary retention etc **Orthostatic hypotension** Weight gain Hyperglycemia Hyperprolactinemia Agranulocytosis (clozapine) Neuroleptic malignant syndrome	

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 LOA: 1 **PARTIAL AGONIST**	In the context of drug-receptor interactions, what is the difference between a full agonist and a partial agonist? Under what circumstances can a partial agonist act as a antagonist? *Prompt: Can you use opioids as an example?*	**High concentrations of full agonists can evoke a maximal response, but partial agonists cannot evoke maximal response at any concentration** In the presence of a full agonist Buprenorphine	
Question 2 LOA: 1 **TRIMETHOPRIM**	Describe the mechanism of action of trimethoprim Can you explain why trimethoprim and sulphonamides when used together are synergistic? How does resistance to trimethoprim occur?	**Inhibition of DNA synthesis**. Selective inhibition of bacterial dihydofolic acid reductase which is required from the step dihydrofolic acid to tetrahydrofolic acid. Much less efficient at inhibiting mammalian enzyme. **Inhibition of** sequential **steps in same pathway**. Sulphonamides inhibit dihydropteroate synthetase (PABA to DHFA), the step before that at which trimethoprim acts Reduced cell permeability Increased production of enzyme DHF reductase Alteration in the enzyme with reduced binding of drug	Any 1 of 3
Question 3 LOA: 1 **CARBAMAZEPINE**	Outline the clinical uses of carbamazepine Describe the mechanism of its anticonvulsant activity Outline some of the side effects of carbamazepine *Prompt: What other organ systems can it effect?* **Optional**: Can you name some drug interactions involving carbamazepine	**Anticonvulsant;** partial and generalised tonic-clonic seizures Treatment of bipolar mood disorder Trigeminal neuralgia **Blocks sodium channels** Inhibits high-frequency repetitive firing of neurons Presynaptic blocker of synaptic transmission (similar to phenytoin) **Ataxia** and diplopia, **drowsiness** (dose related CNS) **GI upsets** and hepatic dysfunction Erythematous **skin rash** Hyponatraemia and water intoxication Blood dyscrasias, including leukopenia common), and rarely aplastic anaemia and agranulocytosis. **Enzyme induction** (all anticonvulsants including itself). Valproic acid + phenytoin may inhibit carbamazepine elimination	Anticonvulsant + 1 other use CNS + one other

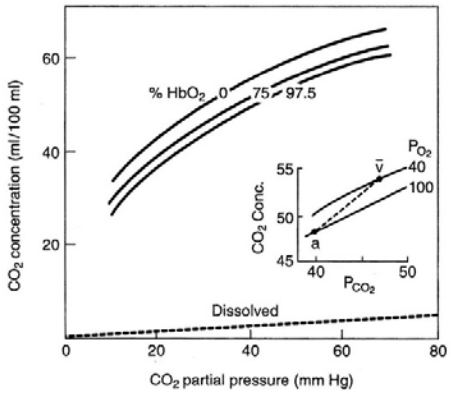
Question 4 LOA: 1 **INSULIN**	Describe the different types of insulin used in the routine management of Type I Diabetes. *Prompt: Please describe in terms of duration of action*	**Rapid and short acting** Clear soln, neutral pH, contain Zn rapid onset, short duration e.g. insulin neutral, insulin lispro, insulin glulusine - **Intermediate acting** Turbid soln, neutral pH, protamine in phosphate buffer (NPH) to prolong action e.g. insulin isophane, insulin aspart protamine **Long acting** Clear solution, soluble Slow onset, prolonged action Daily admin mimics basal insulin secretion e.g. insulin glargine, insuline detemir	Pass criteria: Identify existence of rapid, intermediate and long-acting insulin Aware that combination of therapies required to cover both basal requirements and post-prandial periods
	How are these properties used to achieve optimum glycaemic control?	Tight glycaemic control is achieved by a combination of insulins with different durations of action with an aim of replacing the basal insulin requirements (50%) and meal requirements (50%). This is done with **combinations of insulins with different duration of actions**	
	What type of insulin is used for intravenous infusion and why?	Short-acting regular soluble insulin as it immediately dissociates on dilution and so is able to more precisely delivered.	
	Optional: Describe the principles of operation of a subcutaneous insulin infusion device. PROMPT: Insulin pump.	External open-loop pump for insulin delivery. Delivers individualised basal and bolus insulin replacement doses based on blood glucose monitoring. Programmed by user. Consists of insulin reservoir, program chip, keypad and display screen attached to subcutaneously inserted infusion set.	

Question 5 LOA: 1 **DRUGS IN PROCEDURAL SEDATION**	List the classes of drugs used in emergency department procedural sedation *Prompt: for classes*	**Benzodiazepenes** **Dissociative anaesthetics (** ketamine**)** **Intravenous anaesthetics (** propofol**)** **Inhaled anaesthetics** (N2O ; volatile**)** **Opiates (** morphine**,** fentanyl)	**4 out of 5**
	Describe the elimination pharmacokinetics of propofol *Prompt: Why do patients wake up quickly?*	**Hepatic metabolism** producing inactive watersoluble compounds , excreted renally High plasma clearance exceeding hepatic clearance – thus extrahepatic clearance exists – probably via lungs. Termination of effect by **redistribution** from brain to skeletal muscle (waking after single induction dose at 8-10 mins) "Three compartment model" Short "half – life" making it suitable for infusions – rapid offset.	
	Describe the organ effects of propofol	**CNS: sedative/hypnotic – general depression of CNS activity**, reduced cerebral blood flow and reduction in ICP. Anti convulsant properties.Nil analgesic effect **Cardiovascular effects: hypotension secondary to arterial and venous vasodilatation** (reduced preload and afterload) – incr. effect with age and reduced intravascular volume. Some inhibition of baroreceptor reflex leading to small increase in heart rate response only **Respiratory effects: respiratory depression incl apnoea.** Reduction in tidal volume and rate Reduced response to hypercapnoea and hypoxia Reduction in upper airway reflexes. Other: Antiemetic	**One from CNS, CVS + Respiratory**
	Describe adverse effects of propofol	Effects related to organ system effects • Hypotension • Apnoea, respiratory depression • Loss of airway reflexes – obstruction and aspiration • Pain with injection Allergy – cross reactivity with egg allergy (emulsion) Propofol infusion syndrome (metabolic acidosis & tachycardia)	

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: LOA: 1 **DIFFERENCES IN DRUG METABOLISM**	What factors determine the difference in drug metabolism between individuals?	**Genetic** – enzyme level differences Diet – induce / inhibit enzymes Environmental – exposure to enzyme inducers **Age** – extremes have decreased enzyme activity or decreased levels of cofactors Sex – increased metabolic rate in males **Drug-drug interactions** – enzyme induction or inhibition, substrate competition **Disease states** - hepatic, pulmonary, cardiac, thyroid, inflammatory Liver size & function Circadian rhythm Body temperature	3 of 4 **bold** to pass
	What is meant by "enzyme induction"? *Prompt: What effect does it have on metabolism?* *Prompt: What effect does this have on the pharmacological action of the drug?*	Drug causes an increased rate of synthesis or decreased rate of degradation of enzyme causing: **accelerated substrate metabolism** **decreased pharmacological action** of the inducer or a co-administered drug.	**Bold** to pass
Question 2 LOA: 1 **PARACETAMOL**	Describe the metabolism of paracetamol? *Prompt: Does this change in toxic doses?*	Rapidly absorbed, peak conc at 30-60 minutes Slightly PP bound Partially metabolised by hepatic MEs to paracetamol glucuronide and sulphate (inactive) <5% excreted unchanged Half-life is 2-3 hrs	3 of 5
	What is the toxic dose and how does this cause toxicity?	150-200mg/Kg or >7g in adult. Conjugation AAs (gluthathione in particular) used up, metabolised to toxic metabs NAPQI. Toxic to liver / kidneys.	Reasonable approximation. Must have reasonable understanding of how toxicity is caused
	What are the clinical manifestations of toxicity?	GIT effects: **Hepatic** impairment. N/V, diarrhoea, abdo pain, dizzy, disorientation Renal failure	Hepatic + one other

Question 3 LOA: 1 **SELECTIVE B2 AGONISTS**	What B-receptor types are there?	B1, B2 + B3	Need B1 + B2
	What cellular processes do B-agonist - B-receptor coupling initiate?	Activation of all 3 receptor types results in stimulation of **adenylyl cyclase** and increased conversion of ATP to cAMP. Mediated by stimulatory coupling protein (Gs) via GDP and GTP	Need adenylyl cyclase
	What are the clinical uses of B2 selective agonists?	**Respiratory**, uterine and vascular smooth muscle relaxation Skeletal muscle K+ uptake	Need respiratory bronchodilation + one other
Question 4 LOA: 1 **WARFARIN**	What is the mechanism of action of warfarin?	Warfarin **inhibits reduction of inactive Vit K** epoxide (KO) to active hydroquinone (KH_2) form. Blocks γ-carboxylation of glutamate residues in **prothrombin (Factor II) and factors VII, IX and X** ,as well as endogenous anticoagulant protein C and S.	Need to know role of vitamin k
	Why is there a delay in the onset of action of warfarin?	8-12 hr delay due to partially inhibited synthesis and unaltered degradation of 4 vit k dependent clotting factors and depends on **degradation ½ life** in circulation eg factor VII- 6 hrs, IX 24-hrs, X - 40 hrs and II- 60 hrs)	Need to have some idea of delay in onset
	What pharmacological agents are used in the reversal of warfarin?	Vitamin K. FFP. Prothrombin Complex. Recombinant FVIIa	
	Optional: Describe the mechanisms of drug interactions with warfarin	Pharmacokinetic: Enzyme induction + inhibition. Altered protein binding Pharmacodymanic: Synergism. Competitive antagonism (Vitamin K)	3 required

Question 5 LOA: 1 **DRUGS IN AF** **SOTALOL**	List the classes of drugs used for the management of AF in the emergency department	B-blockers Ca-channel blockers Cardiac glycosides Class 1c antiarrhythmics Class 3 antiarrythmics	3 of 5
	Describe the pharmacodynamics of sotalol:	**Non-selective beta blocker, Class II** **Prolongs plateau phase Class III**	Need class II + III
	List the main side effects	Pro-arrthymic- Esp **prolongation of QT** and Torsades CCF Asthma, AV blockade	Prolonged QT + 1 other
	What drug interactions with Sotalol prolong the QT? *Prompt: What other interactions can occur with sotalol?*	**Drugs which prolong QT**- phenothiazines, Macrolides, eg erythromycin, quinolones antidepressants,- Increased risk of Torsades Drugs which cause hypokalaemia hypomagnesaemia increase risk of Torsades Myocardial depressant drugs- increased LVF Calcium channel blockers, class 1a antiarrythmics, may increase refractory time and contraction	2 examples

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1	1.1 Describe what happens to Cardiac Output during exercise. *Prompt: By what mechanisms?*	Increases (CO= SV x HR) ↑venous return and hence ↑end diastolic volume, ↑myocardial contractility, so ↑stroke volume. ↑ sympathetic drive and heart rate	**Increases + one mechanism stroke vol + one mechanism heart rate**
	1.2 What are the local mechanisms that maintain a high blood flow in exercising muscles?	↓in tissue PO_2, ↑tissue PCO_2, and accumulation of K^+ and other vasodilator metabolites, ↑ temperature in active muscle	**Need 3 to pass.**
Question 2	2.1 In what forms is carbon dioxide transported in the blood?	• Dissolved. • As carbamino compounds with proteins, especially Hb. • Hydrated in red cells – H^+ is buffered and 70% of HCO_3^- enters the plasma.	**Two of three to pass.**
	2.2 Please draw the carbon dioxide dissociation curve for normal arterial blood. Prompt: "Draw a graph showing the relationship between the pressure of carbon dioxide and the total carbon dioxide content in arterial 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.	**Reasonable shape of the curve indicating the near linearity in the physiological range.** Prompt if necessary.
	2.3 Where will the curve lie for venous blood and why?? Prompt: "Does the curve move up or down and why??"	• The **graph moves upwards** indicating greater CO_2 content per unit pressure. • 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.	**The candidate must understand that venous blood is able to carry proportionately more CO_2 than arterial blood.**

Question 3	3.1 By what mechanism is H^+ secreted in the distal tubules and collecting ducts of the kidney?	ATP driven proton pump. Aldosterone acts on this pump to increase H^+ excretion. Abundant carbonic anhydrase in the cells numerous tubulovesicular structures. Pumps in the vesicles $H - K^+$ ATPase	**ATP driven proton pump**
	3.2 In H^+ secretion, what is the limiting urine pH?	A urine pH of 4.5 is the maximal H^+ gradient against which transport mechanisms can secrete H^+	**pH 4-5**
	3.3 Describe the principal urinary buffers and what is their role?	HCO_3 buffer system particularly in the proximal tubules HPO_4^{2-} in the distal tubules NH_3 in the proximal and distal tubules	**2 examples + increased capacity to excrete H+**
Question 4	4.1 What is normal serum osmolality?	~ 290mOsmol/L	**Within the range 280-300**
	4.2 What substances contribute to serum osmolality?	Principally (all but 20mOsmols) the ions (Na, K, Cl, HCO3). Rest is other cations & anions, urea, glucose. Much less so proteins (due to high MW). Possibly alcohols or mannitol.	**Na+, Cl- and one other**
	4.3 How does plasma differ in composition to intracellular fluid?	*Intracellular* K+ and proteins high, many more 'miscellaneous' phosphates Na+, Cl & HCO3 low, (Figure 1-1 page 3)	**Na, K, protein differences**
Question 5	5.1 What is the main hormonal factor that stimulates the release of cortisol from the adrenal cortex?	Adrenocorticotropic hormone (**ACTH**)	
	5.2 What factors determine the rate of ACTH secretion?	**Increased by stress** (pain, emotional), drive for circadian rhythm through the hypothalamus via release of CRH (corticotropin releasing hormone) **Inhibited by circulating glucocorticoids** and afferent from baroreceptors	
	5.3 What happens to ACTH levels after prolonged treatment with high doses of glucocorticoids is stopped abruptly?	**Slowly increases over weeks** (the pituitary may not be able to secrete normal amounts of ACTH for as long as a month. Presumed to be secondary to diminished ACTH synthesis)	
	5.4 How can this be avoided?	This can usually be avoided by **slowly decreasing the dose over a long period of time.**	

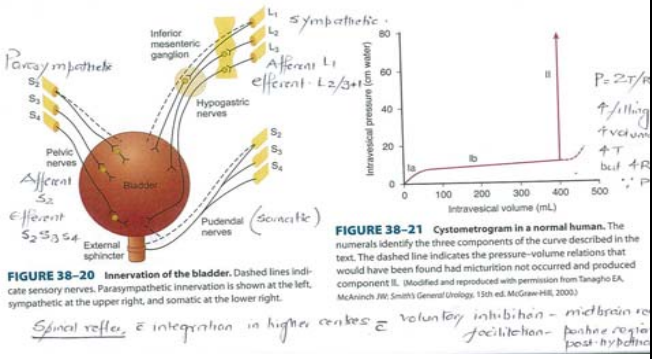
ACEM PRIMARY 2012/1 Physiology VIVA March 29 Thursday Afternoon Session 2 ***Candidate Number……..*** **AGREED MARK……**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 LOA: 1	1.1 What local factors can cause vasoconstriction or vasodilatation? 1.2 What is autoregulation in relation to blood flow?	Vasodilatation: ↑ CO2, ↑ lactate, ↑adenosine, ↑ local temp; ↓ O2 or ↓pH Vasoconstriction: ↓local temp, autoregulation. Autoregulation: blood flow remains constant by compensating pressure changes with peripheral resistance. 1) **Myogenic**: as blood pressure rises, muscle fibres in the blood vessels contract. The muscles correspond to the wall tension which is maintained at fairly constant level. Wall tension is determined by the radius of the blood vessels (pressure x radius). So rise in pressure, leads to a reduction in the radius of the blood vessel. 2) **Metabolic**: active metabolites cause local vasodilatation.	At least 4 to pass, and at least one in each group Need bold & some details to pass.
Question 2 LOA: 1	2.1 What factors determine the work of breathing? 2.2 What variables affect elastic workload? 2.3 What variables affect viscous resistance?	• **Elastic forces** of the lungs and chest wall • **Viscous resistance** of the airways and tissues **Larger tidal volumes** increase elastic workload. Elastic workload is increased by **reduced compliance** due to: o Lung volume - a person with only one lung has halved compliance. o Slightly lesser during inflation than during deflation. o Increased tissue mass - fibrosis or pulmonary congestion or chest wall restriction. o Loss of surfactant • Higher respiratory rates increasing flow rates • Decreased airway radius due to: Lower lung volumes; Bronchoconstriction; • Increased air density (eg SCUBA diving) • Increased air viscosity	Must understand both to pass. Prompt if necessary. Must understand **both major points** Must give at least **two examples** to pass.

Question 3 LOA: 1	3.1 What are the essential features of the loop of Henle countercurrent multiplier?	High permeability of the thin descending limb to water (via aquaporin-1) and active transport of Na^+ and Cl^- out of the thick ascending limb which is not permeable to water. A system in which Na K 2Cl are actively transported, and the inflow runs parallel to, counter to, and in close proximity to the outflow for some distance	Either version
	3.2What is the role of urea in the countercurrent mechanism?	Contributes to the **osmotic gradient** in the medullary pyramids	**Osmotic gradient**
	3.3 How does urea reach the interstitium?	Transported by urea transporters, by **facilitated diffusion** Amount of urea depends on the amount filtered which is influenced by dietary protein	**Facilitated diffusion**
Question 4 LOA: 1	4.1 Describe the body's response to cold?	shivering, hunger, ↑voluntary activity, ↑NA, A, ↓ heat loss, curling up, behaviour change, cutaneous vasoconstriction, horripilation	Give 4
	4.2 Outline the pathogenesis of fever.	Toxins from infective agents act on monocytes, macrophages and Kupffer cells to produce cytokines which act as endogenous pyrogens (EPs), *also IL-1β, IL-6, β-IFN, γ-IFN, TNF act on the OVLT, which in turn activates pre-optic hypothalamus through local release of PGs.*	EPs indirect action on hypothalamus to reset
Question 5 LOA: 1	What is the sequence of events in skeletal muscle excitation contraction coupling?	**Discharge of motor neuron.** **Release of transmitter (acetylcholine) at motor end-plate.** **Binding of acetylcholine to nicotinic acetylcholine receptors.** Increased Na+ and K+ conductance in end-plate membrane. **Generation of end-plate potential.** **Generation of action potential in muscle fibers.** Inward spread of depolarization along T tubules. **Release of Ca2+ from terminal cisterns of sarcoplasmic reticulum and diffusion to thick and thin filaments.** **Binding of Ca2+ to troponin C, uncovering myosin-binding sites on actin.** ATP dependent **Formation of cross-linkages between actin and myosin and sliding of thin on thick filaments, producing movement.**	Need bold to pass

ACEM PRIMARY 2012/1 Physiology VIVA March 30 Friday Morning Session 3 ***Candidate Number……..*** **AGREED MARK……**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: LOA: 1	1.1 Describe the factors affecting Cardiac Output	**CO=SVxHR** SV related to contractility, preload and afterload, HR controlled by intrinsic rate, autonomic, exogenous factors, heat, thyroid	Bold to pass + 2 mechanisms from each SV and HR
	1.2 What are the physiological responses to moderate blood loss?	↓venous return, stimulation of **baroreceptors**, inc catecholmine release, ↓ renal blood flow – activation **of renin angiotensin system** fluid shifts, hepatic synthesis of proteins, inc RBC production	Bold to pass
Question 2 LOA: 1	2.1 What are the effects of exercise on the respiratory system? Prompt(s): "What are the effects on: gas exchange; OR ventilation; OR pulmonary blood flow."	• **Gas exchange**: o ↑Respiratory uptake and consumption of O_2 (VCO_2) and production and excretion of CO_2 (VCO_2) - increases by 10-20 times; o ↑Lung diffusing capacity due to ↑diffusing capacity of the membrane and the pulmonary blood volume; o ↓**Ventilation**–perfusion inequality; • Ventilation: o ↑Respiratory rate; o ↓Functional residual capacity (FRC); o ↑Tidal volume (TV); o ↑Minute ventilation. • **Pulmonary blood flow**: o Distension and recruitment of pulmonary vessels increases total cross-sectional area of the pulmonary vasculature; o ↑Total pulmonary blood volume; o ↑Cardiac output and pulmonary blood flow; o ↑Pulmonary vascular pressures; o ↓Pulmonary vascular resistance. • Other respiratory effects: o ↑Respiratory exchange ratio (R) from 0.8 to 1.0 due to carbohydrate metabolism and may exceed 1.0 due to anaerobic glycolysis; o The Hb-O_2 dissociation curve shifts to the right in the tissues and back to the left in the lungs; o Additional capillaries open in peripheral tissues;	One effect from each **bolded** section and at least six to pass.
	2.2 What changes occur in blood gases during exercise?	• Arterial blood gases are little affected by moderate exercise but at high workloads pH falls due to lactic acidosis, $PaCO_2$ often falls to compensate for the acidosis and PaO_2 rises; • Arteriovenous pH, PaO_2 and $PaCO_2$ differences increase.	Basic understanding of the effects on blood gases.

Question 3 LOA: 1	3.1 Describe the micturition reflex.	**Spinal reflex**, **voluntary facilitation/inhibition** from the higher centres. Micturition centre in the brain stem. Bladder innervation - **sympathetic** L1,2,3; **parasympathetic** S2,3,4; **somatic** S2,3,4.  **FIGURE 38–20** **Innervation of the bladder.** Dashed lines indicate sensory nerves. Parasympathetic innervation is shown at the left, sympathetic at the upper right, and somatic at the lower right. **FIGURE 38–21** **Cystometrogram in a normal human.** The numerals identify the three components of the curve described in the text. The dashed line indicates the pressure–volume relations that would have been found had micturition not occurred and produced component II. (Modified and reproduced with permission from Tanagho EA, McAninch JW: Smith's General Urology, 15th ed. McGraw-Hill, 2000.) **Bladder muscle smooth** and plastic (explanation) **Initial urge at 150mls, fullness 400 mls.** **Detrusor muscle contracts.** **Perineal muscles/external urethral sphincter relax.** In females aided by gravity; in males contraction of bulbocavernous muscle	Need bold to pass – Innervation , sympathetic – inhibitory, parasympathetic- excitatory. Bladder distention, excitation of the mechanoreceptors, afferent projection to the brain stem and efferents via sympathetic, parasympathetic and somatic nerves. cystogram for additional marks Plastic – tension initially produced by filling (distension) is not maintained. P = 2T/R as T increases so does R, i.e. filling and distension therefore P remains constant
Question 4 LOA: 1	4.1 What factors stimulate glucagon release? 4.2 What are the physiological effects of glucagon?	**Hypoglycaemia**; increased sympathetic drive to pancreas; vagal stimulation; protein load; amino acids oral or IV infusion; exercise; stress; starvation; CCK; gastrin; cortisol; theophylline. **Gluconeogenesis**; glycogenolysis (not in muscle); lipolysis; ketogenesis; calorigenic – through hepatic deamination of amino acids; +ve inotropic effect in large doses; stimulates secretion of GH, insulin and pancreatic somatostatin.	Must give hypoglycaemia + 2 others Gluconeogenesis + 1 others
Question 5 LOA: 2	5.1 What is clonus? 5.2 Why does ankle clonus occur with upper motor neuron lesions? 5.3 What are the components of the stretch reflex?	**Regular, repetitive, rhythmic contractions of a muscle subjected to sudden, sustained stretch.** Loss of descending cortical input to inhibitory neurons called Renshaw cells, and therefore **loss of inhibition of antagonists**, resulting in repetitive sequential contractions of ankle flexors and extensors. **Sensor, afferent nerve, Monosynaptic at spinal level, efferent nerve, effector**	Bold to pass

ACEM PRIMARY 2012/2 Anatomy VIVA **Morning Session 1** ***Candidate Number.....*** **AGREED MARK.....**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: **Bone** Bones of the foot LOA: 2	Identify the bones of the tarsus and foot	**Talus** – body, neck, head, trochlea **Calcaneus** – tuberosity ,**Navicular** – tuberosity, **Cuboid** **Cuneiforms** – medial, intermediate & lateral **Metatarsals** - 1-5 **Phalanges** – prox, middle & distal	Name all bold
	What are the major dorsiflexors of the foot and where do the attach	Tib anterior – base 1st met, med cuneiform EHL – middle & distal ph EDL – distal ph F Tertius – base of 5th met	3 of 4
Question 2 **Xray** Elbow LOA: 1	Identify the bony features on this XRay	Medial/lateral epicondyles, capitellum, olecranon, radius-head/ neck, olecranon fossa, coronoid fossa, trochlea, proximal radio-ulnar joint, coronoid process of ulnar	6 to pass
	What factors determines the stability of the elbow joint?	**Bony factors-shape of trochlea /olecrannon fossa** Joint capsule-fibrous joint capsule weak **Ligaments-** ***radial collateral ligament***- lateral epicondyle and blends with the ***annular ligament*** of the radius (holds the radial head in the radial notch of the ulnar). Medial ***ulnar collateral ligament*** *(3 bands)* from medial epicondyle to the coronoid process and olecrannon of the ulnar Muscles- biceps, brachialis, (BR) , triceps RCL and UCL and annular ligament	3 of 4 bolded Prompt – what are the ligaments of the elbow jt

Question 3 **Photo** Extracranial facial nerve	Name the branches of the facial nerve and indicate their position in the photo What is its main function? What else does it supply?	Forms parotid plexus in gland with 5 branches **5 Buccal 15 Marginal mandibular, 25 Temporal, 27 Zygomatic** , cervical (not seen) **Motor nerve to muscles of expression** + digastric, stylohyhoid & stapedius taste anterior 2/3 tongue, skin close to external acoustic meatus, lacrimal gland, sublingual and submandibular glands	4 of 5 Prompt if necessary by Bold to pass Must note one
Question 4 **Model** Posterior compartment of leg LOA: 1	Identify the muscles of the posterior compartment of the leg? What is the nerve supply of these muscles? Using the model describe the course of this nerve in the leg?	Superfical – gastroc, soleus ,plantaris Deep: Popliteus, FHL , FDL , Tibialis post b) Tibial branch of **sciatic nerve** c) Formed at apex of popliteal fossa by **bifurcation of sciatic** Runs **vertically in pop fossa** with pop artery, passing between heads of gastroc and deep to tendinous arch of soleus Runs inferiorly on tib posterior with post tib vessels Divides into medial and lateral plantar nerves under flexor retinaculum	1 superficial, 3 deep Must name nerve Comes from sciatica and terminates as plantar nn
Question 5: **Discussion** Superior mediastinum LOA: 2	Describe the vascular structures which lie in the superior mediastinum Can you name the other structures which lie in the sup mediastinum	Aorta Asc – technically in inf mediastinum. A rch – extends superiorly, posteriorly and left before heading inferiorly. Branches – BC trunk (which becomes RSC and RCC), L CC L SC Veins – L & R IJV and SCV each unite to form L&R BC vein. LBCV passes anterior to Ao arch/branches to meet RBCV and form SVC Thymus, Vagus nerves (R give R rec laryngeal looping around RSC art, phrenic nerves, trachea, oesophagus	**Name all 3 branches of Ao arch & formation of BCVs** Prompt (may well be needed!) Describe the arch aorta Describe the great veins in the upper chest Would you like to draw this? Bonus pts

ACEM PRIMARY 2012/2 Anatomy VIVA **Afternoon Session 2** ***Candidate Number.....*** **AGREED MARK.....**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: **Bones** Scapular LOA: 1	Identify the anatomical landmarks of this bone	**Glenoid, spine, supra/infra spinus fossae, subscap fossa, coracoid and acromion processes** Suprascap notch, supra/infraglenoid tubercles, inf angle, med/lat border	Correct side and 6 of 7 of those in bold And 2 of the rest
	Demonstrate the muscular attachments on the posterior surface	Superior - Lev scap, inf belly o-h, biceps/coracobra Medial – Lev scap, rh min/maj, lat dorsi Lateral – long hd triceps, teres min/maj, lat dors **Supraspinatus** and **Infraspinatus** Spine - **Trapezius** (sup) and **Deltoid** (inf)	3 of 4 in bold plus 3 others
	What are the muscles involved in ABduction of the shoulder joint	Intitiated by supraspinatus, then **deltoid**	Must know deltoid
Question 2 **XRAY** Knee, extra capsular and intra-articular ligs LOA: 1	Indentify bony structures are shown on this x-ray?	**Femur** – condyles (medial & lateral), epicondyles (medial & lateral), Adductor tubercle **Tibia** – condyles (medial & lateral), tibial plateau, intercondylar eminence with intercondylar tubercles (medial & lateral) **Fibular** – head with apex, neck **Patella**	All bold plus 6 others
	What factors stabilise the knee joint	1. Strength & actions of surrounding **muscles and their tendons** – most impt **quadriceps femoris**, esp inferior vastus medialis & lateralis 2. **Ligaments** connecting femur & tibia - **Cruciates & collaterals** (Most stable position = erect extended knee – articular surfaces most congruent, Cruciates and collaterals taut and jt splinted by many tendons)	Must ID muscle groups and all 4 main ligs
	Describe the attachments of the cruciate ligs	ACL attaches ant and runs up & laterally, PCL opposite	Must identify A/P tib attachments

Question 3 **Photo** Median nerve in hand LOA: 1	What structures can you identify in this image? What are the attachments of the flexor retinaculum and what does the carpal tunnel contain? Describe the median nerve supply in the hand	**Median nerve**, FCR, BR, FCU, FDS, FDP, ulnar nerve, ulnar artery, lumbricals, thenar muscles-APL, APB, FPB Boundaries: roof- flexor retinaculum, floor-scaphoid & trapezoid laterally, pisiform and hook hamate medially Contents: Median nerve, FDP, FDS, FPL, FCR Sensory- palmar-thumb and index and middle fingers, dorsal surface- distal aspect thumb, index, middle and half ring fingers Motor- LOAF muscles (lat 2 lumbricals, OP, APB, ,FPB)	Median nerve plus 4 muscles to pass 3 of 4 flex ret attachments and 4 of 5 contents Both motor & sens to pass
Question 4 **Model** Lower limb - buttock region LOA: 1	The gluteus maximus has been removed. Please identify the main structures seen here Can you demonstrate the course of the sciatic nerve and name the muscles that it supplies in the thigh	**Sciatic nerve** **Piriformis** Gamelli – sup/inf Obt internus Gluteus medius Ischial tuberosity/greater troch Quadratus femorus, obt ext **Muscles of the posterior compartment of the thigh** - Common fibular part – supplies short head biceps femoris - Tibial part – supplies the rest, namely; Long head biceps femoris Semitendinosus Semimembranosus Hamstring portion of adductor magnus	2 bold and 2 others to pass 2 of 4 muscles and nerve is deep to hamstrings and bifurcates to named terminal branches
Question 5 **Discussion** Posterior abdomen, retroperitoneal compartment LOA: 2	Describe the course and branches of the abdominal aorta What is the relationship of the IVC to the aorta	aortic hiatus of **diaphragm at T12** Ends at **bifurcation to common iliac aa at L4** Branches: - **Coeliac** (T12), **SMA** (L1), **IMA** (L3); Suprarenal (L1), **renal** (L1), gonadal (L2); Subcostal (L2), Inferior phrenic (T12), Lumbar (L1-L4) (2 of minor branches) **IVC:** lies posterolateral and to the R. Leaves abdomen through **caval opening of diaphragm** at T8 Drains from lower limbs and non-portal blood **Tributaries correspond to paired vessels of Ao**	3of bold, 1 of non-bold Behind and to the R

ACEM PRIMARY 2012/2 Anatomy VIVA Morning Session 3 *Candidate Number.....* AGREED MARK.....

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: **Bone** Clavicle LOA: 2	a) Identify and describe the features of this bone *Prompt- what other bones does it articulate with?*	Name and side bone Medially- **sternal end,** articulates with manubrium Laterally- **articulates with acromion** Inferiorly-conoid tubercle and trapezoid line, for coracoclavicular ligament; subclavian groove; Impression for the costoclavicular ligament	All bold and 3 other features
	b) What structures stabilise the acromioclavicular joint *Prompt – what ligaments?*	Ligaments of the joint **Acromioclavicular ligament** **Corocacoclavicular ligament** – conoid and trapezoid components	Name both ligaments
Question 2 **Xray** Ankle LOA: 1	1. Identify the bony features on this xray	**Fibula/lateral malleolus** **Tibia/medial malleolus** **Talus** head, neck, body **Navicular, Calcaneus,** metatarsals	Bold to pass
	2. Please describe the ligamentous attachments of the ankle joint	**3 lateral ligaments** – anterior talofibular (weak) - post talofibular (runs med, strong) - calcaneofibular (round cord, passes post/inf from tip of fibula)	Bold plus 2 out of 3 lateral ligaments named
		1 Medial ligament – deltoid ligament – medial malleolar attachment fans out to ant/post talus, calcaneus and navicular	Bold to pass
Question 3 **Photo** Femoral artery LOA: 1	Using this photograph describe the course and relationships of the femoral artery Prompt	Continuation of external iliac A. , enters femoral triangle **deep to midpoint of inguinal ligament** (midway between ASIS and pub tub) **lateral to femoral vein**, posterior/ deep to fascia lata, anterior **/ lies on (1 of 2)** iliopsoas and pectineus, **medial to femoral nerve.** Fem artery continues down thigh deep to Sartorius and **pass through adductor canal and becomes popliteal art at adductor hiatus**	All bold

	Describe the branches of the femoral artery Prompt: what branch supplies the head of femur	**Profunda femoris** ("**deep artery of thigh**"!) branches off post-lat in triangle to supply thigh, passes behind add longus. Gives med and lat cx fem arteries. Med cx fem supplies NOF 4 branches anterior part in fem triangle (superf epig, superf cx iliac, superf and deep external pudendal)	Profunda and 1 other.
Question 4 **Model** Extraocular muscles LOA: 1	Identify the muscles responsible for eye movement and describe their function What is the nerve supply to these muscles? What are the effects of an oculomotor nerve palsy?	**Supr (elev**, add), **medial, inferior (dep**, add), **lateral rectus** **Superior oblique (dep,** abd) and **Inf (elev,** abd) **oblique**. **Oculomotor (III) N** to all, except Abducent (VI) N (Lat Rectus) and Trochlear (IV) to Supr oblique. **Dep and Abd – dilated pupil, ptosis.**	All bold 3rd N and one other to pass
Question 5 **Discussion** Lungs LOA: 2 Page 199 Moore 6th	Can you describe the surface anatomy of the lungs and pleura? What are the anatomical structures to consider when inserting a lateral chest tube?	R Lung- Apices of L & R lung begin in **supraclavicular fossa** Lungs and visceral pleura run **parasternal to 6th costal** cartilage – then pass laterally to **MCL 6th rib, MAL 8th** rib, **SL at 10th** rib in contrast to parietal pleura which is at mid-clavicular line at 8th CC, 10th rib at mid-axillary line, 12th rib at scapular line Oblique fissure – spinous process T2 posteriorly – to 6th costal cartilage anteriorly Horizontal fissure R extends from oblique fissure at level of 4th rib & costal cartilage **Above the rib below** to avoid neurovascular bundle The level 5th or 6th Intercostal space to **above diaphragm** **Ant or Mid ax line** to avoid long tx nerve posteriorly	Prompt if necessary 2 of 5 bold

ACEM PRIMARY 2012/2 Pathology VIVA **Thurs AM (Session 1)** ***Candidate Number.....*** **AGREED MARK.....**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Q1 Hyperplasia LOA: 1	1. What is hyperplasia? 2. What are the causes of hyperplasia? 3. Give some examples of hyperplasia *Prompt: can you give me a physiological/pathological example?*	Hyperplasia is an **increase in the number of cells** in an organ/tissue, usually get increased mass of organ/tissue a. Hormonal effects – reversible with withdrawal of hormonal stimulation b. Tissue damage or resection - compensatory hyperplasia c. Growth factors - pathological hyperplasia d. Increased workload (muscle) - as for hypertrophy Physiological: female breast at puberty and during pregnancy, partial hepatectomy, Pathological: endometrium – hyperplasia, dysfunctional uterine bleeding; BPH; Papilloma virus	Bold to pass 2/4 required to pass 1 physiological and 1 pathological cause to pass
Q2 Reperfusion Injury LOA: 1	1. What is reperfusion injury? 2. What are the mechanisms of reperfusion injury?	It is when reperfused tissues sustain loss of cells in addition to the cells that are irreversibly damaged at the end of ischaemia. a. **Reactive O2 and N species** produced from incomplete reduction of the incoming O2 by damaged mitochondria in parenchymal and endothelial cells b. **Inflammation –** increased cytokine production and adhesion molecule expression by hypoxic cells recruits inflammatory cells (neutrophils) causing further injury c. **Activation of complement**. IgM Abs may deposit in ischaemic tissues - complement binds and activate – further injury and inflammation	Broad concept expressed Concept of 2 of 3 bolded
Q3 Heart failure LOA: 1	1. What are the major causes of heart failure? 2. What pathological processes can occur in the myocardium in heart failure? 3. What are the pathological changes in the liver caused by heart failure?	**Ischaemic heart disease, Valvular heart disease, Hypertension,** Cardiomyopathy, Fluid overload, Infarction, Ischaemia of myocardium Calcification, Hypertrophy of cardiac myocytes, interstitial fibrosis Nutmeg liver, Centrilobular necrosis (results from central hypoxia), Centrilobular fibrosis =cardiac sclerosis (due to long standing RHF. Cardiac cirrhosis in extreme cases.	2 Bold and one other3 to pass 2 to pass Congestion/oedema leading to fibrosis or necrosis

Thurs AM Q4 Acute meningitis LOA 2	1. What are the types of meningitis? *Prompt: What other type?* 2. What bacteria cause meningitis in different patient groups? 3. How do the CSF findings differ between bacterial and viral meningitis?	Infectious meningitis: **acute pyogenic, aseptic (inflammatory) viral, parasitic, chronic (TB)** chemical meningeal carcinomatosis Neonates: E. Coli; Gp B Strep Infants: HIB (less with immunisation) Strep Young adults: N. meningitidis Elderly: Strep pneumoniae; Listeria Immunosuppressed: Klebsiella; anaerobe; BACTERIAL / VIRAL Increased pressure / May be normal/slight inc Cloudy or purulent / Often clear **Increased white cells - neutrophils** / **Less increase white cells - lymphocytes** Raised protein / Only moderate increase Reduced glucose / Nearly always normal Bacteria on smear / (PCR)	**Bacterial, viral + 1 other** **3 bacterial causes including N. meningitidis in right age range** **White cell differences x2 + 1 other**
Q5 Cholecystitis LOA 2	1. Describe the pathogenesis of acute cholecystitis Prompt: what is the pathogenesis of acute calculous cholecystitis? *Prompt: What are the risk factors for acalculous cholecystitis?* 2. What is the role of bacterial infection in acute cholecystitis?	**1.** Disruption of protective mucous layer, bile salt detergent action -> irritation and inflammation (**occurs in absence of bacterial infection initially**) **90% due to gallstone obstruction** of neck or cystic duct; 10% acalculus cholecystitis Acalculous - Occurs in severely ill people, thought to be due to ischaemia (risk factors septic shock, immunosuppression, diabetes) burns, trauma 2. Often late	Concept and gallstones and acalculous to pass. Recognition of immunosuppression or critical illness to pass. Initial chemical irritation then bacterial superinfection.

ACEM PRIMARY 2012/2 Pathology VIVA **Thurs PM (Session 2)** ***Candidate Number.....*** **AGREED MARK.....**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Q1 Metaplasia LOA 1	1. What is metaplasia? 2. Describe some examples 3. What are the possible outcomes of metaplasia? 4.	1. Replacement of one normal cell type with another normal cell type; can be adaptive or pathological. 2. Columnar to squamous (respiratory-chronic irritation eg smoking; excretory ducts due to stones eg salivary, bile). Squamous to columnar (Barrett oesophagus). Connective tissue (myositis ossificans). 3. Malignant transformation, reversibility/resolution, ongoing	Correct definition and 2 examples to pass 2 to pass
Q2 Mechanisms of Cellular Injury LOA 1	1.? What happens inside cells when they are injured? *Prompt: mechanisms of cell injury* 2. What is a free radical? 3. What are the pathologic effects of free radicals? *Prompt: At a cellular level.*	1. ATP depletion, mitochondrial damage, calcium influx, accumulation of free radicals or ROS, membrane damage, DNA/protein damage 2. Chemical species that have a **single unpaired electron** in outer orbit eg reactive oxygen species: superoxide, hydrogen peroxide, hydroxyl, ONOO- peroxynitrite 3. Overall can cause **necrosis** or apoptosis or can stimulate production of degrading enzymes Directly can cause: **Lipid peroxidation** (plasma or organelle membrane damage) **Oxidation of proteins** (affect protein structure eg enzymes) **DNA lesions** (breaks in DNA or cross-linkages)	3/6 Principal & one example to pass Necrosis & 1/3 bolded effects
Q3 Staph infections LOA: 1	1. Describe the virulence factors of Staph aureus. What infections do the different species of Staphylococci cause? 2. *Prompt: Name the Staphylococcal species*	a. **Surface proteins involved in adherence** – expresses receptors for fibrinogen (and others) to bind to host endothelial cells. b. **Secreted enzymes that degrade proteins** (promoting invasion and destruction) e.g. lipase degrades skin lipids associated with ability to produce abscesses **c. Secreted toxins that damage host cells** alpha toxin – membrane depolarisation/damage; beta toxin – sphingomyelinase; Exfoliative A & B toxin; Superantigens – TSS and food poisoning **S. aureus** – skin, pneumonia, osteomyelitis etc **S. epidermidis** – opportunistic eg prosthetic valves **S. saprophyticus** – UTI in women	2/3 bolded sections including toxin 2 of the 3 bolded

Thurs PM Q4 Aortic dissection LOA: 2	1. What are the risk factors for aortic dissection? 2. Describe the pathogenesis of aortic dissection? 3. What are the complications of aortic dissection?	**Hypertension;** Connective tissue disease (Marfans, Ehlers-Danlos); Iatrogenic (eg coronary angiography); Pregnancy , Age Medial weakness due to underlying cause, medial hypertrophy of vasa vasorum, intimal tear, **blood flow dissects the media resulting in medial haematoma.** Cystic medial degeneration Depends on type. Both: rupture. Type A: dissects to aortic root involving coronary ostia (myocardial ischaemia/infarction), pericardial tamponade. Dissects into great vessels leading to cerebrovascular accident. Type B: dissects into renal, mesenteric, spinal and distal arterial tree causing ischaemia/infarction.	Bold and one other. At least four complications.
Q5 Thrombocytopaenia LOA: 1	1. What are the causes of thrombocytopaenia? 2. What is the pathogenesis of immune thrombocytopaenic purpura?	**Decreased production of platelets** - Generalised diseases of bone marrow [Aplastic anaemia (congenital / acquired); Marrow infiltration : leukaemia/cancer] - Selective impairment of platelet production [Drug induced (alcohol, thiazides, cytotoxics); Infections (measles, HIV)] - Ineffective megakaryopoieis [Megaloblastic anaemia, Myelodysplastic syndromes ,parox noct Hburia] **Decreased platelet survival** - Immunological destruction [Autoimmune (ITP, SLE); Iso immune (post transfusion, neonatal); Drugs (quinidine, heparin, sulfa); Infections (mono, HIV, CMV)] - Non immunological destruction [DIC, TTP, giant haemangioma, micro-angiopathic haemolytic anaemia; Sequestration] - Hypersplenism; Dilutional Triggers: Primary /Idiopathic ITP : acute / chronic Secondary : drugs ,HIV Chronic – more common – young adult women **Formation of antibodies against platelet** membrane glycoproteins (IIb-IIIa or Ib-IX); Antibodies evident 80% (plasma/platelet surface) Opsonised platelets susceptible to phagocytosis (mononuclear) Spleen probably major site of removal; 80% improve after splenectomy (site destruction + auto antibody synthesis) Acute – disease of childhood Viral illness – abrupt onset; Antiplatelet autoantibodies; Self-limiting, resolves usually within 6 months	2 groups in bold 2 examples from each Bold to pass

CEM PRIMARY 2012/2 Pathology VIVA Fri AM (Session 3) *Candidate Number.....* AGREED MARK.....

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Q1 Morphologic patterns and outcomes of acute inflammation LOA: 1	1. What are the different types of acute inflammation? *Prompt: What are the morphological patterns of acute inflammation?* 2. What are the outcomes of acute inflammation?	**1.a. Serous inflammation:** thin fluid from plasma or mesothelial lining cells e.g. burns, effusions (pericardial, pleural) **b. Fibrinous inflammation:** more severe injuries and greater vascular permeability allows larger molecules such as fibrin e.g. characteristic of inflammation in body cavities (pericardial sac,meninges,pleura) **c. Suppurative / purulent inflammation:** large amounts of pus / purulent exudates – neutrophils,necrotic cells,oedema fluid e.g. organism type (staph) ; site (appendicitis) **d. Ulcers:** local defect in surface of an organ/tissue **2.a. Complete resolution +/- scarring** **b. Abscess formation** (suppurative inflammation) **c. Fibrosis** (fibrinous inflammation) **d. Chronic inflammation**	2 with examples 2 of 4
Q2 Type 1 Hypersensitivity Reaction LOA: 1	1. What is a type I hypersensitivity reaction? 2. What is the immune mechanism that causes it? 3. What pathological effects do the substances released from mast cells have?	1. A **rapid** immunologic reaction due to **antigen and antibody(IgE)** combining. 2. **Previous Ag exposure** results in activation of T_h2 cells results in **IgE Ab production** by B cells. IgE binds to mast cells. **Repeat Ag exposure**, Ag-Ab bind and results in **mast cell degranulation. Vasoactive amines** (Histamine), and lipid mediators (Leukotrienes, PG) released. May have **late phase reaction** (Cytokines) 3. Vascular dilation/ oedema, SM contraction, mucus production	Bold required 3/6 bold with concept 2 to pass

Fri AM Q3 CVA LOA: 1	1 What are the causes of focal cerebral infarction? 2. What are the sources of cerebral thromboemboli? (Prompt: What happens in cerebral embolism?)	**1. Arterial thrombosis, Cerebral embolism** Lacunar- arteriosclerosis of the vessels in the lenticular nucleus, thalamus, internal capsule, deep white matter, caudate nucleus, and pons Arteritis – giant cell (temporal arteritis), PAN, SLE, infectious (CMV, aspergillosis, TB, Syphilis) Arterial dissection Venous infarction – hanging, - venous sinus thrombosis 2.Source (s) - usually from heart (**LAA, mural thrombus, valvular vegetations**) - **plaques from carotid bifurcation**; - **paradoxical emboli** in patent foramen ovale Precipitant (not specifically in text) – Afib / cardioversion Consequence – most commonly **lodges in MCA** , often at branch points, **causes ischaemia** due to poor collateral flow	Need bold (arterial thrombosis, embolism) and one other (underlined) to pass. Need at least 1 cardiac and 2 sources in total to pass.
Q4 Cholelithiasis LOA: 2	1. What are the risk factors for the development of cholesterol stones? 2. Describe the pathogenesis of cholesterol stone formation.	**1.Age, Gender** – 25% in the > 80 yo, women > men; **Environmental factors** – OC, pregnancy – increase expression of hepatic lipoprotein receptors and stimulates hepatic HMG-CoA reductase – enhancing cholesterol uptake and synthesis. Obesity, rapid weight loss.; **Acquired disorders** – gallbladder stasis – neurogenic or hormonal; **Hereditary factors** – e.g. genetic factors encoding for hepatocyte proteins that transport biliary lipids - ATP-binding cassette (ABC) transporters. 2.Requires the following simultaneous conditions: **Bile supersaturated with cholesterol;** Hypomotility of gall bladder; **Cholesterol crystal nucleation** – accelerated; Hypersecretion of mucus in the gall bladder traps crystals – **aggregation into stones**	3 of 5 bolded. Bolded and displays understanding of concept
Q5 Acute Kidney Injury LOA: 2	1. What causes acute kidney injury? 2. How does urine output often change with time following acute kidney injury?	1. Commonest cause of acute renal failure. **Ischaemia**: hypotension, vasoconstriction, capsular tamponade. **Direct toxic injury**: (aspirin), aminoglycosides, contrast, myoglobin, crystals, protein. Acute tubulointerstitial nephritis (infections, heavy metals, hypersensitivity reaction to drugs). Post renal urinary **obstruction**. DIC, sepsis. 2.Highly variable. a. Initiation phase: decreased urine output with elevation of urea (< 36 hours) b. Maintenance phase: sustained decreased output (40 – 400 ml/day), salt and water overload, uraemia, hyperkalaemia, metabolic acidosis. c. Recovery phase: increased output and hypokalaemia. Increased vulnerability to infection. May last for months.	One example for each bolded and then at least one other cause. Know initial decrease followed by diuresis

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Q1 Hypertrophy LOA: 1	1. What is hypertrophy? 2. What are the types of hypertrophy? 3. Describe examples of each type hypertrophy? *Prompt: Can you give examples of physiologic and pathologic hypertrophy?*	**Increased size of a tissue due to increased cell size);** Due to synthesis of structural components. May be **physiological or pathological** depending upon increased functional demand or specific hormonal stimulation. Cell hypertrophy can occur in dividing or non-dividing cells Physiological: skeletal muscles with exercise, uterus in pregnancy (hormonal), breasts in lactation. Pathological: prostate in BPH, heart in chronic hypertension.	Bold One example of each
Q2 Mediators of acute inflammation LOA: 1	1. What are the chemical mediators of acute inflammation? 2. What do they do? -	Histamine: Vasodilation, increased vasc permeability, endothelial activation Serotonin: Vasodilation, increased vasc permeability Prostaglandins: Vasodilation, pain, fever Leukotrienes: Increased vasc permeability, chemotaxis, leukocyte adhesion and activation Platelet-activating factor: Vasodilation, increased vasc permeability, leukocyte adhesion, chemotaxis, degranulation, oxidative burst Reactive oxygen species: Killing of microbes, tissue damage Nitric oxide: Vascular smooth muscle relaxation, killing of microbes Cytokines (TNF, IL-1): Local endothelial activation (expression of adhesion molecules), fever/pain/anorexia/hypotension, decr vascular resistance (shock) Chemokines: Chemotaxis, leukocyte activation Complement (C5a, C3a, C4a): Leukocyte chemotaxis and activation, vasodilation (mast cell stim Kinins: Incr vasc permeability, smth muscle contraction, vasodilation, pain Proteases activated during coagulation: Endothelial activation, leukocyte recruitment	4 to pass 4 general correct actions
Q3 Strep infections LOA: 1	1. What types of infections do Streptococcal bacteria cause? *Prompt: Give examples of the different strep subtypes and the infections they cause?*	1. Acute **suppurative: skin, throat, lungs and heart valves.** Group A S.pyogenes (throat, skin), Group B S.agalactiae (female genital, neonate sepsis), α Haemolytic, S.pneumoniae (CAP), meningitis S.viridans (mouth, SABE), S.mutans (teeth)	Pus >= 2 to pass

Fri PM Q3 Strep (con'td)	2. What post infectious syndromes do streptococci cause?	2. GN, rheumatic fever, erythema nodosum	1 to pass
Q4 Hepatitis C LOA: 2	1. What type of virus causes Hepatitis C? 2. What are the risk factors for acquiring Hepatitis C? 3. What is the natural course of Hepatitis C?	1. **Flaviviridae** family **RNA** virus 2. **IVDU** 54%; Multiple sex partners 36%; Recent surgery 16%; Needle stick 10%; Multiple contacts with HCV infected person10%; Health care workers 1.5% Unknown 32%; Children (perinatal) 6% (cf HBV 20%) 3. Incubation 2 – 26 weeks (mean 6 – 12); **Asymptomatic in 85%** HCV RNA detectable in 1 – 3 weeks Anti HCV Ab 50 – 70% while symptomatic Usually a mild disease **Persistent infection -> chronic hepatitis 80 – 85%** Cirrhosis 20 – 30% (5 – 20 years) Fulminant hepatitis rare	One of bold IVDU and 2 others Bolded
Q5 Consequences of Atherosclerotic Disease LOA: 2	1. Describe the differences between stable and vulnerable atherosclerotic plaque. 2. What pathological changes can occur in these plaques? 3. What are the consequences of these changes?	1. Stable = dense collagenous and **thickened fibrous caps** with **minimal inflammation** and **small underlying atheromatous** core. Vulnerable = **thin fibrous cap, large lipid core and increased inflammation** – prone to rupture. 2. Categories for plaque change: a. **Rupture/fissuring** – exposing highly thrombogenic plaque components – inducing thrombosis. b. **Erosion/ulceration** – exposing thrombogenic subendothelial basement membrane – inducing thrombosis c. **Haemorrhage into atheroma** – expanding volume 3. Consequences a. Small vessels can **occlude – compromising distal perfusion** b. **Ruptured plaque can embolise** atherosclerotic debris and occlude distal circulation or can cause acute **thrombosis**. c. **Destruction of vessel wall can cause aneurysm** formation with secondary **rupture** and/or thrombosis.	1. 2 Bolded parts from each 2. 2 of 3 bold 3. 2 of 3 concepts

ACEM PRIMARY 2012/2PharmacologyVIVA **Morning Session 1** ***Candidate Number.....*** **AGREED MARK.....**

TOPIC	QUESTIONS	KNOWLEDGE	NOTES
Question 1 Clearance-renal and hepatic	What is drug clearance?	Clearance predicts the rate of elimination in relation to drug concentration. **CL=rate of elimination/concentration**	**Bold**
LOA 1	Which organs are involved in drug clearance?	2 main organs are **kidney and liver**, others are blood, muscle, lung. CL systemic= CL liver + CL kidney + CL other	**Bold**
	What factors affect renal clearance?	**Renal function, renal blood flow**, plasma protein binding, ionization	Bold
	Please name drugs that are predominantly cleared by the kidneys?	ampicillin, **gentamicin**, vancomicin, digoxin, enalapril, metformin, lithium	At least bold plus 2 others-prompt: Any drugs that need dose changes in patients with poor renal function?
Question 2 Oral hypoglycaemics	Describe the pharmacokinetics of metformin?	Well absorbed, not protein bound, not metabolised, elimination t1/2: 1.5-3 hours, **excreted by kidney** as unchanged compound	Bold
LOA: 1	What are the side effects of metformin?	Gastrointestinal most common 20%, decreased absorption Vit B12, **lactic acidosis** esp with renal disease, ETOH, chronic cardiopulmonary disease	Bold
	With regard to sulphonylureas, what is	**Increase insulin release from the pancreas** bind	Patients more prone to hypo

	the mechanism of action of glipizide? (prompt: it's a sulphonylurea)	to receptor associated with ATP sensitive K channel, inhibits efflux of K ions, results in depolarization and opens ca channel, influx of Ca causes release of preformed insulin Reduction of serum glucagon levels Closure of potassium channels in extrapancreatic tissues	than with biguanides eg metformin
Question 3 Non Depolarising Muscle Relaxants LOA: 1	What's the mechanism of action of Rocuronium? (Prompt: receptor level) Describe the pharmacokinetics of rocuronium. *Prompt: Describe rocuronium's distribution and elimination.*	**Non-depolarising NM blocker**. In low doses it predominantly acts as a **competitive inhibitor of Acetylcholine** at nicotinic receptors. In larger doses it can enter the pore of the ion channel -> greater NM blockade. It can also block prejunctional sodium channels-> interference with the mobilisation of AChl at nerve endings. Undergoes **rapid distribution.** Highly ionized - so small Vd (80-140ml/kg). Undergoes hepatic metabolism (75-90%) and renal excretion. Duration of action is 20-35mins.	Non-depolarising NM blocker. Initially acts as competitive inhibitor for Ach at nicotinic receptors Rapid distribution. **Short T1/2.**
Question 4 Calcium	Can you give me an example of a preparation of calcium that is taken orally?	**Calcium Carbonate** or Ca -acetate, citrate, glubionate, gluconate, lactate or phosphate	Need to name 1

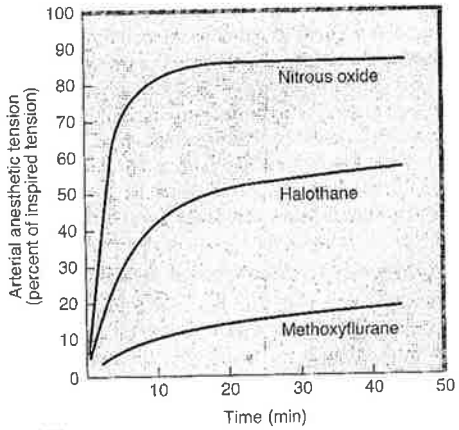
LOA: 1	What are the possible uses of oral calcium preparations?	i) Treatment of **hypocalcaemia** (eg. in patients with hypoparathyroidism, vit D deficiency, chronic renal disease or malabsorption). ii) As an antacid	hypocalcaemia.
	What are the potential adverse effects of giving calcium intravenously?	Irritation of the veins. Cardiac arrhythmias with rapid administration. Hypercalcaemia.	phlebitis
Question 5 Anti-influenza agents LOA: 2	List some anti-influenza agents	Zanamivir, Oseltamivir, Amantadine, Rimantadine	1 to pass
	What is the mechanism of action of zanamivir (relenza) and oseltamivir (tamiflu)?	Neuraminidase (a glycoprotein) inhibitors: disrupt viral replication and release Active against both influenza A and B;	Some concept
	What are the indications for their use?	Approved for treatment of uncomplicated influenza; 5 day course of therapy within 36 – 48 hrs of symptom onset shortens severity and duration of illness; may decrease incidence of respiratory complications	1 to pass
	What is the relevance of these agents to emergency medicine practice? PROMPT: what about during the recent	May be of use to **higher risk groups** eg indigenous, pregnant women, older people and immunocompromised, however primary prevention by **vaccination is preferred**. Used	One of bold

	flu pandemic?	preferably at **early phase of pandemic** to limit spread and numbers infected, and limit severity of disease in those infected.	

ACEM PRIMARY 2012/2Pharmacology VIVA Afternoon Session 2 ***Candidate Number.....*** **AGREED MARK.....**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Volume of distribution LOA: 1	Define the "volume of distribution" of a drug.	Defined as the volume in which the amount of drug in the body would need to be uniformly distributed to produce the observed concentration in blood, plasma or water. Vd = Amt drug in body/C	Pass: either definition or formula
	How is it possible for a drug to have a VD of 2500L in an adult?	**Higher concentrations in extra vascular tissues than in blood – e.g. lipid soluble** (not homogeneously distributed)	Pass: either not homogeneously distributed or extra vascular tissue higher conc
	Give an example of a drug with a: - high VD - low VD	High: Morphine, chloroquine, digoxin, clonidine, fluoexitine, tricyclics, β blockers, diazepam, Low/approximating ECF/TBW: aspirin, frusemide, antibiotics (gentamicin, amoxicillin, cephalexin), tolbutamide, phenytoin, valproic acid, lithium, warfarin, theophylline, indomethacin, sulphamethoxazole.	**One of each** **One of each**
	What is the importance of Vd in the overdose situation PROMPT – for example (drug name)?	**Drugs with large Vd (TCAs) cannot be dialyzed** whereas **drugs with a low Vd (ASA, lithium)** can.	**Bold**– use these to prompt; should be able to designate "high" or "low" VD to pass.
Question 2	Describe the central nervous effects of	1) central	**Bold to pass**

Morphine LOA: 1	Morphine Describe peripheral effects?	• **analgesia** • euphoria • **sedation** • **respiratory depression** • cough suppression • miosis • truncal rigidity • nausea / vomiting • temperature 2) peripheral: • **cardiovascular** • GI- **constipation** • Biliary • Renal • Uterus • Neuroendocrine • Pruritis • immune	candidate should be able To describe in detail of each one in bold
Question 3 Nitrous oxide	Explain the solubility characteristics of nitrous oxide	Nitrous oxide possesses **low solubility** in the blood, reaches **high arterial tension rapidly, Rapid equilibrium in the brain** and **fast onset of**	**Bolded concept to pass**

LOA: 1	Draw the arterial anaesthetic tension vs time for nitrous oxide vs halothane or Methoxyflurane	**action (rapid onset-rapid recovery)**  	A curve
Question 4 Warfarin Interactions LOA: 1	Describe the mechanisms by which drugs interact with Warfarin. *Prompts* *Please describe pharmacokinetic interactions* *Please describe pharmacodynamic interactions* Give some examples of drugs that increase the INR.	**PK - Enz inhibition** (majority), **Enz induction**, altered, plasma protein binding, altered abs **PD** – Synergism (**impaired haemostasis)** Competitive antagonism (**clotting factor synthesis/concentration**) **↑ INR**: aspirin, heparin, corticosteroids metronidazole, fluconazole, trimethoprim-	Must get one example of PK and PD Must give at least 1 example of each

	Give some examples of drugs that decrease the INR.	sulfamethoxazole, third generation cephalosporins, macrolides, amiodarone, SSRIs, tramadol ↓ **INR:** Vit K, diuretics, barbiturates, phenytoin, carbamazepine, rifampicin, diclox, azathioprim	
Question 5 Serotonin Syndrome LOA: 2	Describe the mechanism by which Serotonin Syndrome occurs. *Prompt: What receptors are involved in SS?* How do drugs cause excessive stimulation of serotonin receptors? *Prompt: Can you give an example*	**Excessive stimulation of serotonin receptors in the CNS** due to overdose of single drug or concurrent use of several drugs. Predictable, not idiosyncratic. Inhibition of serotonin metabolism: meclobemide, amphetamines Prevention of serotonin reuptake in nerve terminals: fluoxetine, paroxetine, sertraline, venlafaxine, tramadol, TCA Serotonin release or increased intake of serotonin precursors: tryptophan, lithium,	Must get bold items Must identify at least 1 mechanisms with corresponding example

ACEM PRIMARY 2012/2Pharmacology VIVA **Morning Session 3** ***Candidate Number.....*** **AGREED MARK.....**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Signalling mechanisms LOA: 1	List the various molecular mechanisms of transmembrane signalling.	1. Lipid soluble ligand crosses membrane and binds to intracellular receptor. 2. Transmembrane receptor protein with ligand binding to extracellular domain regulating intracellular enzymatic activity 3. Transmembrane receptor protein that binds and stimulates protein tyrosine kinase 4. Ligand-gated transmembrane ion channels 5. Transmembrane receptor protein, G protein, intracellular second messenger	Describe 3 mechanisms to pass
	Describe the function of the system involving G proteins	Transmembrane signally system with 3 separate components. Extracellular ligand binds to specific **cell surface receptor**. This receptor then activates **G protein** located on cytoplasmic surface of membrane. Activated G protein changes activity of **effector element** (enzyme or ion channel) leading to a change in concentration of **second messenger**.	Bold concepts to pass

	Give an example of a drug that acts via this system.	B agonist: B adrenoreceptor, G_S protein, adenylcyclase, increased concentration cAMP. (other examples include glucagon, thyrotropin, histamine, serotonin, acetylcholine, opioids)	Correct example to pass. Extra points for describing components
Question 2 adenosine LOA: 1	What are the indications for use of Adenosine? How does it work? How do the specific pharmacokinetic properties of adenosine influence the method of administration?	**Conversion of paroxysmal SVT to sinus rhythm.** Activation of inward rectifier K+ currents and inhibition of calcium currents. Leads to marked hyperpolarisation and suppression of calcium-dependent APs. Effect is direct inhibition of AV nodal conduction and increase in AV node RP. This **interrupts re-entry pathway thru AV node**. **Very rapid metabolism** by adenosine deaminase in red cells and vessels walls = very short elimination t1/2 (<10s) and duration of action (~30s). Must be given by **rapid intravenous bolusing.** If initial dose ineffective then subsequent dose should be increased (no accumulation occurs).	Bold to pass AV node conduction interruption Bold to pass
Question 3 Phenothiazines LOA 2	What are the side effects of chlorpromazine? (If required: What are the mechanisms of these side effects?)	**Hypotension – alpha blockade** Parkinson's, akthesia, **dystonic reactions – D2** Lactation – D2 Sedation – antihistamine Neuroleptic malignant syndrome – dopamine Confusion, tachycardia – anti muscarinic	Two bolded side effect any dyskinesia sufficient) and one correct mechanism.

	How do the newer atypical anti psychotic agents differ from chlorpromazine?	Newer agents have less side effects.	
Question 4 Tisssue Plasminogen Activator LOA 1	Describe the mechanism of action of tissue plasminogen activator (tPA)?	Activates **plasminogen to form plasmin**, resulting in fibrin digestion. Preferentially activates plasminogen bound to fibrin by several hundred fold therefore is considered **clot specific.** Short half life therefore heparin is essential adjunct. Naturally occurring.	Bold
	What are the clinical uses of tPA? *Prompt: Are there are any other time-critical indications?*	**AMI, unstable PE, acute ischaemic stroke**, severe DVT, intra arterial peripheral limbs	First 3 to pass
	What are the complications of tPA?	**Haemorrhage.** Physiological hemostatic thrombi at site of vascular injury eg GIH, or systemic lytic state resulting from formation of plasmin, producing fibrinogenolysis and destruction of other coagulation factors esp V and VIII.	Must give more than one site.
Question 5 Seizure medications	Describe the pharmacokinetics of sodium valproate	Well absorbed PO, bioavailability >80% Food may delay abs for several hours. Peak plasma levels 2 hrs if empty stomach 90% **protein bound** (fraction bound reduces as	Highly protein bound and small Vd to pass

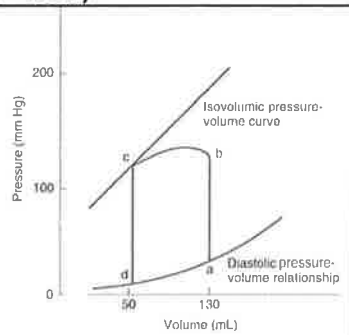
LOA: 1		total dose increases). Highly ionized and highly protein bound, therefore **Small VD**, essentially confined to extracellular water, approx. 0.15L/kg 95% hepatic metabolism, (some to active metabolites), 5% unchanged in urine Clearance is low and dose dependent, T1/2 is approx. 15/24 (9-18) and reduced if taking other antiepileptic drugs	
	Describe the toxic effects of sodium valproate.	Mild : Transient GI inc anorexia, nausea and vomiting. Rash, alopecia and increased appetite. Weight gain. Major Overdose: **CNS:** coma, cerebral oedema (potentially fatal) Bone marrow depression Metabolic effects: hyperNa, hypoCa, hyperammonaemia CVS, renal effects Severe and idiosyncratic **1.** Hepatotoxicity – rarely fatal, usually in under 2 yo, or multiple meds. Elevation of LFTs in 40%. May be reversible **2.** Thrombocytopaenia	CNS to pass

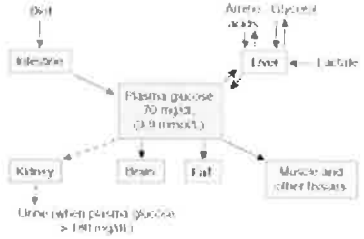
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: Factors affecting drug metabolism LOA: 1	What factors are responsible for differences in drug metabolism between individuals?	Genetic factors Diet and Environmental Age and Gender Drug-Drug Interactions Disease states Induces inhibitors Protein binding Renal clearance Pharmacodynamic interactions	Need 3 to pass
	Can you give an example of a drug-drug interaction?		Must give an example with correct mechanism
Question 2 Atropine LOA: 1	What is the mechanism of action of atropine?	A reversible **muscarinic antagonist** Binds to the muscarinic receptor, preventing the release of inositol trisphosphate (IP_3) and the inhibition of adenyl cyclase which are caused by the muscarinic agonists.	Bold to pass
	Describe the organ effects of atropine.	CNS: ⇓ tremor in Parkinson's Disease, delirium EYE: Mydriasis and cycloplegia CVS: Tachycardia LUNG: Bronchodilation and ⇓ secretions GIT: ⇓salivary secretion, ⇓ gastric secretion acid, pepsin and mucin, ⇓ gastric emptying, ⇑ Gut transit time GUT: relaxes ureteric and bladder wall smooth muscle and slows voiding; ⇓ sweating.	**3/6 organ effects** to pass

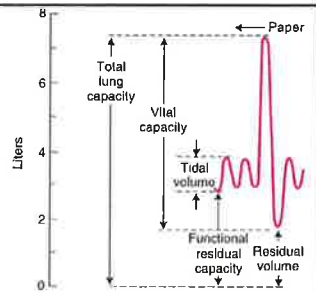
Question 3 Macrolides LOA: 2	Name some macrolide antibiotics? Describe the mechanism of action of macrolides? What organisms are macrolides effective against?	Erythromycin, roxithromycin, azithromycin, clarithromycin. **Inhibits bacterial protein synthesis** by binding to 50S ribosomal RNA, which blocks the aminoacyl translocation reaction and formation of initiation complexes (transpeptidation). May be inhibitory or bactericidal, particularly at higher concentrations. Gram + orgs: pneumococci, streptococci, staphylococci, corynebacteria Mycoplasma, Legionella, Chlamydia sp, listeria, some mycobacteria Gram – orgs: Neisseria sp, Bordatella pertussis, Treponema pallidum, Campylobacter sp, bartonella (Haemophillus less susceptible)	**Pass = 2** **Pass = bold** **Pass = 3**
Question 4 Induction agents LOA: 1	Give some examples of drugs used as anaesthetic induction agents? Describe the onset and recovery of propofol and ketamine? Describe the cardiovascular effects of propofol and ketamine?	Thiopentone, propofol, ketamine, fentanyl, midazolam, etomidate **Both have rapid, Ketamine has a slower recovery** and is often associated with emergence phenomena. Propofol–**marked decrease in BP during induction** via decreased peripheral arterial resistance and venodilation. Also greater direct negative inotropic effects cf other induction agents Ketamine – **produces does-related CV stimulation, increased HR, BP** and CO (by stimulating central symp nervous system +/- inhibiting NA reuptake at symp nerve terminals	**Pass = 2** **Bold to pass**

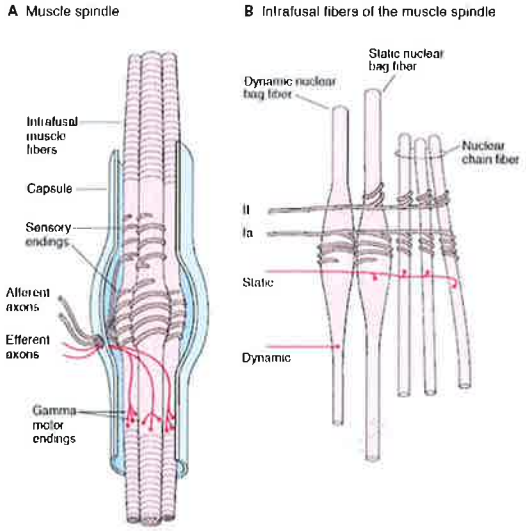
Question 5 Heparin LOA: 1	Describe the mechanism of action of heparin?	Binds to endothelial cell surfaces and plasma proteins and its activity depends on antithrombin Heparin binds to antithrombin, causes a conformational change in the inhibitor, exposing its active site for more rapid interaction with proteases. Heparin acts as a co factor for the antithrombin-proteases reaction Antithrombin inhibits proteases espec thrombin 2a, 9a, 10a by forming stable complexes with them and the presence of heparin accelerates this reaction 1000x The binding of AT III and unfractionated heparin ↑ degradation of both factor Xa and thrombin	**Binds to AT III**
	How is heparin reversed? *Prompt: is there a specific antidote?*	Stop the drug Administer antagonist **protamine** (100 units heparin-1mg protamine) which binds heparin to form a complex devoid of anticoag activity Excess protamine anticoag effect	**Bold**
	What are the potential adverse effects of heparin? Prompt: Are you aware of any less common but serious idiosyncratic effects?	**Bleeding** (elderly women, renal failure more prone) **TCP** (1-4%), rare pregnancy, lower rates in paediatrics. Mortality relates to thrombosis Allergy ↑ hair loss Reversible alopecia Accelerates the clearing of post prandial lipaemia by causing release of lipoprotein lipase from tissues Long term: osteoporosis, spontaneous fracture, mineralocorticoid deficiency	**Bold**

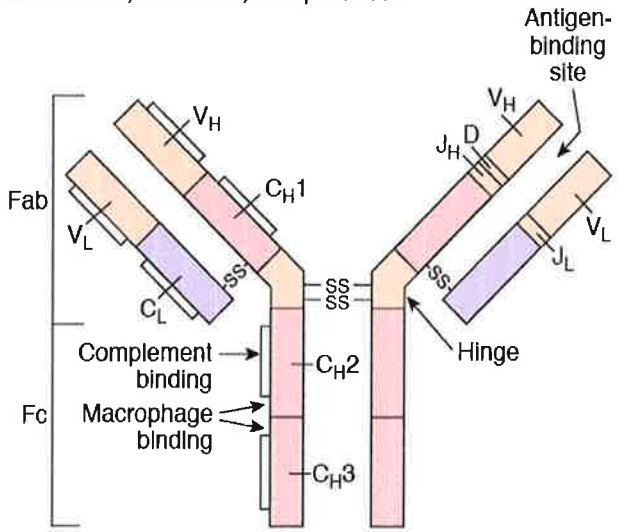
TOPIC	QUESTIONS	KNOWLEDGE (**essential in bold**)	NOTES
Question 1 LOA: 1	Please draw a pressure-volume loop for the left ventricle.	▪ a → b isovolumetric contraction ▪ b → c ventricular systole ▪ c → d isovolumetric relaxation ▪ d → a ventricular filling	**The candidate must be able to label the axes 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**

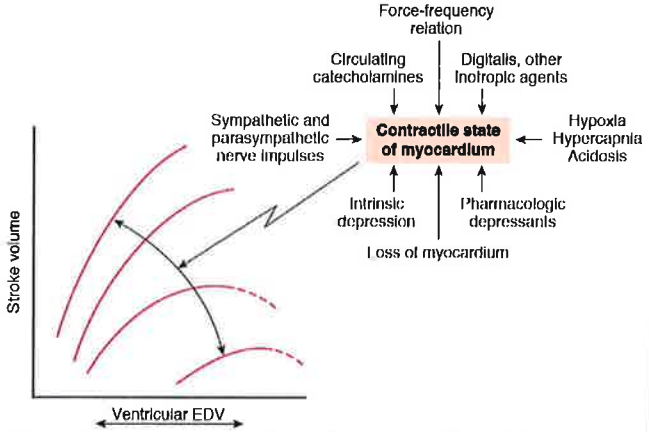
Question 5 Immunoglobulins LOA: 2	1. What are the types of immunoglobulins and what is the clinical significance of each?	1. Five Types A = Secretory D = Antigen recognition by B cells E = Anaphylaxis; release of histamine from basophils & mast cells G = Complement Activation; infections M = Complement 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

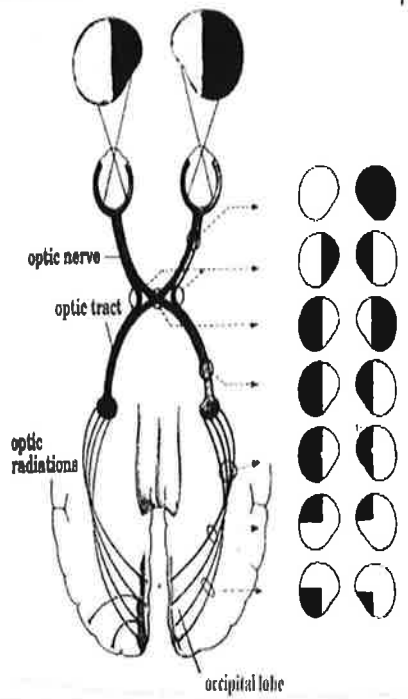
	BONUS
	3. What are the features of innate and acquired immunity?
		Innate immunity
		• triggered by cellular receptors (eg TLRs = "Toll-like Receptors")
		• bind molecular sequences common on MOs (not in eukaryotic cells)
		• activate defence mechanisms (interferons, phagocytosis, production of antibacterial 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.

ACEM PRIMARY 2013/1 Anatomy VIVA **Morning Session 1** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: Femur (Bony Landmarks)	Identify the important features of this bone	**Head**, fovea, **neck**, **greater / lesser trochanters**, trochanteric fossa, intertrochanteric crest (post) / line (ant), **shaft**, linea aspera, **med + lat femoral condyles**, intercondylar fossa, adductor tubercle, patellar surface	Bold + 2 to pass
	Describe the blood supply to the head of the femur	Via posterior retinacular arteries of **medial circumflex femoral artery**. Lateral circumflex femoral artery contributes a little via cruciate anastomosis. Possibly from foveal artery (branch of obturator artery)	Bold to pass
	What is the clinical significance of this	Intracapsular #s (subcapital, transcervical) may lead to **AVN** of head of femur, especially if displaced	Bold to pass
Question 2 CT Brain (Describe Structures)	Identify the anatomical features of the brain shown on this CT	**Lateral ventricles**-anterior and posterior horns Choroid plexus **Lobes- frontal, parietal, occipital and temporal** (3 of 4) + Gyri/ sulci **Falx cerebri** Thalamus, caudate nucleus, internal capsule(ant/post limb) Lentiform nucleus	Bold to pass
	Describe the circulation of CSF in the brain	**Formed in choroid plexus** in lat/3/4 ventricles. Drains from lat to 3rd via iv foramen then 4th via aqueduct then into SA space and brainstem cisterns. **Absorbed from SA space by arachnoid granulations**	Bolded info plus sequence of lat to third to 4th ventricles

Question 3 Photo Left Lung Root/Mediastinum (Describe Structures)	This is a longitudinal section through the hilum of the left lung. What structures can you identify? What are the branches of the aortic arch	**23,Heart** (LV), 26, pericardium, **3,32 aorta**, 18 L subclavian art, 4 L costocx trunk,12 L internal thoracic art,10 L common carotid,22 vagus n, **16 L pulm art**, 15 L main bronchus, 11,21 pulm vv,9 L brachioceph v,31 sympathetic trunk, 14 phrenic nerve.... **Brachiocephalic** trunk (dividing into RCC and RSC), **L common carotid** and **L subclavian**	**Bold** plus 4 others to pass **Bold** to pass
Question 4 Model Female Pelvis (Organs)	Identify the major anatomical structures in this model. Name the potential spaces where free fluid can accumulate in the pelvis and demonstrate their boundaries	Rectum, Uterus, Bladder, Sacrum, Pubic symphysis, Peritoneum, vagina, ovary, fallopian tube, Round ligament Broad ligaments, Int and Ext iliac vessels **Vesicouterine pouch** (anterior to bladder) & **Rectouterine pouch (of Douglas) between ant rectum and post uterus,** open above to peritoneum, close to cx and post fornix of vagina, inferior most ext of peritoneal cavity	8 to pass ID name and location of each in bold
Question 5 Discussion Movements of Thumb	Describe the origins and insertions of the muscles in the thenar eminence Demonstrate the movements produced by these muscles. What nerves innervate these muscles?	**APB, FPB, OP** (all originate flexor retinaculum and scaphoid/trap tubercles) APB inserts lat side base prox phal, OP inserts lat 1st MC FPB both heads insert base lat prox phal. OP opposes (mc to middle palm, rotates), ABD abducts the MCP jt, helps opposition, FPB flexes the MCP jt All recurrent br. **Med n,** except deep head FPB -deep Br ulnar nerve variable ++ or recurrent Br Median nerve	All bold and 1 correct origin and 1 correct insertion Demonstrate 2 correctly Must ID median n

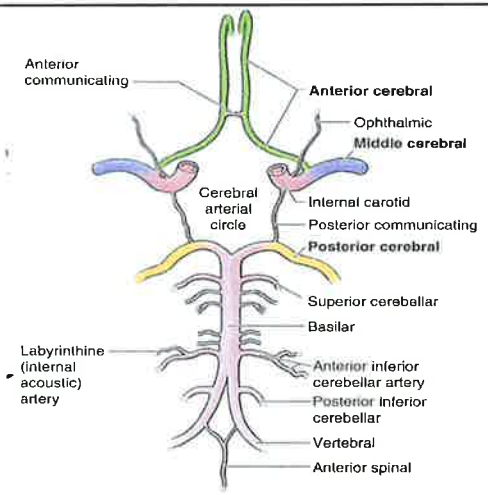
ACEM PRIMARY 2013/1 Anatomy VIVA **Afternoon Session 2** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: Bone Tibia (Bony Landmarks)	Identify this bone and its main features	**Side bone, identify anterior and interosseus borders** Tibial tuberosity, shaft, medial malleolus, tubercles of intercondylar eminence, medial and lateral condyles, facet for fibula, soleal line,	Bold plus 6 features
	Describe the articulations between this bone and the fibula	**Tibiofibular joint** (superiorly) and **tibiofibular syndesmosis** (inferiorly) TF joint- synovial joint b/w facet fibular head and on posterolaterally on lateral tibial condyle. Joint capsule strengthened by ant and posterior ligaments of the fibular head TF syndesmosis-compound fibrous joint, it is the fibrous union of tib & fib by the IOM and the ant and post tibiofibular ligaments	Both joints identified and 1 classified to pass
Question 2 CT Abdomen (Describe Structures)	Identify the structures visible on this CT	**Liver**/duodenum/IVC/**pancreas**/splenic vein/**kidneys/spleen/aorta**/coeliac trunk/crus of diaphragm/small bowel/ribs, vertebral body	**Bold** plus 2 others to pass Prompt for pancreas
	What are the branches of the abdominal aorta?	Single – **coeliac trunk, SMA, IMA** Paired –gonadal, renal, suprarenal, inferior phrenic, lumbar, subcostal. Terminating as common iliacs	**Bold** plus two paired branches to pass
Question 3 Photo Thoracic Inlet (Describe Structures)	Identify the **vascular** structures in this photo.	**Common carotid aa left 14, & right 19, brachio-cephalic trunk 4, , right subclavian a. 21, brachiocephalic vv right 18 & left 13, subclavian vv 24, left internal jugular v 8, thyrocervical trunk 32**	5/8 to pass
	What is the anatomical relationship of the internal jugular vein to the carotid artery?	**Superiorly IJV lies posterior to ICA** **Passes inferiorly in the carotid sheath with vagus n** between IJV and carotid **Inferiorly IJV lies lateral to CCA,** passes deep to heads of SCM	2 of 3 bold to pass
	Describe the surface marking of the internal jugular vein.	From **earlobe/mastoid to medial end of clavicle**	Bold to pass

Question 4 Model Heart (Coronary Artery/Valves)	Using this model, describe the arterial supply of the Heart	Main coronary vessels arise from the corresponding aortic sinuses above the AV **R Coronary** courses inferiorly in AV grove, 3 Branches – SA nodal, Marginal, Posterior Interventricular **L Coronary –** bifurcates into **Circumflex** & **LAD** Cx gives off Marginal Br , LAD gives off Diagonals	Bold and 1 other to pass
	Using this model identify the chambers and valves of the heart	**RA, Tricuspid v, RV, Pulmonary v, LA, Mitral v, LV, Aortic v**	Must identify all chambers and valves
	Identify the components of the Tricuspid V	3 cusps – Anterior, Posterior & Septal Chordae Tendinae Papillary Muscles	2 of 3 to pass
Question 5 Discussion Sensory Innervation (Upper Limb)	Demonstrate the dermatomes of the upper limb	C4 – lateral shoulder C5 – lateral arm C6 – lateral forearm & thumb C7 – middle / ring fingers & center of posterior forearm C8 – little finger, medial hand / forearm T1 – medial forearm, inferior arm T2 – medial arm, axilla	5 to pass. General concept of distribution required
	On your own hand demonstrate which nerves supply sensation to which parts of the hand? Prompt: Demonstrate the peripheral cutaneous innervation of the hand	Median –palmar & distal dorsal tips of lateral 3.5 digits Ulnar – palmar & dorsal surface of medial 1.5 digits Radial – dorsal aspect of lateral 3.5 digits (excluding tips)	All 3 nerves & correct distribution

ACEM PRIMARY 2013/1 Anatomy VIVA **Morning Session 3** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: C1/C2 (Bony Landmarks/Articulation)	C2 – identify the features of this bone.	**C2 (axis).** Body, dens, superior and inferior articular facets, pedicle, transverse process, transverse foramen, lamina, spinous process, vertebral foramen,	Dens and 5 others to pass
	Describe the joints between C1 and C2	Lateral atlanto-axial joints (facet joint) and joint between anterior arch of C1 and dens. Both synovial joints – first one hinge, second one pivot	ID location of both joints to pass
	Which ligaments stabilise these joints?	**Cruciate (cruciform) ligament –** vertical and transverse components **Alar ligaments** Post longitudinal lig continued as **tectorial membrane** Anterior longitudinal ligament, Ligamentum flavum, Nuchal ligament, Interspinous ligament, Joint capsule	2 of 3 bold and 1 other
Question 2 X-Ray Foot AP/lat	Identify the bones in this xray	**Calcaneus, Talus, Navicular, 3 Cuneiforms ,cuboid** and **metatarsals,** phalanges, distal tibia, distal fibula).	4 of 5 bold plus 2 others
	What are the movements that occur in the **foot** and the joints where those movements occur?	**Inversion / eversion - subtalar (talocalcaneal) and calcaneocuboid** Flexion /extension at MTP joints Flexion /extension at IP joints	Must ID eversion /inversion and name one of the two joints involved
Question 3 Photo Palm of Left Hand (Describe Structures)	Using the photograph as a guide describe the vascular supply of the hand Prompt: Can you identify any of them	Ulnar Artery (21) forming the Superficial palmer arch (18) Radial Artery (17) forming deep palmar arch	Identify ulnar artery and superficial palmar arch Describe radial artery and deep palmar arch 3 to pass
	What other major structures can you identify	12-**Median N,** 13-Med N – Palmer Br, 14-Med N – Recurrent Br, 15-Digital nerves, 22-Ulnar N 1- Abductor pollicis brevis, 2- Abductor digiti minimi, 3-Adductor pollicis, 8- Flexor digiti minimi brevis, 9- Flexor pollicis brevis, 11- Lumbrical , 16- Palm Br 6- FCR, 7- FCU, 10-Flex Retinacum, 20-Synvo Sheaths of flex tendons	Bold and 6 others to pass

Question 4 Model Male Pelvis (Urinary System/Testes)	Identify the structures that form the male genitourinary system in this model	Bladder, ureter, prostate gland, seminal gland, spermatic cord, testis, epididymis, penis	6 to pass
	What are the contents of the spermatic cord?	**Ductus deferens**, artery of ductus deferens, **testicular artery**, **testicular vein** → pampiniform plexus, lymphatics, autonomic nerves (sympathetic, parasympathetic)	Bold to pass
	Indicate on the model the location of the named parts of the male urethra	**Intramural** (base of bladder wall), **prostatic** (length of prostatic) , **membranous** (short narrow section surrounded by ext sphincter) and **spongy** (length of corpus spongiosum)	3 of 4 to pass
Question 5 Discussion Circle of Willis	Draw a diagram depicting the Circle of Willis	 (C) Inferior view	ID 3 paired arteries and ICA to pass
	Which arteries supply which parts of the brain?	**Anterior cerebral a – Frontal lobe,** medial and superior surface **Middle cerebral a - Temporal lobe and lateral surface** **Posterior cerebral a - Occipital lobe,** inferior surface Vertebro-Basilar	BOLD TO PASS

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: Infarction LOA: 1	1. What is an infarct? 2. What mechanisms lead to infarction? 3. What factors determine the development of an infarct? Prompt- What influences whether an infarct will develop?	1. Area of **ischaemic necrosis** caused by **arterial** or venous occlusion **2 Arterial thrombosis, embolism**, vasospasm, haemorrhage into plaque, extrinsic vascular compression (by tumour or oedema), torsion of vessel, traumatic rupture, entrapment in hernial sac, venous thrombosis 3. Factors that determine development of an infarct • *Nature of vascular supply eg dual vs end arterial* • Rate of occlusion development – time for collaterals to develop • Vulnerability to hypoxia of the tissue type • Oxygen content of blood	Bold Bold + 2 2 of 4
Question 2 Type 2 Hypersensitivity Reaction LOA: 1	1. What is Type 2 hypersensitivity? 2. Describe the mechanisms involved giving examples for each mechanism.	**1 Hypersensitivity caused by antibodies that react with antigens present on cell surfaces or in the extracellular matrix** Antigens can be intrinsic to the membrane or matrix or extrinsic eg. Drug metabolite **2 a) Opsonisation & phagocytosis: IgG antibodies opsonise cells plus complement activation** generates C3b & C4b recognized by phagocyte Fc & protein receptors resulting in **phagocytosis & destruction of opsonised cells** ADCC- cells coated with Abs killed by monos, neutros, eosinos and NK cells **Examples:** transfusion reaction, erythroblastosis fetalis, autoimmune haemolytic anaemia, agranulocytosis,, thrombocytopaenia, drug reactions when a drug acts as a hapten **b) Complement and Fc receptor mediated inflammation: antibodies bind to fixed tissue** such as basement membranes, extracellular matrix ... **activates complement** ... generate by-products particularly chemotactic agent C5a ... direct PMN migration and C3a and C5a = **increase vascular permeability. PMNs activated** by C3a and Fc receptors... release of pro-inflammatory substances like prostaglandins, production of lysosomal enzymes, reactive O2 species **Examples:** glomerulonephritis, vascular rejection in organ grafts, vasculitis caused by ANCA, Goodpastures c) **Antibody mediated cellular dysfunction:** antibodies directed against cell surface **receptors impair or dysregulate function without causing cell injury or inflammation** **Examples:** myasthenia gravis, Graves's disease, insulin resistant diabetes, pemphigus vulgaris, pernicious anaemia	Bold (concept)_ Bold 2/3 With 1 example in each

Question 3 Community Acquired Pneumonia LOA:1	1.What organisms cause community acquired pneumonia? PROMPTS: What organisms cause atypical pneumonia? What viruses may cause atypical pneumonia?	**1 Bacterial** • **Strep pneumoniae** • Haemophilus influenza • Moraxella catarrhalis • Staph aureus • Legionella pneumophilia • Others eg klebsiella pneumonia, pseudomonas **Atypical pneumonia** • Mycoplasma pneumonia • Chlamydiae spp • Coxielle burnetti (Q fever) • RSV, parainfluenza, influenza A+B, adeno virus. SARS virus	Need • Bacteria bold +2 • Atypical 1
	2. What conditions predispose to the development of pneumonia?	2 Extremes of age, malnutrition, alcoholism Chronic conditions – CCF, COPD, DM Neurological/swallowing disorders-aspiration pneum Congenital or acquired immune deficiencies Decreased or absent splenic function- splenectomy, sickle cell disease Recent viral infection (esp staph). IVDU & staph	4 broad categories 2/3 bold
	3. What are the potential complications of pneumonia Prompt-Pathological sequelae	3 **Abscess formation** (type 3 pneumococcus, Kleb) **Empyema** **Bacteraemic dissemination** – endocarditis, pericarditis, meningitis, abscesses of kidney, spleen, brain, septic arthritis	
Question 4 Infective enterocolitis LOA: 2	1. What are the organisms that cause infectious enterocolitis?	**1. Bacterial**- E.coli, Salmonella, Shigella, Campylobacter, C.difficile, Cholera, Yersinia, Mycobacteria **Viral**- Norovirus, Rotavirus, Adenovirus Parasitic- Giardia, Amoeba, Cryptosporidium, other (nematodes, cestodes, trematodes)	Bold with 1 bact & 1 viral 3 examples total
	2. What is pseudomembranous colitis?	2. Colitis caused by **overgrowth of C. difficile** (also Salmonella, C.perfringens typeA, S.aureus) Associated with antibiotic use Forms a **pseudomembrane** made up of adherent layer of inflammatory cells and debris	Bold
	3. What are the risk factors for development of pseudomembranous colitis?	3. **Risk factors- advanced age, hospitalisation, antibiotic treatment**	2/3 Bold
	What are the clinical features of pseudomembranous colitis?	30% hospitalised patients colonised, but most aymptomatic **Fever**, leucocytosis, **abdominal pain**, cramps, hypoalbuminaemia, watery **diarrhoea**, dehydration, rarely gross bloody diarrhoea Diagnosis-usually detection of toxin Treat with metronidazole, vancomycin	Bold

Question 5 Gout LOA: 2	1. What are the causes of gout?	**Hyperuricaemia:** 1. Primary Gout (90%; often idiopathic): Overproduction (diet, unknown enzyme defects); Reduced filtration/excretion with normal production. 2. Secondary Gout (10%; known cause, secondary effect is gout): Leukaemias/tumor lysis/psoriasis, inborn errors of metabolism (overproduction with increased excretion); Chronic renal disease (reduced excretion).	Hyperuricaemia + 1 Primary and 1 Secondary cause Or 1 overproduction and 1 decreased excretion
	2. Describe the pathogenesis of acute gouty arthritis. Prompt- What are the steps involved?	1. Hyperuricaemia 2. **Precipitation of urate crystals into joints** (in synovium / cartilage) 3. **Release of crystals into synovial fluid** (?trauma) 4. **Inflammatory response initiated** – crystals phagocytosed by macrophages and neutrophils; release of inflammatory mediators by macrophages (interleukins, cytokines (IL-1B)); resulting in further neutrophil chemotaxis; neutrophils also release inflammatory mediators (free radicals, leukotrienes (LT B4), lysosomal enzymes) – acute arthritis.	Bold to pass
	3. (only if needed) What factors contribute to the conversion of asymptomatic hyperuricaemia into gout	Age & duration of hyperuricaemia; genetic predisposition/etoh/obesity/drugs e.g. thiazides/lead toxicity	

ACEM PRIMARY 2013/1 Pathology VIVA **Afternoon Session 2** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: **Oedema formation** LOA: 1	1. What are the mechanisms of oedema formation?	**1.↑ hydrostatic pressure –** impaired venous return, eg CHF,Constrictive pericarditis,ascites,venous obstruction(internal/external +immobility), arteriolar dilatation eg heat **Decr plasm oncotic pressure (hypoproteinaemia)** – nephrotic syndrome, ,malnutrition, protein losing enteropathy. **Lymphatic obstruction** - inflammatory, neoplastic, post-surgery/radiation **Sodium and water retention** –XS salt with renal insufficiency, incr renin-angiotensin-aldosterone secretion **Inflammation** –acute/chronic, anglogenesis	3 out of 5 bold, example from each
	2. What is the pathogenesis of cardiogenic oedema?	**2.** Decreased cardiac output, decr renal perfusion, secondary aldosteronism, Incr blood volume, incr venous pressure	At least 3 steps.
Question 2 Hep B LOA: 2	1. How can Hepatitis B infection be transmitted?	1. Vertical – perinatal during childbirth Horizontal – skin or mucosal breaches - intercourse - shared needles / syringes in IVDU - blood transfusion	**3/5**
	2. What are the potential outcomes following ACUTE Hepatitis B infection?	**2. Recovery** >90% Fulminant hepatitis necrosis <0.5% **Chronic Hepatitis** <5% - cirrhosis 12-20% +/- hepatocellular Ca - healthy **carrier state** - non progressive chronic hepatitis <2%	**Bold to pass**
	3. What are the serum markers of ACUTE infection with Hepatitis B? Prompt: What antigens and antibodies are present during acute hepatitis B?	**3. HBeAg, HBsAg** HBV-DNA, **Anti-HBc IgM** Anti-HBe, (not Anti-HBs)	**2/3 Bold**

Question 3 IHD LOA: 1	1. In myocardial infarction, what sequence of events leads to acute coronary artery occlusion? Prompt- pathological events 2. Describe the time course of myocardial injury after acute coronary artery occlusion. Prompt- What happens to the myocardial tissue over time?	**1. Sudden change in atheromatous plaque** haemorrhage, erosion, ulceration, rupture, fissure **Platelet** adherence, activation & aggregation leading to microthromi Vasospasm from plt released mediators Activation of coagulation pathway causing **thrombus** Vessel occlusion **2. Reversible** • cessation of aerobic metabolism seconds • decreased ATP production • lactic acid production (noxious metabolites) • loss of contractility, acute heart failure 1 min • ultrastructural changes – myofibrillar relaxation, glycogen depletion, cell & mitochondrial swelling few minutes • ATP depletion up to 40 min **Irreversible** • myocyte injury – defects in sarcolemmal membrane and cell leakage 20 - 40min • initially subendocardial then transmural myocyte death • microvascular injury 1 hour • coagulation necrosis > 2 hours (more protracted if collaterals)	**Bold to pass** **Bold to pass with minutes to hours concept**
Question 4 Asthma LOA: 1	1. What are the pathological features of asthma? 2. Asthma may be categorized as atopic or non-atopic. What are the characteristics of each of these types? Prompt - What is the underlying mechanism of atopic asthma? What are some of the triggers? 3. In atopic asthma, what happens in the early-phase reaction?	**1. Increase airway responsiveness** to variety of stimuli; episodic **bronchoconstriction**; bronchial wall **inflammation**; incr mucus 2. Atopic- **IgE mediated type1 hypersensitivity** (allergen sensitisation); **environmental allergen triggers** e.g. dust, pollen, dander e.g. house dust mite, foods. Family Hx common; skin test positive to allergen; RAST shows allergen sensitivity Non-Atopic- **hyperirritability of bronchial tree**-no allergen sensitisation, skin tests usual negative; family Hx uncommon; triggers-resp infection secondary viruses common; inhaled air pollutants may contribute (SO2, ozone, NO2) **3. Allergen exposure**=> IgE. • Reexposure=> Mast cell degranulation with release of cytokines/mediators • =>bronchoconstriction (via subepithelial vagal/parasympathetic receptors), • mucus production, • vasodilation with increased vasc permeability	Bold Bold. One trigger for each Bold plus concept

Question 5 Obstructive uropathy LOA: 2	1. What are the causes of urinary tract obstruction?	1. Congenital- urethral valves & strictures; bladder neck obstruction; ureteropelvic narrowing; reflux **Calculi; Prostatic hypertrophy** **Tumors**- prostate; bladder; cervix/uterus; other Inflammation- prostatitis; urethritis; ureteritis; retroperitoneal fibrosis Sloughed papillae, clots; Pregnancy; Uterine prolapse; cystocele Functional- neurogenic (spinal cord/diabetic); dysfunctional; ureter or bladder	Bold plus one other.
	2. What are the clinical features of acute obstruction?	**2. Pain** due to distension or Sx of underlying process e.g. renal colic, LUTS in prostatic disease asymptomatic (in Unilateral complete or partial) Polyuria and nocturia. Calculi, HT, distal tubular acidosis- (In Bilateral partial) oligo/anuria, hyperkalaemia, incr urea & creat- (in Complete bilateral)	Bold
	3. What are the possible clinical sequelae of urinary tract obstruction?	3. Infection Stone formation Atrophy/hydronephrosis/obstructive uropathy (if chronic)- => renal failure Complications of renal failure.	3/5

ACEM PRIMARY 2013/1 Pathology VIVA **Morning Session 3** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: LOA: 1 Vascular changes of acute inflammation	1. In acute inflammation what changes occur in blood vessels? Prompt: What happens next? 2. What are the mechanisms for the increased vascular permeability seen in acute inflammation?	1. Changes in blood flow: (transient constriction), **vasodilation** (NO mediated) lead to **increased flow** • **Increased permeability,** loss of protein-rich fluid • Fluid loss & dilation lead to **stasis/congestion** • Leukocytes accum at vasc endothelium, endothelium expresses adhesion molecs, leuks adhere & migrate out 2. Chem mediated **endothelial cell contraction** (caused by eg histamine, LKT, sub P) • Endothelial injury direct/microbes/leuks eg burns • Increased transcytosis of fluids/proteins via channels of connected vesicles/vacuoles (vesiculovacuolar organelles) stim by factors eg VEGF	3/4 Bold 2/3 must include bold
Question 2 LOA: 2 The normal immune response	1. What are the major classes of lymphocytes? 2. What is the role of each class of lymphocytes in the normal immune system? Prompt- What is the role of B-cells? What is the role of T-Cells?	1. B lymphocytes CD4+ helper T- Lymphocytes CD8+ Cytotoxic T Lymphocytes Natural Killer (NK) Cells 2. Adaptive immunity – circulate widely & rec-circulate esp Ts - respond to foreign substances/Ag. Can become effector or memory cells B cells: recognise Ag via memb IgM/IgD –plasma cell -secretes Ig/Ab = humoral immunity. (B cells also have compl R, FcR, CD40) T cells: Ag specific T cell R - binds to Ag on cells (on MHC molecules on APCs) – activates cell depending on type = cell-mediated immunity CD4/T helper recog class II MHC bound Ag: cytokine release – leads to macrophage activation, inflam, B cell stimulation CD8/ T cytotoxic recog class I MHC bound Ag: infected cell destruction NK Cells- kill inf&tumor cells. No prior exp needed. Healthy cell Class I MHC=>inhibits NK. Can secrete cytokines=>inflame	B&T B-Humoral plus concept T-Cell mediated plus concept
Question 3 Pulmonary Embolism LOA: 1	1. From where do pulmonary thromboemboli originate? 2. What are some risk factors for thrombus formation? 3. What are the clinical effects of pulmonary thromboemboli?	1/95% **arise in the deep veins of the leg** – pass up to R side of heart and into pulm vasculature. Size determines where they lodge. 2. **Primary** – (genetic factors) – factor 5 Leiden, protein C+S deficiency, antiphospholipid syn **Secondary-** (acquired) – stasis/Immobilisation, long haul flights, active malignancy, trauma/burns/surgery, pregnancy, OCP. Indwelling catheters 3. most clinically silent 60-80%, Cough, SOB, fever, CP, haemoptysis, tachy-cardia/pnoea through to sudden death,cor pulmonale,CVS collapse Pulm haemorrhage / infarction, over time multiple emboli may cause pulm hypertension & cor pulmonale	Bold to pass (exact % not required but rough idea) At least one example from Primary, and 2 from secondary 5 features

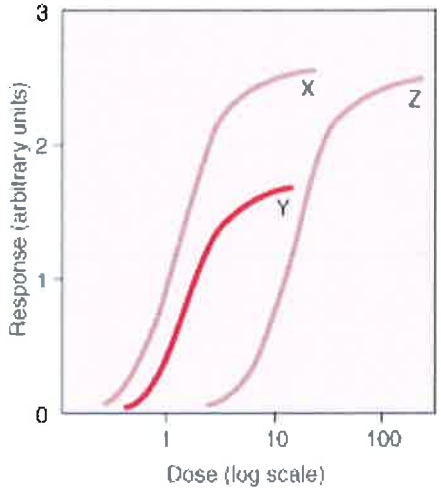
Question 4 Portal Hypertension LOA: 2	1/What are the causes of portal hypertension? May need to prompt for examples/classification. 2/What are the clinical consequences of portal hypertension? 3/What mechanisms are involved in the formation of Ascites?	1/ Incr resistance to portal blood flow Prehepatic – portal vein thrombosis or narrowing Hepatic – (most important)- **cirrhosis**, massive fatty change, schistosomiasis, granulomatous disease eg sarcoid/Tb Post hepatic - severe RHF, constrictive pericarditis hepatic vein occlusion 2/ **Ascites** – with potential for infection **Porto-systemic shunts** : varices, haemorrhoids, spider naevi Congestive **splenomegaly** – thrombocytopaenia/pancytopaenia **Hepatic encephalopathy** 3/ Sinusoidal hypertension – Starling forces : Incr pressure and decr albumin Incr formation of hepatic lymph – exceeds capacity of thoracic duct- percolates into peritoneum Splanchnic vasodilation with dec BP=> Renal retention of sodium and water due to secondary hyperaldosteronism	Bold. One from each other group 2/4 bold 2/3 concepts
Question 5 Traumatic CNS Injury LOA: 1	1/ What types of intracranial bleeding can be seen in a patient with a head injury? 2/What sequence of events occur in an extradural haemorrhage 3/Define concussion and what are its clinical features?	1/ **Extradural** **Subdural** **Subarachnoid (including intraventricular)** **Intra-parenchymal** 2/Dural **artery** (eg. middle meningeal) tear**, usually associated with a skull fracture** Strips off the dura from the skull May be a lucid period before ALOC 3/**Altered consciousness secondary to a head injury** **Transient** neurological dysfunction Transient resp arrest Transient loss of reflexes (pathogenesis is unclear, may be dysregulation of RAS) Features inc headache, amnesia, N&V, Concentration and Memory issues, perseveration, irritability, behaviour/personality changes, dexterity loss, neuropsychiatric syndromes	3 of 4 Must get bold Must get bold 3 features

ACEM PRIMARY 2013/1 Pharmacology VIVA **Morning Session 1** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: **PHARMACOKINETICS** LOA: 2	Describe the pharmacokinetic changes that occur in the elderly	**Absorption**: nutritional deficits; delayed gastric emptying (diabetics); co ingested agents (laxatives, antacids) **Distribution:** ↑ body fat, alpha-acid glycoprotein (bases); ↓ lean body mass, body water, albumin (weak acids); **Metabolism**: ↓ phase 1 reactions P450; ↓ liver blood flow, liver disease, CCF, nutritional defic **Elimination:** ↓ renal CL; renal disease; ↓ resp capacity; resp disease	**Hepatic metabolism↓** **Renal clearance↓** **+ 1 other**
Question 2 **VERAPAMIL** LOA: 1	Describe the effects of verapamil on the heart.	Binds to α1 receptor L-type Ca channel **Blocks Ca influx** **Reduced contractility** CO, O_2 demand **Reduced** impulse generation/**conduction AV node** Reduced coronary artery spasm	**Bolded**
	What are the indications for verapamil?	**Angina; hypertension; atrial arrhythmias** migraine	**2 bolded**
	Name some clinical adverse effects	Extensions of therapeutic action (exacerbated by β blockers) **Bradycardia; AV block; CCF; hypotension** Other Constipation; peripheral oedema; dizziness; flushing; nausea	**2 bolded**

Question 3 **CEPHALOSPORINS** LOA:1	What is the mechanism of action of cephalosporins? How does the spectrum of microbiological activity differ between the cephalosporin generations?	**Inhibit bacterial cell wall synthesis** , cell division and growth (similar to penicillins) Bacteriocidal Work best in rapidly dividing cells **1st generation: very active against GPC**, Ecoli, K.pneumoniae, proteus ok but Pseudomonas not. Anaerobic cocci sensitive **2nd generation:** active against those by 1st generation but **added GN coverage** -klebsiella Some anaerobe cover NO Pseudomonas **3rd generation expanded GN coverage and cross BBB**. Less active re staph . Work against B-lactamase Haemophilis and Neissria. Ceftazadime works re Pseudomonas 4th generation more resistant to B- lactamases, extended coverage against enteric GNR-pseudomonas, enterobacteriaceae, S pneumonia, S aureus, Haemophilis and Neisseria. Cross BBB	**Bolded to pass** **Understand the principles of the 1st, 2nd and 3rd generations**
Question 4 **KETAMINE** LOA: 1	What are the indications for ketamine What are the routes of administration? What is the IV dose used for induction of general anaesthesia? Name some of the adverse effects.	**Induction agent, procedural sedation, analgesia** **IV, IM,** IN, epidural, PO, PR, SC **1-2 mg/kg** **Hypersalivation, larygospasm(peds), vomiting(recovery phase), emergence reactions, Hypertension, tachycardia, raised ICP**	**2 of bolded** **IV, IM + 1 other** **Bolded** **Emergence reactions + 2 other**

Question 5 **N-ACETYLCYSTEINE** LOA: 2	What is the mechanism of action of N-acetylcysteine in paracetamol overdose?	Paracetamol metabolism by hepatic glucuronidation/sulphation is saturated resulting in increased metabolism via cytochrome p450 system to form N –acetylbenzoquinoneimine (NAPQI), a **toxic intermediate**. Elevated NAPQI production leads to depletion of hepatic glutathione stores, resulting in hepatotoxicity. NAC prevents paracetamol induced hepatotoxicy by 4 possible mechanisms: **1) Increased glutathione availability**/Sulfhydryl donor 2) Direct binding to NAPQI 3) Provision of inorganic sulphate 4) Reduction of NAPQI back to paracetamol	**Bold to pass**
	Name an adverse effects of N-acetylcysteine.	Mild **anaphylactoid reactions**(15-20%)- mild flushing, rash and angio-oedema.	**Bold or description**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: **POTENCY & EFFICACY** LOA: 1	Define "potency".	Potency refers to the affinity or attraction between an agonist and its receptor. It reflects the dose axis of dose response curves. A measure of drug potency is the EC_{50} – the conc'n/dose req'd to produce 50% of maximal response.	**Be able to explain potency and efficacy**
	How is this different to Efficacy?	Efficacy is the maximal response that a drug (agonist) can produce (E_{max}) when all receptors are occupied, irrespective of the concentration required to produce that response. Efficacy determines a drugs clinical effectiveness and reflects the response axis
	Draw a concentration-response curve showing 2 drugs with the same potency but different efficacy.	
		X and Z have similar efficacies, X and Y have similar potencies; X and Y are more potent than Z	

Question 2 **PROPRANOLOL** LOA: 1	Describe the pharmacodynamics of propranolol. What are the potential adverse effects?	B antagonist; competitive; non-selective CV ↓BP if high -ve inotrope –ve chronotrope ↑PR interval ↓renin release Resp bronchospasm Eye ↓pressure (↓humour production) Metabolic ↓glycogenolysis ↑VLDL ↓HDL Bradycardia; ↑CCF; ↑PVD ↓hypoglycaemia response Bronchospasm Sedation/depression Abrupt withdrawal effects Exacerbate Ca channel blocker effects	2 CV + 1 other Bradycardia, bronchospasm and 1 other
Question 3 **TRIMETHOPRIM** LOA: 2	Describe the mechanism of action of trimethoprim. What is the rationale for combining trimethoprim with sulphonamides?	Selectively **inhibits bacterial enzyme (**dihydrofolic acid reductase) which is required in the conversion of dihydrofolic acid to tetrafolic acid. **Hence inhibits** purine and **DNA synthesis.** Less efficient in inhibiting mammalian dihydrofolic acid reductase **Enhanced effect** - sulphonamides **inhibit sequential steps (** acts step before triprim). Inhibits dihydropteroate synthase involved in conversion PABA to dihydrofolic acid As sequential steps are blocked in folate synthesis usually bacteriocidial c.f bacteriostatic of 1 alone.	**Inhibits bacterial enzyme Resulting in Inhibition DNA synthesis** **Bold**

Question 4 **MIDAZOLAM** LOA: 1	What are the clinical indications for the use of midazolam?	Anxiolysis, **sedation, anticonvulsant,** antiemetic	**Bold to pass**
	What are the advantages and disadvantages of the various routes of administration?	PO, IV, IM, PR, IN, Buccal	**Reasonable discussion of IV + 1 other**
	What are the adverse effects?	**Excess sedation, respiratory depression,** decreased motor skills, impaired judgment, hypotension + occasionally rashes	**Bold to pass**
Question 5 **OCTREOTIDE** LOA: 2	What are the therapeutic uses for octreotide?	Control of **bleeding gastro-oesophageal varices,** sulphonylurea induced hypoglycaemia, pituitary and carcinoid tumors.	**Bold to pass**
	What is the mechanism of action of octreotide in acute variceal bleeding?	**Reduces splanchnic blood flow/portal venous pressure.** Exact mechanism of how this occurs is not known.	**Bold to pass**
	How is it administered in acute variceal bleeding?	**IV bolus and infusion** (50mcg bolus then 25-50mcg/hr) or SC	**Bold to pass**
	Why is an infusion required?	**Short half-life**	**Bold to pass**

ACEM PRIMARY 2013/1 Pharmacology VIVA **Morning Session 3** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: Bioavailability LOA: 2	What is bioavailability?	Fraction of **unchanged drug** reaching the **systemic circulation** following administration by any route.	**Bold to pass**
	What factors limit drug bioavailability following oral administration?	**Extent of absorption:** **a) Property of the drug** eg hydrophyllic vs lipophyllic b) Gut factors - reverse transporter pumps p-glycoprotein &gut wall metabolism **First pass elimination-** metabolism by liver before reaching systemic circulation or small effect biliary excretion	**Bold to pass**
	How can you overcome the effects of high first pass metabolism?	**Change route of administration** to sublingual, transdermal eg GTN, rectal, inhalation, IV, IM **Increase dose** Use pro-drugs	**Bold**
Question 2 **GLYCERYL TRINITRATE (GTN)** LOA: 1	How does Glyceryl Trinitrate (GTN) exert its effect on smooth muscle?	Nitrate→**Nitric Oxide→↑cGMP→** relaxation→**vasodilation** Also involves Prostaglandin E or prostacyclin	**Nitric Oxide , cGMP/second messenger, vasodilation**
	Describe the Pharmacokinetics of GTN **Prompt**: How is GTN given?	**Low Bioavail** (<10-20%) Sublingual, transdermal or IV S/L: onset 1-3min, lasting 10-30min Liver metabolism and excreted by kidney Tachyphylaxis with continuous use	**Low Bioavailabilty** **Short halflife**

Question 3 **NORFLOXACIN** LOA: 1	Describe the mechanism of action of norfloxacin.	Fluoroquinolone. Bacteriocidal. a. **Inhibition topoisomerase II /DNA Gyrase** → interferes with relaxation of supercoiled DNA, required for normal transcription and replication b. Inhibition topoisomerase IV → interferes with separation of replicated chromosomal DNA	**Bold to pass**
	Describe the anti-bacterial activity of norfloxacin	**Gram negative bacteria** Organisms of atypical pneumonia: mycoplasma, chlamydia Limited gram positive activity	**Bold to pass**
	How does the anti-bacterial activity of norfloxacin compare to that of ciprofloxacin?	**Ciprofloxacin has greater activity** (4-8 times lower MICs) against gram negatives and much greater activity against gram positives	**Bold to pass**
Question 4 PROPOFOL LOA: 1	What are the indications for the use of Propofol?	**Induction agent, maintenance of anaesthesia procedural sedation**	**2 bold to pass**
	What properties of Propofol make it suitable for procedural sedation?	**Rapid onset and offset**	
	What are adverse effects of Propofol?	Localised pain with bolus administration. Dose related **depression of respiratory drive** (central effect) and apnoea. Muscle movements, hypotonus and rarely tremor. **Hypotension** (reduced arterial resistance venodilation and negative inotropism).	**Bold to pass**

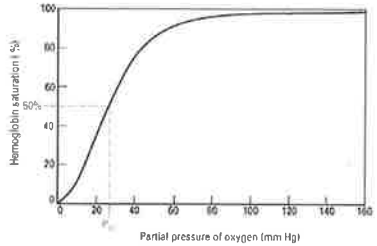
Question 5 **NALOXONE** LOA: 2	What is the mechanism of action of Naloxone?	Pure **opioid antagonist** binds to μ-opioid binding sites.	**Bold to pass**
	What is the time to onset and duration of action when administered intravenously?	**Rapid onset 1-3 minutes** **Duration 1-2 hours**	**Bold to pass**
	What problems may be associated with naloxone administration?	**Opioid withdrawal** **Resedation**	**Bold to pass**
	How can these problems be minimised or avoided?	**Smaller/titrated doses** Infusion Route of administration	**Bold to pass**

ACEM PRIMARY 2013/1 Physiology VIVA **Morning Session 1** ***Candidate Number:*** **AGREED MARK:**

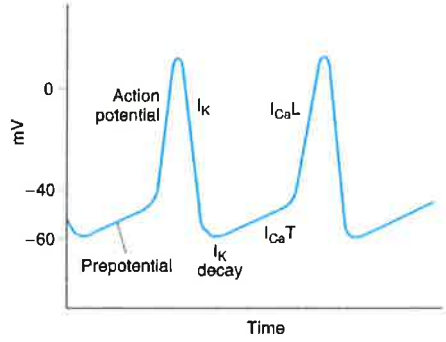
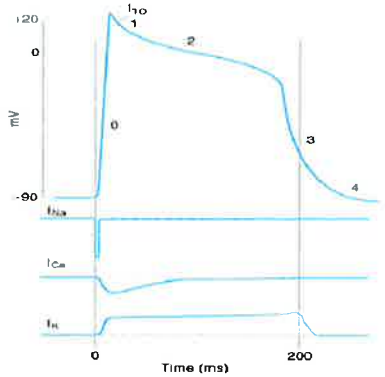
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: LOCAL FLOW REGULATION LOA: 2	a. Describe the autoregulation of tissue blood flow. Prompt: what are the main features of autoregulation	Capacity of tissues to regulate their own blood flow Tissue blood **flow remains relatively constant** despite moderate **changes in perfusion pressure** through alterations in **vascular resistance.**	Three main features to pass
	b. How would this apply to autoregulation of cerebral blood flow?	**Constant flow over arterial pressure range 65-140 mmHg.** Sympathetic stimulation prolongs the plateau.	Bold including approximate range
	c. What are the proposed mechanisms involved in autoregulation? Prompt: What are some important metabolic changes that cause vasodilatation	**Myogenic: intrinsic contractile response of smooth muscle to stretch.** **Metabolic: production of vasodilator metabolites by active tissue**. Accumulation assoc. with decreased flow leads to vasodilation. Examples dec pO2, acidosis, high K, lactate, pCO2(brain and skin), local temp, adenosine (heart)	Both mechanisms & 2/5 metabolites
Question 2 Pulmonary resistance & Compliance LOA: 1	a. Describe the factors that determine the airway resistance in the lung. Prompt: when would airway resistance increase?	<u>Airway resistance</u> • Decreases with stimulation of ß-adrenergic receptors causing bronchodilatation. • Increases with parasympathetic nerve stimulation causing bronchoconstriction. • Increases with histamine • Increases when Lung volume reduces • Increases when pCO2 decrease • Increases with increase density & viscosity of gas	Need 3 factors to pass Poiseuille's Law: Resistance = 8 x viscosity x length / radius^4 x π
	b. With regard to lung **<u>compliance</u>** give examples of diseases that reduce compliance. Prompt define compliance: volume change/unit pressure.	b. Pulmonary fibrosis, pulmonary oedema, pulmonary haemorrhage, atelectasis, loss of surfactants such as respiratory distress syndrome.	Need 3 examples (may be others not listed that are acceptable)
Question 3 Renal H+ regulation LOA: 1	Describe the renal response to metabolic acidosis Prompts: "What prevents H+ secretion stopping when a pH of 4.5 is reached?" "What substances act as buffers in the urine?"	• **Renal compensation aims to normalise blood pH by reabsorbing all filtered HCO_3^-, and generating new HCO_3^-** by titration of filtered acid. • Anions that replace HCO_3^- are filtered at the glomerulus along with corresponding cations • **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 further H^+ elimination) Urinary buffers include **HCO_3^-**, PO_4^-, and NH3	Pass criteria **bold** Buffers need bold and 1 other

Question 4 Glucocorticoids LOA: 1	a. What are the physiological effects of glucocorticoids? Prompt: "Can you expand on non-vascular effects"	1. Essential for survival stress response 2. 'Permissive action' for **catecholamine effects: pressor/ vascular reactivity**, bronchodilation, calorigenesis, lipolysis **3. Metabolic**: protein catabolism, hepatic glycogenesis & gluconeogenesis. Rise in plasma glucose + peripheral anti-insulin effect. Increase plasma lipids. 4. Permit 'free water' excretion: plasma tonicity 5. Immunological: Decrease inflamm + allergic responses. Reduced lymphocytic activity, lymph tissue, cytokines 6. Haematological: increased neutrophils, RBC, platelets. Decreased basophils, eosinophils 7. Mental: EEG slowing, personality changes	**Must get bold, at least 2 metabolic + 1 other**
	b. How is glucocorticoid secretion regulated?	**Released adrenal cortex in response to ACTH from ant pituitary.** ACTH release driven by CRH from hypothalamus (response to low corticoid level or stress) **Glucocorticoid –ve feedback on hypothal/ pit to reduce ACTH secretion**	Must get **bold.**
Question 5 Hearing LOA: 2	a. What are the two major mechanisms of deafness?	**Conductive deafness** – due to impaired sound transmission in external or middle ear, affects all frequencies. **Sensorineural deafness** – due to loss of cochlear hair cells (commonest), or problems with CN VIII or within central auditory pathways, affects some frequencies.	**Bold** Explain both and 2 examples of each to pass
	b. Explain these causes in physiological terms and give examples.	**Examples** Conductive – blockage of extl canals (e.g. wax, FBs), otitis ext or media, perforated eardrum, osteosclerosis Sensorineural – degeneration (presbycusis), damage to outer hair cells (prolonged noise exposure), aminoglycoside antibiotics, CN VIII tumours or cerebellopontine angle, CVA in medulla.	
	Bonus: How can one differentiate between the two forms using a tuning fork?	Weber/ Rinne : 256 tuning fork	Bonus if have time

ACEM PRIMARY 2013/1 Physiology VIVA **Afternoon Session 2** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: CORONARY BLOOD FLOW LOA: 1	a. Describe coronary arterial blood flow during the cardiac cycle. Prompts: How is flow different in the left and right coronary arteries during systole and diastole? Which part of the heart is most at risk due to low coronary flow?	**Greater flow in diastole c/w systole in L coronary** due to higher pressures required in the LV to overcome aortic pressure in systole. **LV subendocardium** most vulnerable as only gets diastolic flow. **R coronary flow throughout systole and diastole** due to lower RV pressures	Three main features to pass
	b. What factors can decrease coronary artery blood flow?	**1.Physiologic**: **Tachycardia:** shorter diastole; reduced L coronary flow in particular **2.Pathologic**: AS: Increased LV pressures req. to overcome stenosis & decreased flow; Vasospasm; Coronary artery disease; Heart failure: increased venous pressure; reduced coronary perfusion press.	Tachycardia and 2 pathological
Question 2 O2 transport LOA: 1	a. Describe how oxygen is carried in the blood.	**Dissolved**: amount dissolved proportional to partial pressure (Henry's law) – 0.3 ml O_2/100 ml blood/100 mm Hg PO_2 **Combine with haemoglobin**: 20.8 mg/100 ml blood.	Need **bold**
	b. Please draw the Oxyhaemoglobin dissociation curve.	See diagram: draw graph to pass, 3 key points (2/3 accurate): examples P50 & 90/60 and 1 other.	 Source: Levitzky MG: *Pulmonary Physiology*, 7th Edition: http://www.accessmedicine.com Copyright © The McGraw-Hill Companies, Inc. All rights reserved.
	c. Describe factors that can affect the oxygen dissociation curve.	**Shift to right** by inc H^+ conc, pCO_2, temp, 2,3 diphosphoglycerate to unload oxygen. **Shift to left** with the opposite changes.	2 factors
Question 3 Renal Tubular Function LOA:	a. How do the ascending and descending limbs of the Loop of Henle differ in function?	Thin **descending limb water permeable** (aquaporins) and tubular **fluid becomes hypertonic**. **Thick ascending limb impermeable to water, and Na^+, K^+,Cl^- actively transported out, so fluid ends up more hypotonic.** K^+ diffuses back passively	Bold, illustrate clear difference
	b. Describe the process of tubuloglomerular feedback in the nephron.	This process **aims to maintain the constancy of the load delivered to the distal tubule**. The macula densa **in the ascending limb of the loop of Henle senses the rate of flow and feeds back to either increase or decrease the rate of filtration** in the glomerulus	Correct concept

Question 4 Anterior Pituitary Hormones including insufficiency LOA: 1	a. What hormones are secreted by the anterior pituitary? b. What are the clinical effects of anterior pituitary insufficiency?	**TSH; ACTH;** Growth hormone; LH; FSH; Prolactin 1. Adrenal cortical atrophy: glucocorticoid + sex hormone levels fall. Mineralocort secretion maintained: salt loss/ hypovolaemic shock does not occur. But unable to mount stress response. 2.Hypothyroidism; 3.Growth inhibition 4.Gonadal atrophy, sexual cycles cease, loss of some secondary sex characteristics 5.Tendency to hypoglycaemia (increased insulin sensitivity)	Bold + 2 other Pass: Adrenocortical effects + 2 other
Question 5 GIT handling of water and electrolytes LOA: 1	a. Explain the mechanisms of absorption of water and electrolytes in the gastrointestinal tract. Prompt: How is sodium absorbed? b. Explain the mechanisms of water and electrolyte secretion in the gastrointestinal tract. Prompt: How is chloride secreted?	**Absorption:** After meals – **fluid reuptake due to coupled transport of nutrients**, e.g. glucose and Na (Water reabsorbed 8800 ml) Between meals – NaCl enters across the apical membrane via the coupled activity of a Na/H exchanger and a Cl/HCO3 exchanger (electroneutral mechanism in small intestine & colon). In distal colon, Na enters the epithelial cell via epithelial Na Channels (electrogenic mechanism). **Secretion:** **Cl secretion occurs continuously in the small intestine & colon**. Cl uptake occurs via Na/K/2Cl co-transporter and is secreted into the lumen via Cl channels (CFTR = cystic fibrosis transmembrane conductance regulator). **Water endogenous secretions** 7000 ml	Bold and 1 mechanism of Na absorption somewhere Bold and 1 mechanism of Cl secretion somewhere Note: Water balance Input: Ingested 2000 ml & Endogenous secretions 7000 ml; Output: Reabsorbed 8800 ml; Balance in stools 200 ml

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: LOA: 1 Cardiac Muscle Action Potential (- incl difference to pacemaker action potential)	a. Please draw and explain the action potential in a cardiac pacemaker cell. Prompt: "What electrolytes are responsible for each phase of the action potential?"	Pre-potential is initially due to a decrease in K^+ efflux, then completed by Ca^{2+} influx through CaT channels The action potential is due to influx of Ca^{2+} via CaL channels Repolarisation is due to K^+ efflux 	Must have the shape to pass and know the ion fluxes. 
	b. Describe the major differences between a ventricular muscle action potential and a pacemaker cell potential.	Greater negative RMP. Fast depolarisation via Na^+ versus slower Ca^{2+} dependent. No prepotential and no automaticity. Plateau phase.	Clear contrast to the above graph, No prepotential as no leaking Ca^{2+} and plateau due to Ca^{2+}.
Question 2 LOA: 1 Lung volumes and capacity	a. Please describe the components of total lung capacity? Prompt: What individual volumes or capacities are described in relation to the total lung capacity or volume.	**Tidal volume**: the volume of gas moved in and out of the lung during normal breathing (500ml) **Vital capacity**: the exhaled gas volume after a maximal inspiration (5.5-6 litres) **Residual volume:** the volume of gas remaining in the lung after maximal expiration (1.5-2 litres) **Functional residual capacity**: the volume of the gas in the lung after a normal expiration (3 litres)	Three of four volumes
	b. Name a method to measure each of these?	**Spirometer can measure tidal volume and vital capacity** Total lung capacity, functional residual capacity and residual volume may be measures by helium dilution or the body plethysmograph	Bold

Question 3 Renin- Angiotensin System LOA: 1	a. What are the actions of the renin-angiotensin system? b. What factors affect renin secretion?	**Mediated through AT II;** - arteriolar constriction with **rise in SBP and DBP; increases secretion of aldosterone;** facilitates release of NAd acting on post-ganglionic neurones; positive feedback loop on brain by decreasing sens. to baroreflex and increase effect of AT II, and secretion of vasopressin and ACTH **Stimulation: sympathetic activity** via renal nerves; increased circ. **Catecholamines;** prostaglandins **Inhibition: - increased Na and Cl reabsorption** across macula densa; **- increased afferent arteriolar pressure;** AT II; vasopressin	**Bold** **Bold**
Question 4 LOA: 1 Vasopressin (hypothalamus)	a. Describe the feedback loop that ensures homeostasis of blood osmolality b. Name the stimuli that affect vasopressin secretion	**increase blood osmolality** triggers: thirst mechanism; **renal conservation of water - via the release of vasopressin from the posterior pituitary** Both outcomes decrease blood osmolality back to normal. Feedback terminates hypothalamic signalling Increase: **increased osmotic pressure** plasma; **decreased ECF volume**; pain emotion stress exercise; nausea vomiting; standing; drugs (carbamezepine, clofibrate); angiotensin II Decrease: **decreased osmotic pressure** plasma; **increased ECF**; Alcohol	Bold to pass Bold & 2
Question 5 LOA: 1 Exocrine pancreas	a. List the enzymes secreted from the exocrine pancreas. b. Give at least 3 examples of substrates that these enzymes work on.	Trypsin – proteins,polypeptides Chymotrypsins– proteins,polypeptides Elastase –elastin and some proteins Carboxypeptidase A - proteins,polypeptides Carboxypeptidase B - proteins,polypeptides Colipase –fat droplets Pancreatic Lipase -triglycerides Bile salt –acid lipase –cholesterol esters Cholesterol ester hydrolase–cholesterol esters Pancreatic alpha amylase -starch Ribonuclease -RNA Deoxyribonuclease -DNA Phospholipase A2 –phospholipds	Lipase and at least 2 examples & matched substrates

Opening stem: An 80 year old man presents with a leaking AAA and is to undergo Emergency Surgery

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Anatomy: Photo - Abdominal Aorta / posterior abdominal wall	1. Identify and name the blood vessels in this image.	**Aorta (1), common iliac arteries (3), femoral artery (9). IVC (23)**, Common iliac veins (4), External iliac arteries (7), External iliac veins (8), Internal iliac arteries (25), IMA (22), Femoral vein (12), lumbar artery (13), testicular vessels (39)	4 in bold PLUS 3 others to pass
	2. Identify the ureters and describe their course	**Ureters (40)** Origin at the renal hilum (PUJ); run inferiorly lying across the psoas (32) ; Lie medially to tips of the lumbar tps (on xray); Cross over the pelvic brim ; Cross anteriorly to the bifurcation of the common iliac artery; lie on the lateral wall of the pelvis, travels medially to bladder; short intramural path at VUJ	Correctly identifies ureters PLUS 3 points
	3. What are the narrowest points of the ureters?	Narrowings at the PUJ, VUJ, & pelvic brim	2 of 3 points
Pathology: Abdominal Aortic Aneurysms	1. What are the risk factors for development of abdominal aortic aneurysms?	Male; Smoking; Age > 60; Family History; Connective tissue disease (eg. Ehlers Danlos); Vasculitis; Hypertension, Diabetes; Atherosclerosis	5 to pass
	2. Describe the pathogenesis of AAA formation	**Atherosclerotic plaque** in intima compresses media with **degeneration and weakness of wall** and cystic medial degradation Local inflammation **Proteolytic enzymes** with collagen degradation -role of matrix metalloproteinases (MMP). Loss of vascular smooth muscle cells. Inappropriate Synthesis of non-elastic ECM	2 of 3 bold to pass
	3. What are the clinical consequences of an AAA?	<u>Rupture</u>: increase with diameter (higher if >5cm) & can be retroperitoneal OR intra peritoneal with rapid fatal haemorrhage <u>Obstruction</u>: ischaemia from branch vessel obstruction eg. mesenteric, vertebral, renal <u>Embolism</u>: plaque or thrombus <u>Impingement or compression</u> of adjacent structure (eg. ureter) <u>Painless mass</u>	Bold and 2 others.

The patient develops renal failure following his surgery			
Physiology: Renal regulation of K+ plus GFR	1. What is normal Glomerular Filtration Rate (GFR) and what factors regulate it? (Prompt: How does it change?) (Prompt: Identify **two clinical factors** that alter Starling Forces) 2. (Optional) How do the kidneys deal with Potassium?	1. **Normal GFR** = 125mls/min (180L/24hrs). 10% lower in females Controlled by **Starling Forces** ie: GFR = $K(P_{GC} - P_T) - (\pi_{GC} - \pi_T)$ P_{GC} = mean **hydrostatic** pressure in glomerular **capillaries**, PT = mean **hydrostatic** press in **tubule**. π_{GC} = **osmotic** press of plasma in glom **caps**, π_T **OP** of filtrate **in tubule.** K = GF coefficient; altered by **mesangial cell** contraction (-> dec area for filtration). Contraction = Angio II, ADH, NA, PAF, TxA2, hista Relaxation – ANP, dopamine, cAMP, PgE2 GFR changes along glomerular cap with Starling forces dropping from 15 mmHg to 0. **Clinical Factors** altering Starling Forces: Alterations in renal blood flow, systemic BP, ureteric obstruction, renal parenchymal oedema, changes in plasma protein concentration, changes in K as above 2. Freely filtered at glomerulus (600 mmol/d) Actively reabsorbed in PCT (560 mmol/d) Secreted in DT – rate proportional to flow Secreted in Collecting Ducts – Aldosterone Excreted = 90 mmol/d Total secreted load averages 50 mmol/d but varies with renal tubular flow and aldosterone lev.	***Pass/Fail*** 1.a) approx **value** for GFR b) Identify **Starling Forces** involved c) Identify central role of **mesangial cells and two factors which change their degree of contraction** d) Identify **two clinical factors** that alter Starling Forces 2. a) freely filtered at glomerulus, b) largely reabsorbed in PCT c) Sites of distal secretion plus influence of aldosterone
While in ICU, he goes into rapid AF and is treated with Amiodarone			
Pharmacology: Amiodarone Indications, mechanism of action, adverse effects	What are the indications for amiodarone? Describe the mechanism of action of amiodarone. Can you describe the possible adverse effects of amiodarone associated with both its short and long term use?	**Treatment of atrial and of ventricular tachyarrhythmias**. Used both to revert VT & prevent recurrence. Used in VF/VT cardiac arrest (after 3 shocks & adrenaline). Has Class I, II, III & IV effects. **Prolongs the AP duration (hence QT interval) by K channel blockade**. **Acute: Bradycardia** & heartblock ; **Hypotension**; Chronic: **Pulmonary fibrosis;** Abnormal LFTs & hepatitis; Skin deposits -> photodermatitis & grey-blue discolouration in sun-exposed areas; Asymptomatic corneal microdeposits; Optic neuritis (rare); Hypo/hyperthyroidism.	Bold to pass. Bold to pass. All bold and 1 other. Especially in those with pre-existing S/AVN disease. Due to peripheral vasodilation.

tem: A 60 yo woman presents with symptoms suggestive of carpal tunnel syndrome

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Anatomy: X-ray - carpal bones / hand Include description of contents of carpal tunnel	Identify the carpal bones.	**Identify all carpal bones in AP view.**	All bones required
	Identify the attachments of the flexor retinaculum.	Scaphoid tubercule, hook of hamate, trapezium and pisiform	2 of 4
	What structures pass through the carpal tunnel?	**FCR/FPL/FDS/FDP** tendons + Synovial sheaths **Median nerve**	**bold** to pass
	Which muscles flex the wrist?	**FCR, FCU, FPL, FDM, FDS, FDL, Palmaris longis**	5 of 7
Her symptoms are due to complications of lung cancer			
Pathology: Clinical effects of tumours	What is the definition of a neoplasm?	**Abnormal growth of a tissue** **Growth exceeds and is uncoordinated with that of the original tissue** **Growth continues in the absence of the stimuli which evoked the change** **(preys on host and serves no purpose)**	Must get the gist of all 3
	How may a malignant tumour affect the 'host'?	**Local and metastatic direct effects.** Pressure, Bleeding, ulceration, rupture and infarction. **Cachexia** **Hormonal**	3 of 4 bold
	(Prompt: what is meant by paraneoplastic syndrome?) Give examples of paraneoplastic endocrinopathies	**Paraneoplastic:** - *Endocrinopathy with 3 examples (Cushings, SIADH, Ca++ up, hypoglycaemia, Carcinoid synd, polycythaemia)* - *Nerve and muscle – myasthenia,* - *Skin - acanthosis nigricans, dermatomyositis* - *Bone: HPOA and clubbing* - *Blood/Vascular: anaemia, venous thrombosis*	3 examples of paraneoplastic syndrome

In the ED, she becomes progressively tachypnoeic			
Physiology: Control of ventilation	What are the major components of the control of ventilation (or respiration)	**Voluntary versus automatic** **Medulla pacemaker cells,** **Pons** pneumotactic centre modifies the medulla actvity **Higher centres** hypothalamus, limbic system, cerebral cortex **Vagal afferents** from lung **Central chemoreceptors** CSF (medulla, floor 4th ventricle)– ⇧H^+, **Peripheral chemoreceptors carotid and aortic bodies** – pO_2, pH ⇩, pCO_2⇧ Integrated response: $PaCO_2$, PO_2, pH **Lung receptors** – stretch, irritant, bronchial C fibres (J receptors),	5 of 7 bold
	How does a rise in CO_2 affect ventilation?	Direct effect on **central and peripheral chemoreceptors**, due to both **high CO_2** and **lower pH.** **Increase in rate and depth of ventilation.**	4 of 5 bold
Due to her worsening respiratory failure, she requires intubation			
Pharmacology Suxamethonium MOA, adverse effects	What is suxamethonium	**depolarising muscle relaxant** producing rapid neuromuscular blockade at **motor endplate nicotinic receptors.** Structurally two acetylcholine molecules linked end to end	Pass = bold
	Describe the mechanism of action of suxamethonium	**Phase I (depolarizing)** binds to nicotinic receptor; **opens channel** and causes **depolarisation of motor end plate**; spreads to adjacent membranes causing contractions of muscle motor units (fasciculations); **depolarised membranes remain depolarised (& unresponsive to subsequent impulses)** causing flaccid paralysis Phase 2 (desensitising) With continued exposure, the initial end plate depolarisation decreases & membrane becomes repolarised; membrane cannot be depolarised again as it is *desensitised* (mechanism unclear however ? due to channel block becoming more important than agonist action at receptor)	Pass = bold
	What are the important adverse effects of suxamethonium?	**hyperkalaemia** (eg burns, trauma patient); cardiac arrhythmias (eg if given with halothane) / **bradycardia** (repeat doses); increased IOP; increased intragastric pressure; muscle pain (likely related to fasciculation); malignant hyperthermia, prolonged paralysis	Pass 2 bold + 2 others

ACEM PRIMARY VIVA B **Thursday Morning Session 1** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 PATHOLOGY LOA: 1	*"An elderly man presents with an acute exacerbation of COPD."* What is the definition of emphysema? Describe the pathogenesis of emphysema. Prompt: What is the mechanism of the destruction? What are the possible complications of emphysema?	• A condition of the lung characterised by **irreversible enlargement** of the airspaces distal to the terminal bronchiole accompanied by **destruction of their walls** without obvious fibrosis. • **Mild chronic inflammation (neutrophils + macrophages)** - **mediator release** (e.g. leukotriene B_4, IL-8, TNF) – causes damage and sustains inflammation • **Protease-antiprotease imbalance – destructive effect of high protease activity** in pts with **low anti-protease activity** - 1% of pts with emphysema have alpha1-antitrypsin deficiency (inhibits proteases, including elastase, secreted by neutrophils) • **Oxidant-antioxidant imbalance – abundant reactive oxygen species** (superoxide dismutase, glutathione)**in smoke depletes antioxidant mechanisms,** incite tissue damage • Bullous lung disease • Expiratory airflow limitation • Infection • Respiratory failure • Pneumothorax • Cor pulmonale, congestive heart failure ("pink puffers")	**BOLD TO PASS** • Irreversible • Destruction **TWO EFFECTS** • Chronic inflammation • High protease activity • Reactive oxygen species **THREE COMPLICATIONS**
Question 2 PHYSIOLOGY LOA: 1	1. What are the possible physiological causes for hypoxemia in this man? 2. What is the alveolar gas equation ? 3. Explain the concept of the A-a gradient.	Hypoventilation Diffusion limitation Shunt V/Q mismatch $PAO_2 = PIO_2 - \frac{PACO_2}{R} + F$ Difference between the measured and the predicted paO_2.	Need 2 /4 to pass or a good understanding of the concepts Numbers ok Need the basic concept
Question 3 PHARMACOLOGY LOA: 1	*"Moving on. He is treated with a cephalosporin."* 1. What is the mechanism of action of cephalosporins? 2. What class of antibiotics do they belong to? 3. How are they classified and give an example of each class ?	1. **Inhibit bacterial cell wall synthesis**, cell division and growth (similar to penicillins) Bactericidal Work best in rapidly dividing cells 2. Beta-lactams 3. Generations – First through Fourth 4. **1st Generation**: very active against GPC, E. coli, K. pneumoniae, Proteus OK but Pseudomonas not. Anaerobic cocci sensitive. Cephalexin, Cephazolin	1. Bold to pass 2. **Beta-lactams** 3. **4 Generations** 4. Concept of increasing activity against gram –ves and example of 2 classes

	Prompt: How does the spectrum of microbiological activity differ between the different generations?	**2nd Generation**: active against those by 1st generation but added GN coverage – Klebsiella, some anaerobe cover. NO Pseudomonas. Cefaclor, Cefuroxime **3rd Generation:** expanded GN coverage and cross BBB. Less active vs Staph. Effective against against B- lactamase producing Haemophilus and Neisseria. Ceftazadime works vs Pseudomonas. Ceftriaxone, Ceftazidine, Cefotaxime. **4th Generation:** more resistant to B- lactamases, extended coverage against enteric GNR, pseudomonas, enterobacteriaceae, S pneumonia, S aureus, Haemophilus and Neisseria. Cross BBB. Cefipime.	
Question 4 ANATOMY LOA: 1	*"Moving on, the patient has limitation of shoulder movement."* What muscles are called the "rotator cuff muscles?" Demonstrate or describe the origins and insertions of the rotator cuff muscles. Note that the model has no rotator cuff muscles. What are the actions of the rotator cuff muscles?	**Subscapularis** Origin – Medial 2/3 costal surface of scapula Insertion – fuses with capsule of shoulder joint and into lesser tuberosity of humerus Nerve – Upper and lower subscapular **Teres minor** Origin – Dorsal surface axillary border of scapula Insertion – Lower facet greater tuberosity humerus Nerve – Posterior branch axillary N **Supra spinatus** Origin – medial2/3 supraspinous fossa scapula Insertion – Upper part of greater tuberosity humerus Nerve – Suprascapular nerve C5,6 **Infraspinatus** Origin – Medial 2/3 infraspinous fossa and deep surface infraspinous fascia which covers muscle. Insertion – Central facet greater tuberosity humerus Nerve – Supra scapular **Supraspinatus** – **initiates abduction** and other muscles hold humeral head down **Subscapularis** – **medial rotation** of humerus **Infraspinatus** and **teres minor** –**lateral rotators** of humerus Supraspinatus – **abducts shoulder** All muscles **stabilise the shoulder joint** by bracing humeral head against glenoid (tendons fuse with capsule)	Must know all 4 to pass Must have knowledge about origins, insertions and actions of 2/4.

ACEM PRIMARY VIVA B **Thursday Afternoon Session 2** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 PATHOLOGY LOA: 1	*"A patient presents with chronic inflammatory arthritis."* 1. What are the characteristics of chronic inflammation? 2. Why does macrophage accumulation persist in chronic inflammation? 3. What are the causes of chronic inflammation? (prompt can you give an eg. of each)	• Inflammation for a **prolonged period** (week or more). • Characterised by **macrophages**, lymphocytes and plasma cells • With simultaneous-active inflammation/ **tissue destruction** and **attempts at repair** by connective tissue, fibrosis **Continued recruitment** of monocytes (continued expression of adhesion molecules and chemotactic factors) Local proliferation of macrophages Immobilisation of macrophages • **Persistent infection**- TB, syphilis • **Autoimmune**-RA, MS, IBD, SLE • **Prolonged exposure to an agent**: exogenous-silica->silicosis , FB, persistent trauma endogenous-lipid->atherosclerosis	¾ Bold to pass Bold 2/3 bold with examples
Question 2 PHYSIOLOGY LOA: 1	Question 2 - Physiology 1. List the physiological effects of glucocorticoids 2. What are the vascular effects of abruptly stopping long term glucocorticoids? Bonus: What is the benefit of elevated glucocorticoid levels in stress?	a) **Inc protein catabolism.** b) Inc hepatic glycogenolysis and gluconeogenesis, inc Glu-6-phosphatase → **inc plasma glucose** c) Antiinsulin effects on peripheral tissues d) Inhibit ACTH secretion e) **Controls vascular reactivity to NAd and Ad** f) Control ability to excrete water load g) Increased neutrophils/ plts/ RBC and dec eosinophils/ lymphocytes/ basophils Vascular smooth muscle becomes **unresponsive to NAd and Ad** Capillaries dilate and inc permeability Failure to respond to NAd **impairs vascular compensation** for hypovolaemia and promotes **vascular collapse** Effect on **vascular activity to catecholamines** plus necessary for catacholamines to mobilise **FFA** for emergency energy source	2 bold and 2 others Must have general concept
Question 3 PHARMACOLOGY LOA: 1	1. Moving on to pharmacology. What is the mechanism of action of the non steroidal anti – inflammatory drugs (NSAIDs)? 2. How does aspirin differ from other NSAIDs in its action on COX?	NSAIDs serve to suppress inflammation chiefly by **inhibiting prostaglandin synthesis.** In so doing they decrease the sensitivity of vessels to bradykinin and reverse the vasodilation of inflammation. Cyclo – oxygenase (COX) is the key catalyst for arachidonic acid conversion to prostaglandins. **NSAIDs inhibit COX,** thus inhibiting this conversion. **Aspirin** (original NSAID**) irreversibly inhibits COX**, whilst the **newer NSAIDS** (ibuprofen, diclofenac) **reversibly** inhibit COX.	**Pass criteria** Inhibit COX, thus decrease prostaglandin synthesis – and in so doing the response to inflammation is modulated. **Irreversible vs reversible**

	2. What are the adverse effects of NSAIDs?	2 types of COX exist – COX 1 is expressed in most cells, and COX 2 is inducible, its expression varies depending on stimulus. Selective COX 2 inhibitors (celecoxib) do not affect platelet function at usual doses, whilst the other NSAIDs do inhibit platelet aggregation. **GI EFFECTS** – GI irritation, ulcers, abdominal pain, N and V **BLEEDING** – secondary to platelet effects **RENAL** – nephrotoxicity, hyperkalaemia ALLERGY – rash, pruritis **CARDIOVASCULAR** – Selective COX 2 inhibitors - implicated in increased risk of c'vasc thrombotic events, - fluid retention, oedema, hypertension CNS – headaches, tinnitus, dizziness, stroke PULMONARY – asthma HAEM - rare – t'cytopaenia, neutropaenia HEPATIC – abnormal LFTs	**¾ Bold plus one other to pass** – namely – GI effects, bleeding, and renal effects...plus any one of the others
Question 4 ANATOMY LOA: 2	Moving on to anatomy a. This is a photograph of the gluteal region. Identify the labelled structures. Point to piriformis if not identified. b. Describe the actions of the gluteus maximus muscle. c. Describe the course of the sciatic nerve in the gluteal region and leg.	**23. Sciatic nerve** – tibial part 1 – common fibular part **2. gluteus maximus** 3. gluteus medius 4. gluteus minimus 5. greater trochanter of femur 6. inferior gamellus 7. inferior gluteal artery 8, 21, 22. inferior gluteal nerve 9. internal pudendal a 10. ischial tuberosity 11. nerve to obturator internus 13. obturator externus 14. obturator internus **15. piriformis** 16. posterior femoral cutaneous nerve 17. pudendal nerve 18. quadratus femoris 19. sacrotuberous ligament 20. superior gamellus **Straightens the leg at the hip** during walking, running, climbing. Assists in raising from a sitting position. Lower part acts as adductor and external rotator of lower limb. Tensor of the fascia lata and by its connection with the iliotibial band, steadies the femur on the tibia during standing when the extensor muscles are relaxed. Enters the gluteal region via the greater sciatic foramen **inferior to piriformis** and **deep to gluteus maximus**; descends in midline of the posterior thigh deep to biceps femoris; **bifurcates into tibial and common fibula nerves at apex of popliteal fossa.**	**All three bolded** to pass and at least two other named structures. **Bold to pass** **Two of three bolded to pass**

STEM: Following administration of anti-venom for a snakebite, a 60 yr old man is noted to be hypotensive. We will begin with Physiology....

TOPIC	QUESTIONS	KNOWLEDGE (essential in bold)	NOTES
Question 1 **PHYSIOLOGY** **Cardiac Output** LOA: 1 (Ganong 24th ed p545-552)	What is cardiac output?	**Output of the heart per unit time.** **HR x SV**	**Bold**
	What factors determine cardiac output?	SV is related to the **preload (degree of stretch prior to contraction) and afterload** (resistance to flow) of the heart and the **intrinsic contractility** of the myocardial cells. HR- Sympathetic vs parasympathetic stimulation.	
	What methods can be used to measure cardiac output?	**Direct Fick method or indicator (or thermal) dilution** Can also measure by **Doppler U/sound** techniques Fick principle; amount of substance taken up by organ per unit time = (A-V conc difference) x blood flow. In the heart can use O_2. LV output =O_2 consumption ml/min/[A_{O2}]-[V_{O2}] (both in ml/L) Indicator dilution; substance injected IV and serial sampling in arterial blood performed, log plotted and extrapolated to find circulation time (indicator must not be lost from circulation)	*2 to pass*
	What causes of decreased cardiac output could be causing this man's hypotension?	1) variation in heart rate due to induction of **arrhythmias or heart block** (too fast or too slow) 2) **Reduced preload** (venodilatation with reduced venous return due to anaphylaxis) 3) Increased afterload (not too likely in this case **4) Reduced contractility** (i.e. ischaemia, venoms, drugs)	

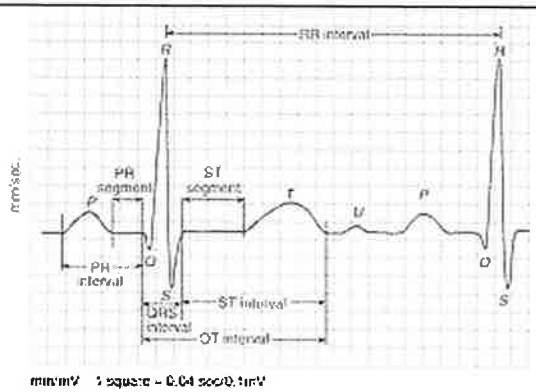
The patient develops airway obstruction and is going to be intubated. We are now moving to Pharmacology.			
Question 2 **PHARMACOLOGY** **PROPOFOL** LOA: 1 (Katzung 12th ed p 438-440)	1. Describe the pharmacokinetics of propofol.	**1. Distribution half life 2-4 minutes** **Elimination half life 4-23 minutes** **Rapid onset and recovery**. Termination of drug effect due to **redistribution** from brain to sk muscle and then fat (rather than metabolism). Duration of action 3-8min Rapidly metabolised in liver and extrahepatic sites (lungs). Water soluble metabolites excreted in urine.	**Bold**
	2. What is the usual induction dose of propofol?	**2. 1-2.5mg/kg adults,** 2.5-3.5mg/kg in kids	**Bold**
	3. What clinical effects are expected after this dose of propofol is administered.	**3. Anaesthesia / Sedation**. Respiratory depression. Transient **apnoea**. **Decreased blood pressure** through vaso and venodilation (most pronounced of induction drugs). Does NOT have analgesic properties Anti-emesis, Metabolic acidosis, Pain at injection site	**Bold**
	4. List some drug interactions of propofol important in the setting of sedation/anaesthesia	**4.** Opioids – enhance respiratory depression Benzodiazepines - enhanced sedation and respiratory depression	**1 of 2**

We are now moving to Anatomy			
Question 3 **ANATOMY** **Model – Larynx (Full model with tongue in situ)** LOA: 1 Model - Tongue & Airway (Somso upper airway models)	1. a) Identify the structures in the upper airway that could lead to airway obstruction b) What other structures are visible 2. What are the bony and cartilaginous components of the larynx 3. What is the innervation of the larynx?	**1.** **Tongue** Tonsils, **pharynx** **Epiglottis,** glottis **Hyoid** bone, floor of mouth - **mylohyoid (Prompt)** **Mandible** Buccal muscles, cheek. Medial pterygoid muscles ary-epiglottic folds & vallecula, Piriform fossa **2. Cartilage:** **Cricoid, Thyroid,** Arytenoids **and Epiglottis** Bone: Hyoid **3. Motor:** **Recurrent laryngeal N** (inferior laryngeal-terminal branch of Rec Laryngeal) except for Cricothyroid which is External Laryngeal N (tenses cords). Both from CN X **Sensory:** **Above cords: Internal Laryngeal N** (branch of superior laryngeal N) **Below cords: Recurrent Laryngeal N** (Inferior laryngeal branch) (Br of CrN X)	**bold 5/6 total** **2 underlined** **All bold** **Bold**

One week later he develops serum sickness. We are now moving to Pathology			
Question 4 **PATHOLOGY** **Type 3 Hypersensitivity** LOA: 1 (Robbins pp 204-205)	1. What is the pathogenesis of serum sickness? Prompt (if required): How is the tissue damage caused?	**1. Type 3 hypersensitivity** Phase 1: **Formation** of **Immune complexes.** Protein Ag, 1/52 -> Ab -> blood -> Ag-Ab complexes Phase 2: **Deposition** of immune complexes. Medium size, Ag excess most pathogenic High pressure filtration , glomeruli, joints Phase 3: **Tissue injury** caused by immune complexes **Acute inflam reaction** ~ day 10 IgG & IgM (C' fixing Ab) bind to leukocyte Fc receptors. Leuk recruitment and activation - release proteases/lysozymal enzymes ->damage. Deposition, activation and Consumption of C' and decreased C3 levels -> inflam reaction and tissue damage	Bold 3 Phases
	2. What are some clinical features?	2. Fever, urticaria, arthralgia, LN enlargement, proteinuria	3 of 5
	3. What are some other examples of Type III hypersensitivity?	3. Acute: post strep G-N, reactive arthritis, Arthus reaction Chronic: SLE, PAN, other vasculitides, possibly membranous G-N,	3 examples

ACEM PRIMARY VIVA C **Thursday Afternoon Session 2** ***Candidate Number:*** **AGREED MARK:**

Stem: A 65 yr old man presents with an inferior myocardial infarction
We are starting with Physiology

TOPIC	QUESTIONS	*KNOWLEDGE* ***(essential in bold)***	NOTES
Question 1 **PHYSIOLOGY** **ECG including MI changes** LOA: 1 Ganong 24th ed pp 524-529, 534-537	1. Draw and describe an ECG tracing of a single normal heart beat Prompt: What produces the waves and segments?	 **P wave- atrial depolarization, PR AV conduction** **QRS- ventricular depolarization, ST- plateau of Vent depolarization, (QT- Ventricular Action potential),** **T wave- Vent repolarization**	Bold 5/6
	2. What features would appear different in this patient's ECG?	**2. ST segment elevation in inferior leads** **ST segment depression in the reciprocal leads**	both
	3. At the myocardial cell membrane level, what causes these changes?	3. Abnormally rapid depolarisation in early phase (accelerated opening of K+ channels) Decreased resting membrane potential (due to loss of intracellular K+) Slowed depolarization of affected cells (cf normal cells)	1 of 3 to pass

We are now moving to Pharmacology			
Question 2 **PHARMACOLOGY GTN** LOA: 1 Katzung 12th ed Chapter 12) MoA, principles of tachyphylaxis	1. By what routes can GTN be administered?	1. **Sublingual, transdermal, IV, oral,** buccal, inhaled	Bold 3/4
	2. Why are parenteral routes favoured?	2. To avoid the **hepatic first pass** effect which significantly decreases bio-availability	bold
	3. What is meant by the term tachyphylaxis as it relates to Glyceryl Trinitrate (**GTN**)	3. Continuous exposure to nitrates – smooth muscle may develop tolerance. Particularly seen with continuous IV infusion or long acting preparations. (oral, transdermal)	Understand concept
	What is the implication of this for the dosing and administration of GTN	Concept of "drug-free" interval – at least 8h between doses	concept
	What is the theoretical basis for this phenomenon? (bonus)	(a) Diminished release of nitric oxide resulting from reduced bioactivation secondary to depletion of tissue thiol compounds, decreased tissue sulphydryl groups, increased generation of O2 free radicals , decreased availability of CGRP. (b) Systemic compensation – after > 1 day of therapy salt and water retention reverse favourable hemodynamic change	for better candidates
	4. When should GTN be used with caution?	4. **hypotension**, those on sildenafil, inferior&posterior MI/RV infarct, Fixed cardiac output (AS, tamponade etc), raised ICP, significant tachy/brady cardia, allergy	Bold +2

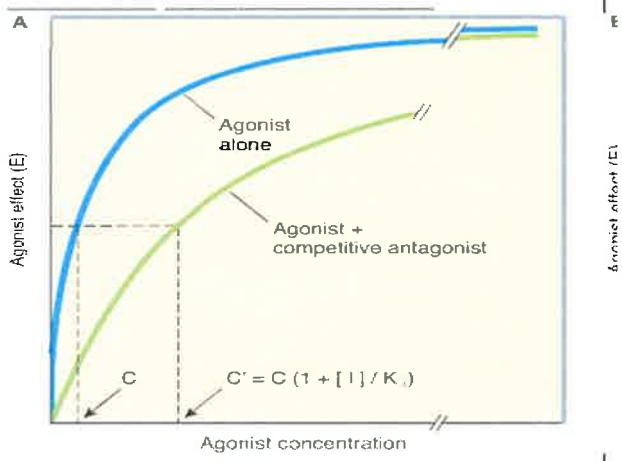
We are now changing to Anatomy			
Question 3 **ANATOMY** LOA: 1 Heart model assembled	1. Identify the arterial supply of the heart	1. L+R coronary arise from corresponding aortic sinuses above AV **R coronary** courses inf in AV groove. Gives off branches to SA node, Marginal, Post interventric, and AV nodal **L coronary** bifurcates into **Circumflex and LAD** (anterior I – V art), then Cx gives off Marginal branch, and LAD gives off diagonals.	**bold** to pass
	2. What does the R Coronary artery supply?	2. R atrium, most of RV, Diaphragmatic surface LV Post 1/3 septum, 60% SA, 80% AV	3 out of 6 to pass
(Take the model back!)	3. Describe the venous drainage of the heart	3. Major drainage is via the **Coronary sinus** 3 main tributaries are: Great cardiac vein (accompanies LAD, then Cx) Middle (accompanies PIV) Small cardiac veins (accompanies R marginal). Oblique vein L atrium marks start of sinus. Ant cardiac vn's start ant surface RV, drain straight into R atrium Smallest cardiac vn's (venae cordis minimae) drain direct into chambers	bold +2
	4. Describe the major components of the conducting system	4. SA Node junction of SVC & RA AV Node near coronary sinus-postero-inferior interatrial septum AV Bundle R & L Bundles	3 of 4

We are now changing to Pathology			
Question 4 **PATHOLOGY** **Healing post MI** LOA: 1 Robbins pp 551-553, 102-106	1. What are the consequences and complications of a myocardial infarction	1. Contractile dysfunction/CCF, Arrhythmias, Myocardial rupture, Pericarditis, R vent infarction & RHF, infarct extension, Infarct expansion, Mural thrombus (=>embolism), Ventricular aneurysm, Papillary muscle dysfunction, Progressive late HF, Remodelling, death	6
	2. What are the main cardiac rupture syndromes	2. Free wall -> tamponade (most common of 3 occurs at 1-10 days) Septum -> VSD and L->R shunt Papillary muscle dysfunction -> severe Mitral Regurg	1 of 3
	3. What changes occur in ventricular remodelling	**3. Hypertrophy** and **dilatation**, increased oxygen demand -> **ischaemia** & **depressed cardiac function**, **scar formation** -> **stiffening** and hypertrophy.	3
	4. What systemic factors affect infarct healing?	4. Nutritional: **protein, Vit C** Metabolic: **diabetes** Circulatory: **arterial** or **venous** Hormonal: **glucocorticoids**	3

ACEM PRIMARY VIVA D Thursday Morning Session 1 *Candidate Number:* AGREED MARK:

Stem: A 30 year old man has had a motor vehicle accident after a heroin overdose, and has been given Naloxone. Commencing with Pharmacology:

TOPIC	QUESTIONS	KNOWLEDGE	NOTES
PHARMACOLOGY Question 1 LOA: 1	1. What is an antagonist?	1. Receptor antagonists **bind to receptors** but do **not activate** them. The primary action of antagonists is to **prevent agonists from activating receptors.**	Bold to pass
	2. What is the difference between a competitive and non-competitive antagonist?	2. Competitive antagonist: In the presence of **increasing concentration of antagonist, higher concentrations of agonist will produce a given effect.** Eg propanolol and noradrenaline / adrenaline. Irreversible or non competitive antagonist Bind via covalent bonds or **just binding so tightly to receptor so receptor unavailable** for agonist. Duration of action of antagonist depend on rate of turnover of receptor-antagonist molecules.	
	What type of antagonist is naloxone?	Competitive	
	3. What effect does a competitive antagonist have on the concentration-effect curve?	3. Shift agonist vs effect **curve to right. Higher concentrations of agonist can overcome competitive antagonist**	



Stem: Moving on to Anatomy: On examination, the patient is very tender over his pelvis.			
ANATOMY Question 2 LOA: 1	1. Demonstrate the major anatomical features of the pelvis. 2. What is this? (AIIS)What attaches here? What is this? (ASIS) What attaches here? 3. Describe the course of the iliac arteries.	Pubic symphysis, inferior and superior pubic rami, obturator foramen, acetabulum, iliac crest, sacro-iliac joint, sacrum. AIIS- Rectus femoris ASIS- Sartorius **Common Iliac origin from aorta L3-5** Follows medial border of Psoas to pelvic brim Divides at level of L5S1 **Internal Iliac artery** enters pelvis **External Iliac artery** follows Iliopsoas ends at the inguinal ligament and becomes **femoral artery** at **mid-inguinal point**	6 major features to pass 3/4 to pass Bold to pass

Stem: Moving on to Pathology: The patient becomes hypotensive.

PATHOLOGY Question 3 LOA: 1	1. What is hypovolaemic shock?	1. **Systemic hypoperfusion due to reduced effective circulating blood volume** resulting in **impaired tissue perfusion** and cellular hypoxia	Bold to pass
	2. Describe the stages of hypovolaemic shock Prompt: What compensatory mechanisms are involved?	2. A. **Non- Progressive phase** – reflex **compensatory mechanisms** activated to **maintain vital organ perfusion.** Variety of neurohumoral mechanisms activated to help maintain cardiac output and blood pressure (baroreceptors reflexes, release of catecholamines, activation of renin-angiotensin axis, ADH release and increased sympathetic output resulting in: tachycardia, peripheral vasoconstriction, and renal conservation of fluid with decreased urine output. Coronary and cerebral vessels less sensitive to sympathetic response and blood flow/ O_2 delivery spared.	All 3 phases to pass. 2A. Bold to pass + 3 features (prompt if necessary)
		B. **Progressive phase- tissue hypoperfusion** and worsening circulatory and metabolic imbalance including **acidosis.** Widespread tissue hypoxia resulting in anaerobic glycolysis with excess lactic acidosis production blunts vasomotor response → peripheral pooling, hypoxic injury, DIC, vital organs begin to failure	2B Bold to pass.
		C. **Irreversible phase** - after body has incurred **cellular and tissue injury so severe** that even if haemodynamic defects are corrected, **survival is not possible**	2C Bold to pass
	3. What happens at the cellular and tissue level during the irreversible phase?	- Widespread cell injury - lysosomal enzyme release - nitric oxide → decreased myocardial contractility - acute tubular necrosis -> acute renal failure, - ischaemic gut→ bacteraemic shock - severe hypotension, unconscious, anuric - pre-cardiac arrest - > death	3 features to pass

Stem: Moving on to Physiology: The patient complains of shoulder tip pain that is thought to be referred from his abdomen.			
PHYSIOLOGY Question 4 LOA: 2	1. Define the term 'referred pain" 2. From which structure is pain referred to the shoulder? 3. Explain this relationship 4. Can you give another example of referred pain? 5. (EXTRA if good candidate) What is the physiological basis/theory for referred pain	1. **Irritation of a visceral organ causing pain in a distant somatic structure** 2. **Diaphragm** 3. Dermatome rule. Referred pain is usually to a structure that **developed from the same embryonic segment or dermatome** as the structure from which the pain originates 4. Cardiac pain to arm. Ureteric pain to testicle. 5. Convergence-Projection Theory. Somatic and visceral pain fibres converge on the same second-order neurons in dorsal horn that then go on to thalamus and sensory cortex via common path. Sensory cortex cannot determine whether the stimulus came from viscera or are of referral	Bold to pass Bold to pass Bold to pass 1 to Pass

ACEM PRIMARY VIVA D **Thursday Afternoon Session 2** ***Candidate Number:*** **AGREED MARK:**

Stem: A 60 yr old woman presents with severe jaw pain following a dental extraction a month earlier and is given IV morphine. Commencing with Pharmacology:

TOPIC	QUESTIONS	KNOWLEDGE	NOTES
PHARMACOLOGY Question 1 LOA: 1	1. Define drug elimination half life Prompt: Is there a formula you can use?	**Time required to change the amount of drug in the body by ½ during elimination** T1/2 = 0.7 x Vd/clearance (0.7 approx log 2) 50% after 1, >90% after 4	Bold to pass
	2. How does knowledge of a drug's half life help us clinically?	Dosing regimens Decay afterdose/overdose Time to steady state after dose change	2 to pass
	3. What disease states cab affect elimination half-life?	Liver, renal, cardiac disease	one organ
	4. What disease state could affect the elimination half-life of morphine?	Liver, renal	one organ
Stem: Moving on to Anatomy: You are planning to do a nerve block to relieve her pain.			
ANATOMY Question 2 LOA: 2	1. Which Nerves run on or within the bony mandible	**Inf alveolar**/mental N (V3- mandibular) **Lingual N ((**V3+ chorda tympani) **+ 1 other of** Auricultemporal N (V3) N to mylohyoid (V3- branch of inf alveolar) Mandibular branch of the facial N (VII)	Bold to pass
	2. Show the course of the inferior alveolar N on this mandible and why it is prone to injury?	Early large branch of mandibular trigeminal after it exits the Foramen ovale, runs on surface of inside mandible ramus to **Mandibular foramen,** (gives off N to mylohyoid), Passes inf and ant thru bone in alveolar canal which is v close to **roots of 3rd molar** supplying all lower teeth and exits as **(mental N)** from **mental foramen** ant/ superior (in edentulous)	
	3. Why is it prone to injury?	Close relationship to bony mandible	
	4. If the lingual N is damaged what deficits would you expect?	**Ant 2/3 tongue- taste + sensory loss** (via the chorda tympani) Loss of secretory function –submandibular salivary glands Sensory loss to floor of mouth and/ or to gums	

Stem: Moving on to Pathology: Her Xray reveals evidence of bony destruction in the mandible.			
PATHOLOGY Question 3 LOA: 1	1. Describe the pathogenesis of osteomyelitis. Prompt: How would this patient have suffered a bony infection of his jaw?	**Local infection** related to extraction of tooth **Blood borne** **Spread from neigbouring** gingival source.	2/3
	2. What organisms cause osteomyelitis?	**Staph Aureus majority** >80% pyogenic E Coli, Kl Pneum, Pseudo A, from GU tract or IVDU H Infl and GBS in neonates Viruses, Fungi, Parasites, TB, syphilis also About 50% no orgs found.	Staph A and 1 other
	3. What changes occur in the bone?	**Acute inflammation and necrosis**, abscess formation Sclerosis and **involucrum** formation Deformity and sequestrum formation, Draining sinus Characteristic lytic focus surrounded by zone of necrosis on X ray, lifting of periosteum 5-25% become chronic inflammation	Bold
	4. What are the clinical consequences of osteomyelitis?	Resolution after Rx with IV antibiotics and drainage Conversion to chronic O myelitis Deformity and bony destruction Severe sepsis syndrome, ARF etc.	2 to pass

Stem: Moving on to Physiology:

PHYSIOLOGY Question 4 LOA: 1	1. What percentage of cardiac output goes to the kidneys?	RBF = 1.2-1.3L/min or approx 25% CO (adult)	**RBF**
	2. How is renal blood flow regulated? Prompts: What other mechanisms are there?	**<u>Substances/Chemicals</u>** Norepeinephrine (noradrenaline) • Constricts renal vessels • Stimulates renal nerves to ↑ rennin secretion Dopamine – renal v/dilatation A II – arteriolar constrictor PG - ↑cortex flow, ↓medulla flow Ach – v/dilatation High protein - ↑ b/flow **<u>Renal Nerves</u>** • Stimulation nerves = ↑renin secretion , ↑JG sensitivity, ↑Na resorption, and renal vasoconstriction • Strong stimulation sympathetic (noradr) ↓flow • Fall in BP = v/constrict **<u>Autoregulation</u>** • Renal vasc resistance varies with pressure to keep RBF fairly constant • Present in denervated kidney, but not if drugs that paralyse vasc sm muscle • Factors = direct contractile response, NO, A II	**3**/6 substances **plus** nerve or auto – with example
	3. How can renal blood flow be measured?	• Fick principle – subs taken up/unit time • PAH used to measure renal plasma flow • Renal b/flow using plasma flow and Hct	**One** example
	4. Describe the differences in regional blood flow within the kidney.	- AV o2 difference for kidney = 14ml - Cortical b/flow = 5mL/g/min • Little o2 consumption - Medulla b/flow low (outer = 2.5ml, inner= 0.6ml) • Maintence of osmotic gradient	One aspect of regional blood flow to pass

ACEM PRIMARY VIVA A **Thursday Morning Session 1** ***Candidate Number:*** **AGREED MARK:**

Stem: A 40 yo man presents with extensive burns to the lower half of his body. A CVC is inserted. **We are starting with Anatomy**			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Photo – Anterior Triangle of Neck (McMinn's p39) **Subject:** Anat LOA: 1	What are the boundaries of the anterior triangle of the neck? SCM has been removed in this photo. Where is the internal jugular vein? Describe its course. What major structures are at risk during insertion of an IJ line.	**SCM, midline, mandible** 22 Internal jugular vein IJV – continuation of sigmoid sinus Contained in **carotid sheath**. Lies lateral and ventral to artery. Goes **deep to SCM** and 2 heads of SCM – sternal and clavicular heads **Joins subclavian vein** posterior to **sternal end of clavicle**. Forms **brachiocephalic vein.** external carotid artery (11) common carotid artery (8) vagus (63) , other nerves, lung, trachea, scm, thyroid, thoracic duct	All 3 to pass Need to identify Concept. 4/5 Bold to pass. 2 to pass
Stem: We are now moving to Pathology			
Question 2 Thermal Injury (Robbins pp 421-422) **Subject:** Path LOA: 1	How are thermal burns classified? What are the potential complications of thermal burns? How do you determine the extent of burns?	According to depth of injury: • **Superficial** – confined to epidermis • **Partial thickness** – extends to dermis • **Full thickness** – involves subcutaneous tissue **Early:** • Hypovolaemic shock (especially with >20% BSA) • Compartment syndrome (circumferential LL burn) • Associated injuries (eg inhalational burn, CO poisoning) • Airway compromise • Hypermetabolic state **Late:** • Infection / sepsis (Pseudomonas) • ARDS • Multi organ failure • Skin grafting, scarring / cosmetic • Psychological TBSA calculation notoriously inaccurate. Does not include superficial burns • Wallace "rule of nines"/Lund & Browder diagram	Bold required 2 early and 2 late Mention 1 method

Stem: We are now moving on to Physiology			
Question 3 Venous Pressure and flow (Ganong 24th ed pp 582-584) **Subject:** Phys LOA: 1	1. Describe the mechanisms of venous return to the heart	a) **Thoracic pump:** inspiration resulting in negative pressure in the thorax and positive pressure in the abdomen. Blood flow towards the heart because of venous valves b) Effect of heart beat: during systole, AV valves are pulled downward → increase the capacity of the atria c) Muscle pump: contraction of muscles around the veins in the limbs during activity d) Differential resistance: resistance of the large veins near the heart is less than peripheral veins	Thoracic pump plus one other
	2. What factors might effect the CVP of this patient?	a) **Decrease CVP**: Fluid loss; blood loss b) **Increase CVP**: Excessive fluid replacement; other pre-existing conditions eg CCF; positive pressure ventilation; increased thoracic pressures	1 example from each bold category
	3. What is the value of mean CVP in normal individuals	4.6-5.8 mmHg or 6-8 cm H2O	Reasonable value
Stem: We are now moving to Pharmacology. He is resuscitated with Hartmann's solution			
Question 4 Compound Sodium Lactate (MIMs & product information) Constitution, Indications, Adverse effects. Comparison to other crystalloids and colloids **Subject:** Pharm LOA: 1	(a) How does Hartmann's solution differ from normal saline?	Addition of **Sodium Lactate, Potassium** Chloride**, Calcium** Chloride (+pH adjustment) Na 131, K 5, Cl 112, Ca 2, Lactate/Bicarb 28 mmol Compare Normal Saline Na 150 Cl 150)	Bold
	(b) What are the potential advantages of Hartmann's solution in resuscitation?	Closer to physiologic – potassium, calcium **Less Hyperchloraemia** **Effective bicarbonate – some (slow) good effect on acidosis** (proof of superiority lacking)	Bold
	(c) What are the potential complications of IV fluid therapy?	overload/under resuscitation, hypothermia, extravasation, acidosis, electrolyte abnormalities, osmo changes, air embolism, infection, cerebral oedema, haemodilution	Bonus

ACEM PRIMARY VIVA A **Thursday Afternoon Session 2** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: An elderly lady presents with acute abdominal pain. **We are starting with Anatomy.**			
Question 1 Photo of Abdominal wall (fig 258A) **Subject:** Anat LOA: 2	1 What structures in this photograph are potential sources of acute abdominal pain? 2 Identify the (other) vascular structures in this photograph (*if not already*) 3 Describe the arterial supply and venous drainage of the gut	**Aorta** (aneurysm), **Coeliac axis and SMA** (mesenteric ischaemic), **kidneys and ureters** (stones/infarcts), Splenic artery (aneurysm/dissection), Lymph nodes (adenitis/pressure), psoas (abscess or bleed) Landmarks and levels**: IVC, left renal vein, right renal vein. Aorta, Coeliac axis** (T12), **superior mesenteric artery** (L1). **Foregut (+hepatobiliary & spleen) - Coeliac axis**: common hepatic (->cystic, hepatic, right gastric, gastro-duodenal), splenic, left gastric (not shown); **Midgut (duodenum to transverse colon)-SMA**: inferior pancreaticoduodenal, jejunal/ileal branches, ileocolic, right and middle colic. **Hindgut-IMA** (small calibre + collaterals, therefore rarely blocked). Venous drainage – superior mesenteric vein (joins splenic vein to form portal vein), inferior mesenteric vein	Bold 4/6 bold Bold
Stem: We are now moving on to Pathology. She has ischaemic bowel.			
Question 2 Thrombosis **Subject:** Path LOA: 1	1. What factors predispose to thrombus formation in a vessel? 2. How are hypercoaguable states categorised? What are some examples of each type? 3. What are the possible outcomes for a vessel thrombus?	Virchows triad. **Endothelial injury; Alteration in blood flow** (stasis or turbulence); **Hypercoaguability** of blood **Primary (Genetic)** • Mutations - Factor V Leiden, Prothrombin • Increased levels - factors VIII, IX, XI, fibrinogen • Deficiencies - AT3, Protein C, S • Fibrinolysis defects, homozygous homocystinuria **Secondary (Acquired)** • Prolonged bed rest, immobilisation, MI, AF, Tissue injury (surgery, #, burn), cancer, prosthetic valves,, DIC, HITS, Anti phospholipid antibody syndrome • Cardiomyopathy, nephrotic syndrome, hyperoestrogenic states (pregnancy, post partum), OCP, sickle cell anemia, smoking • Note: often multifactorial **Propagation** (e.g. resulting occlusion); **Embolization; Dissolution; Organisation and recanalization** (e.g. to variable degree)	3/3 bold 2 categories plus Primary - 2 examples Secondary – 3 examples 2/4 categories

Stem: We are now moving to physiology. Arterial blood gases show a metabolic acidosis			
Question 3 Renal role in the handling of H+ ions **Subject:** Phys LOA: 1	1. Describe how the kidney responds to metabolic acidosis	**Renal tubule cells secrete H^+** into tubular fluid **in exchange for Na^+** **HCO_3^- is actively reabsorbed** into the peritubular capillary (for each H^+ secreted, $1Na^+$ and 1 HCO_3- are added into blood).	Bold
	2. What substances act as urinary buffers for the excretion of H^+	**NH_3** forms NH_4^+; **$HC0_3$-**forms CO_2 and H_20; **HPO_4^{2-}** forms H_2PO_4	2 of 3
	3. How else can the body compensate for a metabolic acidosis? Prompt: What other major system is involved in acidosis compensation?	The **respiratory system** responds by **increasing ventilation** which results in a decrease in PCO2 which causes increase in pH (this is a rapid response)	Bold to pass

Stem: We are now moving to Pharmacology. Her medications include captopril			
Question 4 ACE inhibitors **Subject:** Pharm LOA: 2	What is the mechanism of action of captopril?	**Angiotensin converting enzyme** (kininase II) **inhibitor**: inhibits hydrolysis of A1 to A2. Hence, **inhibits A2** effects (potent vasoconstrictor and increases Aldosterone secretion – salt and H2O retention) and decreases PVR, BP. Also, **inhibits bradykinin inactivation** to cause vasodilatation and decreased PVR, BP.	Bold to pass
	What are the adverse effects of captopril?	**Hypotension**, 1st dose esp. if hypovolaemic, diuretics, NaCl restriction, GI loss **ARF** esp. with bilateral RAS **HyperK+** esp. if renal insuff, DM **Cough, angioedema** (bradykinin, substance P), wheeze Fetal abnormalities (hypotension, anuria, renal failure – 2nd/3rd trim, increased teratogenesis – 1st trim) Altered taste, allergic skin rash, drug fever (10%)	3 of Bold to pass
	What drugs interact with captopril?	**K+ supplements, K+ sparing diuretics** – increase hyperK+ NSAIDs – impair BP reduction (block bradykinin) Other antihypertensives; haemaccel	Bold to pass

ACEM PRIMARY VIVA A **Friday Morning Session 3** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: An 80 year old woman is transferred to your ED following a motor vehicle accident 12 hours ago, where she sustained serious chest injuries. **We are starting with Anatomy.**			
Question 1 Chest X-ray **Subject:** Anat LOA: 2	1. Outline the structures that make up the cardiomediastinal borders on this normal X-Ray	Right: **R Brachiocephalic v, SVC, R Pulmonary Artery, R Atrium, IVC** Left: **Aorta, L Pulmonary Trunk/Artery, L Atrium, L Ventricle**	6 Bold to pass
	2. Which parts of the lungs lie adjacent to the cardiomediastinum?	Right upper mediastinum: **R superior lobe** Right heart border: **R middle lobe** Left upper mediastinum: **L superior lobe** Left heart border: **Lingula segment of L superior lobe**	RML plus one other
	3. In this patient, what injuries may be seen on a CXR? (Prompt: the patient has sustained blunt trauma)	**Chest wall:** # ribs, clavicle, sternum **Lung:** pneumothorax, haemothorax, contusion, **Cardiovascular**: aorta, other vessels (widen mediastinum)	1 example from each bold category to pass
Stem: We are moving to Pathology. She has multiple wounds oozing blood due to DIC			
Question 2 DIC **Subject:** Path LOA: 2	1. On a full blood count and coagulation profile, what would you expect to find?	↓Hb (MAHA – microangiopathic haemolytic anaemia), ↑WCC, **platelets↓**, Fibrinogen↓, PT/INR↑, a/PTT↑ and **fibrin degradation products↑**	Bold to pass
	2. What are the pathological consequences of DIC?	DIC – major trauma releases tissue thromboplastins. Both sides of clotting cascade are activated. 2 major consequences – deposition of fibrin within microcirculation leading to **ischaemia/micro thrombosis** of vulnerable organs; and a **consumptive coagulopathy** - platelets and clotting factors leading to a **bleeding diathesis**.	Bold to pass 3/3
	3. What are the causes of DIC?	**Obstetric** – FDIU, amniotic fluid embolism, preeclampsia, **Sepsis** **Malignancy** – acute promyelocytic leukaemia, adenoca of lung, pancreas, stomach and colon **Trauma**- multi/burns/environmental/snakebite	Must get 3 categories

Stem: We are moving to Physiology. She is shocked			
Question 3 Circulatory Catecholamines **Subject:** Phys LOA: 1	1. Name the endogenous catecholamines? Where are they produced? (prompt to match catechol with source) 2. What are the physiological effects of adrenaline and noradrenaline?	**Adrenal Medulla: Adrenaline, Noradrenaline**, Dopamine. Intrinsic Cardiac Adrenergic Cells: Adrenaline. Sympathetic Nervous System Cells: Dopamine **Metabolic**- Glycogenolysis, increased metabolic rate, mobilisation of free fatty acids, increased lactic acid Cardiovascular- **vasoconstriction and dilation, increase heart rate and strength** α1:Constriction of blood vessels, smooth muscles (esp norad) α2:Mixed smooth muscle effects (esp adren) β1:Cardiac ionotropy and chronotropy, irritability (both) β2:Dilation blood vessels liver & muscle, other smooth muscle relaxation (adrenaline) β3: Lipolysis, detrusor relaxation (esp adren)	Bold One metabolic and bold cardiovascular Extra info only

Stem: We are now moving to pharmacology. You decide to use Bupivicaine as the local anaesthetic to insert a chest tube			
Question 4 Bupivicaine **Subject:** Pharm LOA: 1	1. What is the mechanism of action of bupivacaine? 2. How long will a bupivacaine block last? 3. What are the potential adverse effects from bupivacaine? 4. How can the risk of these effects be minimised in the ED?	1. **Blocks** voltage-gated **sodium channels in nerve.** Threshold for excitation increases, conduction slows, AP rise declines, AP generation abolished. If Na current blocked over length of nerve, propagation is ceased. 2. **3-6 hours** 3. **CNS toxicity** (sedation/light headedness/visual&auditory/tongue&mouth numbness/metallic taste/nystagmus/restlessness/ muscle twitches/seizure/resp depression), **Cardiac toxicity** (arrhythmias/cardiovascular collapse/cardiac arrest), Local toxicity (trauma/neurotoxicity) Allergy 4. Ask re Hx of allergy, Use safe max dose (<2mg/kg), withdraw pre injection, avoid vessels-anatomical consideration (above rib below) & use USS. Ask pt to flag Sx e.g. taste/tongue numb. Avoid hypoxia/acidosis.	Bold Approximate or long duration Bold Extra

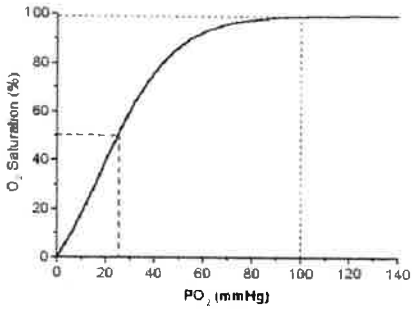
ACEM PRIMARY VIVA B **Thursday Morning Session 1** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: A 40 yo man presents to ED with renal colic			
The first questions is in regard to pathology			
Question 1 Urolithiasis (Robbins pp 962-963) **Subject:** Path LOA: 1	1.What are the main types of renal calculi? *Prompt: What are the common constituents of renal calculi?* 2.What conditions in urine favour stone formation? 3. What are the complications of ureteric calculi?	**1.Calcium** oxalate and phosphate (70%); 2. Struvite or triple (magnesium ammonium phosphate) (15-20%); 3. Uric acid (5-10%); 4. Cystine (1-2%) **2.** Increased concentration of stone constituents; changes in urinary pH; decreased urine volume; bacteria 3. pain, haematuria, **infection, obstructive renal impairment**	**1.Calcium + 1 other to pass** **2. 2 to pass** **3. 1 bold and 1 other.**
Stem: Moving now to your physiology question. The patient is noted to have a low eGFR.			
Question 2 GFR including hydrostatic and osmotic pressure.(Ganong 24th ed pp 678-680) **Subject:** Phys LOA: 1	1.What is normal Glomerular Filtration Rate (GFR) 2. What factors control GFR? Prompt: What agents, mediators & clinical factors affect GFR?	**125ml/min** in normal adult 180L/24h/10% lower in women **Hydrostatic Press/Osmotic press gradient, Size & permeability** of capillary bed (mesangial cell contraction/relaxation & loss of renal tissue) K in Starling Forces=GF coefficient=mesangial cell Increase –ANP Dopamine PGE2 cAMP Decrease – Endothelins, AGII, vasopressin, norepinephrine, PAF,PGF2, leukotrienes Ca/D4, histamine TxA2 Clinical: Systemic BP/Parenchymal odema/Ureteric obstruction/after-efferent arteriolar constriction/plasma proteins	Approx value 2/4 bold Role of mesangial cells Vaso active Agents - 2 Clinical examples - 2
Stem: Moving now to your pharmacology question. You decide to give this patient morphine for analgesia.			
Question 3 Morphine (Katzung 12th edition pp543-556) – pharmacokinetics; pharmacodynamics – in particular, receptors bound to; adverse reactions **Subject:** Pharm LOA: 1	1. What is its mechanism of action? 2. How is morphine metabolised and excreted? 3. What are the possible acute adverse reactions with morphine? Prompt: why are we more cautious in using morphine in renal failure patients?	**1.**Brain and Spinal cord **receptors: mu, delta, kappa.** (Subtypes: 2 mu and delta, 3 kappa). Binding to receptor (particularly mu) >> **reduction of neurotransmitter release from presynaptic nerve terminals** (especially glutamate), and **inhibit postsynaptic neurons** (by opening K channels).Central thalamic action and activation of descending inhibitory pain neurons. **2.** Mostly liver conjugated to morphine-3-glucuronide which has neuroexcitatory properties. 10% is metabolised to morphine-6-glucuronide with 4-6x increased analgesic potency. Excreted renally. **3. Sedation/ resp depression, nausea and vomiting**, hypotension if predisposed, histamine release, dysphoria, biliary colic, pruritis, allergy. In renal failure it can cause seizures, or prolonged analgesia.	Must name mu and 1 other types of receptors, and the 2 bold actions. Liver metabolism & metabolites are renally excreted Bold and 2 more.
Stem: Moving now to your anatomy question. Where would you look for a stone causing this man's pain on this Xray?			
Question 4 AXR- abdomen (outlining ureters) **Subject:** Anat LOA: 2	1.Course of ureter 2. Where is a stone likely to lodge? 3. Where would a staghorn calculus form? If have time – name other structures on XR	**1. Hilum**(~L2/**Tips of Trans Ps** of lumbar vert/**pelvic brim at SI joint or there abouts**(bifurc of Common iliac art.)/Lat wall of pelvis toward ischial spine then medially to **base of bladder** 2. Narrowings of ureter: PUJ; Pelvic brim; VUJ 3. Hilum: Pelvis and calyces	4 Bold 2 of 3

ACEM PRIMARY VIVA B **Thursday Afternoon Session 2** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE (**essential in bold**)	NOTES
Stem: A 3 year old boy presents to ED with measles.			
The first questions is in regard to pathology			
Question 1 Measles **Subject:** Path LOA: 2	1.What organism is responsible for measles infections and how is it transmitted? 2.What type of immune response occurs in measles? 3.What are the clinical features of measles? 4. What are the complications of measles?	1.**Virus,** RNA, Paramyxo >>**respiratory transmission** 2.T cell mediated controls infection and causes rash Antibody mediated protects against reinfection 3. **fever, rash, conjunctivitis, cough/coryza**, Koplik spots, lymph nodes. 4. pneumonia, secondary bacterial infection, delayed – encephalitis, SSPE	Bold to pass Antibody mediated 3 bold to pass 2 as minimum.
Stem: Moving now to your physiology question: He is noted to be hypoxic			
Question 2 Hypoxia **Subject:** Phys LOA: 1	1.Describe the different types of tissue hypoxia. Prompt: Hypoxia is deficiency of O2 at the tissue level 2.Describe the respiratory mechanisms leading to hypoxaemia and give examples? 3. Describe the clinical effects of acute hypoxia	1.**Hypoxaemia (hypoxic hypoxia)** – arterial PO2 reduced 2.**Anaemic hypoxia** – arterial PO2 normal but Hb reduced 3.**Ischaemic/ stagnant hypoxia** – blood flow & O2 delivery decreased 4.**Histotoxic hypoxia** → because of toxin cells cannot use it **Reduced ventilation** (asthma), **VQ mismatch (PE)**. Shunt (CHD), diffusion limitation (APO/LVF/pulmonary fibrosis) Disorientation, confusion, headache, LOC, Tachycardia +/- , hypertension, hypotension, AMI, arrest, diaphoresis, tachypnoea	3 to pass 2 mechanisms and correct example 2 to pass
Stem: Moving to pharmacology: The child's mother has epilepsy and takes valproate.			
Question 3 Valproate **Subject:** Pharm LOA: 1	1.What are the possible pharmacodynamic mechanisms of Na Valproate? Prompt: what ion channels/ neurotransmitters are most likely involved? 2.What are the adverse effects?	**GABA** increased presynaptically by reduced GABA breakdown to succinate (ABAT/ GAT1), (> Cl- inh post synaptic GABR channel)/ possible increased production (GAD) Direct inh actions on post synaptic **Na Channel** particularly high freq gates and Ca+ (membrane stabilisation-reduces voltage gated outflow), Blocked NMDA receptor activation effects? Nausea/vomiting/ GI (v common); **Severe hepatotoxicity**- liver failure (> young/ other hep tox drugs/ liver damaged); Marked fetal abnormality rates (8-9%)/ reduced IQ + other possible developmental effects; Thrombocytopaenia/ bruising; Pancreatitis; alopecia, neuro (asthenia, tremor, nystagmus etc); Hypersensitivity reactions	Bold Bold and 1 other
Stem: Moving now to your anatomy question. The mother has a seizure and falls to the ground hitting her head and face.			
Question 4 Facial Bone CT **Subject:** Anat LOA: 2	1. What air filled structures are visible on this CT? 2. What other structures are visible? 3. What structure passes through the infra-orbital foramen? 4. What is its sensory distribution?	**1. Maxillary,** mastoid, ethmoidal **2.**Bones: Frontal, zygoma, ethmoid, nasal septum, maxilla, nasal concha (middle and inferior), crista galli, Other: orbit, ocular muscles, frontal lobe (coronal slice), temporal lobe and parieto-occipital lobe, **3. Infra-orbital nerve** 4. superior lip, lateral nose, cheek, inferior eyelid, upper teeth and gingiva	bold and 1 other 2 bones and 3 others. Bold 2

ACEM PRIMARY VIVA B **Friday Morning Session 3** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE (**essential in bold**)	NOTES
Stem: An obese 50 year old woman presents to ED with an anaphylactic reaction to penicillin.			
The first questions is in regard to pathology:			
Question 1 Type 1 hypersensitivity reaction **Subject:** Path LOA: 1	1.What type of hypersensitivity reaction is involved? 2.What are the sequence of events involved in type I hypersensitivity reactions following re exposure to an allergen?	**Type 1** **Mast cells** armed with pre formed **IgE antibodies** > on re exposure to **specific antigen** > **release of mediators** from mast cells: **1. preformed mediators – e.g histamine/** proteases/ chemotactic factors, 2. lipid mediators e.g leukotrienes C4 and D4/ PG D2/ PAF and 3. Cytokines e.g TNF and chemokines > **Immediate and late phase reactions**	**Bold**
	3.What changes occur at the tissue level?	1.Vasodilatation 2.increased vascular permeability 3. smooth muscle spasm/ bronchospasm 4. cellular infiltration 5. epithelial damage	**3 of 5 to pass**
Stem: Moving now to your physiology question: She is hypoxic with oxygen saturations of 90% on room air			
Question 2 Oxygen / Haemoglobin dissociation curve **Subject:** Phys LOA: 1	1. Please draw and label the oxygen dissociation curve.		Draw correct shape – have points of 90% (58-60) saturation.
	2. What factors can cause the curve to shift to the right (reduced affinity of Hb for O2)?	• Increased temp, PCO2, 2,3 DPG • Drop in pH (increased H+)	At least 3
	3. What are the physiological advantages of this curved shape?	(UPPER) If pO2 alveolar gas falls, loading of O2 little affected. Also, as RBC takes up O2 along pulmonary capillary, diffusion process hastened as large partial pressure difference maintained when most of O2 has been transferred. (LOWER)Steep lower part means peripheral tissues can withdraw large amounts of O2 for only small drop in capillary pO2	Concept of loading and unloading of oxygen being facilitated

Stem: Moving now to your pharmacology question. Your planned treatment includes IV hydrocortisone.			
Question 3 Corticosteroids **Subject:** Pharm LOA: 1	Describe the mechanism of action of corticosteroids at a cellular level?	• Most of known effects via widely distributed **glucocorticoid receptors** • Present in blood in bound form on Corticosteroid Binding Globulin (CBG) • Enters cell as free molecule • Intracellular receptor bound to stabilizing proteins (most important heat shock protein 90, Hsp90) • Complex binds molecule of cortisol then actively transported into nucleus where binds to **Glucocorticoid Receptor Elements (GRE)** on the gene • Interacts with DNA and nuclear proteins regulating transcription. Resulting mRNA exported to cytoplasm for **protein production** for final hormone response	Bold to pass
	How can corticosteroids be classified? Prompt: How do they differ in their action?	1. length of action (hydrocortisone short to medium-acting, dexamethasone or betamethasone long-acting) 2. **anti-inflammatory activity** (potency: hydrocortisone 1, prednisolone 5, dexamethasone 30) 3. **mineralocorticoid activity** ie., salt retaining (fludrocortisones 250 times that of hydrocortisone) 4. topical vs non topical	bold
	What are the side effects of corticosteroid use? Prompt: what about long term effects?	- Short term: (<2 weeks): insomnia, behaviour changes, acute peptic ulcer, acute pancreatitis, hyperglycaemia - Long term: - Cushing's Syndrome (moon facies, fat redistribution, fine hair growth, acne) secondary to hormonal actions. (Rate of development function of dose and genetic background) - hyperglycaemia, diabetes - myopathy - osteoporosis, aseptic necrosis - psychiatric (hypomania, acute psychosis, depression) - Na,fluid retention, K+ loss - **adrenal suppression** / addisonian crisis - poor wound healing **- immunosuppressant**	Bold and 4 others
Stem: Moving now to your anatomy question. You are inserting an IV in her cubital fossa.			
Question 4 Cubital Fossa **Subject:** Anat LOA: 1	1.please identify and name the superficial veins 2. please identify the arteries and the nerves 3.please identify and name the muscles of the forearm	**1. medial cubital vein (13), cephalic vein(6),** medial forearm Vein(14) **2.median nerve (15)(, radial artery in CF or wrist (21), ulnar artery (22),** brachial artery(4) **3.** pronator teres (20), brachioradialis (5), biceps tendon(2) and aponeurosis (3), FCU(9),FCR (8),PL (18),FDS (10)	bold to pass at one site to pass Name 4

Stem: An 80 year old woman presents with a diabetic foot ulcer. We start with physiology.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Glucose homeostasis (Ganong 24th ed pp 431-432, 433-434, 441-442, 444-445) **Subject:** Phys LOA: 1	1.1 What factors determine glucose homeostasis?	**1.1** Glucose absorption from **intestine** Glucose **uptake** in the **periphery** - muscle, brain, fat, red cells and **liver** Reabsorption in **kidney** **Gluconeogenesis** in liver (Insulin and Glucagon)	1.1 Name at least 3 mechanisms
	1.2 What happens to glucose homeostasis in the absence of insulin?	1.2 Hyperglycaemia due to a) **decreased peripheral uptake** of glucose into muscle and fat (direct effect) b) **reduced glucose uptake by liver** (indirect effect) c) **increased glucose output** by the liver and lack of glycogen synthesis (GIT, renal, brain and red cells glucose uptake unaffected)	1.2 2 out of 3 mechanisms
	1.3 What effect does glucagon have on blood glucose?	1.3 Increase BSL due to increased glycogenolysis and increased gluconeogenesis in liver	1.3 know that glucagon increases liver glucose output
Stem: We now move onto pharmacology.			
Question 2 Insulins (Katzung 12th ed pp 747-753) **Subject:** Pharm LOA: 1	What pharmacological methods are used to optimise blood sugar control when administering insulin? Prompt: what are the different types of insulin?	1. **Titration of dose to BSL** 2. **Pharmacological manipulation of human insulin molecule**: rapid-acting (aa reversal/substitution reducing aggregation properties), intermediate acting (insulin/protamine complexes), long acting (aa substitutions, molecular attachments) 3. Mixing of insulin preparations 4. Continuous subcutaneous insulin infusion devices	Bold to pass

	What are the complications of insulin administration?	**Hypoglycaemia** Hypoglycaemic unawareness Insulin allergy (usually due to non-insulin contaminants) Immune insulin resistance Lipodystrophy at injection sites	Bold + 1 to pass
Stem: We now move onto anatomy.			
Question 3 Model – foot (NS 9), include description of cutaneous nerve supply of foot. **Subject:** Anat LOA: 1	1. identify the structures lying deep to the extensor retinaculum 2. Describe the cutaneous nerve supply of the foot 3. Describe the anatomy of the dorsalis pedis artery (dorsal artery of the foot) Extra question if time allows.	1.Medial to lateral: **Tibialis anterior, EHL, Dorsalis Pedis, Deep fibular nerve, EDL**, fibularis tertius, EDB 2. DORSUM: **Deep Fibular nerve** (1st web space), **Superficial fibular nerve** (becomes dorsal digital nerves) – majority of dorsum of foot **Dorsal lateral cutaneous nerve of foot** (terminal branch of sural nerve) – lateral foot **Saphenous nerve** (medial foot below medial malleolus) PLANTAR: **Medial, lateral plantar nerves** (terminal branches of tibial nerve) **Calcaneal branches** (of tibia & sural nerves) 3. Direct continuation of anterior tibial artery Lies between EHL & EDL & gives off Medial tarsal artery, Lateral tarsal artery (lateral tarsal art. joins the arcuate artery) At the 1st interosseous space divides into the 1st dorsal metatarsal artery & deep plantar artery (the deep pl. artery joins the lateral plantar artery to form the deep plantar arch).	1. 4/5 bold to pass 2. 3/4 dorsal & 2/3 plantar to pass 3. 3 to pass
Stem: We now move onto pathology.			
Question 4 Complications of diabetes mellitus (Robbins pp1138-1143) **Subject:** Path	a) What are the principal complications of Diabetes mellitus? (Prompt: what happens in the pancreas?)	**Vascular-** **- macro** atherosclerosis, CAD, PVD, RAS, HT and CVA - **microangopathic** thickened BM, increased permeability of capillaries to plasma proteins - nephropathy, retinopathy, neuropathy	Bold + 3 of 7 clinical complications.

LOA: 2

b) Outline some of the differences in patients with Type 1 and type 2 diabetes.

Pancreatic changes - loss of islets cells (number and size), amyloid infiltration of islets

Renal - sclerosis, BM thickening, glomerulosclerosis
Occular- prolif and non prolif, haemorrhages, exudates neovascularisation, detachment, glaucoma
Neuropathy

Type 1	Type 2
Onset: childhood, <18	Onset: usually adult
N or under weight	Obese
Dec in insulin	Inc blood insulin
Circulating islet autoantibodies	No islet auto-antibodies
polyuria, polydipsia, polyphagia +/- ketoacidosis	May have HONC
Genetic linkage	No genetic linkage
Dysfunction in T cell resulting in islet Ab	Insulin resistance

<u>Type 1 :-</u>
- typically young < 18 yrs, usually abrupt onset due to exhaustion of b cell reserve - often with a precipitating illness increasing demands on pancreas eg. infection-

<u>Type 2 :-</u>
- often > 40 yrs, obese
- often asymptomatic and incidental finding on routine followup or bloods
- may have DKA or HONC with dehydrating precipitant
- often a longer cause illness due to residual pancreas capacity

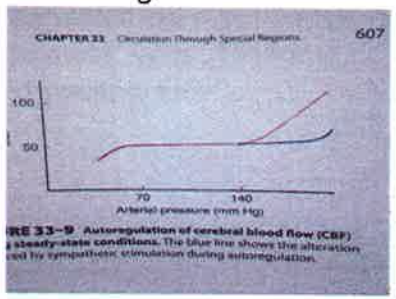
Question b (to pass) - age group and severity of illness + at least 2 symptoms or syndromes associated with each type.

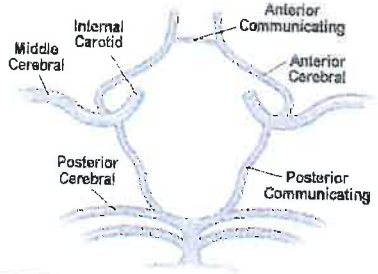
Age + 2 clinical + 1 pathology to pass

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: A 60 year old man with a history of atrial fibrillation on warfarin presents to ED following a motor bike accident. His blood pressure on arrival is 80/40			
Question 1 Baroreceptors **Subject:** Phys LOA: 1	**What are baroreceptors and where are they located?**	**Stretch receptors** Carotid, aortic, cardiopulmonary. In the adventitia of vessels. The **carotid sinus** and **aortic arch** receptors monitor the arterial circulation. Receptors are in the wall of the **right** and **left atria**, at the entrance of SVC and IVC and in the pulmonary veins as well as in **the pulmonary circulation** (collectively the cardiopulmonary receptors).	Bold to pass Carotid and aortic plus one other to pass
	What is their mechanism of action?	Very sensitive to changes in pulse pressure. Exert an **inhibitory** input via the tractus solitarius in the medulla. **Stimulated by distension** of the structures in which they are located, therefore discharge at an increased rate when the pressure in these structures rises. Increased baroreceptor discharge ***inhibits*** **the tonic discharge of sympathetic nerves and *excites* the vagal innervation of the heart.** Result is vasodilatation, venodilation and a fall in BP, bradycardia and decreased cardiac output.	Need mention of inhibitory nature of pathway and nerves affected (vagus, sympathetics)
	What is their action in this setting of acute blood loss?	Decreased blood volume and decreased venous return results in reduced stimulation of arterial baroreceptors and **increased sympathetic output**. The result is **reflex tachycardia and vasoconstriction.**	Bold to pass
Stem: The patient's INR result is 5.5.			
Question 2 Vitamin K **Subject:** Pharm	What methods are available to reverse warfarin induced anti-coagulation? How does vitamin K reverse warfarin	**Cease warfarin** **Vit K – oral or IV 1-10mg** **+/- FFP or prothrombinex**	2/3 bold to pass, must include vitamin K.

LOA: 2	effect? How long does it take for vitamin K to work?	Pharmacodynamic interaction with warfarin to reduce INR ie **reverses the effect of warfarin Re-establishes normal activity of the clotting factors. Vit K dependant clotting factors: II, VII, IX,X** **6 - 24 Hours**	Bold to pass >6 hrs
Stem: He sustained an open ankle injury.			
Question 3 Bones- ankle / foot **Subject:** Anat LOA: 1	1. Identify the bones of the foot and ankle 2. Identify factors that provide stability to the ankle joint (Prompt: Describe the ligament of the ankle in more detail.)	1. **Lat malleolus** (fibula), **Medial malleolus** (tibia), **talus** (dome/head/body),**calcaneus, cuboid, navicular,** med/middle/lat **cuneiforms, MTs** (base shaft/head/neck), **tarsal bones** 2. **Bony**- Ankle mortice around talus (lat/ med malleolus and distal tibial articular surface) held together by ant + posterior tibio-fibular ligament **Ligamentous**- **MCL** (Deltoid)- 4 parts ant + post tibio-talar, tibio-calcaneal, tibio-navicular) / **LCL**- 3 parts (ATFL, PTFL, calcaneo-fibular ligt) / distal tibio-fibular syndesmosis/ IOM **Muscular**- not seen....	**1.Bold to pass** 2/3 bold to pass, some details of one of the ligament.
Stem: Several months after discharge, he develops osteomyelitis. Moving onto pathology.			
Question 4 Osteomyelitis **Subject:** Path LOA: 1	1.Describe pathogenesis of osteomyelitis. (Prompt what organisms cause osteomyelitis?) 2.What changes occur to the bone? 3.What are the pathological sequelae of osteomyelitis?	***Local bone injury** and organism entry, **blood borne** organisms, neighbouring source entry. ***Staph Aureus** > 80% of pyogenic ones Others E coli, Kl Pneum, Ps Aerug from IVDU and GU, haemophilus influenza, Gp B Streptococcus. 50% no orgs found. ***Acute inflammation, necrosis, abscess** Sclerosis, involucrum and sequestrum, lytic focus and surrounding necrosis- periosteal elevation *** Chronic up to 25%, resolve, deformity and bone destruction**, severe sepsis, pathological fracture, endocarditis, SCC, sarcoma.	1.Bold + 1 to pass other organism 2.Bold to pass 3.Bold

ACEM PRIMARY VIVA C Friday Morning Session 3 Candidate Number: AGREED MARK:

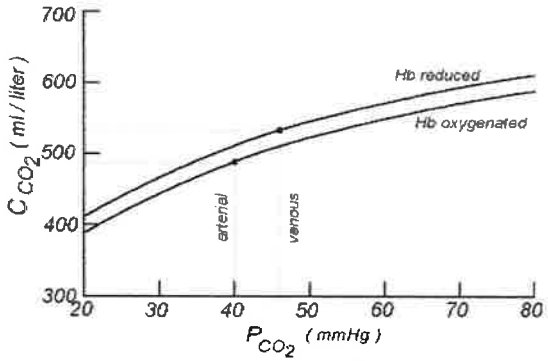
TOPIC	QUESTIONS	KNOWLEDGE (essential in bold)	NOTES
Stem: A 30 year old woman who is 35 weeks gestation presents with a severe headache and a BP of 160/100. We will begin with physiology.			
Question 1 Autoregulation of cerebral circulation **Subject:** Phys LOA: 1	1.1 What factors affect cerebral blood flow? 1.2 Describe autoregulation of cerebral blood flow. You can draw a diagram if you wish. 1.3 What is the Monroe-Kellie doctrine? (optional if run out of time)	1.1 • **Intracranial pressure** • **Mean arterial pressure** • Mean venous pressure • Local factors: pH, pCO2, cause constriction and dilatation of cerebral arterioles • Blood viscosity 1.2 The process by which **CBF is maintained at a constant level despite variation in perfusion pressure.** Average CBF is 54 ml/100g/min between MAP 65- 140 mmHg 1.3 Due to the fact that brain tissue and spinal fluid are essentially incompressible, **the volume of blood, spinal fluid and brain tissue must be relatively constant.** So when ICP rises, the cerebral vessels are compressed resulting in reduced cerebral blood flow (CBF)	1.1 Bold +1 Able to draw a plateau region with a range for MAP of 50 – 150 mm Hg.  Need to pass 2/3 part to pass.
Stem: We are moving onto pharmacology. Her treatment includes Magnesium			
Question 2 Magnesium **Subject:** Pharm LOA: 1	2.1 What are the indications of its use in pregnancy? 2.2 What are the other uses of magnesium in Emergency Medicine? 2.3 What are the toxic effect of magnesium?	2.1 It is indicated **in pre-eclampsia and eclampsia.** for the prevention and treatment of **life threatening seizures.** 2.2 It has an **anti-convulsant effect,** possible **antiarrhythmic effect, bronchodilator effect.** (influence Na+ /K+ -ATPase, Na channels, certain K and Ca channels). 2.3 Hypermagnesaemia include nausea & vomiting, flushing, hypotension, muscle weakness, muscle paralysis, blur or double vision, CNS depression or loss of reflexes, respiratory depression, renal failure, cardiac arrhythmia.	Bold to pass 2/3 bold to pass 3 to pass

Stem: We are moving onto anatomy.			
Question 3 Sagittal model of head looking at the CNS **Subject:** Anat LOA: 2	3.1 Identify the intracranial structures visible on this model. 3.2 Describe the anatomy of the Circle of Willis. You can draw a picture if you wish.	3.1 Brain:- **Cerebrum/ medulla/pons/ cerebellum**/spinal cord/**corpus callosum**/dura/**ventricle** Frontal/parietal/occipital/maxilla/ethmoid *Spine*-Atlas (C1)-ant and post arches/Axis-dens(C2)	Bold 5/6 to pass 4/5 to pass the circle 
Stem: We are moving onto pathology.			
Question 4 Pre-eclampsia **Subject:** Path LOA: 2	4.1 Describe the pathogenesis of pre - eclampsia. 4.2 What is the clinical course of pre eclampsia? 4.3 What morphological changes occur in the placenta?	**4.1 Endothelial dysfunction,** vasoconstriction leads to hypertension, increase vascular permeability causing proteinuria & oedema. 4.2 > 34 weeks typically has **HT, oedema, proteinuria** Headache and visual disturbance **Eclampsia** is progression to seizures and coma 4.3 Infarcts, haematomas, villous ischaemia, syncytial knots, fibrinoid necrosis	Bold + 1 to pass 2/3 bold to pass (prompt: what happens in untreated pre-eclampsia?) 1 to pass

ACEM PRIMARY VIVA D **Thursday Morning Session 1** ***Candidate Number:*** **AGREED MARK:**

Stem: A 70 yo woman undergoes procedural sedation in ED for reduction of a wrist fracture. The first topic is **PHARMACOLOGY**.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Clearance Definition, factors affecting, examples **Subject:** Pharmacology LOA: 1	(a) What is drug clearance?	(a) **Clearance:** • Measure of the ability of the body to eliminate a drug • Rate of elimination in relation to drug concentration • CL = rate of elimination / concentration	(a) Reasonable definition to pass
	(b) What factors affect drug clearance?	• Concentration - **Dose & Bioavailability** • Elimination - **specific organ function / blood flow & protein binding** • Major sites of elimination are kidneys and liver – therefore factors that affect these organs' function and blood flow will have most effect	(b) One for each element
	(c) What is the difference between capacity-limited and flow-dependent drug elimination?	(c) **Capacity-limited is saturable (zero order)** Examples: aspirin, phenytoin, ethanol. **Flow-dependent = non-saturable (1st order)** (organ blood flow, protein binding) Examples: Alprenolol / amitriptyline / Imipramine / isoniazid / labetalol / lignocaine / Morphine / propoxyphene / propranonol / verapamil	(c) Bold to pass
Stem: Moving onto **ANATOMY**			
Question 2 Bone – hand / carpal bones **Subject:** Anatomy LOA: 1	(a) Identify the bones in this hand and wrist.	• **Prox row:** Pisiform, triquetrum, lunate, scaphoid • **Distal row:** Hamate, capitate, trapezoid, trapezium • Metacarpals, and phalanges, prox/middle/distal	(a) All carpal bones to pass
	(b) Identify the boundaries of the carpal tunnel on this model.	(b) Tubercle **scaphoid** and **trapezoid** laterally, and **pisiform** and **hook hamate** medially	(b) 4/4 bony landmarks to pass

	(c) This patient develops median nerve paresis as a consequence of her fracture. What deficits will she develop? (Prompt: what does the median nerve supply in the hand?)	• **Sensory supply:** radial 3 ½ digits and adjacent palm, excluding central palm which is by cutaneous palmar branch passing over flexor retinaculum • **Motor supply:** thenar muscles except add pollicis and deep head fpb; and lateral lumbricals for digits 2 and 3	(c) Correctly identify sensory supply and one group of muscles.
Stem: During the reduction she becomes persistently hypoxic. This topic is **PATHOLOGY.**			
Question 3 Pneumonia including aspiration pneumonia **Subject:** Pathology LOA: 1	(a) Describe the pathogenesis of aspiration pneumonia. (Prompt: predisposing features, organisms, outcomes)	• Aspiration of **gastric** contents • Type of **patient** (↓conscious/debilitated/abnormal gag/repeated vomiting) • **Chemical and bacterial** • **>1 organism** (aerobes>anaerobes) • **Necrotizing** • Death / **abscess**	(a) 4 bold to pass
	(b) How are community-acquired pneumonias different?	• **Bacterial or viral** • **Variable** pneumonia dependent on – etiol., host response etc • **Predispose** – extremes age, chr disease etc • **Agents** – strep pneum, haem. Influenza, etc • Clinical course modified by **ABs** • **Low hosp, low death** • **Complications** – empyema, endo/pericarditis, meningitis	(b) 5 bold to pass

Stem: Moving onto **PHYSIOLOGY**			
Question 4 CO2 carriage and dissociation curve **Subject:** Physiology LOA: 1	(a) How is carbon dioxide transported from the tissues to the lungs?	(a) In plasma: • **Dissolved** • **Carbamino compounds with plasma proteins** • **Hydration – H+ buffered – HCO3- in plasma** In RBC: • Dissolved • Formation of carbamino-Hb • Hydration – H+ buffered – 70% of HCO3- enters plasma Each 49ml CO2/dL arterial blood – 5% dissolved, 5% in carbamino compounds, 90% hydrated as HCO3	(a) Bold to pass
	(b) Draw and explain the carbon dioxide dissociation curve		(b) Concept to pass
	(c) What is meant by the term 'chloride shift'?	(c) 70% of HCO3- formed in red cells enters the plasma in exchange for chloride – exchange is the chloride shift	(c) Reasonable definition to pass

ACEM PRIMARY VIVA D **Thursday Afternoon Session 2** ***Candidate Number:*** **AGREED MARK:**

Stem: A 30 yo man presents with a suspected dislocated right shoulder. He is given IV opiates. The first topic is **PHARMACOLOGY**.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Potency & efficacy with reference to morphine / fentanyl **Subject:** Pharm LOA: 1	(a) What is drug potency? (b) Draw and explain dose-response curves comparing morphine with fentanyl. (c) What are the pharmacokinetics of fentanyl?	(a) **Dose or concentration to achieve 50% maximal effect** (EC_{50} or ED_{50}) (b) **Must graph dose or log dose (X axis) versus response (Y axis).** (c) Highly lipid soluble, Half-life 5 mins, duration 1-1.5 h, low bioavailability, hepatic metabolism	(a) Bold to pass (b) Display differences and explain on graph (c) 3 of 5 to pass
Stem: Moving onto **ANATOMY**			
Question 2 Shoulder Model **Subject:** Anatomy LOA: 1	(a) Identify the features on this model of a shoulder. (b) What anatomical structures confer stability to the shoulder joint?	(a) Bony: **Humerus** / Humeral head **Scapula – coracoid process / acromion** / spine / body **Clavicle** Joints: **glenohumeral** and **acromioclavicular** Ligaments: **Coracoclavicular** – conoid part and trapezoid part – most important for stability AC joint **Acromioclavicular** –top of clavicle to acromion Glenohumeral ligaments – reinforce anterior part of capsule from glenoid labrum to humerus Tendons: **Long head of biceps tendon** • **Joint capsule** with fusion of the tendons of scapular muscles • Ligamentous: **glenohumeral and coracohumeral ligaments** • **Coracoacromial arch** superiorly created by coracoacromial ligament • **Deepening of glenoid cavity by glenoid labrum** • **Tendons** of long head of biceps and triceps	(a) Bold to pass (b) 3/5 to pass

	(c) What structures can be damaged by shoulder dislocation? (Prompt for ax nerve)	(c) Joint capsule and glenoid labrum damage results in recurrent dislocation **Axillary nerve** lies below joint capsule – palsy Associated fracture of greater tubercle	(c) Bold to pass
Stem: Your intern consults you on a 60 yo lady he suspects has acute cholecystitis. This topic is **PATHOLOGY**.			
Question 3 Cholecystitis **Subject:** Path LOA: 1	(a) Describe the pathogenesis of acute calculous cholecystitis. (b) What are the complications of cholecystitis?	(a) **Chemical irritation of obstructed GB** • Mucosal phospholipases hydrolyse luminal lecithins to toxic lysolecithins • Protective glycoprotein mucus layer disrupted • Allows Bile salts to have detergent action on exposed mucosal epithelium • PGs contribute to inflammation • GB dysmotility develops • Distension and increased intraluminal pressure decreases mucosal blood flow (b) **Bacterial infection - cholangitis / sepsis** • Perforation and localised abscess • Rupture and peritonitis • Biliary fistula • Porcelain gallbladder	(a) Bold + 2/6 (b) Bold + 2/4
Stem: Moving onto **PHYSIOLOGY**			
Question 4 Liver metabolic functions especially bilirubin metabolism. **Subject:** Phys LOA: 1	(a) List the principal functions of the liver	(a) **Bile formation** (500ml/day) • **Synthesis** – protein, coag factors, albumin • **Inactivation** / detoxification – drugs, toxins, active circulating substances • Nutrient vitamin **absorption, metabolism / control** (e.g. glucostat), AAs, lipids, fat sol vitamins • **Immunity** (esp. gut organisms) – Kupffer / macrophages in sinusoid endothelium	(a) 3/5 bold with an example to pass

	(b) Describe the metabolism of bilirubin.	(b) • **Formed by breakdown of haeme, Hb** • Bound to albumin • In liver – actively transported (OATP) as dissociates – binds to cytoplasmic proteins • **Conjugated by gluc-transferase in ER with glucuronic acid to H2O sol bil-digluc** • Bil di gluc active transport (MDRP2) against gdt to bile canaliculi – to gut (<5% bil/bdg reflux to blood) • Intestinal mucosa relatively impermeable • Gut bacteria act / convert most to urobilinogens • **Some bile pigments / urobilinogens/unconj bil reabsorbed in portal circulation – most resecreted = enterohepatic circulation** • Small amounts urobil in blood excreted in urine – urobilinogen and faeces – stercobi	(b) Bold to pass

ACEM PRIMARY VIVA D **Friday Morning Session 3** ***Candidate Number:*** **AGREED MARK:**

Stem: You have a 25 yo man with a painful knee. He has received ibuprofen for analgesia. The first topic is **PHARMACOLOGY.**			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Bioavailability with particular reference to NSAIDs **Subject:** Pharm LOA: 1	(a) What is bioavailability? (b) What factors affect bioavailability? (c) What is the bioavailability of ibuprofen?	(a) Fraction of **unchanged drug** reaching the **systemic circulation** following administration by any route (b) 3 factors: **Extent of absorption** • Too hydrophilic or too lipophilic – decr. absorption • Reverse transporter associated with p-glycoprotein – pumps drug back to gut lumen – decr. absorption • Gut wall metabolism – decr. absorption **First pass metabolism** • Metabolism by liver before it reaches systemic circulation • Small additional effect if drug has biliary excretion Rate of absorption • Determined by site of administration and drug formulation (c) **High** - Weak organic acid – **well absorbed rapidly. Minimal first pass metabolism**	(a) Bold to pass (b) Bold with reasonable explanation of each (c) Bold to pass
Stem: Moving onto **ANATOMY**			
Question 2 **Subject:** Anatomy LOA: 1	(a) Identify the features on this model of the knee joint.	**Bones: patella, femur, fibula, tibia, and** **Features:** med/lat fem condyles, med/lat tibial condyles, tibial tuberosity, head/neck fibula, lat /med epicondyle femur, menisci, patellar tendon **Ligaments:** medial/lat collateral, ant / post **cruciates**	(a) All bones + 5 features + 4/4 ligaments

	(b) Describe the cruciate ligaments and their actions. (c) What features confer stability on the knee joint?	**Attachment points:** • Ant cruciate - weaker, ant intercondylar area tibia, extends sup, post and laterally to attach to post part of med side of lat condyle femur • Post cruciate - arises from post intercondylar area of tib and passes sup and anteriorly on med side of ant cruciate to attach to ant part of lat surface of med condyle of femur) **Actions:** • Ant cruciate prevents post movement of femur on tibia (or ant movement of tib on femur) and limits hyperextension of knee • Post cruciate limits ant movement of femur on tibia (or post movement of tib on femur) and prevent hyperflexion of knee (c) **Muscles/tendons, and ligaments connecting femur to tibia – no bony contribution. 2/3** of **quadriceps** (esp. inf. fibres of vast med/lat) **Collateral ligaments** and **cruciate ligaments**	(b) 2/4 attachment points 1/2 actions (c) Bold to pass
Stem: The next patient is a 20 yo woman who is dehydrated secondary to poor oral intake from glandular fever. This topic is **PATHOLOGY**.			
Question 3 EBV **Subject:** Path LOA: 2	(a) Describe the pathogenesis of glandular fever.	• **EBV** transmitted by close contact (saliva) • Envelope g/protein binds to B cells • Viral infection begins naso/oropharyngeal **lymphoid** tissues (esp. tonsils) • EBV accesses submucosal lymphoid tissues • B Cell infection 1) **lysis** infected cells and virion release (minority) or 2) **Latent** infection (EBV genes expressed) • Symptoms appear on initiation host immune response (cellular CD8+ cytotoxic T and NK cells) • Atypical lymphocytes (characteristic) • Reactive T cell proliferation lymphoid tissues – lymphandenopathy and splenomegaly. • IgM Ab (viral capsid Ag) and later IgG • Healthy – cease viral shedding with few resting B cells but Acquired defects may → B lymphomas	(a) To pass: EBV Lymphoid tissue Involves B (latent and lysis) and T cells

	b) What are the clinical features of glandular fever?	(b) Classically – Fever, sore throat, lymphadenitis splenomegaly Atypical presentation common – fatigue, lymphadenopathy, hepatitis, rubella-like rash	(b) 4 clinical features to pass
	(c) What are the outcomes of glandular fever?	4-6 weeks **most resolve** - some fatigue longer **Hepatic** dysfunction – j, abn. LFTs, appetite **Splenic** rupture Other systems – nervous, renal, lungs, heart. Transformation – **lymphomas**	(c) 3 outcomes to pass
Stem: Moving onto **PHYSIOLOGY**			
Question 4 Renal response to dehydration **Subject:** Phys LOA: 1	(a) What is the renal response to dehydration?	(a) **Renin release, converts a-gin to AT1** **ACE converts AT1 to AT2** **AT2 increases aldosterone synthesis, vasoconstriction of aff arteriole** **Aldo - Na and water retention**	(a) Need details re secretion i.e. reduced pressure at JG cells of renin and actions of A-2
	(b) What is the role of vasopressin in dehydration?	(b) Promotes **water resorption** in CD via **aquaporins** insertion. **Vasoconstriction**	(b) Bold to pass

Stem: An 80 year old woman presents to ED following a fall secondary to an episode of melaena.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Anaemia (pp 639-665) **Subject:** Path LOA: 1	1. How are the causes of anaemia classified? Prompt if use RC morphology: How are the causes classified by mechanism? Prompt for example if not volunteered.	1. **Blood loss**: acute, chronic 2. **Increased RC destruction** Inherited genetic: H Spherocytosis, G6PD, Thal, Sickle cell Acq genetic: Parox noct hemo. Ab mediated: transfusion, drugs, Rh disease. Mech trauma: HUS, DIC, TTP, cardiac valves, runners. Infx: malaria; Toxic: envenom, clostridia, Pb. 3. **Decreased RC production** Inherited genetic: Fanconi's, thalassemia. Nutritional: B12/folate, iron. Erythropoietin deficit: renal failure, chronic dis. Immune: aplastic anaemia.	Bold main headings & 1 example of each to pass.
	2. Describe the pathogenesis of iron deficiency anaemia.	Causes: Chronic blood loss, poor diet, impaired absorption, incr reqs Iron stores used up first – **ferritin** haemosiderin. Once reserves depleted **serum iron & transferrin** decr. Erythroid activity increases, no iron in marrow macrophages. RCs become **hypochromic & microcytic.**	Bold to pass.
	3. (Please give examples of anaemias that are more common in specific ethnic groups.) Ask if there is time.	Hereditary spherocytosis: northern Europe G6PD: 10% African American, Africa, Middle East, Med Sickle cell: African descent, up to 30% Thalassemia trait: Africa, Asia, Med, India Pernicious: Scandinavian, Caucasian.	1 correct with example.
Stem: Initial treatment included commencement of a Pantoprazole infusion			
Question 2 Proton Pump Inhibitors (pp 1085-1089) **Subject:** Pharm LOA: 2	1. Describe the MOA of PPIs	**Irreversibly inactivates H^+K^+ATPase**, blocking the proton pump-inhibiting >90% acid secretion, for up to 24 hrs (time taken for synthesis new enzymes).	Bold to pass.
	2. Why is an IV infusion preferred to a single bolus dose?	**Only inactivates actively secreting acid pumps** (<10% in fasting patients). Hence single dose only decreases acid secretion for a few hours.	Bold to pass.
	3. Regarding oral formulations of proton pump inhibitors, please describe strategies used to increase their bioavailability and activity.	Taken as **inactive pro-drugs**, Begin as **acid resistant enteric coated** to prevent gastric elimination. Take on **empty stomach** as food decreases bioavailability. Weak bases so pass into acidified parietal cells, where concentrated 1000x, ecomes activated and binds to H^+K^+ATPase**. Take 1 hour prior to meal** so peak dose drug occurs when most pumps are active.	2 concepts.

Stem: Her BP is low.			
Question 3 Renal response to hypovolaemia (pp 701-706) **Subject:** Phys LOA: 1	1. Explain how hypotension activates the renin-angiotensin system. 2. How does the renin-angiotensin system contribute to the restoration of the blood volume? 3. What other factors increase renin secretion?	1. Hypotension leads to **reduced perfusion pressure of the afferent glomerular arteriole**, stimulating release of **renin** by the juxtaglomerular cells. 2. Renin **converts angiotensionogen to angiotensin I. Angiotensin converting enzyme** converts AG1 to **angiotensin II.** Ang II acts on the adrenal cortex's zona glomerulosa cells to **release aldosterone**. Aldosterone acts on the renal distal tubules to **retain Na and water**, thus increases intravascular volume. Ang II also a potent arteriolar constrictor and contributes to a rise in blood pressure. 3. Renin (protease) release is stimulated by increases in: **catecholamines, sympathetic activity** through renal nerves, **prostaglandins**, low Na states: cardiac failure, liver failure and Na depletion.	1. Bold to pass. 2. 4/5 bold to pass. 3. 1/3 bold to pass.
Stem: Following resuscitation, she complains of a painful hip and X-rays show a fractured neck of femur.			
Question 4 Femur (bone) **Subject:** Anat LOA: 2	a) Identify this bone and the significant bony features at the proximal end (Fig 5.7 p 517). **b)** What is the blood supply of the head and neck of the femur? c) You plan to do a femoral nerve block. What structures does the femoral nerve supply? (Supplementary question if time remaining)	Femur, appropriate side/orientation **Head**, fovea, **neck, greater & lesser trochanters, intertrochanteric crest** & line, quadrate tubercle, pectineal line, gluteal tuberosity. Med and lat **circumflex** femoral aa Usu branches of deep art of thigh (**profunda femoris**) Branch to form **retinacular** aa (from med >lat), feed under post unattached capsule (med) or through iliofemoral lig (lat). Artery to head of femur – br of obturator (less important). **Anterior thigh muscles** (quadriceps) Pectineus, Sartorius, iliacus **Articular branches to hip** and knee joints **Cutaneous branches** to anteromedial thigh Terminal cutaneous branch is saphenous nerve to anteromedial knee, leg, foot.	Bold structures to pass. Bold to pass Need to show understanding of dual supply, and relative contributions (circumflex aa > art to head of femur). 2/3 bold to pass.

ACEM PRIMARY VIVA A Thursday Afternoon Session 2 ***Candidate Number:*** **AGREED MARK:**

Stem: An 80 year old man is brought by ambulance to ED following a syncopal episode with a head injury. You suspect a base of skull fracture.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Bone – Base of skull **Subject:** Anat LOA: 2	1. What are the major bony compartments within the Base of Skull and what are the major bones forming them?	**Anterior cranial fossa – frontal bone** (ant), ethmoid (mid) and lesser wing of Sphenoid (post). **Middle cranial fossa – Sphenoid** plus Squamous Temporal laterally, contains Sella Turcica. **Posterior cranial fossa – Occipital Bone** plus dorsum sella of Sphenoid anteriorly.	Needs skull model as prop Must identify all 3 fossae plus identify major bone in each.
	2. Identify the various foramina in the Base of Skull.	ACF: Cribriform Plate – Olfactory N, MCF: Optic Canal – Optic N, Ophthal A Superior Orbital Fissure – CN III, IV, VI Foramen Lacerum – Int Carotid A plus associated sympathetic Internal acoustic meatus – CN VII, VIII plus labyrinthine a Foramen Rotundum – V2 Foramen Ovale – V3, accessory meningeal A Foramen Spinosum – middle meningeal A Groove for Petrosal N and Petrosal Br Middle Meningeal A	Must identify 5 foramina.
	3. What structures pass through the foramen magnum?	PCF: Foramen Magnum – **Medulla/Brainstem**, plus vert a, XI Jugular Foramen – CN IX, X, XI, sup bulb of IJV Hypoglossal Canal – CN XII Condylar Canal – emissary veins (sigmoid sinus) Mastoid Foramen – Mastoid emissary vein.	Bold to pass
Stem: His heart rate is 40 beats per minute and he takes metoprolol.			
Question 2 **Subject:** Pharm Metoprolol / Beta blockers (Ch 10) LOA: 1	1. Describe the pharmacokinetics of metoprolol **Prompt** what is its bioavailability and why?	Oral or IV, Vd – large, T ½ 3 – 4 hrs, Metabolised in liver Bioavailability 50% due to 1st pass effect.	**Oral & IV & 1st pass** Or **3/5**
	2. How does metoprolol differ from propranolol in its action at beta receptors?	Beta 1 – full agonist Beta 2 - 50 – 100 fold less potent	**B1 Selective**
	3. How do BB control hypertension?	Negative **inotropic and chronotropic** effects Slow a-v node conduction Antagonises release of renin/not fully understood.	**Negative inotropic & chronotropic effect**

<table>
<tr><td colspan="4">Stem: Now we will move on to Physiology</td></tr>
<tr><td>Question 3
Pressure Volume Loop (pp 540-550)
Subject: Phys
LOA: 1</td><td>1. What is the stroke volume in a normal adult at rest?

2. Please draw and label the pressure volume loop of the left ventricle.

Prompt: Describe the changes in pressure and volume that occur during systole and diastole.

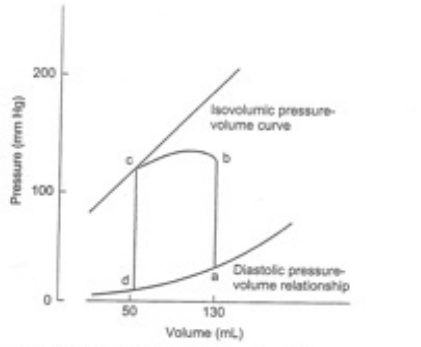
 

FIGURE 30–2 Normal pressure–volume loop of the left ventricle. During diastole, the ventricle fills and pressure increases from d to a. Pressure then rises sharply from a to b during isovolumetric contraction and from b to c during ventricular ejection. At c, the aortic valves close and pressure falls during isovolumetric relaxation from c back to d. (Reproduced with permission from McPhee SJ, Lingappa VR, Ganong WF (editors): Pathophysiology of Disease, 6th ed. McGraw-Hill, 2010.)</td><td>Stroke vol – 70-90ml

A. Start of systole: mitral (and Tric) valves close
Isovolumetric contraction til LVP > Aortic P (80mmHg)
Aortic (and Pulmonary) valves open.
B. Ventricular ejection (rapid at first) peak pressure 120mmHg
End systole: momentum of ejected blood overcome by aortic pressure.
C Aortic valve closes. ESV – 50ml

C-D. Isovolumetric relaxation
LVP drops below atrial pressure – mitral valve opens – ventricle begins to fill (rapidly at first)
EDV – 130ml</td><td>Bold to pass

Correct graph needed to pass. Need to demonstrate reasonable understanding of the loop.</td></tr>
<tr><td colspan="4">Stem: The patient has aortic stenosis.</td></tr>
<tr><td>Question 4
Calcific Aortic Stenosis (pp 561-563)
Subject: Path
LOA: 2</td><td>1. What are the predisposing factors for calcific aortic stenosis?

2. What are the clinical consequences of aortic stenosis?

3. What are the potential complications of a congenital bicuspid aortic valve?</td><td>Age: normal valve 70-90 yrs, bicuspid 50-70
Bicuspid valve or other congenital abnormality
Wear and tear, chronic injury
Hyperlipidemia, hypertension, inflammation
Other factors associated with atherosclerosis

Gradual obstruction of LV outflow leads to concentric LVH – pressure overload
Ischaemia/angina
Can get systolic and diastolic dysfunction
CHF and syncope herald decompensation.

Calcification, stenosis, regurgitation, infective endocarditis, aortic dilatation, dissection</td><td>Bold and one other

3 out of 4 concepts in bold to pass

Bold and 2 other</td></tr>
</table>

ACEM PRIMARY VIVA A **Friday Morning Session 3** ***Candidate Number:*** **AGREED MARK:**

Stem: A 60 yo man presents with fever and dyspnoea. He requires intubation. We will start with Anatomy.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Airway (model) (Somso Model) **Subject:** Anat LOA: 1	1.On the model, identify the structures of the larynx and upper airway (somewhat dependent on model – for this session we take half mandible off and the muscles at the back) 2.Describe the nerve supply to the intrinsic laryngeal muscles (muscles of vocalisation) 3.What is the results of an injury to the recurrent laryngeal nerve	Cartilages: **thyroid,** cricoid**, epiglottis**, arytenoids, corniculate, cuneiform Ligaments: **cricothyroid membrane**, thyrohyoid, **vocal cords** Muscles: cricothyroid muscle thyrohyoid,, cricoarytenoid Spaces & Folds: **vallecula** ,aryepiglottic folds All muscles supplied by branches of X All except cricothyroid supplied by recurrent laryngeal n, cricothyroid supplied by external laryngeal n. Hoarse voice, and if bilateral, stridor due to inability to abduct cords as posterior cricoarytenoids are only abductors.	Must name 4 of 5 bold and 2 others Must name rec laryngeal and X as its source Supplemental
Stem: A CXR shows pneumonia. We will now move onto Pathology			
Question 2 Community Acquired Pneumonia (pp 710-716) **Subject:** Path LOA: 1	1. What organisms cause community acquired pneumonia? **Prompts** What organisms cause atypical pneumonia, and what viruses may cause atypical pneumonia? 2. What are some potential complications of pneumonia? **Prompt** – pathological sequelae 3. How do the clinical features of atypical pneumonias differ from classic (typical pneumonias)?	1. **Bacterial – Step pneumonia**, H influenza, Moxarella catarrhalis, S.aureus, Kelbsiella, and pseudomonas 2. **Atypical orgs** Mycoplasma, chlamydiae spp, coxielle burnetti (Q fever), legionella pneum 3. **Viral** – RSV, parainf, influenza A and B, adenovirus, SARs, H1N1 Abscess formation, Empyema, Bacteraemia/bacterial dissemination (endocarditis, pericarditis, meningitis, kidney, brain abscess), sepsis, respiratory failure Moderate sputum, no physical findings of consolidation, only mod increase in WBC Cough not prominent, typical sx are fever, headache, myalgia. Lower mortality compared with classic pneumonia.	Bacterial – bold plus 2 others Atypical – 1 to pass Viral – 1 to pass 3 complications to pass 2 features to pass

Stem: Blood gases show an acidosis. We will now move onto Physiology			
Question 3 H+ handling in metabolic & respiratory acidosis (pp 711-712) **Subject:** Phys LOA: 1	1.Describe the renal response to acidosis Prompt – Describe the role of buffers in the kidney	Aims to return serum pH to normal by increasing H+ excretion. Kidney retains HCO3 by actively secreting H+ Renal tubule cells excrete carbonic anhydrase converting CO2 to H+ and HCO3, then tubule cells secrete H+ in exchange for Na+ Amount of secreted H+ limited by urinary pH >4.5 (limiting pH) Buffering in tubular fluid pH with HCO2, HPO4 and NH3 allows greater H+ secretion.	Must know that H+ actively secreted into tubular fluid in exchange for Na. Must know about buffering and name 2 buffers.
Stem: We will now move onto Pharmacology. He is a diabetic on metformin.			
Question 4 Metformin (p 757) **Subject:** Pharm LOA: 1	1. Describe the pharmacokinetics of metformin 2. Outline some common side effects of metformin 3. Contrast the mechanism of action of metformin (biguanide) and glipizide (sulfonylurea).	Well absorbed, not protein bound, not metabolised, elimination half-life 1.5 to 3 hours **Excreted by kidney as unchanged compound.** **GI most common (20%) – limits compliance with this drug.** HAGMA **(lactic acidosis)** esp in patients with coexistent renal disease, EtOH, chronic cardiopulmonary disease. Glipizide – **Increases insulin release from pancreas** (patients more prone to hypoglycaemia with glipizide compared with metformin) Decreases serum glucagon levels Metformin Mechanism unclear but: May reduce hepatic gluconeogenesis. **Not dependent on functioning pancreatic B cells – so doesn't influence insulin release from pancreas** May directly simulate glycolysis in tissues with increased glucose removal from blood Decreases glucose absorption in the gut	Bold and one other to pass. Bold to pass. Bold to pass.

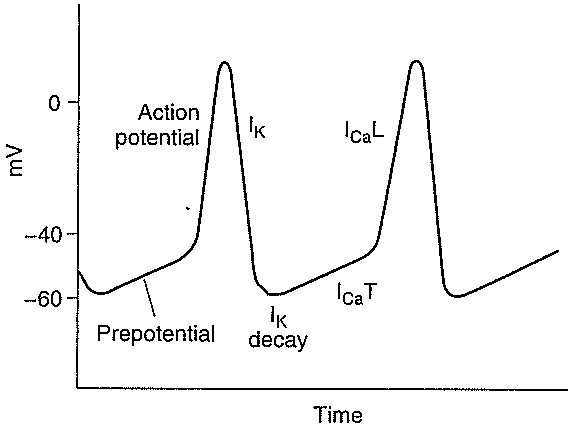
ACEM PRIMARY VIVA B **Thursday Morning Session 1** ***Candidate Number*:** **AGREED MARK:**

Stem: A 70 year old man with a history of Atrial Fibrillation presents with sudden onset of painful index and middle fingers in his left hand. Examination reveals these fingers to be pale and cold. We will start with Anatomy.

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Hand (photo) **Subject:** Anat LOA: 1	1. Please describe the arterial supply of the hand. (without photo) Prompt: What happens to these arteries in the palm? How are they arranged? How do they terminate?	The **Ulnar and Radial Arteries supply all of the blood supply to the hand**. **Radial Artery- Deep Palmar Arch.** Lies deep to long flexor tendons and sits across the metacarpals just distal to their bases. Branches: (of Deep Palmar arch) • 3x Palmar metacarpal arteries • Princeps pollicis artery • Radialis indicis artery **Ulnar Artery-** Two terminal branches Deep Palmar Branch (24) anastomoses with the Radial Artery via the Deep Palmar arch. **Superficial Palmar Arch** is main terminal branch. • 3 Common Palmar Digital Arteries arise. • Each divides into a pair of Proper Palmar digital arteries that run along adjacent sides of 2-4th digits.	Bold + 1 branch of each arch to pass
	2. Can you identify the Ulnar artery on the picture?	**Ulnar Artery (23),**	Bold to pass
	3. Describe the sensory innervation to the index and middle fingers?	**Median nerve (16) & Radial to dorsum to DIP** **1st** (7) **and 2nd** (22) **Lumbricals.** Muscles of the Thenar eminence: Opponens pollicis, **Abductor pollicis brevis (2) and Flexor pollicis brevis (13)**.	Bold to pass Thumb, Index, Middle and half of the ring finger (palmar aspect). Dorsal tips (nail beds) of the thumb, 2nd and 3rd fingers.
	4. Can you identify on the picture muscles in the hand and forearm that are innervated by the median nerve?	Forearm: **Flexor carpi radialis (8),** Palmaris longus, **Flexor digitorum superficialis(12), Radial half Flexor digitorum profundus (2nd and 3rd digits) (11). Flexor pollicis longus (14),** Pronator quadratus	4 of 8 bold to pass FPL and PQ supplied by the Anterior Interosseous nerve (a branch of the Median)

Stem: We will now move on to Pathology.			
Question 2 Embolism (pp 125-127) **Subject:** Path LOA: 1	1. What is an embolus?	A **detached intravascular** solid/liquid/gas **mass** that is carried by the blood stream from its site of origin to a **distant site**.	Bold to pass
	2. Name the different types of embolus?	• **Thromboembolus** **1. Venous: pulmonary** **2. Arterial: systemic** • Fat embolus: from bone marrow • Gas embolus: eg air/nitrogen • Amniotic fluid embolus • Tumour fragment embolus • Foreign body embolus eg catheter	Bold + 2 to pass
	3. What is systemic thromboembolism?	**Definition: Emboli in arterial circulation**	Bold to pass
	4. From where do they arise and where do they lodge?	<u>Sources:</u> 80% from **intracardiac mural thrombi** (2/3 L vent wall infarcts, ¼ L atrial dilation/AF) Other sources: aortic aneurysms, ulcerated atherosclerotic plaques, valvular vegetation, paradoxical emboli, unknown <u>Lodgement Sites:</u> Lower limbs (75%), brain(10%), Other: intestine, kidneys, spleen, upper limbs	Bold + 2/4 sources and 2/4 sites to pass
	Bonus Question Describe the process of infarction from arterial occlusion. Prompt: What are the features that influence the development of an infarct?	Area of ischaemic necrosis: dominant histologic characteristic is ischaemic necrosis - White infarcts occur in solid organs with end-arterial circulation - Acute inflammation happens within hours; reparative response follows - Factors influencing infarct development: nature of vascular supply (end artery vs presence of collateral blood supply), rate of occlusion, vulnerability to hypoxia, oxygen content of blood, calibre of occluded vessel,	

Stem: We will now move on to Pharmacology. A heparin infusion is commenced.			
Question 3 Heparin (pp 604-607) **Subject:** Pharm LOA: 1	1. How does heparin act?	Heparin binds endogenous **antithrombin and enhances its activity. Antithrombin inhibits factors IIa, IXa and Xa** by complexing with them and inducing a conformational change.	Bold to pass
	2. How may heparin be administered?	**IV vs SC.** Continuously (following bolus) vs intermittent. Therapeutic vs prophylactically	Bold to pass
	3. What are the potential adverse effects?	**Bleeding,** allergy, alopecia, osteoporosis, **HIT,** mineralocorticoid deficiency	Bold + 1 to pass
	4. What are the advantages of low molecular weight heparins compared to unfractionated heparin?	Have equal efficacy, increased SC bioavailability, require less frequent dosing, and less monitoring. Shorter chain heparin with less effect on thrombin (IIa).	Demonstrates understanding

Stem: We will now move on to Physiology. The patient has reverted to sinus rhythm.			
Question 4 **Subject:** Phys LOA: 1	1. Describe the normal sequence of electrical excitation of the cardiac conduction system and cardiac muscle?	Normal sequence of depolarisation: **SA Node** **Atria** **AV Node** Bundle of His **Major bundles (Right and left)** Purkinje fibres **Ventricular muscle**	Bold to pass.
	2. What are the common mechanisms that cause abnormalities of cardiac conduction?	Abnormal pacemakers Re-entry circuits Conduction deficits Prolonged repolarisation Accessory pathways Electrolyte disturbance	4 to pass
	3. Please draw and explain the action potential of a cardiac pacemaker cell Prompt: Which electrolytes are responsible for each phase of the action potential?	Pre-potential is initially due to a decrease in K+ efflux, then completed by Ca2+ influx through CaT channels. The action potential is due to influx of Ca2+ via CaL channels. Repolarisation is due to K+ efflux  	To pass: Correct shape of graph Know ion fluxes: • Pre-potential decrease K+ efflux/Ca2+ influx • Action potential Influx Ca2+ • Repolarisation K+ efflux

Stem: 85 year old man presents to your ED in urinary retention, the day after a prostate biopsy. On PR examination, his prostate is extremely tender and you suspect prostatitis. We will start with Pathology.

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Acute inflammation – questions to focus on acute inflammation not prostatitis specifically (as this is an LOA 3 topic) (pp 48-56) **Subject:** Path LOA: 1	1. What are the three major components of acute inflammation?	1. **Dilation of small vessels** leading to increase blood flow. 2. **Increased permeability of the microvasculature** enabling plasma protein and leucocytes to leave the circulation. 3. **Emigration of leucocytes from the microcirculation to the site of injury**.	Bold to pass Neutrophils predominate in the early inflammatory (6 – 24 hours) infiltrate and are later replaced by monocytes and macrophages (24 – 48 hours).
	2. How are leucocytes delivered to the site of injury? PROMPT: What are the three processes that leucocytes undergo to move from the blood to the site of injury?	This is a multistep process mediated and controlled by adhesion molecules and chemokines. 1) **Margination:** Occurs when leucocytes adopt peripheral position along the epithelium. Rolling (transient adherence mediated by selectins), activation and firm attachment (mediated by integrins) to the endothelium. 2) **Transmigration** (diapedesis): across the endothelium. Migration through interendothelial spaces typically in post capillary venules. 3) **Chemotaxis**: Leucocytes move toward the site of injury along a chemical gradient of chemoattractants, which can be exogenous or endogenous.	Bold to pass Polymerisation of actin at the leading edge of the cell establishes a “front wheel “ drive in the direction of the injury
	3. Name some of the chemoattractants responsible for chemotaxis?	Most common exogenous agent **Bacterial products**. Endogenous: IL-8, **C5a**, and Leukotriene B4. All bind to specific receptors and promote polymerisation of actin.	Bold + 1
	4. What chemical mediators are responsible for pain, fever and tissue damage?	**IL-1,** TNF, Prostaglandins, Bradykinin, Neutrophil and Macrophage Lysosomal enzymes, Oxygen metabolites, NO.	Bold + 1

Stem: We will now move on to Anatomy.			
Question 2 Male pelvis model (Model No: MS2) **Subject:** Anat LOA: 2	1. Name the parts of the male pelvis visible on this model. Prompts: What are the… • Skeletal features • Organs of the urogenital system • Vascular structures	1 **Pubic bone**, 2 **Sacrum**, 3 Coccyx, 4 **Urinary bladder** (a. apex, b. fundus, e. ureteral orifice, f. trigone), 5 **Prostate**, 7 Seminal vesicles, 8 Spermatic duct, 9 **Ureter**, 10 Urethral corpus cavernosum, 11 **Penis**, 12 Glans penis, 13 External urethral orifice, 14 Ischio-cavernosus muscle, 15 **Testicle**, 16 **Epididymis**, 17 Pampiniform plexus, 18 Testicular artery, 19 Cremaster muscle, 20 **Rectum**, 21 **Common iliac artery,** 22 **Common iliac vein**, 23 Peritoneum, 24/25 **Inguinal ligament**, 26 Femoral canal	Bold to pass
	2. Name the parts of the urethra	- pre-prostatic part (surrounded by internal urethral sphincter - **prostatic part** - **membranous (intermediate) part** (surrounded by external urethral sphincter) - **penile (spongy) part**	Bold to pass
	3. What is the innervation of the urethra?	- **prostatic nerve plexus** to first 3 parts above - **dorsal n. of the penis** (from pudendal n.) to penile part	1 of 2 bold to pass
	Bonus Describe the anatomy of the prostate	• Surrounds prostatic part of the urethra (about the size of a walnut). • base sits near the neck of the urinary bladder • Apex is next to the urogenital diaphragm. • Covered in a thick fibrous capsule, which houses the prostatic plexuses of nerves and veins. • 5 lobes: anterior, middle, posterior, & 2 lateral • Arterial supply via inferior vesical, internal pudendal, and middle rectal arteries. • Venous drainage via the prostatic venous plexuses, which is located around the base and sides of the prostate.	

Stem: We will now move on to Pharmacology. Treatment with Ciprofloxacin is commenced.			
Question 3 Fluoroquinolones (Chp 46) **Subject:** Pharm LOA: 2	1. What class of drug is Ciprofloxacin?	**Fluoroquinolone**	Bold to pass
	2. What is its mechanism of action?	**Blocks DNA synthesis** by inhibiting bacterial topoisomerase II and IV	Bold to pass
	3. What is its antimicrobial spectrum?	**Excellent Gram neg activity and moderate Gram positive activity**. Methicillin susceptible strains of S *Aureus* are susceptible, but methicillin resistant Staphylococci are resistant. Also active against agents of atypical pneumonia – Mycoplasma and Chlamydiae Intracellular pathogens such as Legionella and Mycobacterium. Ciprofloxacin the drug of choice for anthrax.	Bold + 1 to pass MIC for Gram neg are 1-2 mcg/mL.
	4. What are the potential adverse effects of Fluoroquinolones?	• **Prolonged QT** (with some), • Nausea, vomiting, diarrhoea (inc. C difficile) • Rash • Abnormal LFTs • Photosensitivity • Hyperglycemia in diabetics, • Growing cartilage damage (not routinely recommended for < 18 yo or pregnancies) • Tendonitis • Allergy	Bold + 2 dot points

Stem: We will now move on to Physiology.			
Question 4 Micturition (pp 693-695) **Subject:** Phys LOA: 2	1. Describe the neurological pathways involved in normal micturition.	**Spinal reflex mediated by S2, S3 and S4 nerve roots. Facilitated and inhibited by higher centres; subject to voluntary control.** - First urge to void at 150ml. Marked fullness at 400ml - sudden rise in intra-vesical pressure triggers reflex contraction. **Micturition reflex:** - Stretch receptors in bladder wall. Afferent limb in pelvic nerves. **Parasympathetic efferent fibres** (via same pelvic nerves) mediate contraction of detrusor muscle. **- Pudendal nerve (S2, S3 and S4)** permits voluntary contraction of perineal muscles/external urethral sphincter, to slow or halt flow. - Sympathetic nerves to bladder play no role in micturition	To Pass: Spinal Reflex Parasympathetic control higher centre control
	2. Describe the muscles involved in micturition.	**1. Bladder:** smooth muscle arranged in spiral, longitudinal and circular bundles. Circular bundle is called the **detrusor muscle. Contraction of detrusor is responsible for involuntary emptying.** **2. External urethral sphincter – skeletal muscle sphincter of the membranous urethra. Relaxes during micturition. This is voluntarily controlled.** 3. Perineal muscles. Relaxes during micturition. Also voluntarily controlled. 4. In males, urine left in urethra expelled by several contractions of bulbocavernosus muscle. 5. Contraction of abdominal wall muscles aids expulsion of urine. NB: Internal urethral sphincter (smooth muscle bundles passing on either side of urethra) plays no apparent role in micturition.	Bold to pass
	3. What prevents vesico-ureteric reflux?	**Oblique passage of ureters through bladder wall** keeps ureters closed except during peristaltic waves.	Bold to pass

Stem: A 70 yo woman with metastatic lung cancer presents with polydipsia and polyuria. We will start with Pathology.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Lung Tumours (pp 721-731) **Subject:** Path LOA: 2	1. What are recognised aetiological factors in lung cancer? Prompt for detail: Are you aware of any environmental factors that place you at greater risk for lung cancer?	**Tobacco smoking -** 87% of cancers in recent or current smokers- 10x increase in risk**,** Statistically associated with daily amount; inhalation tendency; duration of habit**,** Histologic changes in respiratory epithelium in smokers **Industrial Hazards** Ionising radiation, Uranium, Asbestos **Air pollution -** Radon **Molecular genetics -** Familial clustering **Precursor lesions -** Squamous dysplasia and CIS, Atypical adenomatous hyperplasia, Diffuse idiopathic pulm neuroendocrine cell hyperplasia	Tobacco smoking and 2 other bold to pass
	2. What are the most common presenting symptoms of lung cancer?	**Cough** (75%), Loss of weight(40%), Chest pain (40%), Dyspnoea (20%), Haemoptysis	3 to pass
	3. What are the clinical effects of local lung tumour spread?	• **Airway obstruction ->pneumonia, abscess, lobar collapse**, Lipoid pneumonia, • **Obstruction of SVC** leading to SVC syndrome • **Pleural effusion**, • **Pericarditis or tamponade**, • **Hoarseness** (r/c laryngeal n), • **Dysphagia** (oesophagus), Rib destruction, • **Diaphragmatic paralysis** (phrenic nerve) • **Horner syndrome** (sympathetic ganglia)	5 of 8 bold to pass
	4. What paraneoplastic syndromes are associated with lung cancer? PROMPT: What hormones might be produced?	Clinically significant in 1-10% of patients ACTH- **Cushing's** (predominantly small cell) ADH—**hyponatraemia** (predominantly small cell) PTH, PTH related peptide, PGE and some cytokines**- hypercalcaemia** (predominantly small cell/squamous cell)**,** Calcitonin-hypocalcaemia, Gonadotrphins-	2/3 bold + 1 other to pass

		gynaecomastia, 5HT and bradykinin- wheeze/flushing	

Stem: We will now move on to Physiology. She has a raised corrected calcium level.			
Question 2 Calcium metabolism (pp 377-378) **Subject:** Phys LOA: 1	1. Where in the body is Ca2+ stored?	**Bone:** 99%, **Plasma – bound to protein**, **Plasma – unbound (free/ionised**) - important second messenger and is required for coagulation, nerve function and muscle contraction.	Bold to pass
	2. How is the plasma Ca2+ level regulated? Prompt: What hormones increase or decrease plasma Calcium?	**Parathyroid Hormone**: Increases plasma Ca2+ by mobilising Ca2+ from bone. Increases Ca2+ reabsorption in kidney. Increases formation of 1,25 DHCC in the kidney. **1, 25 DHCC** (from Vit D) increases Ca2+ absorption from intestine and kidneys. **Calcitonin** (from thyroid) lowers circulating Ca2+ levels. Effect by inhibition of bone reabsorption. It also increases Ca2+ excretion in urine Glucocorticoids – decrease plasma Ca2+ by inhibition osteoclast formation and activity. Oestrogens – inhibit stimulatory effects of cytokines on osteoclasts Growth Hormone – increases Ca2+ excretion in urine & absorption in intestine. Net balance may be positive. Hypercalcaemia is a complication of cancer. Raised Ca2+ from either: - bone erosion (local osteolytic hyperCa2+ - elevated Parathyroid hormone related protein (PTHrP)	Bold and their effects on plasma Ca2+ (increase / decrease)
	3. How does bone resorption occur	**Osteoclasts** are monocytes that develop from stromal cells under influence of RANKL. • Attach to bone via integrins in sealing zone of the membrane. • Hydrogen dependent proton pumps move into cell and acidify the area. • Acid dissolves hydroxyapatite and acid proteases break down collagen.	Osteoclasts + 1 other RANKL – receptor activator of nuclear factor kappa B ligand

		• Products move across osteoclast into interstitial fluid.	

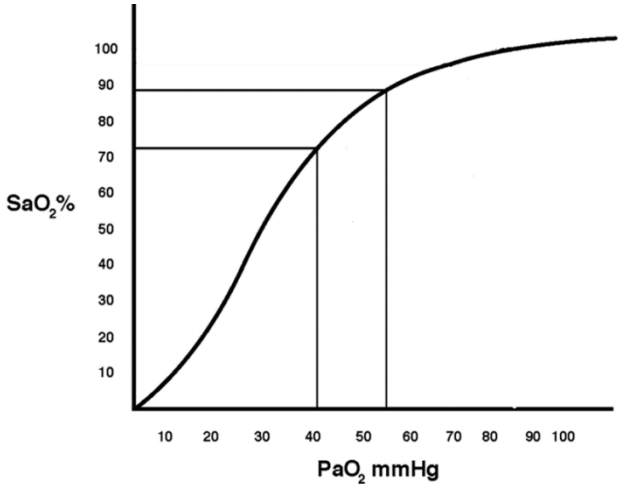
Stem: We will now move on to pharmacology. Treatment is commenced with normal saline and frusemide.			
Question 3 Frusemide (pp 258-260) **Subject:** Pharm LOA: 1	1. How does frusemide exert its action?	**Selectively inhibits Na+-K+-2Cl- transporter in thick ascending limb of loop of Henle thus preventing resorption of Na+ & Cl-** Abolishes counter-current concentrating mechanism leading to dilute urine. Increased prostaglandin synthesis -> inhibition of salt transport in thick ascending limb -> increased renal blood flow, decreased pulmonary congestion, decreased LV filling pressures	Bold to pass
	2. What are the pharmacokinetic properties of frusemide?	• Rapid absorption after oral admin • Oral bioavailability 50% (range 10 –100%) • Highly protein-bound (>95%) • 50% conjugated in kidney & 50% excreted in urine unchanged (tubular secretion) • Elimination t1/2 1.5 – 2 hours • Peak effect 30 minutes IV / 1 hour oral	List 3
	3. What are the potential adverse effects of frusemide? PROMPT: What are the electrolyte disturbances?	• Electrolyte disturbances • **hypokalaemia**, • hyponatraemia, • hypomagnesaemia, • hyperuricaemia • Postural **hypotension** & dizziness • Metabolic Alkalosis • Allergy - rash, eosinophilia, interstitial nephritis • Increased LDL & triglycerides, decreased HDL • Hyperglycaemia • Ototoxicity (high dose IV)	Bold plus 2

Stem: We will now move on to Anatomy. She has limited shoulder movement due to bony metastases.			
Question 4 Shoulder (bone /model) **Subject:** Anat LOA: 1	1. What are the articulating surfaces in the shoulder joint	Ball-and-socket synovial joint, Rounded **head of humerus**, Shallow **glenoid cavity of scapula**, deepened by labrum	Bold to pass
	2. What structures stabilise the shoulder joint?	Fibrocartilaginous glenoid labrum. Coraco-acromial arch, Anterior glenohumeral ligaments Coracohumeral ligament Transverse humeral ligament **Rotator cuff (SITS) muscles, Supraspinatus, Infraspinatus, Teres minor, Subscapularis**	Rotator cuff (3/4 muscles), plus 2 others to pass Need to show understanding that there are different elements that contribute to stability.
	3. What muscles are responsible for abduction and adduction of the shoulder joint?	Abduction -**Deltoid** (esp acromial part)**,** **Supraspinatus** (initiates), + upward movement of scapula Adduction -**Pec major** and **lat dorsi** acting in concert**, Teres major** and **long head of triceps** (synergists)	Bold to pass 2/4 Bold to pass
	Bonus What muscles are responsible for the other movements of the shoulder?	<u>Flexion -</u>Pectoralis major (clav head)**,** Deltoid (clav and anterior acromial parts)**,** Coraco-brachialis (synergist) <u>Extension -</u>Spinal part of deltoid**,** Lat dorsi, teres major, long head of triceps (synergists) <u>Medial rotation-</u> subscapularis, pec major, lat dorsi, teres major, deltoid-clavicular part (synergists) <u>Lateral rotation -</u>infraspinatus, teres minor, deltoid- spinal part (synergists) <u>Circumduction</u>	
	Bonus: Outline the bursae of the shoulder joint.	Subscapular bursa- located between neck of scapula and subscap tendon. Protects tendon. Subacromial (subdeltoid) bursa -Between acromion, CA ligament and deltoid superiorly, and supraspinatus tendon and joint capsule inferiorly Facilitates movement of supraspinatus tendon	

Stem: A 20 year old woman presents with a rash and dyspnoea. Her oxygen saturation is low. **We are starting with physiology**			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 VQ mismatch (West Chp 5) **Subject:** Phys LOA: 1	1. What are the causes of hypoxemia in a patient breathing room air? 2. How does the ventilation/perfusion ratio change in different regions of the lung? 3. What is the effect of ventilation-perfusion inequality on arterial PO2 and arterial PCO2? *Prompt if required* Why does V/Q inequality cause reduced arterial PO2 while arterial PCO2 remains relatively normal?	1. Hypoventilation 2.Diffusion limitation 3. Shunt 4. Ventilation/perfusion (V/Q) inequality **V/Q ratio is high at apex (blood flow minimal) and decreases down the lung to the base.** PO2 highest at apex but blood flow is greatest at the base where PO2 is lowest (can be 40mmHg difference) Respiratory exchange ratio (CO2 output/O2 uptake) highest at apex where blood flow is lower **Much greater influence on PO_2 than CO_2.** **O2 dissociation curve nonlinear**. Areas with high V/Q ratio add relatively little O2 with increased ventilation. Whereas areas with low V/Q ratio have lower PO2 (close to mixed venous) overall PO2 is reduced **CO2 dissociation curve is linear** in the working range. Chemoreceptor stimulation increases ventilation and CO2 output especially in lung areas with high V/Q ratios. normal PCO2 (minimal change)	**3 of 4 to pass** **BOLD + general concepts to pass** **BOLD + demonstrates understanding**
Stem: We are now moving to anatomy. A CXR is performed.			
Question 2 CXR including understanding of pleural reflections **Subject:** Anat LOA: 2	1. Demonstrate the lobes of the lungs 2. What are their immediate relationships (if not answered in Q1) Prompt: what are the boundaries of the lobes	**Right superior mediastinum to apex ; right upper lobe** RUL: apex -horizontal fissure /upper right mediastinum medially **Right heart border; right middle lobe** RML: right heart border & horizontal fissure (superior border 4th rib) to 6th costal cartilage	**Demonstrate all 5 lobes**

		Left upper mediastinum to apex; left upper lobe LUL: Apex- 4LICS parasternal line,6th LICSMCL & 5th LICS AAL **Left heart border ; Lingula** lobe : left heart border **Lower lobes posteriorly, sit over domes of diaphragms** rise as high as 3rd intercostal space posteriorly R & L lower lobes: from Obliques fissures (T2 spinous process-6th costal cartilage anteriorly) to level T10 spinous process posteriorly , 10th ribs at scapular line & 8th ribs in MAL.	
	3. Describe the surface anatomy of the parietal pleura	supraclavicular fossa, medially follow the middle of the sternum to the level of the 6th intercostal cartilage, deviates laterally reaching MCL at 8th rib, MAL at the 10th rib, paravertebral line 12th rib. Notch on Left.	**Reasonable description**
Stem: We are now moving to pathology. The rash is diagnosed as Varicella Zoster			
Question 3 Varicella Zoster (p 353) **Subject:** Path LOA: 1	1. What are the 2 clinical conditions caused by this virus	Chicken pox and shingles	**Both**
	2. Describe the pathogenesis and clinical course of infection with this virus Prompt: start with how the virus is transmitted	Starts with aerosol or direct contact spread →haematogenous dissemination → vesicular skin lesions → vesicles rupture, crust over then heal Some virus lies dormant in dorsal root ganglia and reactivated later with immunosuppression	**Reasonable sequence**
	3. What are the complications of chicken pox	Lung → interstitial pneumonia Nervous system - encephalitis, transverse myelitis Skin and mucous membranes → shingles, bacteria superinfection Gut – necrotising visceral lesions	**3 to pass**

Stem: . We are now moving to pharmacology. Treatment is commenced with Acyclovir			
Question 4 Acyclovir (pp 862-864) **Subject:** Pharm LOA: 2	1. What are the indications for acyclovir in the ED?	**HSV – encephalitis**; VZV, patients with HIV, genital herpes	**Bold**
	2. Describe the mechanism of action of acyclovir.	**Inhibition of viral DNA synthesis** • Irreversible binding to viral DNA polymerase. • Incorporation in to viral DNA with termination Specificity for virus-infected cell (virus-specific thymidine kinase).	**Bold**
	3. Describe the pharmacokinetics of acyclovir?	**Short half life 2.5 hrs** (5xdaily dosing oral); low oral bioavailability; mostly excreted unchanged in urine; CSF 20-50% of plasma; wide distribution	**Bold + 1 other**
	4. Name some side effects of acyclovir	Nausea, vomiting, diarrhoea, headache, reversible renal toxicity Neuro – tremor, delirium, seizures	**2 to pass**

Stem: We are starting with physiology. A 40 year old man with acute pancreatitis Is hypoxic.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Gas transport to the tissues (West Chp 6) **Subject:** Phys LOA: 1	How is oxygen carried in the blood? Prompt: Which dominates Draw and label the oxygen dissociation curve What are the implications of this curved shape? Prompt: what happens to the top + bottom	**Dissolved**: amount dissolved proportional to partial pressure (Henry's law)-0.3ml O2/100ml blood at PO2 100mmHg **Most Combined with Hb**: 20.8 ml O2/100ml blood(at Hb level of 15g/dl)  UPPER- If PO2 alveolar gas falls (eg ARDS in acute pancreatitis) loading of O2 little affected. LOWER- Steep lower part means large amounts of O2 unloaded at peripheral tissues for only small drop in capillary PO2	**Bold** **Draw correct shape and have 2 points of saturations** eg 27mmHg SaO2 50%, 30mmhg SaO2 60% 40mmHg SaO2 75%, 56mmHg SaO2 90%, 80mmHg SaO2 95%, 90mmHg SaO2 97% Explain **concept** of loading and unloading of oxygen
Stem: We are now changing to pathology.			
Question 2 Acute Pancreatitis (pp 893-896) **Subject:** Path LOA: 1	1. What are the potential causes of this man's pancreatitis? 2. What is the likely pathogenesis of acute pancreatitis?	1. **Gallstones, alcohol,** iatrogenic, viral, hyperlipoproteinaemia, hypercalcaemia, drugs, trauma, shock, vasculitis, genetic mutations, scorpion bite, atheroembolism, duct obstruction (tumour, parasites etc) **2. Autodigestion of the pancreatic substance by inappropriately activated pancreatic enzymes,** eg trypsinogen	**1. Bold plus 1** **2. Bold**

	3. What are the acute complications of severe pancreatitis?	Causes interstitial inflammation and oedema, proteolysis, fat necrosis and haemorrhage 3. Haemolysis, DIC, fluid sequestration, ARDS, diffuse fat necrosis. Peripheral vascular collapse; shock; acute renal tubular necrosis	**3. 3 answers to pass**
Stem: Another patient arrives with a stab wound to the back of his right thigh. **We will move now on to anatomy**			
Question 3 Posterior Thigh Muscles photo **Subject:** Anat LOA: 1	Identify the structures in this photo of the posterior thigh. What are the clinical features of a severed sciatic nerve in the upper thigh? Prompt: what does the sciatic nerve supply distal to this point.	**Sciatic nerve (19),** gluteus maximus (5), long head biceps (9), semitendinosus (22), semimembranosus (21), ischial tuberosity (8), gracilis (6), iliotibial tract (7), adductor magnus (1), popliteal artery and vein(16,17), quadratus femoris (18) **Motor**: SN supplies all posterior thigh muscles (depending on level of injury these may be affected), all leg and foot muscles **l**oss of hip extension and knee flexion**.** All ankle (**Flex/Ext, inversion, eversion**) and **toe movements** lost. **Sensory:** skin of most of leg and foot ->**posterior and lateral leg, sole** of **foot, lateral and dorsum of foot.**	**Bold + any 4 others** Prompt bold if required. **Motor 3 bold** **Sensory 3 bold**
Stem: We will now move onto pharmacology. He is agitated. You use Olanzapine to sedate him.			
Question 4 Olanzapine & atypical antipsychotics (Chp 29) **Subject:** Pharm LOA: 2	1. By what routes can Olanzapine be administered? 2. What dose, and route would you use in this situation? 3. What are the advantages of olanzapine over older "typical" antipsychotics? Prompt: e.g. chlorpromazine 4. What are some of its disadvantages? Prompt if needed – what about longer term effects	**1. Oral** (Tab or wafer); **Parenteral**- IMI, Depot IMI 2. Gives dose (10-20mg), same for each route 3. less hypotension; less tachycardia; **less extrapyramidal effect**; high clinical potency; less effect on prolactin; more effective vs neg&pos psychotic symptoms and cognition; multiple routes of admin 4. Anticholinergic effects; lowered seizure threshold; weight gain; DM; Hyperlipidaemia; expense	**Bold** **Reasonable answer** **Bold** **2 disadvantages**

ACEM PRIMARY VIVA C **Friday Morning Session 3** ***Candidate Number:*** **AGREED MARK:**

Stem: A 90 yo lady arrives by ambulance with confusion and agitation. She is hypotensive. **We will start with Physiology.**			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Baroreceptors / regulation of blood pressure (pp 589-592) **Subject:** Phys LOA: 1	1. What are baroreceptors and where are they located?	**1. Stretch receptors** in the walls (adventitia) of the heart & blood vessels, impt in control of BP (esp short term). Arterial- carotid sinus/Ao arch. Low pressure- Atria at entrance of IVC and SVC, Pulm veins and pulm circulation.	**Bold plus 2 locations.**
	2. What stimulates these receptors?	**2. Distension** of the structures above. More sensitive to pulsatile than constant pressure. Maximal firing at 150mmHg (@ Carotid sinus)	**Bold**
	3. What are their effects?	**3.** Inhibit tonic sympathetic drive& inc vagal drive=>**vasodilation, venodilation, hypotension, bradycardia (tachycardia in low pressure baroreceptors), ↓CO**. Allows rapid adjustments in BP in response to abrupt changes in posture, blood volume, cardiac output, or peripheral resistance	**3/5 end effects**
Stem: We will now move on to Pharmacology. Haloperidol is suggested for her agitation.			
Question 2 Haloperidol (pp 503-513) **Subject:** Pharm LOA: 2	What are the pharmacodynamics of haloperidol?	Butyrophenone- **high potency D2 receptor effects (dopamine antagonist)**, **high extra-pyramidal side effects**, **low sedative**, low hypotensive, minimal anticholinergic effects, minimal 5-HT and H1 blockade effects.	**2/3 Bold**
	How does olanzapine differ?	Thienobenzodiazepine- less D2 receptor effects, **high 5-HT receptor blockade effects**, **low extrapyramidal effects, medium sedative**, low hypotensive and anticholinergic effects, low H1 blockade effects	**2/3 Bold**

Stem: We will now move on to Anatomy. A recent CT brain is available.			
Question 3 CT brain **Subject:** Anat LOA: 1	1.Identify the intracranial structures visible on this CT (level of anterior & posterior horns lat ventricles)	Lobes: **frontal temporal parietal occipital** **Lat ventricle** : anterior and posterior horns 3rd ventricle, Caudate nucleus, choroid plexus Lentiform nucleus (putamen & globus pallidus) **Thalamus,** Septum pellucidum, **Falx** Anterior & posterior limbs of **internal capsule** Sylvian fissure	**Bold to pass** **Prompt if required**
	2. What arteries supply the main areas of the cerebral cortex? Prompt: point	**ACA** area anterior to anterior horns lat ventricle (**frontal and parietal lobes medially and superiorly**) **MCA** area between the ant & post horns LV (**most of lateral surface anterior, parietal, and temporal lobes**) **PCA** area posterior to posterior horn LV (**Inferior and medial aspects of occipital and temporal lobes**)	**Ant, Middle and Post CA** **Reasonable distribution**
	3. Describe the venous drainage of the cerebral hemispheres	3. Superior cerebral veins (superolateral surface of the brain) > **superior sagittal sinus**. Inferior and superficial middle cerebral veins (inferior, posterior and deep aspects of cerebral hemispheres) > straight, transverse and superior petrosal sinuses. **Great cerebral vein** (midline vein formed from the paired internal cerebral veins) >merges with inferior sagittal sinus to form the straight sinus . Eventually terminate in **Internal Jugular veins**	**2/3 bold**

Stem: We will now move on to Pathology. On examination she has a rigid, tender abdomen.			
Question 4 Ischaemic Bowel (pp 791-793) **Subject:** Path LOA: 1	What conditions can lead to infarction of bowel	**1. Acute arterial obstruction** Atherosclerosis, Aortic aneurysm, Hypercoagulable state, OCP use, Embolism **2. Intestinal hypoperfusion** cardiac failure, shock, dehydration, vasoactive drugs <u>systemic vasculitis</u> HSP, Wegeners granulomatosis <u>Mesenteric venous thrombosis</u> Hypercoagulable state, Invasive neoplasm, Cirrhosis, Trauma, Abdominal masses <u>Miscellaneous</u> Radiation, Volvulus, Stricture, Amyloid, diabetes	**BOLD to pass** **Minimum 2 from each bolded group** **2 from non-bolded**
	What are the clinical features of ischaemic bowel?	Severe **pain**, may be transient. Tenderness, peritonism, nausea, vomiting, bloody diarrhoea, melaena, shock, hyper/hypothermia, sepsis	**Bold + 3 features**
	What parts of the bowel are most susceptible to ischemic injury And why?	**Watershed zones** - Splenic flexure, sigmoid colon, rectum - Located at end of arterial supply Surface epithelium : Villi more at risk than crypts Intestinal capillaries run from crypts up villi to surface	**Must be able to explain why watershed zones are most at risk**

ACEM PRIMARY VIVA D **Thursday Morning Session 1** ***Candidate Number*:** **AGREED MARK:**

Stem: A patient presents with a penetrating eye injury. He has been given oxycodone. Commencing with pharmacology			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Oxycodone (p 558) **Subject:** Pharm LOA: 1-2	Describe the pharmacokinetics of oxycodone? Prompt: Describe the pharmacokinetics of opiates. What adverse effects might you anticipate? When prescribing oxycodone what prescribing strategies may help in reducing the development of, dependence.	**Oral commonly** Good oral absorption High Vd **Low first pass metabolism CW others** 10 morphine = 4.5mg oxycodone duration 3-4h, longer if CR formulation. Hepatic met Sedation/Respiratory depression/N+V/hypotension/dysphoria/biliary colic/pruritis/caution in renal failure **Smaller doses at longer intervals**/establish goals at start of Rx/**limit doses/use of other analgesics**/frequent evaluation of ongoing need/**use of modified CR formulations**	Bold plus one more N+V a particular concern in context of penetrating eye injury 3 to pass 2 to pass
Stem: Moving on to physiology You assess his visual acuity as 6/24.			
Question 2 Eye / Acuity / Vision (pp 178 -183) **Subject:** Phys LOA: 2	How is visual acuity measured? What does the fractions of a VA of 6/24 represent? What factors influence visual acuity? Why is the fovea important for visual acquity	Measurement from **Snellen chart** viewed at a distance of **6m** or 20 feet; **6/24 indicates reduced VA** **Optical factors** The state of the image forming mechanisms/sharpness of focus **Retinal factors** the state of the cones **Stimulus factors (**illumination, brightness of the stimulus, contrast between stimulus and background, length time exposed to stimulus); sensitivity and interpretative ability of the brain Resolving power of the eye, property of the cones fovea is the point where **VA is greatest**; fovea is the centre of the macula, a thinned out rod free portion of the retina where the **cones** are densely packed & each synapses on a single bipolar cell	numerator is the distance at which the chart is read; the denominator is the **smallest line** that can be read; 6/6 indicates normal vision; 2/3 to pass One of bold

Stem: Moving on to anatomy. He has abnormal eye movement			
Question 3 Eye (Model) – (model no. F 13) **Subject:** Anat LOA: 2	Identify the muscles responsible of eye movement. Describe their actions. What nerves supply these muscles? How are the actions of these muscles tested clinically? *Prompt: Why is the "H" pattern used?*	**Recti: Superior** (**elev**, add, med rot); **Inferior** (**dep**, add, lat rot); **Medial** (**add**); **Lateral** (**abd**) **Obliques: Superior** (**dep**, abd); **Inferior** (**elev,** abd) **Oculomotor (III) N** to all, except Abducent (VI) N to Lateral R, and Trochlear (IV) N to Sup Obl In **Abd** (LR): Elev (SR) and Dep (IR) In **Add** (MR): Elev (IO) and Dep (SO)	All Bold to pass Bold plus one to pass abd and add isolates recti and obliques to pass
Stem: Moving on to pathology: Six weeks later ne develops sympathetic ophthalmia, which is a **Type IV** hypersensitivity reaction			
Question 4 Type 4 hypersensitivity reaction (pp 205-208; 1356) **Subject:** Path LOA: 1	1. Describe the sequence of events that lead to this reaction. Prompt: what cells are involved? 2. What tissue changes would occur 3. Name other examples of Type IV hypersensitivity reactions.	Injury **Initiated by antigen** sensitised CD4+ or CD8+ **T cells** Retinal antigens may be transported in the lymphatics of the damaged eye Reaction may occur in both eyes causing a Pan Uveitis. CD4+ predominate in autoimmune disease CD8+ in post infectious (esp viral) reactions **Can be cytokine** (CD4+ Th1 or TH17 cells involved) **or direct cellular** (Cyotoxic lymphocyte) **mediated** tissue injury (either satisfactory). Perivascular cellular infiltrates, tissue oedema, granuloma formation, cell destruction. Type I diabetes Multiple sclerosis Rh arthritis Inflammatory bowel disease Guillain Barre Contact sensitivity dermatitis Tuberculin reaction Granulomatous diseases Viral hepatitis	Requires antigen and either cytokine or direct cellular mechanisms to pass **(2/4 to pass)** **(2 examples to pass)**

ACEM PRIMARY VIVA D Thursday Afternoon Session 2 ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: An elderly woman is brought to your ED hypothermic and unconscious. She is intubated. Commencing with pharmacology			
Question 1 Biotransformation – Phase 1 and 2 reactions with an emphasis on Suxamethonium (Chp 4) **Subject:** Pharm LOA: 1	What is drug biotransformation? Describe phase 1 and phase 2 reactions? How is Suxamethonium metabolised? Why may a patient have a prolonged paralysis following Sux	Drug metabolism to allow drugs to become **inactive or by increasing excretion** by making them more hydrophilic, or by metabolising them to less active agent**.** Phase 1 – **unmasking functional group** (-OH, -NH2, -SH) to become more polar metabolite. Includes oxidation, deamination, hydrolysis, reductions Phase 2**- conjugation with endogenous substrate** to become highly polar conjugate **Rapid phase 1 hydrolysis** by **butyrycholinesterase and pseudocholinesterase** in liver and plasma Genetically deficient in BCHE so slowed metabolism	Bold Bold One of the bold
Stem:Moving on to physiology			
Question 2 Hypothermia / thermoregulation (pp 316-320) **Subject:** Phys LOA: 1	By what processes does the body lose heat? How does the body produce heat? What temperature-regulating mechanisms are activated by the cold? What part of the brain controls the reflex responses activated by cold?	**Radiation & Conduction** (70% of loss at 21 °C) **Vaporization of sweat** (27%) Respiration (2%) Urination & defecation (1%) **Basal metabolic processes** Food intake **Muscular activity** Shivering Hunger Increased voluntary activity Increased secretion of Adr + NorAdr Decreased heat loss mechanism Cutaneous vasoconstriction Curling up Horripilation The posterior **hypothalamus**	Bold to pass Bold to pass 4 to pass bold
Stem: Moving on to pathology.			

Question 3 Cerebrovascular Disease (pp 1290-1295) **Subject:** Path LOA: 1	What are the types of cerebral ischemic injury? Prompt: Describe the patterns cerebral ischemic injury What are the causes of focal cerebral infarction? Prompt: Give examples What are the pathological effects of hypertension on the brain?	**Global cerebral ischemia** (ischemic/ hypoxic encephalopathy) when there is a **generalised reduction** of cerebral perfusion **Focal cerebral ischemia** follows reduction of blood flow to a **localised** area of the brain **Embolic** (from cardiac mural thrombi; thromboemboli from arteries, esp. carotid; paradoxical assoc with cardiac anomalies; tumour, fat or air), **thrombotic arterial occlusion/** in situ thrombosis (large vessel disease); **Vasculitis** (small vessel disease) infectious (immunosuppression and aspergillus, CMV encephalitis, syphilis, TB); non-infectious eg PAN, primary angiitis; **Others** eg amphetamines, cocaine, heroin; dissecting aneurysm extracranial arteries; hypercoaguable states Lacumar infarcts (in lenticular nucleus, thalamus, internal capsule, deep white matter, caudate nucleus, pons); slit haemorrhages; hypertensive encephalopathy; massive intracerebral haemorrhage)	Both types and description 3 causes plus 1 example of each 4 out of 4
Stem: Moving on to anatomy She has a swollen right elbow.			
Question 4 Elbow X-ray **Subject:** Anat LOA: 1	Identify the bony features on this Xray What factors determine the stability of the elbow joint? Prompt – What are the ligaments of the elbow	Medial / Lateral epicondyles, capitulum, olecranon, radius – head/neck, olecranon fossa, coronoid fossa, trochlea, proximal radio-ulnar joint, coronoid process of ulna **Bony factors – shape of trochlea / olecranon fossa** Joint capsule – fibrous joint capsule weak **Ligaments** **– radial collateral ligament** – lateral epicondyle and blends with the **annular ligament** of the radius (which holds the radial head in the radial notch of the ulna). - medial **ulnar collateral ligament** from medial epicondyle to the coronoid process and olecranon of the ulna - 3 bands 1. Anterior – strongest 2. Posterior – weakest 3. oblique – deepens trochlear notch Muscles – biceps, brachialis,(BR), triceps RCL & UCL & annular ligament	6 to pass Bone and ligaments 3 of 4 bolded

ACEM PRIMARY VIVA D **Friday Morning Session 3** ***Candidate Number:*** **AGREED MARK:**

Stem: A 50 year old woman is brought to the ED with an amitriptyline overdose. Commencing with Pharmacology.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Tricyclics including Volume of distribution (Chp 30) **Subject:** Pharm LOA: 1	Which factors determine the volume of distribution of a drug? Describe the volume of distribution of tricyclic antidepressants How does this influence their toxicity? What therapies for tricyclic toxicity might reduce their tissue distribution?	**Drug factors**; lipid solubility (high in TCA), pKa, pH, protein binding (high in TCA). **Patient factors**; age, gender, comorbid disease (eg. Oedema or ascites), body fat, blood flow to tissues. TCAs have a **large Vd** (5-30L/kg), tissue **concentrations are high** especially in well perfused organs such as **the brain and heart.** **Alkalinisation** (Bicarbonate or hyperventilation) increases **plasma protein binding of the free drug** removing it from the tissues reducing its tox	At least 2 from each group bold bold
Stem: Move onto Anatomy. You insert a femoral venous line.			
Question 2 Femoral Triangle (photo) **Subject:** Anat LOA: 1	Demonstrate the boundaries of the femoral triangle. What are the contents of the femoral triangle. What surface markings help would you look for when trying to locate the femoral vein? Which veins drain into the common femoral vein	**Inguinal ligament** (11), medial border of **Sartorius** (23) and lateral border of **adductor longus** (1) form the triangle, **pectineus** (med) and **iliopsoas** (lat) form the floor. Contents = **femoral vein** (6), **artery** (4) **and nerve** (5) (med to lat) and deep inguinal lymph nodes. **Artery is found below inguinal ligament, midway between ASIS and pubic tubercle, vein is just medial to artery** Continuation of the femoral vein, popliteal vein, receives **profunda femoris** and **great saphenous vein** (7), ends posterior to the inguinal ligament where it becomes the external iliac vein. Also receives superficial epigastric vein (27), superficial circumflex iliac vein (25) and superficial external pudendal vein (28).	3/5 to pass all content 3/3 to pass bold
Stem: Moving on to Pathology. She has a history of chronic alcohol abuse.			
Question 3	1. Describe the pathological effects on the liver long-term alcohol ingestion.	1. **Steatosis**: fatty change, perivenular fibrosis	Bold with 3 morphologic features of each to pass.

Alcoholic Liver Disease (pp 857-860) **Subject:** Path LOA: 1	PROMPT: please describe the morphological features 2. Which of these conditions reversible? 3. What are the possible sequelae of cirrhosis? Prompt: Complications?	2. **Hepatitis**: liver cell necrosis, inflammation, Mallory bodies, fatty change, fibrosis 3. **Cirrhosis**: extensive fibrosis, hyperplastic nodules 4. (Hepatocellular carcinoma) **Steatosis and Hepatitis are reversible**. Cirrhosis irreversible. **Portal Hypertension**, GIT Bleeding, Hepatic Failure, Coagulopathy, Hepatocellular Ca, Hepatorenal Syndrome, Hepatopulmonary Syndrome, Encephalopathy, Infection	 Bold to pass Bold plus 3
Stem: Moving on to Physiology			
Question 4 Dead Space (pp 19-21) **Subject:** Phys LOA: 1	What is DEAD SPACE? 2. What types of DEAD SPACE are there?" Prompt explain difference between the two types 3. How is it measured? (bonus)	Portion of the tidal volume that **does not participate in gas exchange** $V_T = V_D + V_A$ **1. ANATOMICAL** • Volume of **conducting airways** – trachea, bronchi, terminal bronchi (16 gen) • About 150mls of 500ml V_T • Measured by Fowler's method • Determined by: ○ Increased diameter of airways during inspiration ○ Size & posture of individual **2. PHYSIOLOGICAL** • Volume of gas that **does not eliminate CO_2** • Same as anatomical DS in normal individuals • **Increased in lung disease** because of inequality of blood flow and ventilation within the lung Measured by Bohr method	Demonstrate principle of bold to pass Two types dead space and describe what it is

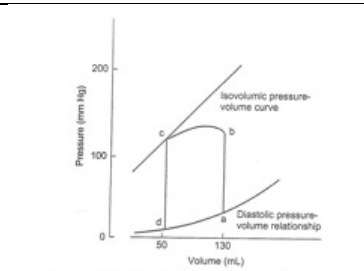
Stem: A 50 year old man presents in septic shock. The cause of this is unclear on initial assessment. We will start with Pathology.			
TOPIC	QUESTIONS	KNOWLEDGE (essential in bold)	NOTES
Question 1 Septic Shock (pp 129-133) Subject: Path LOA: 1	What is Shock?	State where reduced cardiac output or effective blood volume results in **impaired tissue perfusion** and **cellular hypoxia**	Bold concepts
	How do microbes initiate septic shock? *Prompt: What are the mechanisms*	1 Interaction with innate cells of immune system – examples neutrophils, macrophages, monocytes 2 Interaction with Humoral cells of immune system to activate complement & coag pathways 3 Direct action on endothelium (complex, not fully understood) Toll-like receptors recognise microbial elements, and other mechs End result is **mediator release** examples TNF,IL-6, 8, 10, PAF PAI-1, HMGB1	2 of 3 plus examples of each (at least 1) + understand role of mediators
	When DIC develops, what is the process?	Induction of procoagulant state by: **1 Increased TF** production **2** Decreased production of **Protein C** **3** TF pathway inhibitor **Thrombomodulin** **4 Decreased fibrinolysis** by increasing plasminogen activator inhibitor, Combined with stasis (decr washout of activated coag factors) results in activation of thrombin & and fibrin rich thrombi	2 of 4 & understanding of process
	What factors determine the severity and outcome of septic shock in an individual?	Extent and virulence of infection Immune status of host Presence of other co-morbid conditions Pattern and level of mediator production	Bonus Q – no pass criteria

Stem: We will now move to Pharmacology. He is given ceftriaxone.			
Question 2 Ceftriaxone (pp 799-800) Subject: Pharm LOA: 1	What type of antibiotic is ceftriaxone? Describe the pharmacodynamics of ceftriaxone Explain the microbiological spectrum of activity of ceftriaxone What is the clinical relevance of ceftriaxone's half-life?	**Third generation cephalosporin. Beta lactam** **Inhibits** transpeptidation reaction of **bacterial cell wall synthesis.** Halts peptidoglycan synthesis, leading to inhibition of growth, and ultimately **cell death** (**Bacteriocidal)** **Stable to bacterial beta-lactamases**, therefore broader spectrum of activity. **Expanded gram-negative cover** and crosses the blood brain barrier. Effective against B-lactamase producing **Haemophilus and Neisseria** **Half life of 7 to 8 hours**, meaning it **may be administered once daily** at 15 to 50mg/kg	1/2 bold Bold Bold Bold
Stem: We will now move to Physiology. Initial urea and electrolytes show renal failure			
Question 3 Effects of disordered renal function (pp 692-693) Subject: Phys LOA: 1	What are the major physiological features of acute intrinsic renal failure? *(prompt: what happens to urine concentration?)* What are common findings in urinalysis of acute intrinsic renal failure? What are urinary casts?	**Loss of urine concentrating and dilutin**g capacity due to loss of countercurrent mechanism and nephron number. **Polyuria →oliguria → anuria** **Uraemia** due to urea and creatinine and toxins (phenol and acids) build up. **Acidosis.** Anaemia **Na+ retention** and oedema and heart failure **Proteinuria, leucocytes, red cells and casts** **Proteinaceous** material precipitated in tubules washed into bladder.	3/5 bold ones 3 bold Bold
Stem: We will now move to Anatomy. An abdominal CT scan is done			
Question 4 CT abdomen Subject: Anat LOA: 2	Identify the structures visible.	**Liver**, portal vessels, part of gallbladder (not obvious), R Kidney (top), diaphragms, **aorta**, IVC (not clearly differentiated), **L kidney**, **spleen**, splenic vein (not tortuous), bowel loops, pancreas, antrum. Vertebra, ribs, paravertebral muscles, intercostal and abdominal wall muscles, fat, skin.	**4 Bold** + 2 others

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: A 10 yo boy presents with a headache and fever. We will start with Anatomy.			
Question 1 L-spine (bone) **Subject:** Anat LOA: 1	1. What type of vertebral body is this, outline its features? *Prompt: how is it different to other vertebra*	**Lumbar.** Large kidney shaped body. Transverse processes are long and slender. Vertebral foramen triangular, larger than thoracic/smaller than cervical. Spinous process is short, thick, hatchet shaped. Articular processes/facets extend vertically	Bold to pass 2 of 4 to pass
	2. What levels should a Lumbar Puncture be performed at, and what landmarks are used?	**L 3-4, 4-5 or L 5-S1**. Spinal cord ends at L2. **Top of Iliac crests at L4 spinous process**. Hence space above or below this avoids the cord.	2 levels + landmark to pass
	3. What structures does the needle pass through in order?	Skin/sub cut fat/supraspinous lig./interspinous lig./lig. flavum/epidural space/dura/subarachnoid space	5 of 8 to pass
Stem: Moving onto Pharmacology. You use lignocaine as the local anaesthetic			
Question 2 Local Anaesthetics **Subject:** Pharm LOA: 1	Describe the mechanism of action of lignocaine?	Na channel blocker, Class 1B. **Blocks** (activated & inactivated) **Na channels = Blocks nerve conduction**. Less effect in infected tissue	Bold
	What factor affect systemic absorption after local infiltration	Dose/ Site of injection/ Drug tissue binding/ Tissue blood flow/ Vaso constrictors (combine preparation)	3 of 5
	What are the toxic effects of Lignocaine	**CNS -** Early: **tongue/oral numbness/metallic taste**, parathesia, sedation. Moderate: nystagmus, muscle twitching, N&V, Tinnitus, visual disturbance Severe: **Seizures**, sedation **CVS- Cardiovascular collapse** Hypotension, bradycardia, rarely arrhythmia, worsen CCF or conduction blocks GIT Anorexia, N&V (thru CNS effects)	Bold

Stem: These are his CSF results.			
Clinical Building Block:	What is the likely diagnosis and why?	Turbid, low sugar, high protein, pleocytosis with neutrophil predominance, no bacteria **Acute bacterial meningitis**	Diagnosis + 2 reasons
Stem: Moving onto Pathology.			
Question 3 Meningitis **Subject:** Path LOA: 1	What are the other types of meningitis? What organisms commonly cause bacterial meningitis in the different age groups?	**Viral**, chronic (tuberculosis), fungal, chemical / drug induced, carcinomatous Neonates: Escherichia Coli and Group B Streptococcus Children: **Streptococcus Pneumoniae**, Haemophilus Influenza (now less common) Adolescent / young adult**: Neisseria Meningiditis, Streptococcus Pneumoniae** Older adults: Strep Pneum, Listeria	3 out of 5 including bold 1 per age group, must mention Bold.
Stem: Moving onto Physiology. The child appears very unwell.			
Question 4 Effects of Glucocorticoids **Subject:** Physiol LOA: 1	What are the physiological effects of glucocorticoids? How is glucocorticoid secretion regulated?	Permissive action – **catecholamine effects – pressor / vascular reactivity**; bronchodilation **Metabolic** - Increase protein catabolism; increase hepatic glycogenolysis and gluconeogenesis -> increase in plasma glucose; anti-insulin effects on peripheral tissues; increase lipolysis. Free water excretion (decreased vasopressin) Immunological – decreased inflammation/ allergic response/ lymphocyte activity Haematological – increase neutrophils/ platelets/ red blood cells CNS – EEG slowing, personality changes **Glucocorticoids** (cortisol) - **secreted from the adrenal cortex**; secretion **dependant on ACTH secretion** from the **anterior pituitary** ACTH secretion is regulated by CRH released from the hypothalamus (in response to low cortisol levels or stress) **Glucocorticoids** provide a **negative feedback loop on the hypothalamus and the anterior pituitary** to **reduce ACTH secretion**	Bold + 2 metabolic + 1 other Bold

Stem: A 60 year old man presents to ED with palpitations.			
TOPIC	QUESTIONS	**KNOWLEDGE (essential in bold)**	**NOTES**
Stem: An ECG is done.			
Clinical Building Block: ECG Atrial Flutter	Please describe and interpret his ECG. *Prompt: what is the rhythm and rate*	Rate: **Ventricular 75-100**, atrial approx. 300/min Rhythm: **Irregular**. Variable block (3&4:1) P waves: **Atrial flutter waves** (sawtooth) Axis: Normal. QRS: Narrow complex, anterior Q waves. T-waves: difficult to comment. = A flutter, variable block	Bold to pass
Stem: Moving onto Pathology. He has an underlying cardiomyopathy			
Question 1 Cardiomyopathy Subject: Path LOA: 2	Name the types of cardiomyopathy. *(Prompt: based on function/pathology)* What are the causes of acquired cardiomyopathy? How do dilated and hypertrophic cardiomyopathy differ? *Prompt: left ventricular structure and function*	**Dilated cardiomyopathy (DCM), Hypertrophic cardiomyopathy (HCM),** Restrictive cardiomyopathy **Infections** (viral, bacterial, fungal, protozoal); **Metabolic** (hyperthyroidism, nutritional) **Infiltrative** (sarcoid, carcinoma) **Immunological** (autoimmune myocarditis) **Drugs/toxins** *(alcohol, chemotherapy)* Ischaemic, hypertensive, valvular. DCM: cardiac dilatation**, poor LV EF** (<40%). I**mpaired contractility** (systolic dysfunction) HCM: myocardial hypertrophy, **normal or high LV EF**. I**mpaired compliance** (diastolic dysfunction)	Bold 3/5 bold + and examples Bold for each
Stem: Moving onto Physiology. His blood pressure is 100/60			
Question 2 Cardiac cycle; pressure / volume Subject: Phys LOA: 1	Please draw and label the pressure volume curve of the left ventricle Describe the pressure and volume changes in the left ventricle at the onset of systole *Prompt : What is meant by isovolumetric contraction.*	Graph with appropriate axis, curves and approximate pressures **(a to b)**Start of systole**, mitral valve closes**. **Isovolumetric contraction** until LVP>Aortic P (80mmHg) then **Aortic valve opens**. ESV 50ml	Correct graph & bold to pass with reasonable understanding of the loop

	Describe the pressure and volume changes in the left ventricle at the onset of diastole *Prompt : What is meant by isovolumetric relaxation*	**(c to d)** Momentum of ejected blood is overcome by arterial pressure, then the **Aortic valve closes**. **Isovolumetric relaxation** as the ventricular pressure drops rapidly until below atrial pressure. Then **AV valve opens** to start ventricular filling. EDV 130ml, Stroke volume 70-90ml	 **FIGURE 30–2 Normal pressure–volume loop of the left ventricle.** During diastole, the ventricle fills and pressure increases from d to a. Pressure then rises sharply from a to b during isovolumetric contraction and from b to c during ventricular ejection. At c, the aortic valves close and pressure falls during isovolumetric relaxation from c back to d. (Reproduced with permission from McPhee SJ, Lingappa VR, Ganong WF (editors): *Pathophysiology of Disease*, 6th ed. McGraw-Hill, 2010.)
Stem: Moving onto Pharmacology. It is decided to treat him with Amiodarone			
Question 3 Amiodarone Subject: Pharm LOA: 1	What anti-arrhythmic class does amiodarone belong to? What are the effects of amiodarone on the heart? What other arrhythmias is amiodarone used for? What arrhythmias may amiodarone cause?	**Class 3**: also class I,II,IV effects **Increases Action potential duration (APD) due to blockade** of rapid component of delayed **K^+ current**(I_{kr}). Chronic use also blocks slow K^+ rectifier. **Prolongs QT** (due to above effect) Blocks inactivated Na^+ channels. Weak adrenergic and Ca^{++} channel blocker Atrial Fibrillation/ Ventricular tachycardia/Ventricular fibrillation/ Supraventricular (re-entrant/ accessory) Torsades de pointes **(**rare < 1%), Bradycardia, Heart block	Bold to pass Bold to pass 2 to pass 1 to pass
Stem: Moving onto Anatomy.			
Question 4 Heart Model Subject: Anat LOA: 1	Using this model please describe the arterial supply of the heart What does the Right Coronary artery supply? Describe the cardiac conduction system.	Coronary arteries arise from coronary sinuses. **L Coronary artery** divides into **LAD** and **Circumflex**. LAD - diagonal branches. Circumflex – marginal branches. **RCA** inferior in AV groove – SA nodal, AV nodal, marginal, posterior IV (2/3). RA, most of RV, diaphragmatic (inferior) part of LV, post 1/3 IV septum, SA node in 60%, AV node in 80% **SA Node** – Junction of SVC and RA. **AV Node** – Postero-inferior AV septum near coronary sinus. AV bundle of His. Left and Right Bundles	**Bold** + 1 other 3/6 to pass Bold + 1 other

ACEM PRIMARY VIVA B Thursday Morning Session 1 ***Candidate Number:*** **AGREED MARK:**

Stem: A 25 year old man is brought to the Emergency Department following a motor bike accident. He cannot move his limbs. Here is his cervical spine X-ray.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Clinical Building Block:	Please describe this x-ray.	Xray C1-C7. C7/T1 not visualised **Step at C5/C6** consistent with bi-facet dislocation Disruption of all 4 lines: Soft tissue, Anterior, Posterior, Spinolaminar lines	Bold. PROMPT: What are the radiological lines to examine? 3
Stem: Staying with Anatomy			
Question 1 Cervical Vertebrae **Subject:** Anat LOA: 1	Please describe these cervical vertebrae PROMPT: Demonstrate their anatomical orientation and function. Show C1 and C2 What are the characteristics of a typical cervical vertebra?	**C1** "Atlas": Anterior and posterior arch **Lateral mass** with Transverse processes **Articular facet for Dens** **Superior articular facet**- articulates with occipital condyles Inferior articular facet- articulates with axis Anterior and Posterior tubercles **Foramina**: vertebral and transverse **C2** "Axis" **Dens** with anterior and posterior articular facet Body Pedicle Lateral mass **Transverse and spinous process** Superior and Inferior articular facet **Small, oval body** with **large vertebral canal**, **concave** on **superior surface** and **convex** on **inferior surface**. Superior surfaces of bodies have raised processes (**uncinate**), each of which articulates with a depressed area on inferior lateral aspect of the superior vertebral body **Spinous processes** are **short, bifid**, and **downward sloping** (C7 usually non bifid) **Facet joints are more horizontal** allowing a greater range of movement **Anterior and posterior transverse process** with a **foramen transversarium** allowing passage of vertebral artery, vein and sympathetic plexus	**Must display correct anatomical position and articulation** And name the bold 3 to pass

Stem: We will now move to Pathology. An MRI shows acute swelling of his cord in the region of his injury			
Question 2 Mediators of inflammation (pp 56-66) **Subject:** Path LOA: 1	What stimuli cause production of inflammatory mediators? What are the chemical mediators of acute inflammation and what are their actions?	Substances released from necrotic cells, microbial products, cell injury, mechanical irritation. **Histamine**: vasodilation, inc vasc perm, endoth activation **PG**: vasodilation, inc vasc perm **Leukotrienes**: inc vasc perm, chemotaxis, WC adhesion & activation **PAF**: vasodil, inc vasc perm, chemotaxis, WC adhesion, degran **Complement**: WC chemo and activation, vasodilat **Cytokines** (TNF, IL-1): endo activation (adhesion), fever, pain, hypotension, dec vasc resist **Chemokines**: chemotaxis, WC activation **Kinins**: inc vasc perm, vasodil, pain, sm m contraction	2 to pass. 4 to pass (including names and actions)
Stem: We will now move to Pharmacology. He develops neurogenic shock and is treated with metaraminol.			
Question 3 Metaraminol (chp 9) **Subject:** Pharm LOA: 1	What is the mechanism of action of Metaraminol? What are its effects on the cardiovascular system? What role do sympathomimetics have in management of shock?	Direct **alpha** 1 **receptor agonist** – some indirect effect through increased noradrenaline. **Vaso and arterio – constriction** in vascular beds. Arterioconstriction → **↑BP** Direct cardiac effects less important HR slows due to vagal feedback CO unchanged or slight decrease as ↑VR and hence SV Temporising only While other treatment instituted – fluids, etc Efficacy not proven Useful in 'failure' sympathetic NS (eg/ spinal injury or anaesthesia)	Bold Bold Understanding of temporary only

Stem: We will now move to Physiology.			
Question 4 Spinal Tracts (pp 228-229) **Subject:** Phys LOA: 2	What are upper motor neurons?	Upper motor neurons usually refer to **corticospinal neurons that innervate spinal motor neuron**s (also include brain stem neurons that control spinal motor neurons).	Bold
	What clinical features are seen when they are injured?	Damage **initially causes muscles to become weak and flaccid** but eventually leads to **spasticity, hypertonia, hyperactive stretch reflexes and an abnormal plantar extensor** reflex (upwards))	2 of bold findings
	What is the physiological basis to clonus?	Loss of descending cortical input to inhibitory neurons called Renshaw cells, and therefore **loss of inhibition of antagonists,** resulting in repetitive sequential contractions of ankle flexors and extensors.	Bold
	List the long term complications of spinal cord injury	Ulcers Protein /muscle degradation Hypercalcaemia Renal stones (calcium) Urinary tract infection	2

ACEM PRIMARY VIVA B **Thursday Afternoon Session 2** ***Candidate Number:*** **AGREED MARK:**

Stem: An 80 year old man is sent to ED from his nursing home after a fall. He has multiple injuries and has been given IV morphine by the ambulance officers. On arrival in ED he is hypoventilating. We will start with Physiology.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 CO_2 transport **Subject:** Phys LOA: 1	Q1. How is CO2 carried in the blood?	CO2 is carried in the blood in 3 forms: 1. dissolved - approx 5-10% **2. as bicarbonate - approx 90%** 3. combined with proteins as carbamino compounds, approx 5-10%	Bold + 1 other
	Q2. How is bicarbonate formed in the blood? Prompt: can you write an equation	CA CO2 + H2O ←→ H2CO3 ←→ H+ + HCO3- The 1st reaction is very slow in plasma but fast within the red blood cell because of the presence there of an enzyme **Carbonic Anhydrase (CA).** The 2nd reaction, ionic dissociation is fast without an enzyme.	Talk through equation and Bold.
	Q3. What is the chloride shift?	HCO3- diffuses easily out of the cell. H+ doesn't because the **cell membrane is relatively impermeable to cations**. Therefore to **maintain cell neutrality** Cl- diffuses from the plasma into the cell.	At least 1 bold
	Q4. What is the Haldane effect?	H+ + HbO2 <–> H+-Hb + O2 The Haldane effect: DeoxyHb binds more H+ than oxyHb and forms carbamino compounds more readily. Binding of O2 to Hb reduces its affinity for CO2 1. **Enhances the removal of CO2** from O2 consuming **tissues** (eg muscles) into the blood. CO2 can bind to amino groups on Hb to form carbaminoHb. CarbaminoHb is the major contributor to the Haldane effect. 2. Promotes the **dissociation of CO2 from Hb** in the **presence of O2** (eg the lungs) which is vital for alveolar gas exchange	Additional question if time permits

Stem: Moving onto Anatomy. On secondary survey, he has bilateral painful shoulders. This is a normal shoulder x-ray.			
Question 2 Shoulder X-ray **Subject:** Anat LOA: 1	2.1 Indicate on the X-ray the anatomy of the shoulder joint	**Xray description** **HUMERUS:** **Head** – articulates with glenoid **Anatomical neck** **Surgical neck** **Greater Tubercle** Lesser tubercle **SCAPULA** **Glenoid** cavity Neck **Acromion** process **Coracoid** process Spine Superior, medial and inferior angle **Clavicle** Distal portion articulates with acromion	Bold to pass
	2.2 Name the ligaments and describe how they stabilise the shoulder joint	**1. Glenohumeral ligaments** (superior, middle and inferior) consists of three bands, which runs with joint capsule from glenoid fossa to anatomical neck of humerus. They act to **stablise the anterior aspect of the joint.** **2. Coroacohumeral ligament** – Attaches the base of the coracoid process to the greater tubercle of the humerus. It **supports the superior part of the joint capsule**. **3.Transverse humeral ligament** – Spans distance between two tubercles of humerus. H**olds the tendon of long head of biceps in the intertubecular groove.** **4. Coracoacromial ligament -**runs between the acromion and coracoid process of the scapula, forming the coraco-acromial arch. This overlies the shoulder joint, **preventing superior displacement of the humeral head.**	2/4 to pass.

Stem: Moving on to Pharmacology. There is a fracture dislocation of his left shoulder. His shoulder is reduced under Propofol sedation			
Question 3 Propofol **Subject:** Pharm LOA: 1	Q1. Please outline the pharmakokinetics of propofol	IV administration only, **Distribution half life 2 - 4 minutes**, **Elimination half life 4 -23 minutes,** Duration of action 3 - 8 minutes - **Rapid onset and recovery due to redistribution of drug** from brain to skeletal muscle and then fat (rather than metabolism), Rapidly metabolised in the liver but as total body plasma clearance > hepatic flow, likely some extrahepatic mechanism (mostly lung), Excretion in the urine as glucuronides and sulphates < 1% unchanged	**Bold, reasonable understanding of redistribution of drug**
	Q2. What dose of propofol is used for induction of general anaesthesia? How does this differ from a procedural sedation dose?	PROCEDURAL SEDATION DOSE: 0.5 - 1.0 mg/kg single bolus dose or titrate in 10 - 20 mg aliquots particularly in conjunction with morphine, INDUCTION DOSE: **1 - 2.5mg/kg (adults)** and 2.5-3.5mg/kg in kids	**Bold**
	Q3. What clinical effects should be anticipated when using propofol?	anaesthesia/sedation, respiratory depression, transient **apnoea**, **hypotension** through vaso and venodilation, no analgesic properties, potential allergic reaction (soy, eggs), pain at injection site, metabolic acidosis when given as an infusion, antiemetic properties	**Bold + 2 more**
	Q4. How can you limit adverse effects when administering propofol?	smaller total doses, titrated doses, no opiates or benzodiazepines given simultaneously, IV fluid bolus, caution in the elderly and in those with poor cardiovascular reserve	**2**

Stem: Moving onto Pathology. A junior doctor asks you about the healing process for the fracture			
Question 4 Fracture Healing **Subject:** Path LOA: 1	Describe the steps in fracture repair process	1 **haematoma** fills fracture gap – provides fibrin mesh framework (hrs) 2 **influx** inflam cells, fibroblasts, new vessels (days) 3 haematoma organising -> **procallus** 4 osteoprogenitors deposit trabeculae of woven bone **– ossification -> bony callus** (2-3 weeks) 5 callus matures, **remodelling** (6 weeks)	4 of 5 steps Logical sequence
	How does remodelling of callus occur?	Initial large volume of callus – portions not physically stressed are resorbed, reducing callus size/altering contour	Physical stress, resorption
	What factors can impede the healing of a fracture?	**Inadequate immobilisation**, marked displacement, **infection** (open fractures/FBs), systemic factors (nutrition, smoking…)	2 bold and 1 other
	(Supplementary – if time remaining) How are fractures classified?	Complete/incomplete, open/closed, comminuted, displaced, pathologic, stress	

ACEM PRIMARY VIVA B **Friday Morning Session 3** ***Candidate Number:*** **AGREED MARK:**

Stem: A 70 year old lady with a history of bipolar disorder is found on the floor many hours after a fall. On arrival in ED, she is tachycardiac and complaining of severe thirst and thigh pain. Starting with Physiology			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Thirst **Subject:** Phys LOA: 1	Q1. Where is thirst regulated? Q2. What factors increase thirst?

Stem: Moving onto Pharmacology. Her medications include Lithium			
Question 2 Lithium **Subject:** Pharm LOA: 1	Q1. What are the adverse and/or toxic effects of lithium?	Neuro - tremor**,** choreoathetosis, **ataxia,** dysarthria, hyperactivity, **confusion,** withdrawal. Thyroid - reversible **hypothyroidism.** Renal - **polyuria, polydipsia** (nephrogenic diabetes insipidus), chronic interstitial nephritis, nephrotic syndrome. Cardiovascular - oedema, worsening of sick sinus syndrome	**At least 3 bold**
	Q2. Describe the pharmacokinetics of lithium	**Oral absorption** (peak 0.5-2 h but complete 6-8 h). **Distributes in TBW. Excreted unchanged in urine.** Plasma half-life 20 h. Therapeutic concentration 0.6-1.4 mmol/L	**Bold, plus some appreciation of longer half-life.**
	Q3. How can you assess lithium toxicity and how do you treat it?	**Measure levels** (should be 10-12 h after last dose) >2 mmol/L should be considered toxic. Treatment is supportive and haemodialysis (Prompt that Li is an ion).	**Bold, plus some concept that levels should be measured well after last dose.**

Stem: Moving onto Anatomy. On secondary survey, you suspect a fractured femur			
Question 3 Femur (bone) **Subject:** Anat LOA: 1	Please demonstrate the main bony features of the proximal femur	**Femoral head** / fovea for lig of head **/ Greater trochanter / lesser trochanter** / Neck / intertrochanteric line (anterior)/ Intertrochanteric crest (posterior) / quadrate tubercle /pectineal line / Gluteal tuberosity / linear aspera with medial and lateral lips	Pass = bold + 3 others
	Demonstrate on the model the muscular attachments to the greater trochanter	MM originating ; 1. Vastus lateralis MM Inserting ; 1. Gluteus max (some fibres only, most to ileotib tract) 2. Glut med; To lat surface 3. Glut min; to ant surface 4. Piriformis; to sup border 5. Obturator internus; to med surface (trochanteric fossa) 6. Sup and inf gemelli; to med surface	Pass = 4/7
	The patient has a subcapital (intracapsular) fractured neck of femur. What is the most concerning complication of this type of fracture and why does this occur? *Prompt ; What is the blood supply to the hip joint and how would this be disrupted*	**Avascular necrosis of femoral head** Hip joint has **dual supply** ; 1**. Med & lat circumflex femoral aa**, usually branches of deep aa of thigh (sometimes can arise direct from fem aa) 2**. aa to head of femur**, branch of obturator aa, traverses lig of head (often small/inadequate) Main supply is via retinacular **aa, from branches of circumflex femoral** (esp medial circumflex femoral, because these are able to pass freely under unattached post border of joint capsule. Branches from lat Cx must penetrate thick iliofemoral lig and are smaller and fewer). Retinacular **aa are torn or disrupted in intracapsular #**	Pass = bold

Stem: Moving onto Pathology. Her biochemistry results show an acute kidney injury			
Question 4 Acute tubular necrosis	Define Acute Kidney Injury	Clinico-path entity, **acute reduction of renal function** with morphologic **tubular injury (usually)**	Bold
Subject: Path LOA: 2	What are the causes of AKI (please give examples)?	1 **Ischaemia**/abnormal blood flow. Systemic – thrombosis (HUS, TTP, DIC) or hypovolaemia. Intra-renal – angiopathies, malignant HT 2 **Toxic injury to tubules**– drugs, radio-dye, myoglobin 3 Acute tub.int nephritis – reaction to drugs 4 Obstruction ("post-renal") –tumour, clot	Bold and 1 other category 1 example for each
	Describe the typical clinical course of AKI	Variable 1 Initiation 36 hours – decr UO, incr urea 2 Maintenance – **oliguria**, salt/H2O overload, incr urea/K/H 3 Recovery - **incr urine vol** (up to 3L/d), H2O/Na/K loss. Ur/Cr r/t normal	Oliguric phase, polyuric recovery
	(Supplementary – if time remaining) What are the most likely causes in this 70 year old lady?	Ischaemic injury from hypovol/hypotension from femur # +/- inability to get to water Myoglobin deposition from rhabdo	

ACEM PRIMARY VIVA C **Thursday Morning Session 1** ***Candidate Number:*** **AGREED MARK:**

Stem: A 60 year old woman with a history of hypertension presents with chest pain radiating into her back. An ECG is done.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Clinical Building Block ECG with AMI	Please describe and interpret the significant abnormalities in this ECG.	• Sinus, rate ~100/min, normal axis • **ST elevation (STEMI)** • **Inferior leads** • ST depression and inverted T waves in I, aVL, V2, V3 (Reciprocal changes)	Bold
Stem: We will now move to Physiology.			
Question 1 ECG – myocardial infarction **Subject:** Phys LOA: 1	Explain the electrophysiological changes that cause the ST segment elevation seen in a myocardial infarction? *Prompt for time course* *Second prompt for cellular mechanism*	• Abnormally rapid repolarisation of the infarcted muscle (accelerated opening of K+ channels). Current flow out of infarct (normal region negative relative to infarct). Occurs within seconds of infarction and last a few minutes. • Decreased resting membrane potential (due to loss of intracellular K+). Begins in first few minutes secondary to process above. Current flow into infarct during diastole (ECG configured to record as ST elevation). • Slowed depolarisation of affected cells cf normal cells. Occurs @ 30 minutes into infarct process. Current flow out of infarct.	**2 of 3 to pass**
Stem: We will now move to Anatomy. She needs vascular access.			
Question 2 Upper limb model **Subject:** Anat LOA: 1	Demonstrate the boundaries of the cubital fossa What are the contents of the cubital fossa ? Identify the brachial a and its branches in the forearm	**Lateral:** med border of brachioradialis (20) **Medial:** Lat border of pronator teres (12) **Floor:** Brachialis (10) Superior: Line between 2 epicondyles of humerus Roof: Skin, deep fascia reinforced by bicipital apon FYI: 13 = FCR Med to lat: **median n (71), brachial a (49), biceps tendon (9)**, radial n deep to brachioradialis (20) **Brachial (49), Radial** (55), **Ulnar** (59), Common Interosseous (60), post interosseous (61) Median (62)	Bold to pass Bold to pass Bold to pass

Stem: Moving to Pathology. You suspect aortic dissection			
Question 3 Aortic Dissection **Subject:** Path LOA: 1	What sequence of changes occur in the vessel wall in aortic dissection?	**Intimal tear** into media of aorta, **strips along laminar planes**, formation of blood filled channel which may then rupture outwards.	Bold (conceptually)
	What are the risk factors?	Men aged 40-60 with **hypertension** Connective tissue disorders eg Marfans Complication of arterial cannulation Trauma	**Hypertension + one other**
	What are the types of aortic dissection? Prompt = classification?	Stanford Type A – proximal ascending + (DeBakey I)/-(DeBakey II) distal, may rupture back through Ao Valve . B is Stanford Type B – beyond subclavian artery (DeBakey III)	**Concept (prox & distal)**
Stem: We will now move to Pharmacology. She is now hypertensive. You commence a glyceryl trinitrate (GTN) infusion			
Question 4 Glyceryl Trinitrate **Subject:** Pharm LOA: 1	What is the mechanism of action of GTN	Nitrite -> **NO** -> ^ cGMP -> **Smooth m relaxation**. Prostaglandins may be involved	**Bold**
	What are its clinical effects?	**1.** Beneficial effects-**venodilation**, reduced venous return, decr ventricular pre-load, reduced LVEDV, reduced LV wall tension, **reduced myocardial oxygen consumption**. Vasodilation of epicardial coronary arteries, increased coronary collateral flow. **Decrease systemic BP** 2. Adverse effects - hypotension, tachycardia, headache	**2 of 3 Bold** **2 adverse effects**
	What are the indications for GTN use in the ED?	**Angina,** acute coronary syndrome, hypertensive urgencies/emergencies, APO, aortic dissection (with beta-blockade)	**Bold plus two others**

ACEM PRIMARY VIVA C **Thursday Afternoon Session 2** ***Candidate Number:*** **AGREED MARK:**

Stem: A 60 yo man has a very high blood glucose. We will start with Pathology.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Pathogenesis of Diabetes Mellitus **Subject:** Path LOA: 2	What is the pathogenesis of Type 2 Diabetes Mellitus?	**Insulin resistance** - decreased ability of the peripheral tissues to respond to the secreted insulin - secondary to either genetic predisposition or obesity/lifestyle factors Quantitative and qualitative **beta cell dysfunction** - manifests as inadequate insulin secretion in the face of insulin resistance and hyperglycaemia - initial beta cell hyperplasia maintains normoglycaemia with increased levels of insulin secretion - early and subsequently late failure manifests as impaired glucose tolerance and diabetes - genetic predisposition to B-cell failure.	Bold to pass
	What are the complications of diabetes?	1. **Macrovascular** - coronary, peripheral vascular, cerebral and other large vessel atherosclerosis, hypertension 2. **Microangiopathy-** nephropathy, peripheral neuropathy, autonomic neuropathy, cerebral microangiopathy 3. **Diabetic ocular complications**- retinopathy, cataracts, glaucoma 4. Increased susceptibility to infections 5. HONK, DKA, hypoglycaemia, hyperglycaemia	Two from 1 & 2 and one from the third group.

Stem: Moving onto Physiology. Urine analysis reveals glucose.			
Question 2 (Renal handling of glucose **Subject:** Phys LOA: 1	Describe the way the kidney handles glucose.	1. **Freely filtered** the glomerulus 2. **Resorbed in the early part of the PCT** by secondary active transport 3. **Na dependent** co transportation (SGLT2 into cells then GLUT 2 facilitated diffusion into interstitial fluid) 4. Excreted in the urine if **renal threshold** is exceeded.	1 & 2 plus understanding of 3 & 4
	What are the potential consequences of glycosuria	Osmotic diuresis – dehydration, electrolyte loss (Na, K)	Understanding

Stem: Moving onto Anatomy. On examination, he has a swollen knee			
Question 3 Knee model **Subject:** Anat LOA: 1	1. Please identify the ligaments of the knee joint and their attachments	**Patellar ligament –** apex of patella to tibial tuberosity **Fibular collateral ligament-** lateral epicondyle of femur to – lateral surface of fibula head **Tibial collateral ligament –** medial epicondyle of femur – lateral and superior aspect of tibia **Anterior cruciate –** anterior intercondylar area of tibia to anterior aspect of the lateral surface of medial condyle of femur **Posterior cruciate –** posterior intercondylar area of the tibia to anterior aspect of the lateral surface of medial condyle of femur Posterior meniscofemoral ligament	Identify attachments of 3/5
	2. Describe the main movements of the knee joint and the muscles involved.	**Extension – quadriceps femoris** **Flexion – semitendinosis, semimembranosus, biceps femoris** Medial rotation – when flexed – ST, SM When non wt bearing knee extended – popliteus	Bold
	3. Describe the locking and unlocking process as the joint is extended and flexed	When knee extended and weight bearing – knee passively locks due to medial rotation of femoral condyles on tibial plateau Knee unlocks through contraction of popliteus rotating femur laterally on the tibial plateau allowing flexion	Understand concept
Stem: Moving onto Pharmacology. He has been on Gliclazide for his Diabetes			
Question 4 Sulfonyl Ureas **Subject:** Pharm LOA: 1	1. What class of drug is gliclazide?	**1. Sulphonylurea**	Bold to pass
	2. Describe the mechanism of action of sulfonylureas.	**2. Stimulates insulin secretion from functional pancreatic beta cells** • Binding of sulphonylurea to receptor inhibits potassium efflux causing extracellular depolarisation • Results in opening of voltage gated calcium channels • Calcium influx causes release of preformed insulin	Bold
	3. What are the pharmacokinetic properties of gliclazide?	3. Administered orally – good oral bioavailability (80%) Protein bound – volume of distribution ~ 20L **Hepatic metabolism** to inactive metabolites **Half life approx. 12 hours** Predominantly renally excreted (80%)	Bold
	4. What are potential adverse effects of gliclazide?	**4. Hypoglycaemia** **GI upset** – nausea, vomiting, abdominal pain, diarrhoea Rash/pruritis	Hypoglycaemia plus one

ACEM PRIMARY VIVA C Friday Morning Session 3 ***Candidate Number:*** **AGREED MARK:**

Stem: A 40 year-old man develops a dystonic reaction following a metoclopramide injection. Starting with pharmacology			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Benztropine Subject: Pharm LOA: 1	How does metoclopramide cause a dystonic reaction? You treat the dystonic reaction with benztropine. What is its mechanism of action? What are the potential side effects of benztropine?	Metoclopramide is a **dopamine antagonist** and causes an imbalance in the anticholinergic/ dopamine transmission in the basal ganglia. **Blocks the** muscarinic **cholinergic receptors**; an antimuscarinic agent. Tachycardia, sedation, mydriasis, urinary retention, dry mouth	Bold Bold **Knows 3**
Stem: Moving on to Anatomy. Intravenous access is obtained			
Question 2 Forearm (photo) Subject: Anat LOA: 1	On the photo please identify the major veins that can be seen. What other vascular structures can you identify? Describe the venous drainage of the hand and forearm	From proximal to distal 1; **basilic** v 6; **cephalic** v 13; **median cubital** v 14; **median forearm** v. 4; **brachial artery** 21; **radial** a 22; **ulnar** a a) **Superficial** Dorsal venous network, and superficial palmar venous arch drains to either the basilic v ulnar side or cephalic v radial side of the forearm (highly variable distribution). b) **Deep** Deep venous palmar arch drains to paired radial veins and paired ulnar veins which accompany the arteries of the same name. Interosseous veins unite with radial and ulnar veins All terminate into the brachial veins as they leave the forearm	**2 req'd** **Brachial and one other req'd** **Mention of superficial and deep systems and mention of venae comitantes (or principle)**

Stem: Moving onto Pathology. The man has sickle cell disease.			
Question 3 Sickle Cell Disease Subject: Path LOA: 2	1. What is sickle cell disease?	**Hereditary** blood disorder **Haemoglobinopathy**	**Bold** (Prompt: is it congenital or acquired?)
	2. What are the major pathological manifestations of sickle cell disease?	• Haemolysis/Haemolytic anaemia • Microvascular occlusions (crises/Tissue ischaemia = severe pain in affected organs eg bones, lungs, liver, spleen) • Splenic enlargement, infarct and dysfunction (Increased susceptibility to infection **–** encapsulated organisms [eg strep pneumonia, haemophilus influenza])	**2 of 3 to pass**
	3. In general how are haemolytic anaemias classified?	**Inherited genetic defects** (RBC Membrane [spherocytosis], enzyme deficiencies [G6PD], haemoglobinopathies [thalassaemia, sickle cell disease]) **Antibody mediated destruction** (transfusion reactions, autoimmune) **Mechanical trauma** (Microangiopathic haemolytic anaemias [HUS, DIC, TTP], cardiac valves) **Infections of red cells** (malaria) **Toxic** (envenomation)	**2 of 5 Bold** to pass

Stem: Moving onto Physiology in a NORMAL lung.			
Question 4 Regional Gas Exchange Subject: Phys LOA: 1	1. What happens to the V/Q ratio from top to bottom of the upright lung? Prompt: What happens to the relative values of ventilation and perfusion? 2. Explain the reasons for the normal Alveolar-arterial 0_2 difference? (Extra question = formula for A-a Gradient)	**1. 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.** 2. Normally 5-10 mmHg. A-a Gradient = measure of the difference between alveolar and arterial concentration of O_2 • Even though P Alv 0_2 at apex 40 mm Hg above base, **most of blood flow (Q) comes from base where P Alv 0_2 is low** -> decrease in P Art 02 • **Shunt:** Bronchial blood & coronary blood Also non-linear shape of 02 dissociation curve means that addition of small amount of shunted blood with low 0_2 concentration greatly decreases $P0_2$ of arterial blood and units with high $P0_2$ have little effect on 0_2 concentration because curve is flat at high 0_2 concentration $PA02 = PI02 - \frac{PAC02}{R} + F$	1. 3 of 3 bold to pass (be able to explain concept). 2. Bold.

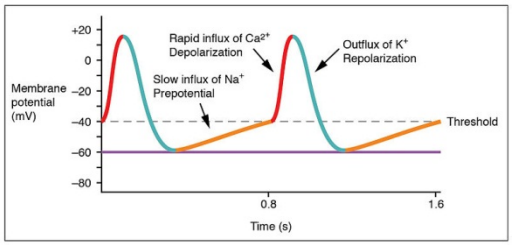
ACEM PRIMARY VIVA D **Thursday Morning Session 1** ***Candidate Number:*** **AGREED MARK:**

Stem: A 6 year old boy has sustained a laceration to the sole of his foot. It is to be repaired under ketamine sedation. We will start with Pharmacology.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Ketamine (pp 444-445) **Subject:** Pharm LOA: 1	1. What is the mechanism of action of ketamine?	**Antagonism of NMDA (**subtype of glutamate) **receptors**. Inhibits reuptake of catecholamines and serotonin	Bold
	2. What are its clinical effects?	**Dissociative anaesthetics**. Profound analgesia, stimulate sympathetic nervous system, bronchodilatation, minimal respiratory depression, stable CVS. increased Cerebral bd flow, partial amnesia, nystagmus	Bold +2
	3. What are its adverse effects? [Prompt- Are there any airway concerns?]	**Unpleasant emergence reaction (**eg vivid dreams or hallucination)**, laryngospasm**, increased salivation, vomiting, myoclonus	Bold
	4. Give an appropriate route and dose for procedural sedation in this child? [What other routes are available?]	**1-2 mg/kg IV, 4-10 mg/kg IMI**	Can state either IV or IM dose
Stem: During the procedure, he becomes hypoxic and requires assisted ventilation. We will now move to Physiology.			
Question 2 Lung volumes and curves (West pp 13-16) **Subject:** Phys LOA: 1	1. Draw a diagram that demonstrates the components of total lung volume.	Should correctly include TLC, VC, FRC, TV, RV, IRV, ERV	Bold to pass **TLC, VC, FRC, TV, RV** correct
	2. **In an adult**, what are the typical volumes of these components? [TLC, VC, RV, FRC and TV]	TLC ~7000ml, VC ~4500 to 5000 mL, RV ~1200 mL, FRC ~2400 mL, TV ~500mL	**2/4** (reasonable approximations)
	3. Which lung volumes can be measured in the ED? Extra:How are the other lung volumes measured?	Spirometer for FEV1 and FVC. TV on ventilator Helium dilution or body plethysmography for TLC, FRC and RV	**1/2 spiromoter**

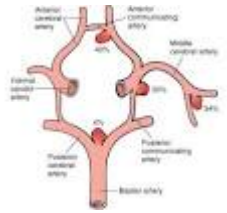
Stem: Once he has stabilised the repair continues. We will now move to Anatomy.			
Question 3 Sole of foot (photo) **Subject:** Anat LOA: 1	1. Identify the structures in this photograph?	<u>Medial to lateral</u> Abd hallucis (2); Fibrous flexor sheath (5); Flexor hallucis longus (11); Medial plantar nn (19/20); lateral plantar nn (14); Lateral plantar aa (13); Flexor digiti minimi brevis (7); Abd. Digiti minimi (1)	4/8 of this list
	2. You decide to perform a nerve block at the ankle. Describe the cutaneous nerve supply of the sole of the foot	**Posterior tibial nerve** - supplies sensation to most of volar foot and toes Medial, and lateral plantar nerves (terminal brs of tibial nn) **Sural nerve** **-** supplies lateral border (volar and dorsal) of foot Calcaneal branches (tibial and sural nn) supply heel	Bold
	3. What is the surface anatomy of these nerves at the ankle joint?	**Posterior tibial nerve-** runs with posterior tibial aa - located on **medial aspect ankle** between medial malleolus and Achilles tendon **Sural nerve** - located on **lateral aspect of ankle** between Achilles tendon and lateral malleolus	Bold
Stem: Once the procedure has finished, his mother asks you about the healing process. We will now move to Pathology.			
Question 4 Cutaneous wound healing (pp102-108) **Subject:** Path LOA:1	**1. Describe the phases of cutaneous wound healing?**	**1.Inflammation, proliferation, and maturation**. Phases overlap, and separation arbitrary. The initial injury -> platelet adhesion and aggregation + formation of clot on wound surface -> ***inflammation***. ***Proliferative*** phase -> formation of granulation tissue, proliferation and migration of connective tissue cells, and re-epithelialization of the wound surface. ***Maturation*** involves ECM deposition, tissue remodelling + wound contraction.	**2 of 3 phases in bold with correct descriptions to pass**
	2. What factors influence cutaneous wound healing?	***2.Systemic factors:*** **•Nutrition**. Protein deficiency and vitamin C deficiency, -> retard healing. **•Metabolic status** : Diabetes mellitus, -> delayed healing **•Circulatory status**: Inadequate blood supply or drainage (arteriosclerosis or varicose veins. **•Hormones** eg. glucocorticoids influence various components of inflammation, also inhibit collagen synthesis. ***Local factors: •Infection*** single most important cause of delay in healing, ***•Mechanical factors*****,** (early motion of wounds). ***•Foreign bodies*** impede healing. ***•Size, location, and type of wound (mechanism of injury)*****.**	**2 systemic and 2 local factors to pass**
	3. What is wound contraction?	***3.Wound contraction generally occurs in large surface wounds***. The contraction **helps to close the wound by** decreasing the gap between its dermal edges + **reducing the wound surface area**. Important feature in healing by secondary union. Initial steps of wound contraction involve formation, at the edge of the wound, of **a network of *myofibroblasts***.	**Bold to pass**

ACEM PRIMARY VIVA D **Thursday Afternoon Session 2** ***Candidate Number:*** **AGREED MARK:**

Stem: A 70 yo woman calls an ambulance for chest pain. She is administered Aspirin en route to hospital. We will start with Pharmacology			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Aspirin **Subject:** Pharm LOA: 2	1. Outline the mechanisms of action for aspirin.	**Irreversible** non-selective **cyclooxygenase inhibition** (Cox 1 and 2) resulting in (a) In platelets irreversible **inhibition of COX 1 results in reduction in** thromboxane A2 and **inhibition of platelet aggregation** for the life of the platelet (10 days), (b) **In tissues inhibits prostaglandin synthesis** (COX2). Results in **anti-inflammatory action,** Analgesic, and antipyretic effects.	Bold Need to mention platelet effect (Cox1) **AND** tissue (COX2) anti-inflammatory or analgesic effect.
	2. Describe the pharmacokinetics of aspirin.	Rapidly absorbed from stomach and intestine, aspirin hydrolysed to salicylic acid in plasma and blood, peak plasma level within 1-2 hrs. Serum half- life of aspirin 15 minutes, low protein binding, **saturable metabolism with increasing doses (switches from first to zero order metabolism).** Urinary alkalinisation increases excretion of salicylate and it's conjugates.	Bold plus 2
	3. Outline the adverse effects of aspirin.	GI upset, **Gastrointestinal bleeding from gastritis or peptic ulceration**, hepatotoxicity, hypersensitivity reactions (asthma, angioedema, rash), prolonged bleeding time from platelet inhibition.	Bold + 1 other.
Stem: She has a history of coronary artery disease. Moving onto Pathology.			
Question 2 Atherosclerosis **Subject:** Path LOA: 1	1. What are the systemic and local factors that lead to atherosclerosis?	1. **Hypertension, hyperlipidemia**, toxins from **cigarette smoke**, homocysteine, infectious agents. Inflammatory cytokines (e.g., tumor necrosis factor [TNF]) can also stimulate pro-atherogenic patterns of endothelial cell gene expression. The two most important causes of endothelial dysfunction are ***hemodynamic disturbances and hypercholesterolemia***. **Local flow disturbances (e.g., turbulence at branch points**) leads to increased susceptibility of certain portions of a vessel wall to plaque formation.	1. Bold to pass
	2. Which arteries are most often affected by atherosclerosis?	2. **Lower abdominal aorta, the coronary arteries, the popliteal arteries, the internal carotid arteries, and the vessels of the circle of Willis**.	2. 3 of 5 bold to pass
	3. How does an atherosclerotic plaque suddenly cause symptoms?	3. **Rupture, ulceration, or erosion** of the intimal surface of atheromatous plaques exposes the blood to highly thrombogenic substances and induces **thrombosis.** Such thrombosis can partially or completely occlude the lumen and lead to downstream ischemia **Haemorrhage into a plaque**. Rupture of the overlying fibrous cap, or of the thin-walled vessels in the areas of neovascularization, can cause intra-plaque haemorrhage. **Atheroembolism**: Plaque rupture can discharge atherosclerotic debris into the bloodstream, producing microemboli. **Aneurysm formation**: Atherosclerosis-induced pressure or ischemic atrophy of the underlying media, with loss of elastic tissue, causes weakness resulting in aneurysmal dilation and potential vessel rupture	3. 2 of 4 bold to pass

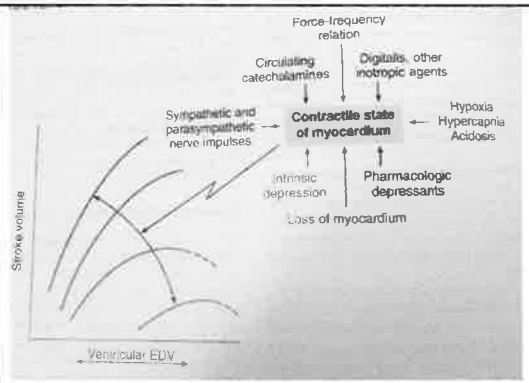
Stem: She has a history of palpitations. Moving onto Physiology.			
Question 3 Ventricular Tachycardia **Subject:** Phys LOA: 1	Draw and label the membrane potential of normal pacemaker tissue		Must identify fast upslope being due to **Ca influx** and repolarisation due to **K efflux plus presence of pre-potentia**l
	By what mechanisms can tachyarrhythmias be generated?	Increased automaticity (AT,VT) Accessory pathways (WPW) Re-entry loops (VT) Early afterdepolarisations (torsade de pointes) Delayed afterdepolarisations (as in digoxin toxicity)	Automaticity plus one other
	What conditions may predispose to increased automaticity?	IHD Previous repair of congenital heart disease (scar tissue) Structural heart disease Channelopathies (congen or acquired) Electrolyte imbalances (K, Mg, Ca) Sympathomimetic agents Infiltrative cardiac diseases	Mention at least one condition
Stem: She is hypotensive and this ECG is performed.			
Clinical Building Block – ECG	What rhythm does it show?	Broad complex regular tachycardia consistent with VT. Rate approximately 180bpm.	Must identify that broad complex, regular tachycardia or VT
Stem: Following successful DC cardioversion, she is still hypotensive and a central venous catheter is inserted for inotrope administration. Moving onto Anatomy.			
Question 4 Thoracic Inlet (photo) **Subject:** Anat LOA: 2	1. Identify the **vascular** structures in this photo.	1. Left common carotid aa (14); Right common carotid (19); Brachiocephalic trunk (4); Right subclavian aa (21); Right brachiocephalic vv(18); Left brachiocephalic vv (13); Subclavian vv (24); Left Internal jugular vv (8) Thyrocervical trunk (32)	5/9 to pass
	2. What *important* structures may be damaged during insertion of an IJ line?	2. **Carotid artery**, Phrenic Nerve, Brachial plexus, Pulmonary dome, Thoracic duct, Trachea, Subclavian vessels	Bold plus 1
	3. What clinical complications may occur from damage to these structures?	Ptx, Haemothorax, hydro/chylothorax, stroke, Air embolism,, bleeding, Haematoma=> airway obstruction., AV fistula, etc	Appropriate example for each
	[What other complications can occur from central line insertion?]	Line misplacement/misdirection, arrhythmias, Infection, Thrombosis/PE pericardial tamponade, catheter loss/embolus, wire knotting	

ACEM PRIMARY VIVA D **Friday Moring Session 3** ***Candidate Number:*** **AGREED MARK:**

Stem: A 75 year old woman sustains a head injury following a fall. Her GCS is13.			
TOPIC	QUESTIONS	KNOWLEDGE (essential in bold)	NOTES
Stem: A CT brain is performed			
Clinical Building Block: CT Brain	What is the major abnormality shown on her CT?	Right sided Subdural with midline shift	Side Subdural
Stem: We will now move onto Anatomy (use the abnormal CT brain for Anatomy)			
Question 1 CT Brain Subject: Anat LOA: 1	1. Could you identify some normal structures on this head CT? 2. What is the arterial supply of the brain?	Lobes: frontal, temporal, parietal, occipital Lat ventricle : anterior and posterior horns; 3rd ventricle Caudate nucleus; Lentiform nucleus (putamen & globus pallidus) Thalamus; Anterior & posterior limbs of internal capsule Septum pellucidum; Falcx **ACA** area anterior to anterior horns lat ventricle (**frontal and parietal lobes medially and superiorly**) **MCA** area between the ant & post horns LV (**most of lateral surface anterior, parietal, and temporal lobes**) **PCA** area posterior to posterior horn LV (**Inferior and medial aspects of occipital and temporal lobes**)	5 structures 3 major vessels + detail 
Stem: Moving onto Pathology			
Question 2 Traumatic Brain Injury Subject: Path LOA: 1	1. Which type of vessels have been damaged to produce the subdural blood seen on this CT? 2. Which groups of patients are most at risk for SDH and why? 3. How does an extradural haematoma occur? 4. Define and describe diffuse axonal injury?	1. Subdural blood comes from damage to **bridging veins between the brain and the venous sinuses** (displacement of the brain with in trauma can tear the veins at the point where they penetrate the dura to enter the sinuses) -> **blood between the dura and the arachnoid**. 2. **Elderly**- veins stretched and more movement due to brain atrophy Infants- thin walled bridging veins 3. Extradural hematoma occurs with **rupture of a meningeal artery**, usually associated with a skull fracture, leads to accumulation of arterial blood **between the dura and the skull**. 4. **Axonal microscopic injury** Micro findings include **axonal swelling and focal haemorrhagic** lesions. Believed to **damage the integrity of the axon at the node of Ranvier, -> alterations in axoplasmic flow. Commonly found with 'coma' but no cerebral contusions.**	Bridging **veins** Elderly **Meningeal (often middle) artery** **Microscopic damage to deep brain white matter**

Stem: Her GCS has fallen to 8. We will now move onto Physiology			
Question 3 CNS Autoregulation / Cushing response Subject: Phys LOA: 1	1. What factors affect cerebral blood flow?	1. MAP at brain level MVP at brain level ICP Viscosity of the blood Local constriction/dilatation of cerebral arterioles	Pass in bold **3/5**
	2. What is the mechanism of the Cushing response?	2. Increase in ICP results in Decr CBF – ischaemia of VMA – SNS output incr - Incr systemic BP – stimulation of baroreceptors – stimulation of vagal outflow – decr HR and RR	**Explains concept**
	3. What is the Monro-Kellie doctrine?	3. The volume of blood (75mL), CSF (75mL) and brain (1400g) in cranium must be relatively constant. Negative effects on these therefore if additional intracranial volume eg SDH / EDH occurs	**Explains concept**
Stem: She has a seizure and you decide to treat her with Phenytoin. We will now move onto Pharmacology.			
Question 4 Phenytoin Subject: Pharm LOA: 1	1. What is the mechanism of action of phenytoin?	Primarily **Na+ channel blockade/reduced neuronal Na+ conductance** and prolongation of inactivated state of Na+ channel. Reduces Ca++ influx into cells and decreases glutamate release and enhances GABA release. Inhibit the generation of rapidly repetitive action potentials	Bold
	2. What are the risks associated with intravenous phenytoin administration?	**Hypotension and bradycardia with rapid infusion** (due to diluent). Allergic reactions. Limit rate of infusion to maximum 50mg/min (30-60 minutes). Less likely with fosphenytoin.. Local necrosis if extravasation	Bold to pass.
	3. Describe the elimination kinetics of phenytoin and why it is important clinically?	**Dose-dependent elimination.** First order elimination at low serum concentrations, however elimination **becomes zero-order as concentration rises** with prolongation of elimination half-life. Implication- Small recurrent dose increase may => toxicity	Explains concepts
	4. What are the common features of acute overdose/intoxication with phenytoin?	Sedation, coma, nystagmus, ataxia, cerebellar toxicity. No cardiac toxicity with ingested overdoses of phenytoin.	2 to pass

ACEM PRIMARY VIVA A **Thursday Morning Session 1** ***Candidate Number:*** **AGREED MARK:**

Stem: An 80 year old man who is on warfarin is brought in following a motor vehicle accident in which he sustained multiple injuries. On arrival in ED, his blood pressure is 80/40 and pulse rate is 130 / minute. A chest X-ray is done.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 **Clinical Building Block:**	Please describe the abnormalities on this CXR	**Surgical emphysema, Pneumothorax**, RML changes ? consolidation or contusion	Bold to pass
Moving on to Physiology			
Question 2 Frank- Starling Curve **Subject:** Phys LOA: 1	1. Please draw the Frank Starling curve as it relates to human cardiac muscle Prompt: What effect does EDV have on SV? 2. What factors influence the Frank-Starling curve?	 2. Circulating catecholamines; inotropes, hypoxia, hypercarbia, acidosis, pharmacol depressants; loss of myocardium; intrinsic depressing; symp NS & PSym, fluid status	Q2.1 – to pass must be able to draw the FS curve including the hump and correctly label axes (SV or Pressure on y axis) Q2.2 – 4 factors with correct influence

Stem: Moving onto Pathology. He has multiple facial lacerations which are bleeding			
Question 3 Haemostasis **Subject:** Path LOA: 1	1. What are the sequence of events in haemostasis after a vascular injury? Prompt: Is there any particular sequence to the events?	a. **Vasoconstriction**: arteriolar, reflex neurogenic, enhanced by endothelin b. **Primary haemostasis**: extracellular matrix exposed, pl adherence/activation - - pl aggregates & forms plug c. **Secondary haemostasis**: Tissue factors exposed, Fac III, thromboplastin, Fac VII, platelet plug consolidated - thrombin/fibrin generated d. **Thrombus & antithrombotic effect** – fibrin polymerises to form permanent plug, tPA regulates	Q3.1 – to pass identify 3/4 steps of hemostasis (bold) in correct sequence
	2. What laboratory tests are used to assess the function of the different pathways of the coagulation cascade? Prompt: Which one is vitamin K dependant	**Prothrombin time – extrinsic pathway** factors VII, X, II, V, fibrinogen (including vit K dependent factors) **Partial thromboplastin time – intrinsic pathway** factors XII. XI, IX, VIII,X, V, II, fibrinogen	Q3.2 – to pass identify test, what pathway it is testing and identify which one is vit K dependant
Stem: Moving onto Pharmacology. It is decided to reverse his anticoagulation and Vitamin K is administered.			
Question 4 Vitamin K and warfarin **Subject:** Pharm LOA: 2 and 1	1. What is vitamin K?	**Fat-soluble substance** in leafy vegetables; **usually synthesised by gut bacteria**. Vit K1(food) & K2(bact)	Q4.1 Bold to pass
	2. Please describe its mechanism of action in reversal of warfarin anticoagulation Prompt: How long does it take for the onset of action	**Warfarin** – coumarin anticoagulant, prevents reductive metabolism of inactive vit K to active form so produces **biologically inactive** VII, IX, X, prothrombin, protein C&S **Vit K1 confers biologic activity** upon prothrombin and factors VII, IX, X by participating in their postribosomal modification. **Onset of action 6 hours**, complete by 24 hours	Q4.2 to pass need concept of warfarin producing biologically inactive factors, vit K overcoming this, & delayed onset of action

Stem: Moving onto Anatomy. Following stabilisation, a secondary survey is undertaken and his facial wounds are closely inspected.			
Question 5 Face Dissection (model) **Subject:** Anat LOA: 2	Demonstrate on the model the arterial supply to the face?	**Facial artery (61)** – arises External Carotid a. – contacts submandibular gland, hooks up over mandible anterior to masseter m. then a tortuous course to the medial angle of the eye. **Transverse facial artery**(62)- anastomoses with above	Bold to pass and demonstrate on model
	You are concerned about injury to his facial nerve. Using the model can you demonstrate the branches of the facial nerve? Prompt: Start from the parotid gland	Facial n. (66) – motor supply of the face **5 branches** – Temporal n(67), Zygomatic n (68), Buccal n (69), Marginal Mandibular n (70), Cervical n (Exits BOS at stylomastoid foramen)	Bold + 3/5 branches
	What is the function of the facial n.?	**Motor supply to the face** Muscles of facial expression Taste anterior 2/3 rds tongue	Bold
	What is the sensory nerve supply of the face? Prompt: What are the branches?	**Trigeminal n.** (5th Cranial n) - **3 branches**: Ophthalmic , maxillary, mandibular NB – not on model – but candidate can map sensory division supply	Bold + 2/3 branches

ACEM PRIMARY VIVA A **Thursday Afternoon Session 2** ***Candidate Number:*** **AGREED MARK:**

Stem: An 80 year old woman is noted to be in heart failure. Starting with Pathology			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Heart Failure **Subject:** Path LOA: 1	What is heart failure?	When **cardiac function is impaired** and/or the **heart is unable to maintain a cardiac output** sufficient for the body's metabolic needs.	Bold to pass
	Please classify the types of heart failure? Prompt: Examples?	**Pump failure:** **Systolic dysfunction (Contractile dysfunction)** eg myocardial contractile dysfunction secondary to ischaemia, AMI, pressure or volume overload, dilated cardiomyopathy. **Diastolic dysfunction (Inadequate filling)** eg LV hypertrophy, myocardial fibrosis, amyloidosis, pericarditis. **Others:** arrhythmias, regurgitant flow eg MR, outflow obstruction eg AS, HOCM **Left heart failure** (IHD, HT, Valvular diseases eg AS, rheumatic heart disease, myocardial disease) **Right heart Failure** (eg secondary to left heart failure, PE, Pulmonary HT etc)	One of the classifications with examples
	What are the clinical features of heart failure? Prompt: What symptoms or signs from other organ systems might occur with heart failure?	Lung –breathlessness, orthopnoea, PND, APO, pleural effusions Cardiac – 3rd HS, gallop, displaced apex beat, AF, murmur, JVP elevation Renal – RAA activation – with fluid retention, pedal oedema, AKI Brain – confusion secondary to hypoxia Hepatic –engorgement, ascites, cirrhosis(late)	3/5 organ system symptoms to pass

This woman also has chronic renal failure. An ECG is obtained.			
Question 2 **Clinical Building Block:** ECG – hyperkalaemia	What are the abnormalities on the ECG? What is the likely diagnosis?	**Widespread peaked T waves**, mild tachycardia, some inverted T waves, ST elevation **Suggestive of hyperkalaemia**	Bold
Stem: Moving onto Physiology			
Question 3 ECG and hyperkalaemia & Renal handling of K+ **Subject:** Phys LOA: 1	What are the ECG changes in Hyperkalaemia?	Peaked T waves (repolarisation abnormality) P wave flattening, Loss of P waves (progressive atrial paralysis) QRS widening / bizarre QRS (Conduction abnormality) Sinusoidal ECG Ventricular arrhythmias Asystole	3/6 to pass
	Describe the way the kidney handles K^+ Prompt: What happens to K^+ in Proximal Tubule? Prompt: What happens to K^+ in distal tubule?	K^+ is filtered at the glomerulus. **Most filtered K^+ is actively reabsorbed at the proximal tubules.** **K^+ is then secreted into the fluid by the distal tubules.** (Rate of K^+ secretion is proportionate to distal tubular fluid flow) At Distal tubule: K+ secretion is passive and Na^+ is reabsorbed. In a healthy person: the amount of K^+ secreted = K^+ intake and K balance is maintained Normally > 93% K^+ is reabsorbed by the kidneys. K^+ secretion & excretion alter depending on serum K^+ and H^+	Bold to pass

Stem: Moving on to pharmacology. Her medications include digoxin.			
Question 4 Digoxin **Subject:** Pharm LOA: 1	What is digoxin's mechanism of action in heart failure	**Ca accumulation in cells** (due **Na- K+ ATP block**, Na in cells drive Na/Ca exchange) leads to a) **increased contraction strength,** b) > stroke vol/ CO per beat- with smaller EDSV, small heart, reduced RHt pressures/ volume c) slower HR- >er stroke volume (partic if AF), via effects on parasympathetic fibres/AV node	2/3 bold + one other
	Why are patients in heart failure prone to digoxin toxicity?	a) **poor renal function from low C/O,** b) **potential dehydration and/or other drug interactions** (e.g. ACE/ diuretics/ spironalactone/ ca channel blockers) c) potential effects on effective vol of distribution d)low K+ from other ht failure meds esp diuretics (makes pts higher risk from dig/toxicity) e) poor cardiac reserve/ output, altered digoxin handling during acute HF/ fluid distribution changes/other major illnesses	To pass 2 including 1 bold
	What are the features of digoxin toxicity Prompt: Any features from other organ systems	a) **high K (assocd strongly with mortality)** b) yellow/ green (or other) colour vision c) GI- D and V, nausea/ malaise-anorexia/ d) arrhythmias from > automaticity and also Av node block (partic brady but R on T as well) e) severe heart blocks- partic if previous blocks , worsening failure, low BP f) CNS, tiredness -lethargy- headaches, paraesthesias, Candidate may differentiate acute vs chronic	To pass hyperkalaemia + at least 2 others from 2 different groups.

Stem: Moving on to anatomy			
Question 5 Heart Model **Subject:** Anat LOA: 1	1.Using the model identify the great vessels and branches which enter and exit the heart	**Superior vena cava** - R brachiocephalic v, L brachiocephalic v **Inferior vena cava** **Ascending aorta** - brachiocephalic trunk, L common carotid artery, L subclavian artery **Pulmonary trunk** and pulmonary arteries **Pulmonary veins**	**(bold to pass)**
	2. Identify the main coronary arteries and their branches	RCA LCA Circumflex LAD/ant interventricular Marginal	4/5 to pass
	3. Which areas of the heart is supplied by the LCA?	Most of the left atrium **Most of left ventricle** Part of right ventricle Intraventricular septum **AV bundle** (SA node in 40%)	Bold +2 to pass
	4. (If required)Describe the position of the heart in the left hemithorax	Inferior border lies on the diaphragm Apex is in the 5th ICS Base is against the Thoracic vertebrae T6 to T9	

ACEM PRIMARY VIVA A **Friday Morning Session 3** ***Candidate Number:*** **AGREED MARK:**

Stem: A 40 year old woman presents with left loin pain and fevers. Urine microscopy is performed			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 **Clinical Building Block:** Urine Microscopy	Please describe the abnormalities. What is the most likely diagnosis?	**High poly and RBC counts** with +ve protein and blood (in the absence of epi-clean catch) indicates **infection** In the clinical context c/w **pyelonephritis** +/- stone	Bold to pass
Question 2 Pyelonephritis **Subject:** Path LOA: 2	What organisms cause acute pyelonephritis? Prompt: what are the most common? What steps are involved in ascending infection of the urinary tract? What conditions predispose to acute pyelonephritis?	**G-ve bacilli** (>85%), endogenous organisms E Coli, proteus, klebsiella, enterobacter, strep faecalis(enterococcus) Other: staph, fungi, (viruses in immunocompromised and renal transplant patients) **5 steps:** 1. colonisation distal urethra 2. entry into bladder 3 . urinary tract obstruction / stasis of urine 4. vesicoureteric reflux 5. intrarenal reflux Urinary tract obstruction Instrumentation Vesico-ureteric reflux Pregnancy Female upto 50yrs Males >50 yrs Abnormalities- congenital/acquired DM, Immunosuppression	G-ve & 3 organisms pass Need to explain the concept clearly 4/9 to pass

Stem: Moving onto Physiology			
Question 3 Renal Circulation **Subject:** Phys LOA: 1	What is normal renal blood flow?	**Renal blood flow = approx 1250 mL/min**	Bold (accept 1000 – 1500)
	What substances influence renal blood flow and how?	Noradrenaline-constriction, Dopamine, ACh -dilatation Angiotensin II – constricts afferent and efferent arterioles PGs-increase flow in cortex and decrease in medulla	2/5 substances + correct action
	How can renal blood flow be measured? Prompt: What substance can be used to measure renal plasma flow?	1. Fick principle (amount of a substance taken up per unit time divided by arterio-venous concentration difference) 2. PAH (or any substance that is 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 3. Actual renal plasma flow = ERPF/0.9 = 700 mL/min 4. Renal blood flow = RPF x 1/1-Hct (Hct = 0.45)	Concept/Principle

Stem: Moving onto Pharmacology. She is treated with Gentamicin			
Question 4 Gentamicin **Subject:** Pharm LOA: 1	1. Describe the mechanism of action of gentamicin	**Irreversible inhibitor of protein synthesis.** **Binds** 30S **ribosome** & inhibits protein synthesis by: 1) interfering with initiation complex of peptide formation 2) Inducing misreading of mRNA thus producing non functional protein; 3) causing break up of polysomes into non-functional monosomes *Additional information*: Enters cell by passive diffusion via porin channels across outer membrane, then enters cytoplasm by o2 dependant active transport process (transport coupled to a proton pump the transmembrane electrochem gradient supplies the energy) Low ecf pH & anaerobic conditions inhibit transport as reduces gradient; transport enhanced by cell wall active drugs eg penicillin, vancomycin.	Bold to pass
	2. What are the benefits of once daily dosing of gentamicin? *Prompt how does this improve clinical effectiveness?*	**Concentration dependant killing** (at increased conc kill increased no of bacteria at a more rapid rate); Post antibiotic effect (effect lasts longer than detectable serum levels); **Reduced toxicity** (as toxicity is time & conc dependant –time above critical level will be longer with multi dose than single dose schedule); less nursing time; OPD therapy possible; convenience	Bold to pass
	3. What micro-organisms is it effective against? Prompt: What group of organisms	**Gram –ve bacteria** – E. coli, Pseudomonas, Proteus, Klebsiella, Serratia Gram +ve- Staph, Strep- with beta lactams, vancomycin No anaerobic activity	Bold + 3 organisms

Stem: Moving onto Anatomy. A KUB X-ray is performed			
Question 5 AXR - ureters **Subject:** Anat LOA: 2	1. Could you point out on the xray the course of the L ureter	From **hila of kidney L1-2**, along transverse processes, just medial to **tips of transverse processes of lumbar vertebrae**, on ant surface of psoas muscles, pass over pelvic brim around SI joint, run along lateral wall of pelvis till **ischial spine, then medially to enter bladder**	Bold
	2. Where in the ureters is a stone likely to lodge	PUJ, pelvic brim, VUJ	2/3
	3. Where else could a stone be present	Kidneys, bladder	1/2
	4. (only if required) What other structures can you identify on the xray (not required for pass)	Liver, Large Bowel, Lumbar spine, Pelvis, Femoral heads, Ribs, Psoas	

ACEM PRIMARY VIVA B **Thursday Morning Session 1** ***Candidate Number:*** **AGREED MARK:**

Stem: An 18 year old woman presents to the ED, 3 days following a self-inflicted wrist injury. She complains of numbness in her hand. We will start with Anatomy			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Wrist dissection (photo) **Subject:** Anat LOA: 1	(a) Identify the structures seen in this image	**Median nerve** (16) + branches (17, 18, 19) **Ulnar nerve** (25) + branches (24, 26, 27, 28) Ulnar artery (23) Thenar muscles – APB (2), FPB (13) Adductor pollicis (4), Lumbricals (7, 22) FCR (8), FCU (9), BR (5), FDS (12). FDP (11)	Median nerve, ulnar nerve, AND 6 out of 10 structures
	(b) Describe the median nerve supply in the hand	Motor – **LOAF (1st & 2nd lumbricals, OP, APB, FPB** [superficial head]) Sensory – **palmar surface of lateral 3½ digits** & dorsum of distal halves of these digits	Both motor (LOAF, 2 out of 4 muscles needed to pass) and sensory
	(c) How would you clinically test the median nerve function in the hand?	Motor – **thumb opposition** (OP) or **thumb abduction** (APB) [thumb flexion not reliable as deep head of FPB supplied by ulnar nerve] Sensory – sensation over volar aspect of **lateral 3½ digits** [sensation over thenar eminence preserved as supplied by palmar cutaneous branch of median nerve]	Both motor and sensory
Stem: Moving onto Physiology.			
Question 2 Resting Membrane Potential **Subject:** Phys LOA: 1	(a) Define resting membrane potential of a neuron	**Potential difference** across cell at rest, as a result of separation of positive and negative electronic charges across cell membrane (**inside negative** relative to outside of cell). Normal RMP of neuron = -70mV	Bold
	(b) Explain how resting membrane potential is created Prompt: Why is RMP negative on the inside of a cell?	Main ions involved – **Na^+ & K^+** **Na^+-K^+-ATPase pump** creates **electrochemical gradient** by pumping out 3 Na^+ for every 2 K^+ pumped in **Na^+ & K^+ diffuse down concentration gradient** across permeable cell membrane (K^+ diffuses from inside to outside of cell; opposite for Na^+) Cell membrane **more permeable to K^+** at rest → that's why RMP is close to equilibrium potential for K^+ RMP represents an equilibrium state; driving force for ions down concentration gradient = driving force down electrical gradient	Bold
	(c) Why is a cell more excitable in hyperkalaemia	RMP moves **closer to threshold potential for eliciting action potential** (becomes less negative on the inside of cell).	Bold

Stem: Moving onto Pathology. On examination there is a purulent discharge coming from the wound.			
Question 3 Staph aureus **Subject:** Path LOA: 1	(a) Name some common bacteria that cause wound infections	**Staphylococcus aureus** **Streptococcus pyogenes** Clostridium perfringens Aerobic Gram negative bacilli Pseudomonas aeruginosa Clostridium tetani	Staph aureus, Strep and 1 other
	(b) What diseases are caused by Staphylococcus aureus?	Skin / soft tissue : cellulitis, impetigo, abscess (furuncle, carbuncle), folliculitis, paronychia, felon, lymphadenitis, necrotising soft tissue infection, scalded skin syndrome Pneumonia Endocarditis Osteomyelitis / septic arthritis Food poisoning Toxic shock syndrome	3 skin and 3 non-skin infections
	(c) Describe the clinical features of Staph. Aureus toxic shock syndrome.	Hypotension (shock), renal failure, coagulopathy, liver disease, respiratory failure, generalised erythematous rash, soft tissue necrosis at site of infection	4 out of 7 (must have specific organs)
Stem: Moving onto Pharmacology. Prior to surgery for debridement, Flucloxacillin is administered			
Question 4 Flucloxacillin **Subject:** Pharm LOA: 1	(a) What micro-organisms are susceptible to flucloxacillin Prompt: is fluclox active against all Staph?	**Staphylococci** (including B lactamase producing**), streptococci** (not active against enterococci, anaerobes, Gram negatives, MRSA)	Bold
	(b) What is the mechanism of action of flucloxacillin Prompt : how does penicillin work	Inhibits bacterial growth by binding to active site of PBPs, **interfering with transpeptidation of bacterial cell wall synthesis** → cell death **(bactericidal)**	Bold
	(c) Why is oral flucloxacillin given before meals	It is acid labile (inactivated by gastric acid), and binds to food proteins (decreasing absorption)	1 out of 2
	(d) What are the important side effects of flucloxacillin?	**Liver (cholestasis),** GI upset (Nausea, vomiting, etc), renal interstitial nephritis, neutropenia/thrombocytopenia, **allergy/anaphylaxis**, serum sickness.	Both bold to pass
	Extra question: What is the frequency of cross allergenicity between flucloxacillin and cephalosporins	Around 5-10%	Any % in range to pass

ACEM PRIMARY VIVA B **Thursday Afternoon Session 2** ***Candidate Number:*** **AGREED MARK:**

Stem: A 50 year old alcoholic presents with a GCS of 7 and respiratory depression. Starting with Physiology			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Control of Ventilation **Subject:** Phys LOA: 1	(a) What are the receptors involved in the control of ventilation?	**Central chemoreceptors, Peripheral chemoreceptors,** Pulmonary stretch receptors, Irritant receptors, J receptors, Bronchial C fibres, Nose and upper airway receptors, Joint and muscle receptors, Gamma system, Arterial baroreceptors, Pain & temperature receptors	Bold & 3 others to pass
	(b) Where are the central chemoreceptors located?	200-400 μm below ventral surface of **medulla.**	Medulla must be stated
	(c) How do these receptors function? Prompt : How do H^+ ions affect their function	BBB permeable to CO_2; relatively impermeable to HCO_3^- **↑blood pCO_2 → ↑CSF pCO_2 → ↑H^+ in CSF** **↑H^+ in CSF stimulates ventilation** ↓H^+ in CSF inhibits ventilation; causes cerebral vasodilation → enhance diffusion of pCO_2 into CSF CSF pH 7.32. Less buffering than blood, CSF pH changes more for given pCO_2 Prolonged pH changes compensated by HCO_3^- transport across BBB. (Chronic CO_2 retention has near normal CSF H^+)	Bold concepts to pass
Stem: He requires intubation and vecuronium is administered. Moving on to pharmacology.			
Question 2 Vecuronium **Subject:** Pharm LOA: 1	(a)What is the mechanism of action of vecuronium	**Non depolarising neuromuscular blockade** **Competitive antagonist** for acethylcholine at **nicotinic receptors** of **neuromuscular junction** Large doses will enter ion channel's pore directly → more intense blockade Also blocks prejunctional Na channels → interfere with Ach mobilization at nerve endings	Must mention blockade type, & either receptor type or ACh.
	(b) Describe the pharmacokinetics of vecuronium Prompt : what is its onset time, what is its duration of action, how is it eliminated	Highly polar/ionic Poorly absorbed from GIT Given IV **Onset within 1 min;** **Max effect at 3-5 mins** **Duration of action : 20-35 mins** **Short half life** **Rapidly distributed** to extracellular space Small volume of distribution (~blood vol), Plasma protein binding : 60-90%, **Eliminated by liver** (75-90%), rest by kidney	4 of 6 bold to pass

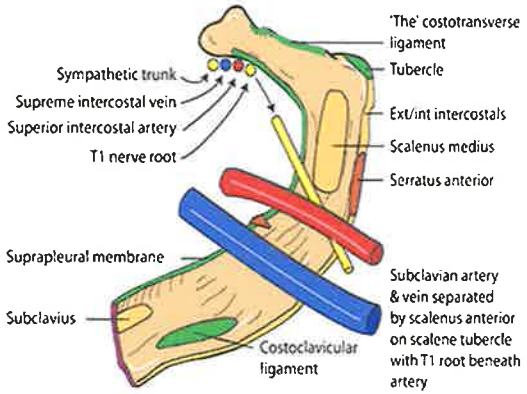
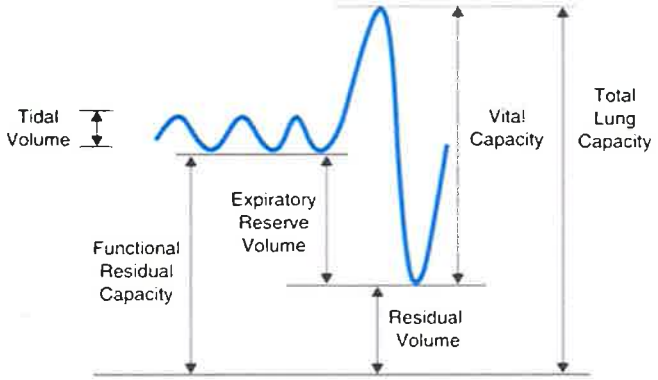
Stem: Following intubation he requires inotropic support and a central line is inserted. Moving onto Anatomy			
Question 3 Anterior Neck Photo **Subject:** Anat LOA: 1	(a) Identify the **venous** structures in this photo	SVC (26), right brachiocephalic v (18), left brachiocephalic v (13), subclavian v (24), internal jugular v (8), inferior thyroid v (7)	4 to pass
	(b) Identify the **nerves** in this photo	Phrenic nerve (17), right vagus nerve (22), right recurrent laryngeal nerve (20), left vagus nerve (15), sympathetic trunk (28)	3 to pass
	(c) What is the difference between the course of the right and left recurrent laryngeal nerve	Right : hooks around subclavian artery Left : hooks around aorta After looping, they ascend in trachea-oesophageal groove to supply intrinsic muscles of larynx (except cricothyroid)	Both to pass
Stem: These are his coagulation blood results.			
Question 4 **Clinical Building Block:** Coagulopathy	What is the abnormality on this coagulation profile	Delayed clot formation in both the extrinsic (PT / INR) and intrinsic (APTT) systems. Fibrinogen low. Consistent with a consumptive coagulopathy/DIC	Must state coagulopathy / DIC with one example of possible cause
	What could cause this	Sepsis, liver failure, malignancy, trauma, envenoming (Brown / Tiger / Taipan) etc	
Stem: Moving onto Pathology			
Question 5 Cirrhosis **Subject:** Path LOA: 1	(a) What types of liver disease may result from chronic excessive alcohol consumption	Hepatocellular steatosis (fatty change) – reversible Alcoholic hepatitis – reversible Cirrhosis – non reversible Hepatocellular carcinoma – non reversible	1 reversible and 1 non-reversible
	(b) What are the morphological features of cirrhosis Prompt : what happens to liver cells when chronically exposed to toxins or injurious agent	Occurs **diffusely** throughout the liver, **parenchymal nodules (regenerating hepatocytes)** surrounded by dense **bands of fibrous scar, disorganised architecture**, variable degrees of **vascular / portosystemic shunting,** elements of progression and regression	3 out of 5 bold to pass
	(c) What are the possible sequelae of cirrhosis	**Portal Hypertension**, GIT Bleeding, Hepatic Failure, Coagulopathy, Hepatocellular Ca, Hepatorenal Syndrome, Hepatopulmonary Syndrome, Encephalopathy, Infection	Bold plus 3 others

ACEM PRIMARY VIVA B **Friday Morning Session 3** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: A motor bike accident victim is transferred from a rural ED to a trauma centre. A chest X-ray is performed post intubation			
Question 1 **Clinical Building Block:** CXR- Pul contusions	Describe the positive findings in this CXR. What is the likely cause?	Portable supine CXR, ETT insitu (2cm above carina), **increased opacities in both lungs** (interstitial & alveolar) – increased opacity in RLL & obliteration of right hemidiaphragm **Pulmonary contusion** (+/- haemothorax)	Must be able to describe CXR, opacities. Pneumothorax difficult to exclude on supine film. Must say pulmonary contusion
Stem: He is hypoxic. Moving on to physiology			
Question 2 Oxygen uptake along the pulmonary capillary **Subject:** Phys LOA: 1	(a) In an alveolus, what factors affect oxygenation (b) Describe the oxygen uptake along a pulmonary capillary (c) How does hypoxia affect oxygenation	**Ventilation, perfusion, diffusion across the blood gas barrier** and alveolar-pulmonary capillary pO_2 gradient **Alveolar pulmonary capillary O_2 gradient** (Alveolar pO_2 = 100mmHg, pulmonary capillary pO_2 = 40mmHg), blood gas barrier thickness 0.3 microns, **RBC transit time = 0.75s** Under normal circumstances, **O_2 uptake is perfusion-limited** (complete in 0.25s) **& alveolar end capillary O_2 difference is minimal.** Rate of rise of end capillary pO_2 is steep – O2-Hb dissociation curve **Alveolar pulmonary capillary O_2 gradient is decreased, O_2 diffusion is decreased** & rate of rise of pO_2 for given O_2 concentration in blood is less	3 Bold to pass Must have knowledge of 3 of 4 concepts in bold. Numbers not required to pass. Can draw graph to explain (West pages 28-29)
Stem: He is quadriplegic and hypotensive. Moving onto Pharmacology. A Noradrenaline infusion is commenced			
Question 3 Noradrenaline **Subject:** Pharm LOA: 1	(a) What receptors do NA act on (b) How does NA increase blood pressure Prompt : what is the effect of NA on blood vessels (c) How does NA affect the heart rate?	**Predominantly α 1 receptor** → vascular smooth muscle constriction Also α 2 receptor (presynaptic) – inhibits NA release (negative feedback) Some effect on β 1&2 receptors (more potent effect on β 1) α 1 activity → vasoconstriction → **↑ total peripheral resistance** → ↑DBP β 1 activity → **↑ myocardial contractility** → ↑ SBP Overall rise in both DBP & SBP β 1 activity ↑heart rate. However compensatory baroreflex causes reflex bradycardia → **therefore minimal change in HR**	Need to mention predominant α 1 and one other receptor. Bold Bold

Stem: You suspect that he must have a cervical spinal cord injury. Moving onto Anatomy			
Question 4 Bones – C1, 2 **Subject:** Anat LOA: 1	(a) Identify this bone	**C2 (axis)**	Must say C2.
	(b) Describe its features	On C2; (1)body,**(2)dens**, (3)impression for alar ligament, (4)superior and inferior articular surface, (5)pedicle, (6)lamina, (7)bifid spinous process, (8)transverse process with foramen, (9)vertebral foramen	Bold + 4 other features
	(b) Name the ligaments that stabilize the atlanto-axial joint Prompt : how is the dens kept in place	**Transverse ligament** Superior & inferior longitudinal band } Cruciate lig Alar ligaments Tectorial membrane (continuation of post long lig) Anterior atlanto-axial membrane (continuation of ant long lig) Posterior atlanto-axial membrane (continuation of lig flavum) Apical ligament	Transverse lig & 2 others
	(c) What movement occurs at the atlanto-axial joint	**Rotation** around vertical axis	Bold
Stem: Moving onto Pathology			
Question 5 Spinal Cord injury including cellular injury as it relates to spinal cord **Subject:** Path LOA: 2 and 1	(a) What changes occur in the spinal cord after a traumatic injury	**Acute phase :** haemorrhage, necrosis, axonal swelling in the surrounding white matter at level of injury **Late phase :** area of neuronal destruction becomes cystic & gliotic, 2° wallerian degeneration involving long white matter tracts, liquefactive necrosis often seen in CNS	1 acute, 1 late
	(b) What are the features of irreversible injury at the cellular level	**Mitochondrial damage:** Failure of oxidative phosphorylation → **ATP depletion** → failure of energy dependent cellular functions **Membrane damage:** Plasma membrane → loss of osmotic balance Lysosomal membrane → **enzyme leakage** → cell necrosis	3 out of 4 bold
	(c) What are the acute clinical consequences of a cervical spinal cord injury Prompt: what happens in a high cervical level injury?	Complete or incomplete Spinal shock - **Quadriplegia**/flaccid paralysis, total anaesthesia, areflexia If above C4 → **respiratory compromise** (diaphragmatic paralysis) Neurogenic shock : hypotension, bradycardia, warm dry skin etc Incomplete syndromes, eg anterior cord, central cord etc	Bold

Stem: A 20 year old motor cyclist is brought to the ED with chest injuries. He is asthmatic.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: Let's start with Pharmacology. He has been given Salbutamol nebulisers for his wheeze.			
Question 1 Salbutamol **Subject:** Pharm LOA: 1	1. What is Salbutamol? 2. Describe the **pharmacokinetics** of salbutamol? (Prompt for t1/2) 3. Describe the **pros and cons** of the different **routes** of delivery of salbutamol? Prompt: Is there any other route? (non-inhaled)	Salbutamol is a selective B2 agonist and used as a Bronchodilator 1. **Absorption – Fast and complete (inhaled)** a. GIT – rapidly absorbed b. Inhaled – Bronchodilation maximal within 15-30 min and persists for 3-4 hours. 2. Metabolism – 50% 1st pass. Sulphated in the liver and metabolites excreted in the kidneys (also excreted unchanged in renal. No metabolism in lungs 3. t1/2 – 3-6 hours **1. Inhaled** a. Spacer/inhaler Pro: Targeted, low dose, minimise systemic side effects. As effective as nebulised. No 1st pass metabolism Con: Coordination and education required b. Nebulised Pro: Less coordination required and minimal education Con: Larger particles and hence dose required, noisy (children get frightened), higher incidence of systemic SE 2. Oral Pro: Easier in very young/disabled. Longer t1/2 Cons: Big doses, high SE profile (tachycardia, tremor, nervousness and weakness). Minimal advantage to inhaled. 50% first pass metabolism 3. IV/IMI/SC – useful in severe asthma Pro: No first pass metabolism Con: Needle, painful, higher cost and SE profile	**A selective B2 agonist** **1. Absorption – Fast or complete (inhaled).** **2. Rapid onset of action** **3. t1/2 3-6 hours** **(2 of 3 to pass)** **Need to describe pros and cons of Inhaled plus 1 other route**

Stem: Arterial blood gases are done as part of his initial trauma work up.			
Question 2 **Clinical Building Block:**	Please **describe** this ABG. On O2 – Fi02 60% P02 85 pCO2 123 pH 6.99 HCO3 28	Primary respiratory acidosis with CO2 retention and hypoxia	**Primary respiratory acidosis with CO2 retention and hypoxia**
Stem: Moving onto Anatomy. Chest X-ray shows multiple rib fractures.			
Question 3 First rib **Subject:** Anat LOA: 1	1. Please identify this bone and demonstrate its features **(bold to pass)** Prompt: What's this? (scalene tubercle) 2. What are the important relations?	**First rib** Head/neck/shaft/ tubercle (articulates with TP of T1) /articulation with costal cartilage to manubrium **/groove for subclavian vein** (ant) **and artery** (posterior to **scalene tubercle**) **Apex of lung** **Subclavian vessels**, intercostal vessels & ns Sympathetic trunk Lower trunk of brachial plexus (sup.) Scalenus ant/ medius Intercostals, Serratus anterior, Subclavius	**RELATIONS AND ATTACHMENTS OF LEFT FIRST RIB**  The under surface of the 1st rib is smoother. When the rib is laid on a flat surface, the head touches the flat surface when the rib is the correct way up
Stem: Moving onto Physiology.			
Question 4 Lung Volumes and Curves **Subject:** Phys LOA: 1	1. **Draw** a diagram that demonstrates the components of total lung volume.	Should correctly include TLC, VC, FRC, TV, RV, ERV  	TLC, VC, FRC, TV, RV, ERV **(3/6 to pass)**

	2. What are the typical volumes? Optional: Which of these volumes can be measured in the ED?	TLC ~7000ml, VC ~4500 to 5000 mL, RV ~1200 mL, FRC ~2400 mL, TV ~500mL FEV1, FVC or TV.	**2/4** (reasonable approximations)
Stem: Moving onto Pathology.			
Question 5 Asthma **Subject:** Path LOA: 1	1. What are the pathological features of acute asthma? 2. What is the underlying mechanism of atopic asthma? Prompt: What may trigger an exacerbation? 3. What happens in the early-phase reaction in atopic asthma?	1. **Increased airway responsiveness; episodic bronchoconstriction; bronchial wall inflammation; increased mucus** 2. **IgE mediated type 1 hypersensitivity**; **Environmental allergens/triggers** (eg dust, pollens, foods, drugs) 3. **Allergen exposure** produces IgE a. re-exposure triggers mast cell degranulation/cytokines b. bronchoconstriction c. mucus production d. vasodilation/incr vasc permeability	**3/4 to pass** **Bold and one trigger** Bold & concept

Stem: A 60 year old lady presents to ED with a painful arm following a fall.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: She has significant pain and is given morphine			
Question 1 Morphine **Subject:** Pharm LOA: 1	1. What is the mechanism of action of morphine?	**Act on receptors: mu**/delta/kappa **Reduce presynaptic neurotransmission (esp glutamate)** **Inhibit post-synaptic neurons** Central (thalamic action)	Mu + 1 other mechanism of action to pass
	2. Why do opiates cause respiratory depression?	**Inhibition of brainstem respiratory controls** allowing less response to hypercapnoea	Bold to pass
	3. How is morphine metabolised?	**Conjugated in liver** (morphine-3-glucuronide = most) Small amount (10%) morphine-6-glucuronide = increased analgesic potency Renal excretion	Bold to pass
Stem: Here is her xray.			
Question 2 **Clinical Building Block:** (# humerus)	Describe the abnormality	**Spiral/oblique fracture mid-shaft L humerus** with displacement.	
	What structure may be injured in this fracture?	**Radial nerve**	
Stem: Moving on to normal Anatomy.			
Question 3 Humerus X-ray **Subject:** Anat LOA: 1	1. Identify the features of the **humerus** on this x-ray	Prox: **Head**, Anat and **Surg neck, Shaft** **Gt tuberosity**/Lesser Tuberosity Distal: **Medial + Lateral** epicondyles, Trochlea, Capitulum, Lateral and medial supracondylar ridges	6 bold to pass
	2. What are the rotator cuff muscles and describe their actions	**Subscapularis** medial rotation of humerus **Supraspinatus** initiates abduction and abducts shoulder **Infraspinatus** and **teres minor** – lateral rotators of humerus All 4 muscles stabilise shoulder joint	4 muscles + 1 action to pass

Stem: Moving on to Pathology.			
Question 4 Fracture Healing **Subject:** Path LOA: 1	1. How do fractures heal? Prompt: What are the stages of fracture healing?	1. **Haematoma** 2. **Influx of Inflammatory cells, platelets, fibroblasts, new vessels and osteoprogenitor cells = procallus** 3. **Fibrocartilagenous callus**: mesenchymal cells + new cartilage along fracture line undergoes **endochondral ossification =** 4. **Bony Callus** 5. **Remodelling**	3/5 to pass + detail
	2. What factors inhibit fracture healing	Inadequate immobilisation/severe displacement/poor reduction/soft tissue Vascular compromise Infection, foreign body Systemic – nutrition, osteoporosis etc.	3 to pass
Stem: Moving on to Physiology.			
Question 5 Calcium metabolism **Subject:** Phys LOA: 1	1. How is plasma calcium regulated?	**1,25-dihydroxycholecalciferol (from Vit D) incr Ca absorption from gut & kidneys.** **PTH mobilises Ca from bone**, incr Ca reabs in kidneys, incr 1,25 DHCC formation in kidneys **Calcitonin** (from thyroid) **inhibits bone resorption**, incr Ca excretion in urine.	2/3 to pass
	2. How is the synthesis of 1,25-dihydroxycholecalciferol (vit D) regulated?	**1,25-DHCC formed in kidneys by 1α-hydroxylase.** **High Ca/high PO4 inh 1,25-DHCC** (incr inactive 24,25-DHCC instead). **Low Ca incr PTH which stimulates 1α-hydroxylase** (low PO4 directly stimulates 1α-hydroxylase).	Bold to pass

ACEM PRIMARY VIVA C **Friday Morning Session 3** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: A patient presents with a Verapamil overdose.			
Question 1 Verapamil **Subject:** Pharm LOA: 1	1. Describe the mechanism of action of verapamil	**Block** voltage-gated L-type **Ca channels** (α1 subunit), reduced frequency of opening when depolarised, resulting in **decreased** transmembrane Ca current, and **Ca influx**: Vascular smooth muscle relaxation (< Dihydropyridines) **Cardiac – decrease AVN conduction, contractility**, CO	Bold to pass
	2. What are the toxic effects of verapamil?	CVS: bradycardia, AV block, cardiac arrest, heart failure, hypotension Minor: flushing, dizziness, nausea, constipation, peripheral oedema	3 to pass 1 to pass
	3. What antidotes can be used to treat verapamil toxicity?	Calcium iv, high-dose insulin (euglycaemia) therapy	1/2 to pass
Stem: Moving onto Anatomy. Intravenous access is obtained.			
Question 2 Cubital fossa/ forearm photo **Subject:** Anat LOA: 1	1. Describe the boundaries of the cubital fossa	**Lateral: Brachioradialis** (5), (extensors from lat epicondyle) **Medial: Pronator teres** (20), (flexors of forearm from CFO) Floor: Brachialis, supinator **Superior: Line between 2 epicondyles of humerus** Roof: Skin, deep fascia reinforced by bicipital aponeurosis (3)	Bold to pass
	2. Please identify the major veins that can be seen in the photo	Basilic vn (1); cephalic vn (6); median cubital vn (13); median forearm vn (14)	2/4 to pass
	3. Identify the major arteries that can be seen in the photo (Bonus: which is the larger terminal branch?)	**Brachial a** (4), radial a (21), ulnar a (22) Ulnar a	Bold to pass
Stem:			

Question 3 **Clinical Building Block:** Photo myoglobinuria and biochemistry	Interpret her biochemistry results? Why is her urine dark?	**Renal failure** likely Acute kidney injury in clinical context - elevated urea + Cr **Markedly elevated CK - rhabdomyolysis** Normal K^+ Rhabdomyolysis Breakdown of skeletal muscle -> **myoglobinuria**	Essential in bold
Stem: Moving onto Pathology			
Question 4 Acute Kidney injury / rhabdomyolsysis **Subject:** Path LOA: 2	1. Define Acute Kidney Injury 2. What are the causes of AKI (please give examples)?	Clinico-path entity, **acute reduction of renal function** with morphologic **tubular injury (usually).** Reversible. 1. **Ischaemia**/abnormal blood flow. Systemic – assoc with thrombosis (HUS, TTP, DIC) or hypovolaemia. Intra-renal – angiopathies, malignant HT 2. **Toxic injury to glomeruli/tubules** – myoglobin, drugs, contrast 3. Acute tub.int nephritis – hypersensitivity reaction to drugs, IgA nephropathy 4. Obstruction ("post-renal") – tumour, clot, stones	Bold Bold and 1 other category 1 example for each
Stem: Moving onto Physiology.			
Question 5 Cardiovascular Regulatory Mechanisms **Subject:** Phys LOA: 1	1. What are baroreceptors? 2. Where are they located? 3. What is their mechanism of action in hypotension?	**Stretch** receptors in the adventitia layer of vessels Located at **aortic arch** and **carotid sinus**, walls of right and left atria (SVC and IVC entrances) and pulmonary circulation. In response to hypotension, the arterial **baroreceptors are less stimulated** because they are less stretched. Reduced baroreceptor discharge travels via glossopharyngeal and vagus nerves to the medulla resulting in an **overall increase in sympathetic discharge** to increase heart rate and stimulate vasoconstriction and **reduce vagal drive**.	Bold Bold and 1 other Bold to pass and understand inhibitory concept

ACEM PRIMARY VIVA D Thursday Morning Session 1 ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: A 70 year old man presents to ED as he has become jaundiced following his return from a trip to India			
Stem: Here are his blood results.			
Question 1 **Clinical Building Block:** (hepatic and renal failure)	Please interpret these biochemistry results Prompt: what is the pattern of the liver enzyme abnormality?	Bicarb 6 - Metabolic acidosis eGFR 31 mL/min Creatinine 151 µmol/L - Moderate-Severe **renal impairment** Bilirubin 32 µmol/L (reduced excretion) Albumin 22 g/L (reduced synthesis) - Mild hepatic impairment ALT 1778 U/L AST 5314 U/L ALP 272 U/L GGT 471 U/L **-Abnormal liver enzymes c/w hepatitis**	Must recognise renal failure and hepatitic LFTs to pass. **(bold to pass)**
Stem: Moving onto Pathology. You suspect Hepatitis A .			
Question 2 Hepatitis A **Subject:** Path LOA: 2	What is the causative agent of Hepatitis A? How is hepatitis A transmitted? How do the clinical outcomes of Hepatitis A differ from Hepatitis B? (Prompt- How are the long term outcomes different?) How is Hepatitis A diagnosed serologically?	**Hep A virus** – small unenveloped single stranded RNA picornavirus, icosahedral capsid **Faecal oral spread** Self-limiting illness no carrier state no chronic state no association with hepatocellular Ca rarely leads to fulminant disease low fatality rate of 0.1% **Acutely IgM-anti- HAV**, followed by appearance / persistence of IgG-anti HAV	Bold to pass Bold to pass 3/6 to pass Bold to pass

Stem: Moving onto Pharmacology. His regular medications include hydrochlorothiazide			
Question 3 Thiazide diuretics **Subject:** Pharm LOA: 2	Describe the mechanism of action of thiazides?	**Inhibition of Na/Cl transporter in the distal convoluted tubule** leading to increased NaCl excretion and **diuresis**	**Bold to pass**
	What are the major clinical indications for thiazide diuretic use?	**Hypertension** **Heart failure** Nephrolithiasis Nephrogenic Diabetes Insipidus Generalised oedema Nephrotic syndrome cirrhosis	**2 bold to pass**
	What are the potential adverse effects of thiazide diuretics?	**Hypokalaemia** **Dehydration/post hypotension/hypovolaemia** **Hyponatraemia** Metabolic alkalosis Hyperuricaemia Hyperlipidaemia Allergic Reactions – x- reactivity with sulphonamides Impaired carbohydrate tolerance – Hyperglycaemia Hypercalcaemia Pancreatitis	**2 bold plus 1 other**

Stem: Moving onto Anatomy. A CT abdomen is done to exclude renal obstruction as the cause of his renal failure			
Question 4 CT abdomen **Subject:** Anatomy LOA: 2	Identify the structures on this CT. (Axial image)	**Liver**, portal vessels, **R Kidney** (top), **aorta** **L kidney**, spleen, splenic vein (not tortuous), bowel loops, **pancreas**, IVC, Vertebra, ribs, paravertebral muscles, intercostal and abdominal wall muscles, fat, skin.	**5 Bold** + 2 others
	Describe the course of the ureters	Originate at renal hilum (PUJ) – approx. L2 Run inferiorly lying across psoas Near tips of transverse process of lumbar vertebra (L3 – L4) Cross over pelvic brim Cross anterior to bifurcation of common iliac artery Lie on lateral wall of pelvis Travel medially to bladder Short intramural path at VUJ	4/8 points to pass
	What are the 3 narrowest points of the ureters?	PUJ VUJ Pelvic brim	2 of 3

Stem: Moving onto Physiology.			
Question 5 GFR **Subject:** Phys LOA: 1	What is the definition of the glomerular filtration rate?	The amount of fluid (plasma filtrate) filtered by the glomerulus per unit time	**Concept of filtration and time to pass.**
	What is the normal GFR?	Usually 125mL/min (180L/day) 10% less in women.	**+/- 20 % to pass (either per min or per day)**
	What are mesangial cells? (Prompt – Where are mesangial cells found? What do mesangial cells do?) (Prompt if "in nephron" stated – where in nephron?)	**Contractile cells** that help to **regulate GFR**. Located between the basal lamina and the endothelium, **in the glomerulus** Common between neighbouring capillaries, and in these locations the basal membrane forms a sheath shared by both capillaries Also secrete the extracellular matrix, take up immune complexes, and are involved in the progression of glomerular disease.	**Bold to pass**
	What factors influence GFR?	Age Afferent arterial (renal artery) pressure (however autoregulation keeps this stable between about 90-210mmHg) Afferent arteriolar pressure Efferent arteriolar pressure Efferent venous pressure Intra-renal (interstitial) pressure (obstruction, oedema) Oncotic pressure Glomerular filtration fraction	**Any 3 to pass**
	What substances act on mesangial cells to change GFR? (Prompt - What substances act on mesangial cells to alter their function?)	Glomerular filtration fraction (mesangial cell function) – influenced by: Increased – ANP, dopamine, PGE2, cAMP Decreased – noradrenaline, vasopressin, AII, PGF2, endothelins, TXA2, Leukotrienes	**BONUS!**

Stem: A 30 year old man has sustained a fractured femur in a motor bike accident. Starting with anatomy.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Femur - Bone **Subject:** Anat LOA: 1	Describe the bony features of the middle and lower end of the femur.	Linea aspera Medial supracondylar ridge/line - inferior continuation of the medial lip of the linea aspera, interrupted to allow passage of the femoral artery, ends in the adductor tubercle Lateral supracondylar ridge/line- descends to the lateral epicondyle. Medial condyle Lateral condyle Intercondylar fossa Adductor tubercle Attachment of the medial ligament	**5/8 to pass**
	Which muscles attach to the linea aspera?	Vastus medialis, vastus lateralis, adductor brevis, adductor longus, adductor magnus, and short head of the biceps femoris	**3/6 to pass**
	Which artery is most likely to be damaged by a fracture of the midshaft of the femur?	**Profunda femoris**	**Bold**
	(Prompt : what is the course of the femoral artery through the thigh?)	(use this as supplemental question for better candidates)	

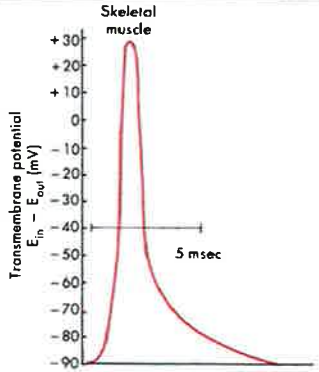
Stem: Moving onto Pathology. He has a head injury and a CT brain reveals gross cerebral oedema.			
Question 2 Cerebral Oedema and raised ICP **Subject:** Path LOA: 1	Describe the pathological mechanisms which cause cerebral oedema. (prompt if specific examples used – can you describe the difference between vasogenic and cytotoxic oedema?)	**Vasogenic.** BBB disruption, increased vascular permeability. Fluid shift intravascular to intercellular spaces of brain May be generalised or localised (inflammation or neoplasm) **Cytotoxic.** Increased intracellular fluid due to neuronal, glial, or endothelial injury eg generalised hypoxic/ ischaemic insult or metabolic damage Interstitial or ependymal oedema around (lateral) ventricles due to the high pressure of hydrocephalus	**Bold to pass or basic understanding of two mechanisms**
	What are the morphological findings of generalised cerebral oedema. (Prompt: What would be the CT findings?)	Flattened gyri, narrowing of sulci, compression of ventricles and/or basal cisterns, herniation	3 of 4 to pass
	Describe the major herniation locations associated with raised intracranial pressure	**Subfalcine** herniation- Asymmetric expansion of cerebrum displaces the cingulate gyrus under the falx cerebri **Transtentorial** or Uncal herniation -Medial aspect of the temporal lobe is compressed against the free margin of the tentorium **Tonsillar herniation**- Displacement of the cerebellar tonsils through the foramen magnum.	2 of 3 bold plus correct description

Stem: Moving onto Physiology. He is becoming progressively hypertensive and bradycardic.			
Question 3 Cerebral Circulation **Subject:** Phys LOA: 1	What are the factors that determine cerebral blood flow?	**Intracranial pressure** **Mean arterial pressure** **Mean venous pressure** at brain level Blood viscosity Local constriction/dilation of arterioles	**Bold** and 1 other to pass
	Describe the autoregulation of cerebral blood flow (Prompt: what happens to cerebral blood flow when blood pressure changes?)	**Maintains CBF at constant rate** (~750ml/min) **across a range of perfusion pressures** (MAP 65-140mmHg)	**Bold** to pass
	The patient's bradycardia and hypertension is caused by the head injury. Describe the mechanism responsible.	Cushing reflex – **increased ICP compromises blood flow to medulla** → **sympathetic outflow** from vasomotor centre → increases BP in attempt to restore medullary flow → **stretch of baroreceptors** → vagal stimulation→ bradycardia	**Bold** to pass Vagal stimulation OK instead of stretched baroreceptors

Stem: Moving onto Pharmacology. He is given Mannitol.			
Question 4 Mannitol **Subject:** Pharm LOA: 2	Why is mannitol used in the management of head injury?	Mannitol is used to **reduce intracranial pressure** after head injury.	**Bold** to pass
	What is the mechanism of action of mannitol?	Mannitol is an osmotic diuretic, it alters Starling forces as it does not cross the intact blood-brain barrier and thus **draws water out of cells** and **reduces intracellular volume** (hence reduces intracranial volume and intracranial pressure)	**Bold** to pass
	What are the other clinical effects?	Reduces intraocular pressure Diuresis / dehydration / hypovolaemia Hypernatraemia Hyperkalaemia	2/4 to pass
	Supplemental Question; What is an appropriate dose of mannitol in this clinical situation?	1-2g/kg as an IV bolus over 15 mins (**0.25-2g/kg IV bolus**).	

Stem: A 25 year old man has ruptured his Achilles tendon. Starting with Anatomy.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Model – lower leg **Subject:** Anat LOA: 1	Identify the muscles of the posterior compartment of the leg	Superficial posterior compartment: **24a,b Gastrocnemius m.** **24c Soleus m.** **24 Plantaris m.** Deep posterior compartment: **26. Popliteus m.** **27 Flexor digitorum longus m.** **28 Tibialis posterior m** **29 Flexor hallucis longus m**	24 a,b,c triceps surae muscle 51 popliteal a. 56 posterior tibial a **Must get 6/8 bold**
	Which muscles form the Achilles tendon	**gastrocnemius and soleus**, +/- plantaris	**Bold** to pass
	Where does the Achilles tendon insert?	supero-posterior aspect of the **calcaneus**	**Bold** to pass
	Can you identify the nerve supply of these muscles?	**tibial n.**	
	Can you identify the structures posterior to the medial malleolus?	Anterior to posterior: Tibialis posterior Flexor Digitorum Longus Posterior Tibial Artery Tibial Nerve Flexor Hall Longus	2/3 muscles and neurovasc bundle to pass
	Supp: What is the blood supply of these mm.?	gastrocnemius - sural a. (branch of popliteal a.); soleus - posterior tibial a. and peroneal a.)	

Stem: Moving onto Pathology.			
Question 2 Repair by healing, scar formation and fibrosis **Subject:** Path LOA: 2	What is the sequence of events for tissue healing by scar formation?	1) Blood Clot (stop bleeding, create scaffold) 2) Granulation tissue (angiogenesis, migration and proliferation of fibroblasts) 3) Cell Proliferation and Collagen Deposition (extracellular matrix (ECM) deposition) 4) Scar formation (blanching, increased collagen: type 3 then type 1) 5) Wound contraction (myofibroblasts) 6) Connective tissue remodelling (ECM synthesis and degradation) 7) Recovery of tensile strength	**5/7 to pass**
	How do skin wounds recover tensile strength?	**Increase in collagen synthesis** (type 1) **and reduction in collagen degradation** (first 2/12) then **structural modification** of collagen with cross linking & increased fibre size	**Bold to pass**
	What is the approximate time frame for recovery of tensile strength in skin wounds? (prompt : what is the strength of skin wounds when sutures are removed?)	Skin wound has **10% tensile strength at 1/52,** and continues to improve over next 3 weeks and **plateaus at ~3/12 when tensile strength is 70-80%.** May never recover to 100%	**Concept that very weak at time of suture removal and months to attain plateau phase**

Stem: Moving onto Physiology.			
Question 3 Skeletal Muscle action potential **Subject:** Phys LOA: 1	Draw a skeletal muscle action potential **(**Prompt if draw cardiac musc AP**)**		**Correct shape, axes, resting membrane potentials and durations (+/- 25%).**
	What is the sequence of events in the contraction of a skeletal muscle fibre, starting at the motor end-plate?	1.Discharge of motor neuron 2.Release of transmitter (acetylcholine) at motor endplate 3.Binding of ACh to Nicotinic Ach 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 depolarisation along T tubules 8.Releases of Ca^{2+} from terminal cisterns of sarcoplasmic reticulum and diffusion to thick and thin filaments 9.Binding of Ca^{2+} 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	**5/10** to pass
	What is the sequence of events in the relaxation of a skeletal muscle fibre?	1.**Ca^{2+} pumped back into sarcoplasmic reticulum** 2.Release of Ca^{2+} from troponin 3.**Cessation of interaction between actin and myosin**	**Bold to pass**

Stem: Moving onto Pharmacology. He is given Ondansetron for nausea.			
Question 4 Ondansetron **Subject:** Pharm LOA: 1	What is the mechanism of action of Ondansetron? Prompt- Where are these receptors found?	**5-HT$_3$ receptor antagonist**; Effect brought about at peripheral (Gut) > central receptors (chemoreceptor trigger zone and vomiting centre)	**Bold,** plus 1 receptor location
	What are the doses and routes of administration of Ondansetron ?	**4-8mg** SL , PO, IV , SC, IM	**Bold**, plus 3/5
	What are the adverse effects of Ondansetron?	Constipation, headache, dizziness, QT prolongation	**1/4** to pass
	In which disease state would you need to modify the dosing?	**Hepatic failure** Not with renal failure or age	**Bold**
	What are some other classes of antiemetic drugs? (ask for drug class if just name a drug)	Phenothiazines Antihistamines Cannabinoids Benzodiazepines Butyrophenones (Droperidol) Benzamides (eg Metoclopramide) Neurokinin receptor antagonists Corticosteroids	**3/8** to pass

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: A 50 year old man presents with dyspnoea to the Emergency Department. A Chest X-Ray is performed.			
Question 1 CXR – lung consolidation **Clinical Building Block**	What abnormalities are present?	**Multiple** (at least 3) **left sided** (patchy) **opacities** the largest of which appears to be pleurally based. Indistinct left heart border. Slight rightward tracheal deviation (?rotation). Relatively normal appearing right lung fields.	**Bold required**
	What is your differential diagnosis	Pneumonia, PE, Less likely in this scenario; contusion, pulmonary haemorrhage, heart failure, tumours	Infective plus one other
Stem: Moving onto Anatomy			
Question 2 Lobes of the lung Mediastinum and cardiac borders (CXR) **Subject**: Anat LOA: 1	a) What structures make up the mediastinal contours? (you can point to the CXR if you want) b) Which lobes of the lung lie adjacent to the right and left cardio-mediastinum? Prompt : Which part of the lung forms the right heart border? etc	**Right:** R Brachiocephalic v**, SVC,** R pulmonary trunk **, R atrium** **Left: Aorta,** Pulm trunk**, L atrium, L Ventricle** Right upper mediastinum - right superior lobe **Right heart border** **- right middle lobe** Left upper mediastinum - left superior lobe **Left heart border** **- left superior lobe (lingula segment)**	**Bold required** Plus one other **Bold required**

Stem: Moving onto Pathology. A pulmonary embolus is diagnosed			
Question 3 Pulmonary Embolus **Subject:** Path LOA: 1	What are the clinical features of PE?	(60-80% are silent) Usually present with respiratory compromise – SOB hypoxia, dyspnoea, tachypnoea shock , collapse, hypotension Right heart failure, pleural rub/pleuritic pain, fever, cough, haemoptysis Death	5 to pass
	Name some risk factors for Pulmonary embolism?	Primary – factor V Leiden, Antiphospholipid syndrome, Prothrombin mutations Secondary- obesity, OCP, cancer, immobilisation, long haul flights , preg , indwelling CVL, hip fractures,	1 primary and 3 secondary factors
	What factors determine the severity of the pathophysiological response to pulmonary embolism? Prompt: features of the emboli…	1. **Extent of Pulmonary artery blood flow obstructed** 2. Size of the vessel occluded 3. Number of emboli 4. Overall CVS status 5. Release of vasoactive factors ie. (thromboxane A2)	Bold to pass Accept 2 of the others also as a pass
Stem: Moving onto Physiology. The patient is hypoxic			
Question 4 Ventilation-perfusion relationships **Subject:** Phys LOA: 1	a) What are the physiological causes of hypoxaemia?	Hypoventilation, diffusion, shunt, ventilation-perfusion inequality.	Need 3 to pass
	b) How does ventilation-perfusion inequality result in hypoxaemia?	Lung units with low V/Q ratio have effluent blood with low pO2 (close to mixed venous). Units with high V/Q ratio have relatively high PO2 but **because of non-linear O2 dissociation curve add little to O2 concentration** (compared to the decrement caused by the low V/Q areas). Overall mixed return has lower O2.	Bold + demonstrate understanding of concepts
	c) How can ventilation-perfusion inequality be measured? (Prompt: is there a formula used to quantify V/Q inequality?)	Using the **alveolar-arterial PO2 difference (the A-a gradient)** – subtracting the (measured) arterial PO2 from the "ideal" alveolar PO2 as given by the alveolar gas equation.	Bold + understanding of concepts

	Extra Q: Why does the PCO2 remain relatively normal in the setting of ventilation-perfusion inequality?	PAO2 = PiO2 – PaCO2/0.8 Normal is 5-10mmHg (increases with age) The CO2 dissociation curve is linear in the working range. An increase in PCO2 stimulates chemoreceptors → increase ventilation	
Stem: Moving onto Pharmacology. The patient is commenced on oral anticoagulants.			
Question 5 Rivaroxaban – mechanism of action Pharmacokinetics **Subject**: Pharm LOA: 1	a) Describe the mechanism of action of rivaroxaban. b) Describe the pharmacokinetics of rivaroxaban. c) What clinical advantages does rivaroxaban offer over warfarin? Extra Q: Do the pharmacokinetics of rivaroxaban present any clinical disadvantages relative to warfarin?	Inhibits both free and prothrombinase-bound forms of activated factor X. Oral bioavailability >80% Maximal plasma levels 3 hours post-ingestion Small volumes of distribution (<50L) Highly protein bound Elimination renal (predominant) and hepatic (CYP3A4) with steady state half-life 5-14 hours and prolonged with renal impairment. More rapid onset/offset of action More predictable effect = easier dosing, wider therapeutic index INR monitoring not required Fewer drug and dietary interactions Predominant renal excretion means dose must be adjusted in renal failure and not suitable for dialysis patients.	Inhibits factor Xa 2 things including predominant renal excretion to pass 2 to pass. Better candidates will be able to correlate differing MOA and pharmacokinetics to advantages Supplementary question, only use if sufficient time.

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: A 78 year old lady is found collapsed at home on the floor. She is found to be dehydrated. Starting with Physiology.....			
Question 1 Response to fluid bolus **Subject:** Phys LOA: 1	What are the physiological effects of dehydration?	Water loss lowers ECF and ICF leading to **↓BP, ↑HR, ↑ADH, ↓UO**, ↓GFR, ↑Renin/Angiotensin, ↑Thirst aiming to maintain IV volume. In adrenal insufficiency Na is lost not only in urine but also into cells.	Na movement is key. Bold elements.
	Describe the effects of a rapid IV infusion of 1000 ml of Normal Saline. Prompt: what is the cardiovascular effect you would see?	↑Cl and acidosis, ↑Baroreceptor firing, **↓HR**, **↑BP,** increased UO, ↓Renin/Angiotensin and improved capillary return. (Bainbridge reflex described initial increase HR if slow initially)	Bold plus one more.
	What is an alternative physiological fluid replacement?	**Hartmann's** (lactated Ringer's) or **Plasmalyte**.	1 of these.
Stem: Moving onto Pharmacology, she is given Atropine			
Question 2 Atropine including Pharmacokinetics **Subject:** Pharm LOA: 1	What is the mechanism of action of atropine?	A competitive, reversible **muscarinic ACh receptor antagonist** Binds to muscarinic receptors, preventing the release of IP3 (inositol triphosphate), DAG (diacyglycerol) and the inhibition of adenylyl cyclase caused by muscarinic agonists. Anticholinergic agent (equipotent at M_1,M_2,M_3Rc)	Bold to pass
	Describe the organ effects of atropine	Eye – mydriasis & cycloplegia CNS – delirium, decrease tremor in Parkinson's disease CVS - tachycardia Resp – bronchodilation & decrease secretions GIT – decrease saliva secretion, decrease gastric acid secretion, decrease mucin production, decrease gastric emptying, decreased gut motility and intestinal transit time increases GUT – relaxes ureteric and bladder wall smooth muscle, urine retention Skin – decreased sweating	Need 3 organ systems with an example to pass
	What is atropine used for clinically?	**Rx symptomatic bradyarrhythmias / bradycardia** Ophthalmology – as a mydriatic & cyloplegic Occas. in paediatric RSI using suxamethonium (not routine any more) esp 2nd dose Drying of secretions eg in cholinergic nerve agent / OP poisoning or in palliative care Traveller's diarrhoea	Bold plus 1 to pass
	Extra question: Describe the	Route of admin: IV, oral, nebulized, topical	

	pharmacokinetics of atropine?	Absorption: well absorbed orally Distribution: **wide Vd** (including CNS) Half life = 2 hrs Metabolism & Excretion: 40% phase I and phase II metabolism and renally excreted 60% excreted renally unchanged	
Stem: Moving onto Anatomy. She complains of a painful wrist			
Question 3 Carpal bones, stability and movements **Subject:** Anatomy LOA: 1	Name and identify the carpal bones on this model What movements occur at the wrist joint (demonstrate them) and which muscles produce them?	Scaphoid, Lunate, triquetrum, pisiform, hamate, capitate, trapezoid, trapezium. **Flexion** – **FCR, FCU** + flexors of fingers & thumb, Palmaris longus & AbPL **Extension** – **ECRL or ECRB, ECU** and extensors of fingers & thumb **Abduction** – FCR, ECRL or ECRB, APL **Adduction** – ECU and FCU (Circumduction)	6 to pass. Bold to pass
Stem: Blood tests were sent on arrival. Here are her coagulation and platelet results.			
Question 4 Coags - DIC **Clinical Building Block** (30 sec)	What is the most likely diagnosis? Prompt: What are the abnormalities in this set of results? What other coagulation test is likely to be abnormal?	**Disseminated Intravascular Coagulation** **D-Dimer** is markedly raised.	All required.
Stem: Moving onto Pathology. You suspect she has disseminated intravascular coagulation.			
Question 5 DIC **Subject:** Path LOA: 1 (2 min)	List some common triggers DIC? How does endothelial injury initiate DIC?? Extra question: Draw the extrinsic pathway of the coagulation cascade.	**Sepsis** (bacterial endotoxins and AgAb complexes), **major trauma/burns/surgery**, **certain cancers** (AML (promyelocitic), adenoca of lung, colon, stomach, pancreas), **obstetric complications** (placenta, amniotic fluid, dead fetal tissue) • Exposure of sub endothelial matrix activates plts and the coag cascade • TNF causes tissue factor to be expressed from endothelial cells • TNF up-regulates the expression of adhesion molecules on endothelial cells to allow leucocytes to bind and damage endothelial cells. • Direct trauma to endothelial cells from AgAb complexes, temperature extremes, or microorganisms.	3 of 4 categories required. Will accept examples. 3 points required for pass

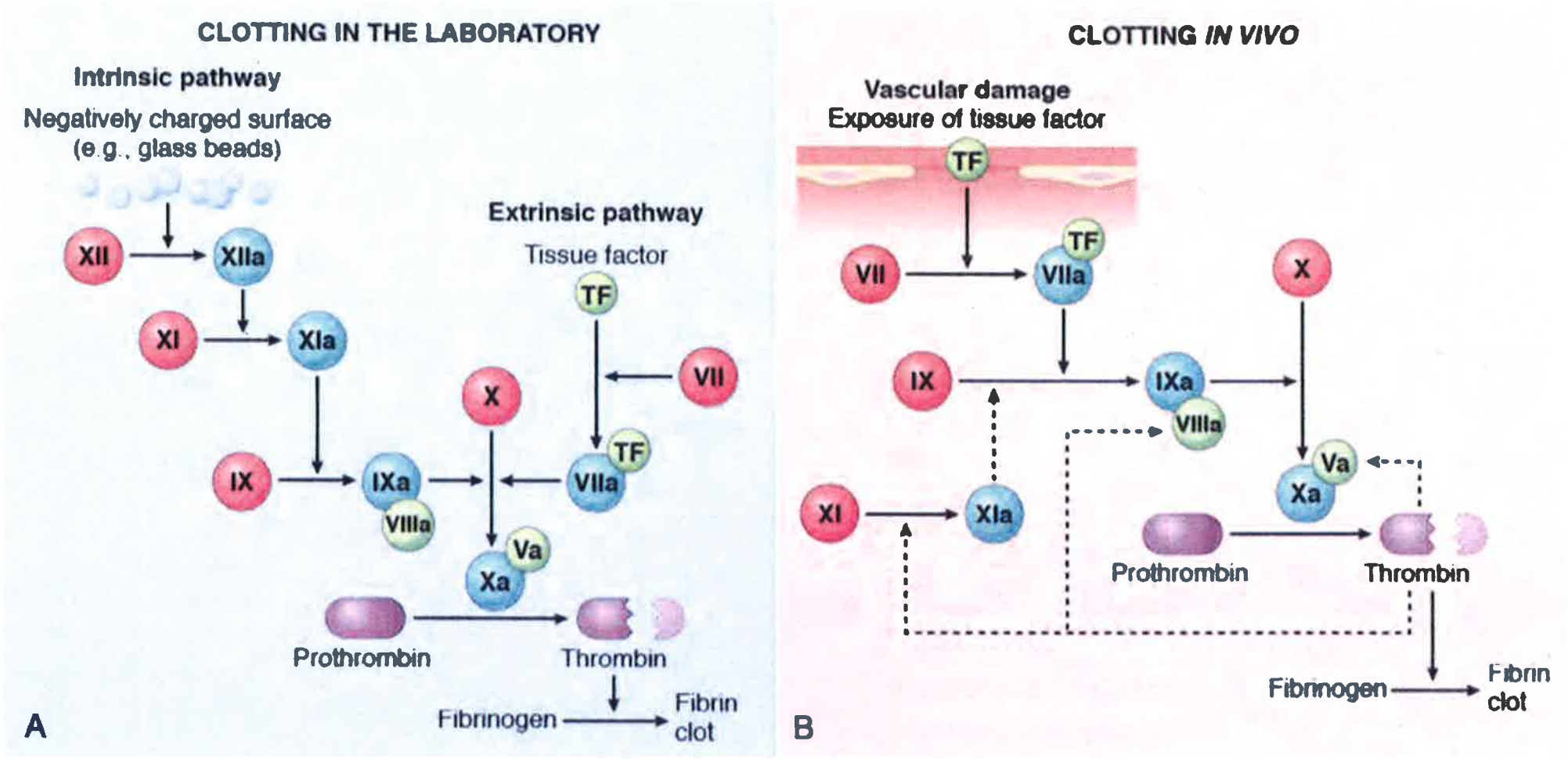


Figure 4-6 The coagulation cascade in the laboratory and in vivo. **A,** Clotting is initiated in the laboratory by adding phospholipids, calcium, and either a negative charged substance such as glass beads (intrinsic pathway) or a source of tissue factor (extrinsic pathway). **B,** In vivo, tissue factor is the major initiator of coagulation, which is amplified by feedback loops involving thrombin *(dotted lines)*. The red polypeptides are inactive factors, the dark green polypeptides are active factors, while the light green polypeptides correspond to cofactors..

ACEM PRIMARY VIVA A **Friday Morning Session 3** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: A 60 year old alcoholic man is brought to the Emergency Department after being found slumped in a chair. He is noted to have a wrist drop.			
Question 1 Brachial Plexus (photo) **Subject:** Anat LOA: 1	Here is a photo of the right brachial plexus. 1) Please identify the nerve most likely injured. 2) Please identify some of the other numbered nerves in the plexus. Prompt: please show us the median and ulnar nerves. 3) Apart from wrist extensor weakness, what other functions may be lost with a radial nerve lesion in the axilla?	**Radial nerve (21)** Axiliary nerve (1), Lateral cord (6), lat root median n (8), medial cord (12), medial cut n of arm (13), medial cut n of forearm (14), Medial root of median n (16), **Median n (17),** Posterior cord (20), **Ulnar n (26).** Finger extension and sensory loss over radial side of dorsum of hand. **Need at least one of these to pass**	**Bold to pass** **Requires median and ulnar n to pass. 4 in total incl these 2** **Need to name and identify to pass.** Can prompt by asking what nerve innervates the extensor compartment of the forearm.
Stem: Moving onto Pathology. He is found to be in heart failure, and on bedside echocardiogram is found to have a grossly dilated heart with poor contractility in all chambers.			
Question 2 Cardiomyopathy – focussing on alcoholic **Subject:** Path LOA: 2	1) What pathological process is likely to be causing his heart failure? 2) Name some causes of dilated cardiomyopathy 3) What are potential pathologic consequences of dilated cardiomyopathy.	Alcohol related **Dilated Cardiomyopathy** Causes include **myocarditis** (viral causes?), **toxins** (alcohol, chemo, cobalt), **congenital**, **pregnancy**. **Valve dysfunction** (incompetent mitral/tricuspid), **mural thrombi** and **embolization**, lethal **arrhythmia**, **atrial fibrillation**, **death** from progressive failure	Bold to pass Any 2 bolded to pass Any 2 bolded to pass
Stem: He is also noted to be jaundiced. Here are his liver function tests.			
Question 3 LFTs **Clinical Building Block**	Please comment on these results.	Expect comments on **raised AST/ALT**, with AST nearly 5 fold increase, slight rise in **ALP**, and **marked raised in GGT**...all consistent with **Alcohol induced hepatitis**	Expect recognition of marked rise in transaminases, with little rise in ALP suggesting **hepatitic picture.** Raised GGT suggests alcohol being cause.

Stem: Moving onto Physiology.			
Question 4 Bilirubin metabolism / jaundice **Subject:** Phys LOA: 1	1) Describe the metabolism of bilirubin	**Formed from breakdown of Hb** **Bound to albumin** Free bilirubin enters liver cells via OATP family (organic anion transporting polypeptide) binds to cytoplasmic proteins **Conjugated** by glucuronyl-transferase in ER with glucuronic acid to H2O soluble bilirubin diglucuronide Bilirubin diglucuronide actively transported against conc gradient by MDRP-2 to bile canaliculi; small amount escapes into blood, bound to albumin, excreted in urine Intestinal mucosa relatively impermeable to conj bilirubin, gut bacteria convert most to urobilinogens **Enterohepatic circulation:** Some reabsorbed in portal circulation and resecreted. Small amt urobilinogens excreted in urine and faeces (uro and stercobilinogens)	Bold plus one more cause
	2) What are the causes of jaundice?	**Excess production of bilirubin (eg haemolytic anaemia)** Decreased uptake of bilirubin into hepatic cells Disturbed intracellular protein binding or conjugation Disturbed secretion of conjugated bilirubin into the bile canniliculi **Intra or extrahepatic bile duct obstruction** (1st three liberate free bilirubin; last 2 result in elevated conjugated bilirubin in blood)	Bold to pass
Stem: Moving onto Pharmacology.			
Question 5 Ethanol **Subject:** Pharm LOA: 1	1) What are the clinical features of acute ethanol consumption?	**CNS**-sedation, disinhibited, impaired judgement, impaired motor skills, ataxia, slurred speech->coma, resp depression Heart-depressed myocardial contractility Smooth muscle-vasodilator ->hypothermia in OD, + uterine SM relaxation	Must mention CNS + one other
	2) Describe the pharmacokinetics of ethanol	Absorption-**rapid from GI tract** (water soluble), peak levels within 30 minutes Distribution –rapid Vol Distribution ~TBW (0.5-0.7 L/kg) **Predominantly liver metabolism -zero order kinetics** (over 90% oxidised liver-to acetaldehyde). Mainly via **Alcohol dehydrogenase (ADH),** less by microsomal ethanol oxidising system (MEOS) Excreted –lungs, urine	Bold to pass-must mention ADH
	3) Name other drugs that have zero order kinetic metabolism	Phenytoin, theophylline, warfarin, salicylates, heparin, paracetamol	One to pass

ACEM PRIMARY VIVA B **Thursday Morning Session 1** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: A 55 year-old man presents to the ED with haematemesis. Hepatitis B serology results from a previous admission are available.			
Question 1: Hepatitis B Serology	*What is the most likely diagnosis, and why? Prompt: "Is this acute or chronic?"*	**HBsAg positive – indicates current infection.** Anti-HBc total positive – exposure to HBV. IgM anti-HBc negative – exposure not acute or recent. **Anti-HBs negative – no current immunity to HBV. Diagnosis: Chronic Hepatitis B.**	Bold to pass.
Stem: Moving onto Pathology.			
Question 2 Hepatitis B LOA: 1	*a. How may Hepatitis B lead to upper gastrointestinal bleeding?*	Cirrhosis and **portal hypertension** with development of **oesophageal varices.** **Coagulopathy** due to loss of synthetic function (unable to produce coag proteins)	2 out of 3 bold to pass
	b. What are the other complications of Hepatitis B-induced cirrhosis?	Jaundice; Hepatorenal syndrome; Hepatic encephalopathy; Ascites/pleural effusions Splenomegaly; Hypogonadism (testicular atrophy, amenorrhoea etc); Hepatocellular carcinoma	3 to pass
	c. In general, how may a patient acquire Hepatitis B?	Congenital (ie vertical; most common worldwide) Contaminated blood products – IVDU, transfusions (from many years ago), needlestick injury. Bodily fluids – eg sexual.	2 to pass
	d. What are the other possible outcomes of hepatitis B exposure?. Prompt: 'Apart from progressive chronic hepatitis'	Asymptomatic Acute hepatitis Non progressive chronic hepatitis Carrier state	2 to pass
Stem: Moving onto Physiology. Despite blood loss, he maintains adequate perfusion.			
Question 3 General principles of autoregulation (local, circulating, myogenic, neurological). LOA: 1	*a. What is autoregulation of tissue blood flow? Prompt: "What are the main features of autoregulation?"*	Capacity of tissues to regulate their own blood flow, which **remains relatively constant** despite moderate **changes in perfusion pressure.** This is achieved by **altering vascular resistance.**	Bold concepts to pass
	b. What are the proposed mechanisms involved in autoregulation?	**Myogenic:** Intrinsic contractile response of smooth muscle to stretch. As pressure rises, vascular smooth muscles surrounding the vessels contract to maintain wall tension (La Place Law, T = P x r). **Metabolic:** Production of vasodilator metabolites by active tissues → vessel vasodilation → ↑flow **Endothelial products** : vasoconstrictors (endothelin, thromboxane A2) and vasodilators (nitric oxide, prostacyclin). **Circulating neurohumoral substances**: vasoconstrictors (adrenaline, noradrenaline, vasopressin, angiotensin II) and vasodilators (kinins, VIP, ANP). **Neural :** Sympathetic (α-adrenergic receptors- vasoconstriction, β-adrenergic receptors – vasodilation) & parasympathetic (muscarinic receptors – vasodilation).	3 bold to pass with explanation
	*c. What are some **local** factors that lead to vasodilation?*	Hypoxia, hypercarbia, increased local temperature, hyperkalaemia, adenosine, acidosis, lactate, prostaglandins, histamine.	4 to pass
Stem: Moving onto Pharmacology. He is treated with octreotide.			
Question 4 Octreotide – mechanism of action, pharmacokineti	*a. Explain the mechanism of action of octreotide.*	A somatostatin analogue that inhibits the release of GH, TSH, glucagon, **insulin and gastrin.** **[Reduces splanchnic blood flow / portal pressure].**	2 bold
	b. Describe the pharmacokinetics of octreotide.	Plasma elimination half-life is 80 minutes. Metabolised by liver (30-40%) & 20% excreted unchanged by kidney.	Know $T_{1/2}$ (range 40-120 min)

cs. LOA: 2	*c. What are some of its clinical uses?* *Bonus: What are its adverse effects?"*	Acute control of **bleeding from oesophageal varices,** sulphonylurea overdose, reduce symptoms caused by hormone secreting tumours eg: acromegaly, carcinoid, gastrinoma, locating endocrine tumours using radiolabelled octreotide. Side effects include nausea, vomiting, abdo cramps, flatulence, steatorrhoea.	Bold plus 1 1
Stem: Moving onto Anatomy. He also complains of a sore wrist after a fall.			
Question 5 Bones- radius and ulna LOA: 1	*a. Identify this bone and describe its anatomical features.*	Identify **radius**. Candidates can mention the side and why. **Head-** articulates with the capitellum of the humerus. **Neck** **Radial tuberosity**- demarcates the head/neck from the shaft, attachment of biceps brachii. **Shaft**- triangular in cross section, anterior and posterior oblique lines. Distal end- **styloid process, ulnar notch and dorsal tubercle of the radius.**	Radius, and 5/7
	b. Which carpal bones articulate with its distal end?	The distal end articulates with the **lunate and scaphoid.**	Both to pass
	c. Describe the normal relative relationship between the radial and ulnar styloid processes. Prompt: "Which is normally more distal?"	Radial styloid projects further (ie more distal) than the ulna styloid.	
	d. Demonstrate the movement of the radius during supination.	The axis of supination passes through the centre of the head of the radius proximally and the ulnar styloid distally. The head of the radius rotates within the collar formed by the annular ligament and the ulnar radial notch. Supination rotates the radial head laterally around its axis (pronation rotates it medially).	Pages 804-5, 819 678-9 7th edition.

ACEM PRIMARY VIVA B **Thursday Afternoon Session 2** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: A 75 year-old man injured his lower limb after a fall. Starting with Pharmacology: He was given fentanyl by the paramedics.			
Question 1 Fentanyl LOA: 1	*a. Describe the mechanism of action of fentanyl.*	Synthetic opioid that acts on the **μ receptor.**	Bold
	b. Describe the pharmacokinetics of fentanyl.	**High first pass metabolism, duration of action 1-2 h**, metabolised by P450 CYP 3A4 with no active metabolites. Transdermal, mucosal and IM absorption are good. Fentanyl may be given IV, IM, IN, SC, SL/buccal (with lozenge), transdermal patch, epidural.	Bold plus 2 routes.
	c. Describe its potency relative to morphine	100 times more potent. 0.1mg fentanyl = 10mg morphine	Range 100 to 200.
	d. List the adverse effects of fentanyl.	Respiratory depression, nausea, vomiting, dysphoria, cough, sedation, constipation, urinary retention, itch, urticaria, chest wall & laryngeal rigidity.	Name 4
Stem: An x-ray of his injured leg is performed.			
Question 2 Tibial X-ray (#)	*a. Describe the abnormalities.*	**Transverse fractures of left tibial and fibular shafts (diaphyses), at junction of distal and middle thirds.** Medial displacement and approximately 3cm shortening/overlap of fractured ends. Also 90 degrees external rotation of distal fragments.	Bold and one other.
	b. What are potential complications of this injury within the first week?	Haemorrhage, compartment syndrome, **neurovascular compromise**, infection, pain, fat embolism syndrome.	Bold plus 2 others.
Stem: Moving onto Anatomy. His pain seems out of proportion to the injury. A compartment syndrome is suspected.			
Question 3 Lower Limb compartments (model) LOA: 1	*a. Identify the muscles of the posterior compartment of the leg, using this model.*	**Superficial** – gastrocnemius (medial and lateral heads), soleus, plantaris. **Deep**- Popliteus, FHL, FDL, Tibialis posterior.	2 superficial + 2 deep.
	b. What is the nerve supply of these muscles?	Tibial branch of sciatic nerve. Aka **tibial nerve**, or posterior tibial nerve.	Must name nerve.
	c. Using the model, describe the course of this nerve in the leg.	**Formed at apex of popliteal fossa by bifurcation of sciatic nerve** (other branch is common peroneal, or common fibular nerve). Runs vertically in popliteal fossa with popliteal artery, passing between heads of gastroc and deep to tendinous arch of soleus. Runs inferiorly on tib posterior with post tib vessels. **Divides into medial and lateral planter nerves under flexor retinaculum.**	Bold to pass.
Stem: Moving onto Pathology. His past history includes severe aortic stenosis.			
Question 4 Calcific aortic stenosis LOA: 2	*a. What are the pathological consequences of aortic stenosis?* *Prompts – 'What type of ventricular hypertrophy?'* *'Which ventricle is hypertrophied?'*	**Concentric left ventricular hypertrophy.** Left ventricular outflow obstruction. Myocardial ischaemia (without coronary artery disease needing to be present) Syncope; Aortic dissection; Heart failure (diastolic or systolic); Endocarditis (uncommon)	Bold and 3 others
	b. What are the likely causes of aortic stenosis in this man?	Calcific/degenerative Bicuspid valve Rheumatic heart disease	2 to pass (do not accept congenital)

	BONUS: What clinical signs may differentiate calcific aortic stenosis from rheumatic aortic stenosis?	Rheumatic disease involves more than one valve (ie aortic and mitral) Absence of features of MS/MR Absence of features of aortic regurgitation	
Stem: Moving onto Physiology. His blood pressure is low.			
Question 5 Renin- angiotensin system. LOA: 1	*a. List some conditions which activate the renin-angiotensin system.*	Activated in response to decrease in BP / ECF volume or increased sympathetic activity. Examples: Hypotension, haemorrhage, dehydration, cardiac failure, cirrhosis, Na^+ depletion / diuretics, upright posture. Pain, fear and arousal (fight, fright, flight) may trigger it too.	4 conditions
	b. What are the principal effects of angiotension II? *Prompt: "Where does angiotensin II act?"*	**Arterioles (AT_1 receptor)** : **vasoconstriction** → ↑TPR. **Adrenal cortex (AT_1 receptor)** : **↑aldosterone** production → **↑Na^+ / H_2O reabsorption** Kidney : direct effect to ↓GFR & ↑Na^+ reabsorption. Brain : ↓sensitivity of brain baroreceptor reflex → potentiates pressor effect Pituitary : ↑ADH & ↑ACTH secretion.	Bold to pass

ACEM PRIMARY VIVA B **Friday Morning Session 3** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: A 65 year-old man, with a long history of smoking, presents with acute dyspnoea.			
Question 1 CXR	*What do you see on the patient's chest x-ray?*	Collapse of right lung with approximately 50% loss of volume. **Right-sided pneumothorax.** >2cm between lung and chest wall at hilum, making it **moderate to large**, per BTS guidelines.	Bold.
Stem: Moving onto Pathology.			
Question 2 Chronic Obstructive Pulmonary Disease LOA: 1	*a. What is emphysema?*	Chronic lung condition characterised by **irreversible** enlargement of the airspaces **distal to the terminal bronchiole,** accompanied by destruction of alveolar walls **without fibrosis.**	2 out of 3 bold
	b. Describe the pathogenesis of emphysema.	- Loss of cellular homeostasis - caused by **exposure to toxic substances** such as tobacco smoke and inhaled pollutants which induces **ongoing inflammation, epithelial cell death and extracellular matrix proteolysis.** - Accumulation of neutrophils, macrophages and lymphocytes results in release of **elastases, cytokines** (including IL-8) and **oxidants** that cause epithelial injury and proteolysis of the extracellular matrix. - **Elastin degradation** products further increase the inflammation. - End result is destruction of the alveolar walls without fibrosis.	2 bold
	c. How do the clinical features of emphysema differ from those with chronic bronchitis?	- "Pink Puffer" = emphysema. Barrel chested, dyspnoeic, prolonged expiration, hyperventilation. Relatively normal gas exchange until late in disease. - "Blue Bloater" = chronic bronchitis. Hx of recurrent chest infections with purulent sputum, less dyspnoea, decreased respiratory drive. Patient is hypoxic and cyanotic. Peripheral oedema results from *cor pulmonale* and RV failure.	2 distinguishing clinical features.
Stem: Moving onto Physiology.			
Question 3 Pulmonary Compliance LOA: 1	*a. What is lung compliance?*	**Change in lung volume per unit change in airway pressure (ΔV/ΔP) – measure of lung "distensibility"** Normally 200mls/cm H_2O. It occurs because of the opposing inward elastic recoil of the lungs and outward recoil of the chest wall. It is represented by the slope of the nonlinear lung pressure-volume curve.	Concept to pass
	b. What physiologic factors affect lung compliance?	Age, volume of the lung, phase of respiration (lower in deflation/expiration than inflation/inspiration), surfactant.	3 to pass
	c. How is lung compliance affected in emphysema?	**Compliance is increased** because of **loss of lung elasticity** / destruction of lung connective tissue & elastin (easy to inflate but reduced capacity to recoil). Patients have to force their expiration to expel air from lungs. Resultant increase in FRC.	Bold to pass
	d. What are the physiologic effects of pulmonary surfactant?	Lowers alveolar surface tension, increases lung compliance, reduces work of breathing, improves the stability of alveoli and keeps the alveoli "dry".	3 to pass
Stem: Moving onto Pharmacology. You use bupivacaine as the local anaesthetic prior to intercostal catheter insertion.			
Question 4 Bupivacaine LOA: 1	*a. Describe the mechanism of action of bupivacaine.*	Amide local anaesthetic, **blocks voltage-gated Na channels.**	Bold
	b. Describe the pharmacokinetics of bupivacaine.	**Metabolised by the liver**, Distribution half-life 28 min, elimination half-life 3.5h, large V_D of 72 L, 95% protein bound, lipophilic. **Duration of action 4 to 8 hours** (longer than lignocaine or ropivicaine).	Bold
	c. Give examples of its clinical use.	Use as a nerve block in low conc 0.25% for local infiltration, digital ring block, femoral, intercostal, intrapleural, epidural (post-op), brachial plexus, sciatic nerve, intra-articular.	Name 2 (do not accept IVRA)
	d. List some of its toxic effects.	Sedation, visual and auditory disturbance, **cardiac arrhythmia**, hypotension & arrest, **seizure.**	Bold

Stem: Moving onto Anatomy.			
Question 5 Chest X-ray with focus on lung and pleura LOA: 1	*a. Describe the expected positions of the fissures of the lung on a normal CXR.*	Right lung has 3 lobes, left 2 lobes. Oblique fissures separate upper from lower lobes, horizontal fissure separates the right upper and middle lobes. **Oblique fissures** follow the 5th ribs (run from 4th thoracic vertebrae to 3 cm posterior of the junction between the diaphragm and the sternum on the left, and to the sternodiaphragmatic junction on the right). **Horizontal fissure on right** at level of 4th costal cartilage or hilum.	Bold (within 1 space either way)
	b. Describe the position of the neurovascular structures in the intercostal space.	Between the middle and innermost layers, protected by the **costal groove of the superior rib of each intercostal space. Ordered vein, artery, nerve from superior to inferior.**	Bold
	c. When placing an intercostal catheter (ICC) in the 5th intercostal space laterally, what anatomical structures are traversed?	Skin -> subcutaneous tissues -> **external intercostal** muscle -> **internal intercostal** muscles -> **innermost intercostals** -> parietal pleura.	2 bold
	d. What structures may be at risk from an ICC inserted laterally?	**Neurovascular bundle; long thoracic nerve** (lies in serratus anterior behind the mid axillary line); **lung, diaphragm, pericardium/heart** and spleen if on the left; **liver** if on right.	2 bold

ACEM PRIMARY VIVA C Thursday Morning Session 1 *Candidate Number:* AGREED MARK:

Stem: A 35 year old woman presents to ED with a neck mass. Her GP has done thyroid function tests			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: TFTs **Clinical Building Block**	1. Please interpret these results.	**Raised FT4, FT3 and suppressed TSH consistent with hyperthyroidism**	Bold
Moving onto Physiology			
Question 2 Regulation of thyroid hormones **Subject** Phys LOA: 2	1. How are thyroid hormones regulated? (Prompt Describe the feedback mechanism) (Prompt What factors affect TSH secretion?) 2. Other than cardiovascular, what are the physiological effects of thyroid hormones?	TRH from hypothalamus=> TSH from ant pituitary=> T4 (& small amount T3)=> T3 in periphery. **Negative feedback on TSH by free T3 & T4** (In hypothalamus and pituitary. Effect of **T3>T4**). Both secretion & synthesis of TSH affected ↑ by cold, ↓by warmth (esp in infants. Effect in adults not clear) ↓by stress (TRH) and glucocorticoids (TSH). ↓ by dopamine & somatostatin (TSH) **Calorigenic** (↑ metabolic rate, ↑ stimulation O2 consumption) Adipose tissue: catabolic (stimulated lipolysis); Muscle: catabolic (↑protein breakdown); Bone: developmental (promote normal growth(Cretin) and skeletal development); Nervous system: promote normal brain development & mentation Gut: metabolic (↑ carbohydrate absorption); Cholesterol: formation of LDL receptors and removal of circulating cholesterol	Bold & concept One factor Bold plus one other system

Stem: Moving onto Pharmacology. She is treated with propranolol.			
Question 3 Propranolol (Beta Blockers) **Subject:** Pharm LOA: 1	1. Describe the pharmacodynamics of propranolol that make it useful in thyrotoxicosis. (Prompt: What are the cardiovascular effects?)	**B-Blocker: Competitive non selective B Blocker, blocking both B1 and B2 receptors** CVS: ↓BP, ↓HR (esp rate control of AF), both negatively inotropic and chronotropic. ↓catecholamine effects which are prominent in hyperthyroid. Inhibition of peripheral conversion of thyroxine (T4) to triiodothyronine (T3) (esp in propranolol cw other B-blockers) Has Na-channel blocking action ("membrane stabilisation")	Bold and 2 CVS effect
	2. What are the adverse effects of propranolol?	**CVS**: Bradycardia, Hypotension, worsening CCF, worsening ischaemia in PVD, QRS widening & arrhythmias in toxicity **CNS**: sedation, depression, dreams, In toxicity-coma/seizure/delerium **Resp**: worsening asthma/COPD, Other: decreased exercise tolerance, fatigue, impotence, ↓libido, mask symptoms of hypoglycaemia	1 example from each bold system

Stem: Moving onto Anatomy.			
Question 4 Neck Model (sagittal section) **Subject**: Anat LOA: 1	1. Identify the boundaries of the anterior triangle of the neck on this model. 2. a) Describe the surface markings of the carotid sheath in the neck b) What are its contents? 3. Describe the position and features of the Thyroid gland	**Anterior border of SCM, midline of neck and inferior border mandible** a) Runs along a **line joining the SCJ and a point midway between the mastoid process and angle of mandible** b) **Common Carotid a., IJV and Vagus** **Anterior in neck at level C5-T1**, deep to Sternohyoid and sternothyoid m. **R and L lobes sit anterolateral to the Larynx and Trachea**. The **isthmus joins these at level approx 2-3 tracheal rings**	All 3 bold for pass a) all b) all 2/3
Stem: Moving onto Pathology. On examination her thyroid gland is enlarged.			
Question 5 Cellular injury – adaptation and hyperplasia **Subject:** Path LOA: 1	1. Define hyperplasia 2. What are the different types of hyperplasia and give some examples for each of them 3. Name some clinical manifestations of diffuse toxic hyperplasia of the thyroid (Prompt: Graves' disease)	Hyperplasia is **an increase in the number of cells** in an organ or tissue resulting in increased mass. **Physiologic** hyperplasia a. hormonal-female breast at puberty and during pregnancy b. compensatory-post partial hepatectomy **Pathologic** hyperplasia a. excess hormones-Benign prostatic hypertrophy, dysfunctional uterine bleeding b. response to viral infection – papillomavirus Cardiac-tachycardia, palpitations, heart failure Eye-staring, lid lag, proptosis GI-malabsorption, diarrhoea Neuro-tremor, anxiety, poor concentration Other	Bold to pass Bold and one example of each to pass 4 to pass

ACEM PRIMARY VIVA C **Thursday Afternoon Session 2** ***Candidate Number:*** **AGREED MARK:**

Stem: A 70-year-old man has presented with left sided limb weakness. Here is his CT brain.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 CT – middle cerebral artery stroke **Clinical Building Block**	1. What is the major abnormality on his CT?	Non contrast Head CT shows area of hypodensity in right MCA region/territory	**Right** thromboembolic **MCA stroke**
Moving onto Anatomy			
Question 2 Cerebral circulation (CT) **Subject:** Anat LOA: 1	2. What is the arterial supply of the cerebral cortex? Name the corresponding parts they supply (Prompt- demonstrate on the CT)	**ACA** area anterior to anterior horns lat ventricle (frontal and parietal lobes medially and superiorly) **MCA** area between the ant & post horns LV (most of lateral surface, parietal, and temporal lobes) **PCA** area posterior to posterior horn LV (Inferior and medial aspects of occipital and temporal lobes)	**Anterior, Middle and posterior cerebral artery with reasonable distribution**
	3. Describe the venous drainage of the cerebral hemispheres?	Superior cerebral veins (superolateral surface of the brain) > **superior sagittal sinus.** Inferior and superficial middle cerebral veins (inferior, posterior and deep aspects of cerebral hemispheres) > **straight, transverse and superior petrosal sinuses.** **Great cerebral vein** (midline vein formed from the paired internal cerebral veins) >merges with inferior sagittal sinus to form the straight sinus. Eventually terminate in **Internal Jugular veins**	**General concept**

Stem: Moving onto Pathology			
Question 3 Ischaemic Stroke / Reperfusion injury **Subject:** Path LOA: 1	1. What are the main pathological processes causing ischaemic stroke? *(Give examples for each category)*	**Thrombotic occlusion – atherosclerosis** most common **Embolism** – AMI, mural thrombus, Valvular heart disease, AF, vascular surgery and shower embolism, fat embolism, endocarditis Inflammatory process leading to luminal narrowing - Infectious vasculitis, autoimmune vasculitis, primary angiitis of the CNS	**Bold to pass** and at least 2 causes of embolism plus one other (embolic or inflammatory).
	2. What are the distinguishing pathological features of haemorrhagic and non-haemorrhagic ischaemic cerebral infarcts? Prompt: why does haemorrhagic change occur in ischaemic stroke.	Haemorrhagic (**red**) - multiple, sometimes confluent, petechial haemorrhages typically associated with **embolic** events. Thought to be secondary to **reperfusion** either via collaterals or dissolution of materials. Greater risk if anticoagulated. Non-Haemorrhagic (**pale/bland anaemic**) – usually associated with **thrombosis.**	Bold causes and concepts
	3. How are these pathological processes important in relation to stroke thrombolysis?	Complications higher with embolic/haemorrhagic CVAs. Trying to reverse injury in ischaemic penumbra. In non- haemorrhagic CVA little macroscopic change can be seen within the first 6 hours. Earlier treatment leads to better outcome and less haemorrhagic risk.	Reversible ischaemic penumbra (term or concept) Red Vs pale –Red CI

Stem: Moving onto Physiology. The patient is febrile.			
Question 4 Temperature regulation **Subject:** Phys LOA: 1	1. What is the pathogenesis of fever (Prompt: What causes a febrile response?) (Prompt: Which area of the brain is activated in a febrile response?)	Bacterial **toxins** eg endotoxin act on monocytes, macrophages, and Kupffer cells to produce cytokines that act as **endogenous pyrogens** IL-1β, IL-6, IFN-β, IFN-γ, and TNF-α can act independently to produce fever. Cytokines also produced by cells in CNS when these are stimulated by infection -may act directly on the thermoregulatory centres. Activates the preoptic area of the **hypothalamus**. Causes release of **prostaglandins** eg PGE2. This causes a raise in **temp set point** resulting in fever	Concept to pass
	2. What is the body's response to hot and cold environments? (Prompt: What happens to heat production & loss?)	Mechanisms activated by **cold:** (post hypothalamus) **Inc Heat Production:** Shivering, Hunger, Voluntary activity, NA/A release **Dec Heat Loss,** Skin vasoconstriction, Curling up, Horripilation. Mechanisms activated by **heat**: (ant hypothalamus) **Dec Heat Production** Anorexia, Apathy & Inertia. **Inc Heat Loss,** Cutaneous vasodilation, Sweating, Respiration.	1 mechanism for each of heat production & loss for cold 1 mechanism for each of heat production & loss for hot environment

Stem: Moving onto Pharmacology. He is given paracetamol for his fever.			
Question 5 Paracetamol **Subject:** Pharm LOA: 1	1. Describe the pharmacokinetics of paracetamol.	Rapid absorption. Bioavailability 70-90%. Peak concentration after 30-60 mins. Slight ppb. Partial metabolism by hepatic MEs to paracetamol glucuronide and sulphate (90%). First order kinetics. T1/2 2-3 hours. <5% excreted unchanged.	4 points to pass (out of total 8)
	2. What is its mechanism of action?	Selective **COX-2 inhibitor**.	bold to pass
	3. What is the mechanism of paracetamol toxicity?	Zero order kinetics. paracetamol is conjugated with glucuronide and sulfate (by transferase enzymes) - this **pathway becomes saturated** in OD, allowing increasing paracetamol to be metabolized by the smaller CYP 2E1 pathway to NAPQI. NAPQI is detoxified by **glutathione** which becomes depleted resulting in high levels of **toxic metabolite (NAPQI).**	bold & concept to pass
	4. *[If time only]* What are the clinical manifestations of toxicity?	Nausea, vomiting, abdominal pain, liver failure, renal failure (tubular necrosis), HAGMA, massive doses - coma.	4 to pass

ACEM PRIMARY VIVA C **Friday Morning Session 3** ***Candidate Number:*** **AGREED MARK:**

Stem: An 80 year old lady with lung cancer presents with increasing dyspnoea and lethargy. Please review her biochemistry results			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 **Clinical Building Block:** Hyopnatraemia & Osmolarity	1. What are the abnormalities? 2. What are some causes in this patient?	Hyponatraemia, hypo-osmolar plasma SIADH; CCF; Water intoxication; drugs/other	 Two causes
Stem: Moving onto Physiology			
Question 2 Tonicity, vasopressin **Subject:** Phys LOA: 1	1. Describe the process by which extracellular fluid tonicity is regulated. *(Prompt lead in: where is plasma osmotic pressure sensed?)*	As plasma osmotic pressure rises → **thirst ↑** + sensed via **osmoreceptors** in anterior hypothalamus (mainly organum vasculosum of the lamina terminalis OVLT) → **vasopressin (ADH) secretion rises** (from posterior pituitary) → renal **V_2 receptor stimulation** → insertion of water channels (**aquaporins)** in luminal membranes of renal collecting tubules, allowing more water to return to body. Conversely as plasma osmotic pressure falls (285mosm/kg is the critical point) ADH secretion suppressed.	Thirst increases water intake. ADH reduces water excretion by kidneys -> dilution of ECF Bold + correct understanding of concept to pass
	2. What factors *other* than rising osmotic pressure increase vasopressin secretion?	**Decreased ECF vol**, **pain**, emotion, surgical stress, exercise, nausea & vomiting, standing, angiotensin II, meds (clofibrate & carbamazepine).	Bold + 1 more to pass

Stem: Moving onto Pharmacology. Her chest X-ray shows consolidation and she is commenced on Azithromycin			
Question 3 Azithromycin (Macrolides) **Subject:** Pharm LOA: 2	1. What class of antibiotic is Azithromycin	**Macrolide**	**Bold to pass**
	2. Describe the mechanism of action of azithromycin?	**Inhibition of bacterial protein synthesis** by binding 50S ribosomal RNA, blocking aminoacyl translocation and formation of initiation complexes (transpeptidation), may be inhibitory or bactericidal (esp at higher concentrations)	**Bold to pass**
	3. Against which micro-organisms is azithromycin effective?	Gm+ pneumococci,strep,staph,corynebact Mycoplasma legionella chlamydia sp, listeria Some mycobacteria Gm- Neis sp, Bordatella pert, Trep pall. Campylobacter sp, Bartonella	**3 to pass** but must include one atypical
	4. How does Azithromycin differ from other macrolides? *(Prompt eg Compared to erythromycin & clarithromycin)*	Higher tissue penetration (tissue conc >>>>serum conc) **Long elimination T1/2** (2-4 days) vs 2-5 hr **Single daily dosing** More effective against haemophillus m.catarr. neiss Highly effective against chlamydia sp Less active against staph & strep Excreted unchanged in urine Absorption impeded by food Doesn't inhibit hepatic cytochrome P450 so drug interactions are uncommon	**Bold** plus one other

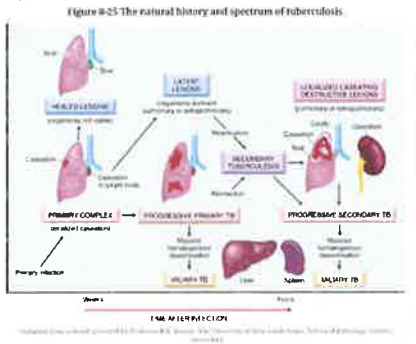
Stem: Moving onto Anatomy. On further questioning, she has been having increasing hip pain			
Question 4 Pelvis - bone **Subject:** Anat LOA: 1	1. Identify the bony landmarks of the pelvis	**3 bones – ilium, ischium, pubis** Surface – **iliac crest, ASIS, AIIS, ischial tuberosity**, ischial spine, PSIS, PIIS, **symphysis pubis** Joints – **SI joints, acetabulum/femur, symphysis pubis** Other – **pubic rami (4)**, ala of ilium, greater/lesser sciatic notches, obturator foramen	Bold
	2. Identify on the model the ligaments of the hip joint and their attachments.	**Iliofemoral (AIIS/acetabular rim – intertrochanteric line), pubofemoral (obturator crest – capsule & iliofemoral lig.),** ischiofemoral (acetabular rim – base greater trochanter)	Bold
	3. Where might you find a pathological fracture on the model?	Pubic rami, neck of femur, proximal shaft	one
Stem: Moving onto Pathology			
Question 5 Lung Neoplasia and principles of neoplasia **Subject:** Path LOA: 2	1. What are the main categories of primary lung cancer?	**Adenocarcinoma** (more common in F) **Squamous cell carcinoma** (more common in M) **Small cell carcinoma** (v. malignant) Large cell carcinoma (undifferentiated)	Bold
	2. What are the pathways by which a malignant tumour may spread?	**Local invasion** **Direct seeding** of cavities/surfaces **Lymphatics** **Haematogenous** Surgical instruments/nerves	3 of 4 Bold
	3. What paraneoplastic syndromes can be associated with lung carcinomas?	**SIADH** (HypoNa as per CBB, small cell Ca) ACTH (Cushing's) PTH, PTH-related peptide, PGE (HyperCa) Calcitonin (HypoCa) Gonadotropins (gynaecomastia) Serotonin/bradykinin (carcinoid syndrome)	SIADH and 1 other

ACEM PRIMARY VIVA D **Thursday Morning Session 1** ***Candidate Number:*** **AGREED MARK:**

Stem: A 65 year old man presents with ischaemic pain in his leg. He is on amitriptyline. Starting with Pharmacology.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Amitriptyline– mechanism of action, side effects **Subject:** Pharm LOA: 1	Describe the pharmacodynamics of amitriptyline What are the toxic effects of amitriptyline and how are they mediated?	Blocks reuptake of **serotonin and noradrenaline**, and blocks muscarinic, sympathetic α_1, $GABA_A$, Na+ channel and histamine receptors. Monoamine vs neurotrophic vs neuroendocrine theories. • Blurred vision, dry mouth, tachycardia, retention, delirium (anticholinergic) • Sedation (antihistamine) • Hypotension (anti-alpha effects), • Wide QRS and bradycardia (Na channel blockade) • Seizures (direct central effect)	**Bold** and 2 other receptors Must name effects and cause for at least 3 groups
Stem: Moving onto Anatomy			
Question 2 Lower leg – popliteal fossa – vascular supply (Photo) **Subject:** Anat LOA: 1	Identify the superficial boundaries and the contents of the popliteal fossa How does the popliteal artery supply the leg and foot? PROMPTS: What are the main branches of the popliteal artery? Prompt: What pulses can you palpate below the knee?	Superiorly: biceps femoris (1), semitendinosus (12) and semimembranosus (11). Inferiorly : lat (3) and med (4) heads of gastrocnemius Contents: Popliteal Art (8) & V (10), Small saphenous V (13) ; Tibial n. (15), common fibular n. (2) & sural n. (14); Divides into **Post and Ant tibial arteries** at lower border of popliteus. Post Tib runs in **post compartment then palpable post to med malleolus** and divides into med and lat plantar arteries to sole of foot. Main branch is fibular art to post and lat compartments **Ant Tib runs ant compartment then crosses anteriorly over ankle to become dorsalis pedis**	To pass – biceps, one of the semis, both heads of gastrocnemius; Common fibular & tibial nerve, popliteal artery and vein **Bold to pass**

Stem: Moving onto Pathology. He is found to have absent right pedal pulses.			
Question 3 Ischaemic injury **Subject:** Path LOA: 1	Describe the sequence of events that occur in **reversible ischaemic** cellular injury PROMPT: What occurs within the cell after delivery of oxygen and substrate is compromised?	↓ox phos & **↓ATP prodⁿ →** **Failure Na pump**: K efflux, Na influx, Cell swelling →**Ca influx:** Further ↓ATP prodⁿ; enzymes activated→ ↓glycogen & ↓protein synthesis → **Cytoskeleton Δs**: loss of microvilli, "bleb" formation, "myelin figures" from degenerating cell membranes → Mitochondrial swelling	3 of 4 **bold**
	List the morphological changes of **irreversible** cellular injury	1.Severe mitochondrial swelling 2.Extensive damage to plasma membrane (including "myelin figures") 3.Lysosomal swelling 4.Necrosis or apoptosis	Any 2
	Describe reperfusion injury (time permitting)	**Increase injury to ischaemic cells with restoration of perfusion**. Due to generation of **reactive O_2** and nitrogen species (NO), Ca++ re-entering cell, **activation inflamm and complement cascades**	At least one concept
Stem: CBB A venous blood gas is done.			
Question 4 Blood gas with metabolic acidosis **Clinical Building Block**	Describe the abnormalities on this venous gas PROMPT: What type of acidosis is it?	**Low pH, low HCO_3^-, metabolic acidosis (**AG 22) with **respiratory compensation**	Bold
Stem: Moving onto Physiology			
Question 5 Renal buffers in acid-base regulation **Subject:** Phys LOA: 1	How will the kidneys respond to a metabolic acidosis?	Aims to **return serum pH to normal by increasing H+ excretion.** **Kidney reabsorbs HCO_3^- by actively secreting H+.** Renal tubule cells contain carbonic anhydrase converting CO_2 to H+ and HCO_3^-, then PCT cells secrete H+ in exchange for Na+ In the DCT, H+ is secreted by a proton pump, limited by urinary pH >4.5 (limiting pH).	Must know that H+ actively secreted into tubular fluid in exchange for Na. Must know about buffering and name 2 buffers.
	PROMPT: Describe the role of buffers in the kidney	**Buffering in tubular fluid** pH with H_2CO_3, HPO_4 and NH_3 **allows greater H+ secretion.**	

ACEM PRIMARY VIVA D **Thursday Afternoon Session 2** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: A 50 year old woman from Papua New Guinea presents with a chronic cough and fever. We will start with a CBB			
Question 1 CXR – RUL collapse /consolidation **Clinical Building Block**	Describe and interpret this CXR	**Opacification involving lower half of right upper lobe**, consistent with **consolidation.** Well defined fissure – slightly elevated. Relevant negatives: no collapse (no shift of trachea / mediastinum / diaphragm), no effusion, no pneumothorax, no #, no invasive lines/tubes, no monitoring, normal heart size	Bold to pass
	What are the possible diagnoses?	**DDx: Pneumonia** (bacterial**,** viral, fungal, TB) Less likely atypical infection, abscess, aspiration, PE, malignancy	Must have bold and 1 other
Stem: Moving onto Pathology. TB is suspected			
Question 2 TB **Subject:** Path LOA: 1	1. Outline the natural history and spectrum of TB. PROMPT: What can happen after primary infection?	1. Primary infection 2. **Primary complex** (localised caseation) (Ghon complex is primary TB with mediastinal nodes) 3. Primary complex may heal (orgs not viable) or lead to latent lesion (orgs viable) 4. **Latent period** OR progressive primary TB (latter of which may lead to miliary TB) 5. Latent lesion reactivated leading to **secondary TB**. (Reinfection may also lead to secondary TB) 6. Secondary TB occurs as localised (pulm or extra-pulm) caseating destructive lesions OR progressive secondary TB 7. Progressive secondary TB may lead to **miliary TB**	4/4 in bold 
	2. How is TB diagnosed?	1. **Clinical features** in at risk patients (Hx and Ex) and apical lung consolidation/cavitation on **CXR** **2. Microbiological confirmation** a. Acid fast smears and cultures (3-6 wks solid agar media, 2 weeks liquid media) b. PCR 3. Other eg Mantoux test (TST)	Must have bold

Stem: Moving onto Physiology. The patient is short of breath			
Question 3 Physiological vs anatomical dead space **Subject:** Physiology LOA: 1	What is "dead space"? What types of DEAD SPACE are there?" PROMPT: Explain difference between the two types How is it measured? (bonus)	**Portion of the tidal volume that does not participate in gas exchange.** VT = VD + VA **ANATOMICAL** • **Volume of conducting airways** (without alveoli) – trachea, bronchi, terminal bronchi. • About 150mls of 500ml VT • Determined by: Increased diameter of airways during inspiration and Size & posture of individual **PHYSIOLOGICAL** • **Volume of gas that does not eliminate CO2/not equilibrating with blood** • Same as anatomical DS in normal individuals • **Increased in lung disease** because of inequality of blood flow and ventilation within the lung Anatomical – Fowlers method, Physiological - Bohr method	Demonstrate principle **in bold to pass** Two types of dead space and describe what it is. May mention VQ mismatch Will accept either
Stem: Moving onto Pharmacology. Cultures grow an organism which is sensitive to ciprofloxacin			
Question 4 Ciprofloxacin- route of administration, dose and mechanism of action **Subject:** Pharm LOA: 2	Describe the pharmacokinetics of ciprofloxacin PROMPT: Which patients will need a dose adjustment? Describe its mechanism of action What is its antimicrobial spectrum?	**PO or IV**. PO bioavailability > 80% 20-40% protein bound **Elimination half-life 3-5 hours** PO 250 - 500mg bd (max 1.5gm) IV 200 – 300 mg bd (max 1.2 gm) **Renal elimination** – dose adjustment if Cr Cr < 50ml/min **Blocks DNA synthesis** by inhibiting bacterial topoisomerase II (DNA gyrase) and IV- prevents normal transcription and replication **Excellent Gram neg activity, moderate Gram pos activity**. S Aureus, Mycoplasma, Chlamydia, Legionella, Pseudomonas, Mycobacterium and Anthrax.	Bold to pass

Stem: Moving onto Anatomy. For ongoing treatment a central venous line is needed.			
Question 5 Thoracic inlet (photo) **Subject:** Anatomy LOA: 1	1. Identify the venous structures in the image.	**Left internal jugular vein (8), Right and Left subclavian veins (24), Left brachiocephalic vein (13), SVC (26), Right brachiocephalic vein (18),** Inferior thyroid veins (7)	Must get bold
	2. Identify the major arterial structures in the image.	**Brachiocephalic trunk (4), Right common carotid artery (19), Left common carotid artery (14), Right subclavian artery (21),** Internal thoracic artery (9)	
	3. What other structures can you identify	Cricoid, thyroid gland, trachea, lung, pleura, phrenic nerve, scalenus anterior, thyrocervical trunk	Must get 3
	4. What are some complications of an internal jugular line insertion?	Pneumothorax Misplacement/arterial puncture Haemorrhage Infection Injury to thoracic duct on left	Must get 3

ACEM PRIMARY VIVA D **Friday Morning Session 3** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: A 30 year old woman is septic and requires intubation. You use suxamethonium in your rapid sequence intubation			
Question 1 Suxamethonium including pharmacokinetics **Subject:** Pharm LOA: 1	1. What is the mechanism of action of suxamethonium? Prompt. Where does it act?	**Depolarizing neuromuscular blocker** Phase I (depolarising)- Reacts with nicotinic receptor, opens channel, depolarizes NM endplate with spread to adjacent membranes, causing fasciculation prior to paralysis. Phase II (desensitising) – Continued or repeated exposure to sux – end plate depolarisation decreases – membrane repolarises but cannot be depolarised as is desensitised	Must get bold with understanding concept
	2. What are the pharmacokinetic properties of suxamethonium?	**Rapid onset (30-60 seconds)** **Short duration of action (2-8 minutes)** **Hydrolysed rapidly** by plasma cholinesterase	2 of 3
	3. What are the adverse effects of suxamethonium?	Muscle pain from fasciculation Bradycardia Potassium release – burns, CHI, trauma, stroke Raised Intraocular Pressure Raised Intragastric pressure Malignant hyperthermia Prolonged paralysis (reduced or abnormal cholinesterase)	4 of 7
Stem: Moving onto Anatomy			
Question 2 Airway - model **Subject:** Anat LOA: 1	1. On the model, identify the structures of the larynx	Cartilages: **thyroid (20), cricoid, epiglottis (54), (**arytenoids, corniculate, cuneiform) Ligaments: **cricothyroid membrane (23),** thyrohyoid(21,22), **vocal cords (60)** Muscles: cricothyroid muscle (18), cricoarytenoid Spaces and folds: **vallecula,** aryepiglottic folds (57), piriform recess	5 of 6 bold plus 2 others
	2. What is the innervation of the larynx?	**Cranial Nerve X (Vagus)** Inferior laryngeal N – (terminal branch of **Recurrent laryngeal nerve**): All intrinsic muscles of larynx except for Cricothyroid M, sensory below cords. External Laryngeal N supplies Cricothyroid M Internal Laryngeal N sensory above cords (Int and Ext Laryngeal Ns both branches of Superior Laryngeal Nerve	Bold with details of one
Stem: This is a CBB question. A lumbar puncture is performed. This is the CSF result			
Question 3 **CBB:** CSF	Interpret this result Prompt. What is the likely diagnosis?	Turbid with **low gluc, high protein and high WCC – mostly PMNL. Likely bacterial meningitis**	Bold to pass

Stem: Moving onto Pathology			
Question 4 Neisseria meningitidis **Subject:** Path LOA: 1	1. How does Neisseria meningitidis cause infection	1. Common coloniser of oropharynx - 10 % of popn at any one time, carry it for months 2. Spread by resp droplets 3. Most people develop immune response and clear it – protected against later infection from this serotype (>/13 serotypes). 4. Invasive dx when encounter new serotype 5. Invades resp epithelium, then blood stream 6. Capsule allows evade immune response by prevention opsonisation and complement destruction 7. Mortality still approx. 10% despite AB Rx.	Need 4/7 to pass
	2. What are the clinical consequences of N. meningitidis infection	Death, gen sepsis, necrotising vasculitis, seizures, SIADH, CVA, hydrocephalus, meningitis, sensorineural hearing loss, cognitive impairment	Need 4 to pass
	3. Apart from Neisseria, what else can cause meningitis?	Other bacteria: E coli & Group B Strep (infants), Strep pneumonia, Listeria, Haemophilus, Listeria Viral: Enterovirus, measles Other: TB, Rickettsial, Carcinoma, Auto-immune, chemical	Must have two specific bacteria plus viral as a group
Stem: Moving onto Physiology.			
Question 5 Venous pressure and flow **Subject:** Phys LOA: 1	Describe the mechanism of venous return to the heart Prompt. In a healthy person	1. **Thoracic pump** : insp generates neg (-) intra-throracic P and pos (+) intra-abdo P. 2. Venous valves : one way flow 3. Heart beat : AV valves pulled downwards in systole – inc size atria – blood sucked into atria 4. Muscle/arterial pump : contraction musc/arteries adj to veins compresses veins 5. Differential resistance – less resistance in more prox (larger) veins	**Thoracic pump** plus one other
	What factors might affect CVP in this patient?	1. **Decrease CVP** - fluid loss (third spacing), loss arterial tone, loss muscle pump (ventilated), myocardial depression (acidosis), poor ventricular filling (tachycardia) 2. **Increase CVP** – positive P ventilation (but will decrease venous return), fluid replacement, vasopressor use	1 example from each bolded category
	What is the mean CVP in a healthy adult?	6-8 cm H2O or 4.6 to 5.8 mmHg	Reasonable value

ACEM PRIMARY VIVA A **Thursday Morning Session 1** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: A 30-year-old woman is brought to the ED with severe dyspnoea. An intraosseous needle has been inserted by the paramedics. We will start with Anatomy			
Question 1 Bones - Tibia **Subject:** Anatomy LOA: 1	Identify this bone and its main features. Please identify the attachments of the proximal end of this bone. Prompt "What attaches to the superior surface of the bone"	L or R **tibia.** **Medial and lateral condyles.** Medial and lateral tibial plateaus, separated by intercondylar tubercles of the intercondylar eminence. **Tibial tuberosity, shaft**, medial border, anterior border, interosseous border, soleal line (post), **medial malleolus,** fibula notch. Ant and post horns of the medial meniscus. Ant and post horns of the lateral meniscus. **Ant + post cruciate ligaments. Patellar ligament.** Semimembranosus & Vastus medialis (med). Iliotibial tract, ED longus + peroneus longus (lat).	Bold to pass Bold to pass
Stem: A set of arterial blood gases are obtained			
Question 2: Blood gas with acute respiratory acidosis **Clinical Building block**	Please describe the abnormalities and interpret these results. *Prompt* "What is the main acid-base disturbance?"	**Low pH** **High CO_2** High base excess Severe **respiratory acidosis** without expected metabolic compensation. Low pO_2 for FiO_2 (likely A-a gradient high)	Bold to pass
Stem: Moving onto Pathology. You suspect that she is developing Acute Respiratory Distress Syndrome.			
Question 3 ARDS **Subject:** Pathology LOA: 2	Describe the pathogenesis of ARDS What conditions are associated with the development of ARDS?	Type of acute lung injury. Initial **injury to alveolar capillary membrane** (epithelium) or vascular endothelium-> **acute inflammatory response** (neutrophil mediated), resulting in **increased vascular permeability** & alveolar flooding; fibrin deposition; formation of hyaline membranes; & widespread **surfactant abnormalities** (with damage to Type II pneumonocytes). Eventually organisation with scarring. **Infection** (sepsis, diffuse pulmonary infection) **Physical / Injury** (trauma – head, pulmonary, fractures, near drowning, burns, radiation). **Inhaled irritants** (O2 toxicity, smoke, irritant gases and chemicals). **Chemical injury** (Opiates, barbiturates, paraquat, acetylsalicylic acid, , gastric aspiration). **Haematological conditions** (multiple transfusions, DIC).	3 of 4 bold Need 1 example from 3 categories + must include infection

		Other (pancreatitis, uraemia, cardiopulmonary bypass, hypersensitivity – organic solvents, drugs)	
Stem: Moving onto Physiology.			
Question 4 CO_2 transport **Subject:** Physiology LOA: 1	Q1) How is CO_2 transported in the blood? *Prompt:Are there any other mechanisms?* Q2) What is the most important mechanism? *Prompt:What proportion does each mechanism contribute.* Q3) What is the role of Red Blood Cells in CO_2 transport?	**Diffusion, Carbamino-proteins and CO_2 to Bicarbonate buffering** Diffusion 5 (a)-10 (v)% Carbamino 5 (a)-30%(v) Bicarbonate 60 (v)-90 (a)% a) Carbonic anhydrase only found significantly in Red cells, major buffer for CO_2 and H+ b) Haldane Effect -Hb (partic de-oxy) is also major H+ buffer allowing ->/faster H+/HCO3dissociation -Chloride shift (allowing 70% HCO_3- diffusion into plasma maintaining ionic neutrality/ enhanced diffusion) is mediated by Band 3 Cl- transporter in RBC membrane c) - Hb protein is the major carbamino protein (better when deoxyHb as more negative charge)	2/3 bold required Bic is the most important 1/3 for pass
Stem: Moving onto Pharmacology. She has been treated with Salbutamol for her dyspnoea.			
Question 5 Salbutamol **Subject:** Pharmacology LOA: 1	1) What class of drug is salbutamol 2) What are the different routes of administration of salbutamol *Prompt:Any other route?* 3) What are the advantages of the different methods of administration of inhaled salbutamol 4) What are the advantages and disadvantages of IV salbutamol in asthma	A **selective beta 2 agonist** that is used as a bronchodilator **Inhaled- nebulized, puffer +/- spacer** Oral, **IV,** IM, SC Nebulized: Rapidly absorbed, no co-ordination required, not much education required, no first pass metabolism Puffer +/- spacer: **As effective as nebulized** when used properly, targeted, lower dose, **less side effects** Pros- no first pass metabolism, maybe useful in severe/life threatening asthma Cons- Requires IV (disadvantage in children), more systemic side effects	Bold to pass Bold to pass Bold to pass 2/4 to pass

ACEM PRIMARY VIVA A **Thursday Afternoon Session 2** ***Candidate Number:*** **AGREED MARK**

Stem: An 80-year-old woman presents with pain radiating down her arm following a fall. We will start with anatomy.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Brachial plexus (photo, McMinn's) **Subject:** Anatomy LOA1	Here is a photo of the right brachial plexus. 1) Please identify its main features. PROMPT: Please list, and identify where you can, the branches of each cord. 2) What does the musculocutaneous N supply?	Deltoid (4) Biceps (2) Cords - Lateral (6), posterior (20) & medial (12) **Musculocutanous nerve (18)**, Axillary nerve (1), **Radial N (21) Median N (17) Ulnar N (26)** Lateral cord-> musculocutaneous N, lat pectoral N + **lateral root of median nerve (8).** Post C -> Upper (27) +lower (11) subscapular Ns, the thoracodorsal N(25), axillary N(1) + radial N(21) Medial C->**the medial root of the median N (16),** the ulnar N, the medial cutaneous nerve of the arm (13) & forearm (14), the medial pectoral nerve. Motor supply-> 3 muscles of ant compartment of the arm (biceps, brachialis & coracobrachialis) Sensory supply -> skin over the lat aspect of the forearm (becomes the lat cutaneous N of the forearm after giving off its motor supply)	Bold to pass To pass – list at least 2 branches from each cord & identify those in bold To pass – 1 motor & sensory distribution
Stem: Clinical Building Block. Laboratory investigations as part of her workup of her fall have been performed.			
Question 2 FBC and Fe studies **Subject:** CBB	Please describe & interpret these blood test results.	**Severe anaemia** **Microcytic** Low MCHC Low serum Ferritin **Low serum iron** High TIBC Low TF **Interpretation: Fe deficient anemia**	Bold for pass
Stem: Moving onto Pathology.			
Question 3 Iron deficiency anaemia **Subject:** Pathology LOA: 1	1) What are the causes of Fe deficiency anaemia 2) What are the symptoms of Fe deficiency anaemia 3) Are there any specific features of Fe deficient anaemia?	**Chronic blood loss – GI tract**, Menorrhagia. Increased requirements – pregnancy, children. Dietary Lack –developing world, infants (prolonged breastfeeding) elderly, extreme diet Impaired absorption –celiac, gastrectomy General –fatigue, weakness, dyspnoea, angina Features of cause – melaena, menorrhagia Koilonychia, alopecia, glossitis,pica, pharyngeal web	Bold + 3 other examples from any categories 4 for pass 1 for pass

Stem: Moving onto Physiology.			
Question 4 Iron metabolism and haemoglobin **Subject:** Physiology LOA: 1	How is Iron absorbed from the gastro-intestinal tract?	• **Gastric acid** aids reduction **Fe 3+ to Fe2+** (ferrous) & formation of soluble complexes • **Duodenum= major site of absorption** • **Fe3+ converted to Fe 2+ by ferric reductase** • **Fe2+ transported into enterocytes via** apical membrane iron **transporter** (DMT1) • **Dietary Heme** transported into enterocyte by Heme Transporter (HT) • Heme oxidase releases Fe 2+ from heme • Some intracellular Fe 2+ converted to Fe 3+ & bound to ferritin • Remainder binds to basolateral Fe 2+ transporter ferroportin (FP) and transported to interstitial fluid aided by hephaestin (Hp)	**Pass Criteria: 2 Bold** + 1 other
	What factors reduce Iron absorption from the gastro-intestinal tract? (Hint- what foods, medications or procedures may result in reduced Iron absorption?)	**Dietary**-Phytic acid (cereals), oxalates & phosphates bind Fe to produce insoluble compounds **Surgical**- Partial Gastrectomy (decreased acid)/ duodenal surgery-loss or illnesses (ulcers, sprue) **Physiological**- high iron stores, high recent FE diet, amount of erythropoiesis **Drugs**-antacids, acid lowering, some antibiotics	**Pass Criteria: 1 Bold**
	How is Iron transported in the plasma?	Fe 2+ converted to Fe 3+ & bound to **transferrin**	**Pass Criteria: 1 Bold**
Stem: Moving onto Pharmacology. Oxycodone is prescribed for her arm pain.			
Question 5 Oxycodone-pharmacokinetics and pharmacodynamics **Subject:** Pharmacology LOA: 1	1) Describe the pharmacokinetics of oxycodone	**Good oral absorption** High Vd Low first pass metabolism (cf morphine) **Duration of action 3-4 hours, longer if controlled release formulation** Hepatic metabolism by P450 Metabolites excreted by kidneys	Bold + 1
	2) How does oxycodone produce its analgesic effects	Opioid agonist that acts mainly on **mu receptors** in brain and spinal cord, but also outside CNS	Bold
	3)What strategies may be used when prescribing oxycodone to reduce the development of dependence	Establish goals at start of Rx Combine with non-opioid analgesics Smaller doses at longer intervals Use of controlled release preparations Frequent evaluation of ongoing requirements	3/5

ACEM PRIMARY VIVA A **Friday Morning Session 3** ***Candidate Number:*** **AGREED MARK:**

Stem: A 46-year-old man is referred by his GP with poorly controlled diabetes. Starting with Pharmacology. He is on Gliclazide			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Gliclazide (Pharmacodynamics / kinetics) **Subject:** Pharm LOA: 1	1) What type (class) of drug is Gliclazide? 2) Describe the mechanism of action of the sulfonylureas? *Prompt: What ion channels are involved?* 3) What are the pharmacokinetics of the sulfonylureas?	**A sulphonylurea.** **They increase the release of insulin from the pancreas** (specifically from pancreatic beta cells). They bind to a receptor -> inhibition of efflux of K^+ ions through a linked ATP-sensitive potassium channel -> (extracellular) depolarization. The depolarization opens a voltage-gated calcium channel -> Ca influx -> release of preformed insulin. Long term use also -> reduced serum glucagon levels. Mechanism for effect unclear, but may involve indirect inhibition due to enhanced release of insulin and somatostatin which inhibit alpha-cell secretion. **Hepatically metabolized** to products which are inactive or have very low activity. **Renally** excreted. Variable (but moderate) T ½ (Gliclazide 8hrs, Glimepiride 12-24hrs, Glipizide 12-24hrs)	Bold to pass To pass bold + concept of ion channels Bold to pass
Stem: Moving onto Physiology.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 2: Glucose homeostasis **Subject:** Phys: LOA: 1	1) What factors influence glucose homeostasis? 2) What happens to glucose homeostasis in the absence of insulin? *Prompt if only give hyperglycaemia -By what mechanisms does this occur?* 3) By what mechanism does glucose cause the release of insulin?	Glucose absorption of the **intestine** Glucose **uptake** from the **periphery** – muscle, brain, fat, RBC's and **liver**. Reabsorption in the **kidney** **Gluconeogenesis in liver** – actions of insulin & glucagon. Hyperglycemia due to: 1) Decreasing peripheral uptake of glucose into muscle and fat (direct effect). 2) Reduced glucose uptake by the liver (indirect effect) 3) Increased glucose output by the liver & lack of glycogen synthesis. (GIT, Renal, Brain, RBC uptake remains unaffected.) Specific GLUT 2 transporter in beta cells of the pancreas, converted to pyruvate, metabolized to glutamate via citric acid cycle (CAC) which primes insulin granules for release. Production of ATP also triggers (via K efflux) Ca influx which causes granules to be released.	Name 3 mechanisms for a pass. For a pass name 2/3 consequences. Basic concept to pass.

Stem: The patient asks you to look at his leg, which is painful.			
Question 3 **Leg Ulcer (Photo)** Clinical building block	Please describe this image. What could be the cause of this?	There are 2 areas of **ulceration** over the medial malleolar region of the right leg. The distal but larger ulceration has a sloughy base. The more proximal smaller ulcer has some bleeding points. Both ulcers have raised edges. There is no oedema. There is surrounding **pigmentation** secondary to chronic venous disease. 1) Trauma or 2) Infection on **background of chronic venous disease**	Pass: Bold Pass: Bold + either 1 or 2
Stem: Moving to Pathology. His ulcer is chronic.			
Question 4 Chronic inflammation **Subject:** Path: LOA 1	1) What cell types are present in chronic inflammation? 2) What processes mediate the persistent accumulation of macrophages seen in chronic inflammation? 3) What clinical conditions can cause chronic inflammation? *Prompt: Anything other than--*	**Macrophages** Multinucleate giant cells Lymphocytes Plasma cells Eosinophils Mast cells Neutrophil polymorphs (scarce) 1) **Continued recruitment** of monocytes (continued expression of adhesion molecules and chemotactic factors- Macrophage Activation Factor) 2) Local proliferation of macrophages 3) Immobilisation of macrophages (Migration inhibition factor) **Persistent infection:** Tuberculosis Syphilis Abscess Empyema Osteomyelitis **Prolonged exposure to an agent:** Exog –foreign body, persistent trauma, silica -> silicosis Endogenous – lipid -> atherosclerosis **Autoimmune:** Rheumatoid Arthritis Multiple Sclerosis Inflammatory Bowel Disease Systemic Lupus Erythematosus	*Bold PLUS 2 others* *Bold to pass* *At least 3 examples from at least 2 categories*

Stem: Moving onto Anatomy			
Question 5 Foot and Ankle Model **Subject:** Anat LOA: 1	1) What bones can you identify in this model.	**Fibula + Tibia** **Talus Calcaneus Cuboid Navicular** **Cuneiforms** – medial, intermediate, lateral First to fifth **metatarsals, phalanges**.	Bold to pass
	2) What structures make up the ankle joint?	Distal ends of the **tibia** & **fibula**, the **talus** + inf transverse part of post tibiofib lig.	Bold to pass
	3)What movements occur through this joint?	**Plantarflexion** (ext)+ **Dorsiflexion** (flex) of the foot.	Bold to pass
	4)In what position of the foot is the ankle joint most unstable and why?	**Plantarflexion** as then the trochlea (sup surface of the talus) which is narrower post, lies relatively loosely within the mortise between the malleoli.	Bold to pass
	Extra Q if time: Demonstrate the joints through which eversion / inversion of the foot occur.	Subtalar **(Talocalcanean)** joint, and **Transverse** (or mid) **tarsal joint** – consisting of the Calcaneocuboid + talonavicular joints.	

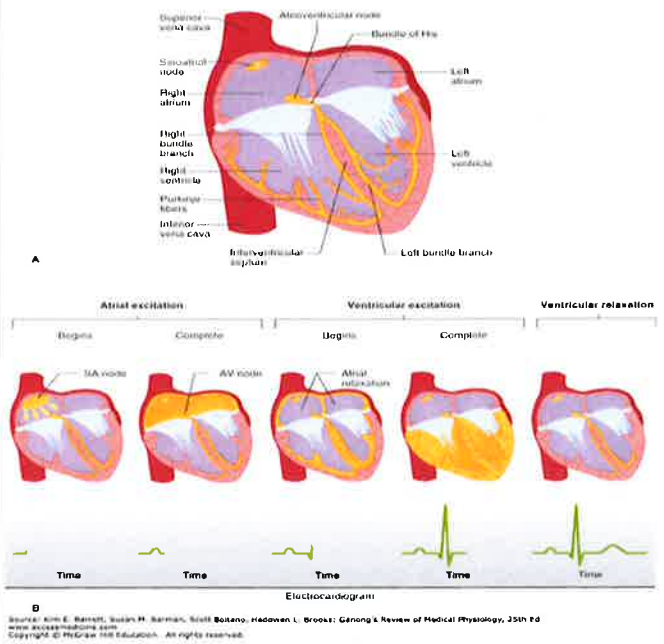
ACEM PRIMARY VIVA A **Friday PM Session 4** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: A 30-year-old male presents with a hand injury following getting his hand caught in a machine at work. An X-ray of his hand is performed.			
Question 1 Metacarpal # (Xray) Clinical Building Block	Please describe the abnormality on this x-ray.	Comminuted, spiral **fracture of the shaft of the 4th metacarpal** (ring finger).	Bold to pass.
Stem: Moving onto Anatomy			
Question 2 **Hand (bone)** **Subject:** Anat LOA: 1	1)Please name the bones in this model. 2)Please demonstrate where the Dorsal and Palmar interossei muscles attach. *Extra if time:* *What do the interossei muscles do*	Prox: Scaphoid, lunate, triquetrum, **pisiform**. Distal: Trapezium, trapezoid, capitate, hamate. Metacarpals, prox, middle and distal phalanges. Dorsal –From adjacent sides of 2 MCs (bipennate) -> bases of the prox phalanges (+ ext expansions of 2nd-4th digits). Palmar – From palmar surfaces of 2nd, 4th & 5th MCs (unipennate)-> bases of prox phalanges (+ ext expansions of digits 2,4+5). **Palmr adduct/Dorsal abduct** digits around axial line. Flex MCPJ & extend IPJ.	All except bold to pass To pass: MCs -> phalanges Dorsal bipennate Palmar unipennate
Stem: Moving on Pathology. He has multiple wounds which are bleeding.			
Question 3 Haemostasis, platelet aggregation **Subject:** Path LOA: 1	1) What is the sequence of events that occurs to produce haemostasis after a vascular injury. Prompt: What happens first? 2) What laboratory tests are used to assess the function of the different pathways of the coagulation cascade? *Prompt: Which one is vitamin K dep.*	**1) Vasoconstriction**: arteriolar, reflex neurogenic, enhanced by endothelin 2) **Primary haemostasis**: extracellular matrix exposed, platelet adherence/activation -- platelets aggregate & forms a plug 3) **Secondary haemostasis**: Tissue factors exposed, Fac III, thromboplastin, Fac VII , platelet plug consolidated – thrombin/fibrin generated **4) Thrombus & antithrombotic effect** – fibrin polymerises to form permanent plug, tPA regulates **Prothrombin time – extrinsic pathway** factors VII, X, II, V, fibrinogen (including vit K dependent factors) **Partial thromboplastin time – intrinsic pathway** factors XII. XI, IX, VIII, X, V, II fibrinogen	To pass identify 3/4 steps & demonstrate understanding of concepts To pass identify test, what pathway it is testing & identify which one is vit k dependent.

Stem: Moving onto Pharmacology. He requires sedation to have his arm attended to and Propofol is used			
Question 4 Propofol (Pharmacokinetics and Pharmacodynamics **Subject:** Pharm LOA: 1	1) Please describe the pharmacokinetics of propofol?	IV admin only. Rapid onset/recovery due to **redistribution** from brain-> skeletal muscle-> fat, rather than metabolism. **Distribution t ½ 2-4 mins** Elimination t ½ 4-23 mins Duration of action 3-8 mins Metabolism- rapidly in liver, some extra-hepatic (lung) Excretion - Urinary as glucuronides & sulphates, < 1% unchanged	**Bold** with reasonable understanding of drug redistribution
	2) What are the adverse effects of propofol?	**Hypotension**- vaso/venodilatation and -ve inotropic effect **Apnoea**- dose-related central depression of respiratory drive. Pain on injection Soy/egg allergy	**Bold**
	3) How can you limit adverse effects when using propofol?	Caution with simultaneous co-administration of opiates/benzodiazepines. Titrate small doses (10-20mg aliquots) slowly to effect. Reduce doses in the elderly or with poor cardiovascular reserve. Caution with haemodynamically unstable patients	**Any 2**
Stem: Moving on to Physiology. His blood pressure falls to 80/40. Blood loss is a contributory factor			
Question 5 Physiological responses to shock **Subject:** Phys LOA: 1	1) What is hypovolaemic shock?	**Systemic hypoperfusion due to reduced effective circulating blood volume** resulting in **impaired tissue perfusion** and cellular hypoxia.	Bold to pass
	2) What are the physiological compensatory reactions to hypotension in acute blood loss? **Prompt:** What are the immediate responses?	<u>Rapid (Seconds/minutes)</u> -**Baroreceptors** (decreased discharge with reduced arterial stretch, reducing the baroreceptor inhibition in medulla -> increased sympathetic discharge with vasoconstriction, venoconstriction and tachycardia) -**Chemoreceptors** (stimulation leads to peripheral vasoconstriction and rise in BP) -CNS receptors	Bold to pass with understanding of baroreceptor and renin angiotensin function.
	Prompt: What about intermediate or longer term?	<u>Early (Minutes/hrs):</u> **Renin-angiotensin system activated** -Blood volume changes -Capillary fluid shifts (isolovaemic anaemia) <u>Longer term:</u> **Renal compensation via aldosterone** - Renin- angiotensin system - Blood volume changes -Salt intake	

Stem: A 40 year old lady presents with suspected pericarditis. We will start with Pathology

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Acute Pericarditis **Subject:** Pathology LOA: 2	Describe the characteristic clinical features of pericarditis	**Chest pain** (dull or sharp, pleuritic, positional), fever, congestive failure, p*ericardial* ***friction rub*** Constrictive pericarditis: distant or muffled heart sounds, elevated JVP, peripheral edema.	2 Features to pass Prompt "History and examination features?"
	What are the causes of pericarditis?	**Infectious: viral**, pyogenic bacteria, TB, fungal **Immune mediated**: Rheumatic fever, SLE, Scleroderma, post cardiotomy. Post MI (Dressler's), Drug hypersensitivity reaction. AMI, uraemia, post cardiac surgery, neoplastic, trauma, radiation	Need viral, immune example and one other
	What types of pericardial fluid exudate occur?	1. Serous: usually non-infectious inflammation (RF, SLE), but also viral, uraemia, tumours 2. Fibrinous/serofibrinous; (most common) post MI, Dressler's, trauma, post surgery but also as in 1. 3. Purulent/suppurative: almost always bacterial invasion from local infection, lymphatic or blood seeding, or at operation 4. Haemorrhagic 5. Caseous (TB)	2/5 types to pass

Stem: Moving onto Physiology.			
Question 2 Electrical activity of the heart / atrial arrhythmias **Subject:** Physiology **LOA:** 1	Please draw and describe a normal ECG complex. Describe the normal sequence of electrical excitation of the cardiac conduction system and cardiac muscle	 **P** = Depolarization initiated in the **SA node** Spreads radially through the atria, Converges on the AV node. (Atrial depolarization 0.1 s.) **PR** = **Atrial depolarization & AV nodal delay (**Delay of about 0.1 s) **QRS** = Bundle of His, R&L Bundles & Purkinje fibers (**ventricles** 0.08-0.1 s) (L) to (R) across IV septum then down septum to apex along ventricular walls to AV groove from endocardial to epicardial surface last parts to be depolarized are posterobasal portion of LV, pulmonary conus, and uppermost septum	Draw ECG and accurately identify all waves and intervals **(P,PR, QRS, T, QT & ST)** Prompt – Intervals? Bold to pass 2 depolarization directions to pass

Stem: She develops palpitations. An ECG is done			
Question 3 ECG – SVT **Clinical Building Block**	Please describe this ECG. What is the diagnosis?	12 Lead ECG, standard calibration and assume standard paper speed. Axis (N), Rate ~220, Essentially Regular Rhythm, no P waves visible QRS morphology: no Q-waves, good R-wave progression, wide spread ST depression (up to 3 mm), T-waves upright (except aVR & V1), no fusion or capture beats, no A-V dissociation **Narrow complex tachycardia (SVT)**	**Rate** (>200) **Rhythm** and 2 more features Bold to pass
Stem: Moving onto Pharmacology. You plan to treat her with Adenosine.			
Question 4 Adenosine **Subject:** Pharmacology LOA: 1	What is adenosine and how does it work? Describe its pharmacokinetics What are its side effects? What are the possible drug interactions with adenosine?	Naturally occurring **nucleoside** **Blocks AV conduction** (activates inward rectifier K+ current, ie hyperpolarises the AV node) **Short ½ life** (less than 30 seconds) Uptake by endothelial and red cells Chest tightness / burning, flushing, headache, nausea, hypotension, parasthesiae, arrhythmia, bronchospasm, "sense of impending doom" ODD Theophylline inhibits - Adenosine receptor blocker Dipyridamole enhances - Adenosine uptake blocker Interaction with other AVN blocking drugs	Bold to pass **Bold** **4 of 9** **1 of 3**

Stem: Moving onto Anatomy. You insert an Intravenous line.			
Question 5 Cubital fossa (photo from Mc Minns) **Subject:** Anatomy LOA: 1	Identify the superficial veins	1 **Basilic vein** 6 **Cephalic vein** 13 Median cubital vein 14 Median forearm vein	**Bold plus 1 vein** Prompt – point Show candidate "medial"
	Identify other neurovascular structures in the cubital fossa	15 **Median Nerve** 4 **Brachial artery** 22 Ulnar artery 21 Radial artery	**Bold plus 1**
	Identify the tendons at the wrist	9 FCU 10 FDS 18 Palmaris longis 8 FCR 11 FPL 5 Brachioradialis	**4 of 6**
	Which structures lie deep to flexor retinaculum at the wrist?	FDS, FDP. FPL, median nerve (+/- FCR, debatable)	**3 of 4**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: A 60-year-old man presents with central chest pain, diaphoresis and shortness of breath. An ECG is performed.			
Question 1 STEMI ECG **Clinical Building block**	Please describe this ECG. What is the diagnosis?	12 lead ECG, (no calibration for paper speed or rhythm strip) **ST elevation in anterior leads** reciprocity (infero-lateral TWI and ST depression) Rate 90 (80-100) SR LAD PR160 (140-200) QRS 100ms (80-120) QT 360ms (320-400) **Ant STEMI**	Bold + 4 others (of 8) Don't need to count mm of STElevation Accept normal if within range **Bold**
Stem: Moving onto Pathology.			
Question 2 Acute coronary syndromes **Subject: Pathology** LOA: 1	1. Describe the pathogenesis of Myocardial infarction due to atherosclerosis	**1. Acute plaque change** a. Rupture / fissuring b. Erosion / ulceration c. Haemorrhage into atheroma **2. Thrombosis** a. Platelet adhesion, aggregation & micro-thrombi formation b. Platelet release of mediators causing vasospasm c. Activation of coagulation pathway leading to thrombus **3. Vasoconstriction** stimulated by: a. Circulating adrenergic agonists b. Locally released platelet contents c. Endothelial cell dysfunction causing decrease NO d. Perivascular inflammatory cell mediators **4. Vessel occlusion leading to:** a. decreased myocardial blood flow b. myocyte necrosis	**3 of 4 Bold** and demonstrate understanding of processes "Can you describe the process" – for each bold

	2. What are the complications of acute myocardial infarction?	• Contractile dysfunction
		  o LVF / RVF / cardiogenic shock
		• Arrhythmias
		• Myocardial rupture
		  o Free wall / Vent septum / Pap muscle
		• Ventricular aneurysm
		• Pericarditis/effusion/tamponade
		• Mural thrombus
		• Papillary muscle dysfunction
			3 to pass

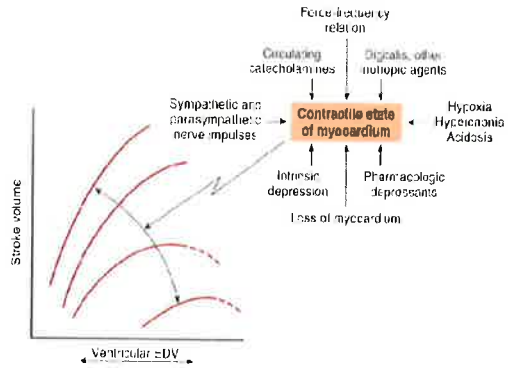
Stem: Moving onto Physiology			

Question 3
Cardiac cycle - ventricular volume relationship to ECG
 	
Subject: Physiology
LOA: 1
	Describe how the waveforms of an ECG relate to the cardiac cycle
	
	Describe the changes in left ventricular volume through the cardiac cycle starting from atrial systole
		Atrial systole starts just after the **P wave**
		Ventricular systole starts near the end of the R wave and ends just after the T wave
		
		Atrial systole
		• Phase 1 – P to R wave
		  o **Small** amount of increased **ventricular filling** due to atrial contraction
		Ventricular systole:
		• Phase 2 = **isovolumetric contraction** – R wave to ST segment (130ml)
		  o Mitral valve closes
		  o Ventricular contraction occurs with no change to volume
		• Phase 3 = **ventricular ejection** – ST segment to end T wave (65ml at end)
		  o Aortic valve opens
		  o Ventricular systole
		Diastole:
		• Phase 4 = **isovolumetric ventricular relaxation**
		  o Aortic valve closes
		• Phase 5 = **ventricular filling**
		Mitral valve opens
			Bold concepts to pass
			Prompt – how do the waveforms relate to atrial and ventricular systole
			
			Bold concepts to pass
			
			Prompt – During atrial systole, what happens to ventricular volume

Stem: Moving onto Pharmacology. As part of the patient's ongoing management, you prescribe Metoprolol.			
Question 4 Metoprolol **Subject:** Pharm LOA 1	Describe the pharmacokinetics of metoprolol? *Prompt: What's the bioavailability? Why is this so?*	Oral or IV, Well absorbed Bioavailability 50% due to **first-pass effect** **Large volume of distribution** (>200L) Half-life, 3-4 hours Metabolised in the liver	Bold and 2 others to pass
	What are the cardiovascular effects of metoprolol?	1 **Negative inotropic and chronotropic effects** 2 Slow a-v node conduction with increased PR on ECG 3 decrease BP by a mechanism not fully understood but probably includes suppression of renin release and CNS effects	Bold and 1 to pass
	How does metoprolol differ from propranolol in its action at beta receptors?	B1 equipotent B2 50-100 fold less potent ie **metoprolol is B1 specific and propranolol is not** (equipotent at B1 and B2) metoprolol at higher doses is less specific	Bold to pass

Stem: Moving onto Anatomy			
Question 5 Heart (model) – coronary supply **Subject:** Anatomy LOA: 1	1. Using the model, identify the great vessels which enter and exit the heart	Ascending **aorta** **Superior vena cava, IVC** **Pulmonary trunk / pulmonary arteries** **Pulmonary veins**	**(Bold to pass)** Prompt - point
	2. Identify the arteries that supply of the heart	Coronary arteries arise from the aortic sinuses **Left (Main) Coronary artery** **Left anterior descending** Diagonal branches **Circumflex** Marginal arteries **Right Coronary artery** Posterior descending (interventricular) artery	**Bold** + 1 to pass Prompt to LCA, where does the LAD arise from?
	3. Which areas of the heart are supplied by the Left Coronary Artery and its branches?	Most of left atrium Most of **left ventricle** Part of right ventricle (anterior wall) **Interventricular septum** Ventricular apex **AV Bundle (His)** SA node in 40% (from circumflex)	**Bold** + 1 to pass prompt - What part of the conducting system does it supply?
	BONUS Q: Describe the position of the heart in the left Hemithorax	Inferior border lies on the diaphragm Apex is in the 5th intercostal space Base is against the thoracic vertebrae (T6 –T9)	

ACEM PRIMARY VIVA B **Friday Morning Session 3** ***Candidate Number:*** **AGREED MARK:**

Stem: A adult male presents with fever and dyspnea. Starting with Physiology			
TOPIC	QUESTIONS	KNOWLEDGE	NOTES
Question 1 Cardiac contractility (Frank Starling curve) **Subject:** Physiology: LOA: 1	1. Please draw or describe the Frank Starling law as it relates to cardiac muscle.	 Curve of SV against ventricular EDV (energy of contraction is proportional to initial length of cardiac muscle fibre (represented as EDV)	**Pass criteria: Must be able to draw or describe the curve and correctly label/discuss the axes and the dotted lines** Prompt - describe
	2. What factors influence the Frank Starling curve?	Circulating catecholamines, inotropes: (eg digoxin), sympathetic input - (positive, shifting curve up and to left) Acidosis, hypercarbia, hypoxia, vagal /psymp stim, pharm depressants (barbits), intrinsic depression –negative, shifting curve down and to right	**For a pass:** **2 positive factors with correct influence** **2 negative factors with correct influence** Does anything move the curve LEFT/RIGHT?

Stem: Moving onto Pathology. He has multiple track marks and has a loud cardiac murmur			
Question 2 Endocarditis **Subject:** Pathology LOA: 2	1. What factors predispose to infective endocarditis?	**Cardiac Factors** – Degenerative mitral valve prolapse (myxomatous), calcific aortic stenosis, bicuspid aortic valve, prosthetic valves, congenital valve defects, rheumatic heart disease **Host Factors** – Bacteraemia (dental or surgical procedure, loss of skin integrity), Intravenous drug use, immunodeficiency, drug induced immunosuppression, malignancy, neutropaenia, diabetes, alcohol	**4 to pass** (2 from each group) Prompt "Any other factors for infective endocarditis in general?"
	2. Which organisms commonly cause infective endocarditis?	**Streptococcus viridans, Staph aureus**, staph epidermidis, enterococci, gram negative bacilli, HACEK (Haemophilus, Actinobacillus/Aggregatibacter, Cardiobacterium, Eikenella, Kingella); Fungal	**Bold** + 1 other to pass
	3. What are the complications of infective endocarditis?	**Local** – erosion/destruction of tissue (valve or myocardium, abscess formation (ring abscess) **Systemic** – Septic infarcts – brain, lung, kidneys, mycotic aneurysms. Embolic phenomena – Subcutaneous tissues (splinter haemorrhages, janeway lesions, oslers nodes) Retina (roth spots) Glomerulonephritis (immune mediated)	**1 local and 1 systemic** to pass Prompt "Any local/systemic complications"
Stem: Clinical building block. A CXR is performed.			
Question 3 X-ray with large pleural effusion **Subject CBB**	Describe and interpret this Xray?	**Left sided pleural effusion** Cardiomegaly (but limited inspiration) Sternal wires – previous sternotomy Blunting right costophrenic angle Calcification of aortic arch (end on)	BOLD + organized approach to describing whole Xray
	What is your differential diagnosis	Congestive cardiac failure, Empyema Pneumonia, PE, Cirrhosis/nephrotic syndrome	2 Causes

Stem: Moving onto Anatomy. You decide to drain his pleural effusion.			
Question 4 Surface anatomy of the chest / pleural reflections (photo) **Subject:** Anat: LOA: 1	Describe the surface anatomy of the lungs and the pleura	Apices of both lungs begin in **supraclavicular fossa** Lungs and visceral pleura run **parasternal to 6th costal cartilage on R ,4th costal cartilage on L, then pass laterally to MCL 6th rib, MAL 8th rib, SL**(scapular line**) at 10th rib** , T10, then paravertebrally to T12 (in contrast to parietal pleura which is at MCL at 8th rib, MAL at 10th rib and SL at 12 the rib **(2 ribs below)** Oblique fissure – spinous process T2 posteriorly –to 6th costal cartilage anteriorly Horizontal fissure - R – from oblique fissure at level of 4th rib to costal cartilage	(Candidate to use photo to demonstrate surface anatomy/pleural reflections) 4 of 6 bold Understanding of left and R variance Understanding of parietal vs visceral pleura (2 ribs below)
	What are the anatomical structures to consider when inserting a lateral intercostal catheter	**Above the rib** below to avoid neurovascular bundle **Above 5th - 6th intercostal space** to avoid diaphragm (nipple line) Anterior to mid axillary line or lat dorsi Posterior to pect major	Bold plus 1 to pass Prompt "What are the borders of the triangle of safety?"

Stem: Moving onto Pharmacology. Lignocaine is used as the local anaesthetic.			
Question 5 Lignocaine **Subject:** Pharm LOA: 1	Describe the mechanism of action of Lignocaine?	Na channel blocker, Class 1 B **Blocks** (activated and inactivated **Na Channels** = **blocks nerve conduction**. Less effect in infected tissue	Bold to pass
	What are the toxic effects of lignocaine?	CNS – Early : **tongue/oral numbness/metallic taste**. Nystagmus, muscle twitching, N+V, Tinnitus, visual disturbance. Severe: **Seizures**, sedation. CVS – **cardiovascular collapse**, hypotension, bradycardia, arrhythmia (rare), worsen CCF, conduction blocks GIT – anorexia, N+V (through CNS effects)	3 BOLD to pass Prompt "Any other systems affected?"
	What factors affect systemic absorption after local infiltration?	Dose, site of injection, drug tissue binding, tissue blood flow, vasoconstrictors (combined preparation)	3 of 5

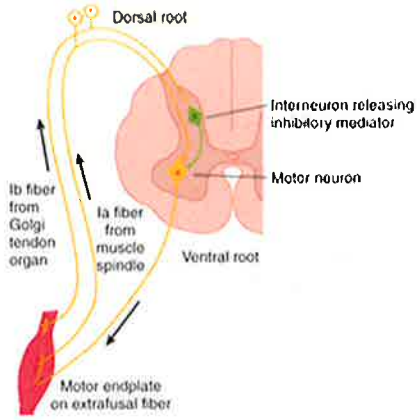
Stem: A 40-year-old woman presents with a blood pressure of 220/160. Starting with Pathology			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Hypertension **Subject:** Path LOA: 2	How is hypertension classified? What are the causes of secondary hypertension?	**Primary (essential)** **Secondary** **Renal** (Acute glomerulonephritis, Chronic renal disease, Polycystic disease, Renal artery stenosis, Renal vasculitis, Renin-producing tumors) **Endocrine** (Adrenocortical hyperfunction [Cushing syndrome, primary aldosteronism, congenital adrenal hyperplasia, licorice ingestion], Exogenous hormones [glucocorticoids, estrogen {including pregnancy-induced and oral contraceptives}, sympathomimetics and tyramine-containing foods, monoamine oxidase inhibitors], Pheochromocytoma, Acromegaly, Hypothyroidism, Hyperthyroidism, Pregnancy-induced **Cardiovascular** (Coarctation of aorta, Polyarteritis nodosa,, Increased intravascular volume, Increased cardiac output, Rigidity of the aorta. **Neurologic** (Increased intracranial pressure, Sleep apnea) **Psychogenic** (Acute stress, including surgery, Pain)	**Bold** **6 examples from at least 3 different systems.** Prompt "Diseases in what (other) systems cause secondary hypertension?"

Stem: Moving on to Pharmacology. She was recently commenced on Irbesartan.			
Question 2 Angiotensin receptor blockers including Pharmacodynamics **Subject:** Pharm LOA: 2	Describe the pharmacodynamics of irbesartan	Competitive selective **antagonist of the AT1 receptor.** Vasodilation, inhibition of aldosterone secretion	**Bold** And 1 of 2
	What are the benefits over ACE inhibitors	**No effect on bradykinin so reduced incidence of cough, angioedema** More complete inhibition of actions of angiotensin 2 compared to ACE inhibitors May increase angiotensin 2 which then has actions on AT2 receptors- vasodilation, additional benefit	**Bold**
	What are specific contraindications	Non-diabetic renal failure Pregnancy Allergy/previous adverse reaction Hyperkalaemia, Renal artery stenosis	2 of 5
Stem: Moving onto Physiology			
Question 3 Renin angiotensin system **Subject** Phys LOA: 1	1 What leads to activation of the renin-angiotensin system. Prompt "List some conditions which activate the renin-angiotensin system"	**Activated in response to decrease in BP/ ECF or increased sympathetic activity** eg hypotension, haemorrhage, dehydration, cardiac failure, cirrhosis, Na depletion, diuretics, upright posture, pain, fear, arousal	Bold + 4 conditions
	2 What are the principal effects of angiotensin II? Prompt Where does angiotensin II act?	**Arterioles** (AT1 receptor) – **vasoconstriction** – increases TPR **Adrenal cortex - increase aldosterone production – increased Na and H2O resorption** Kidney – direct effect to decrease GFR and increase Na reabsorption Brain – decreased sensitivity of brain baroreceptor reflex – potentiates pressor effect Pituitary – increase ADH and increase ACTH scretion	Bold to pass Prompt "What causes that or what effect does that have?"

Stem: An ECG is performed.			
Question 4 Left ventricular hypertrophy **Clinical Building Block**	Please describe the ECG What is the likely cause of the ECG abnormalities?	**Sinus Rhythm** Rate around 75/minute Left axis deviation Normal PR interval **Large QRS voltage** with broad QRS (Voltages – S wave in V2 + R wave in V6 >>35 mm) ST elevation V1-3 LV strain (ST depression/T wave inversion) in leads I, aVL, V5, V6 **Left Ventricular Hypertrophy**	**Bold** + 2 others Prompt – What do you think about the size of the QRS in some of the leads? **Bold**

Stem: Moving on to Anatomy. A CXR is performed.			
Question 5 CXR (mediastinal structures and boundaries) **Subject:** Anat LOA: 1	Outline the structures that make up the right and left cardiomediastinal borders on this X-ray (you can point on the Xray)	Right - Right brachiocephalic vein - Superior vena cava - Right pulmonary trunk - Right Atrium - Inferior vena cava Left - Left subclavian artery/left brachiocephalic vein - Aortic arch - Left pulmonary trunk - Left atrial appendage - Left Ventricle	6/10 to pass
	Describe the lobes of the lungs and their fissures.	**Both lungs: oblique fissures** separate upper and lower lobes (T2 posteriorly to 6th costal cartilage ant) **Right lung** – upper and middle lobes separated by the **transverse fissure** (at level of right lung hilum along line of 4th rib) Left lung – prominent cardiac notch in lower lobe	Bold to pass Bold to pass
	Which part of the lung forms the right heart border?	**RML**	Bold

ACEM PRIMARY VIVA C **Thursday Morning Session 1** ***Candidate Number:*** **AGREED MARK:**

Stem: A 4-year-old girl presents following a febrile convulsion. She is hyperreflexic. We will start with Physiology.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	
Question 1 Stretch reflexes **Subject:** Physiology LOA: 2	1. Please describe the components of the stretch reflex: 2. What is a typical example of this?	1. **Monosynaptic**; skeletal muscle stretched with contraction muscle as the response Stretch receptor sensory neuron makes an excitatory connection with the extensor motor neuron of the same muscle and an inhibitory interneuron projecting to the antagonistic muscle. Sense organ is muscle spindle. Impulses from spindle are transmitted to the CNS by fast sensory fibres that pass directly to the motor neurons that supply the same muscle. The neurotransmitter at the central synapse is glutamate. Eg The Knee **jerk**  	Simple explanation +/- diagram Bold

Stem: Moving onto Pharmacology. She has another seizure and is given Midazolam.			
Question 2 Midazolam including pharmacokinetics **Subject:** Pharmacology LOA: 1	1. What are the pharmacokinetics of Midazolam? 2. What is the mechanism of action of midazolam? 3.What are the clinical effects of midazolam?	1. -**Water soluble** hence oral/IM/intranasal -**Poor oral bioavailability** -**Highly Protein Bound** -Crosses BB barrier easily at body pH. -**Short elimination half-life** 1.5-2.5 hours. -hepatic metabolism/renal excretion **2.** -Binds to GABA-A receptor (or complex) -**Potentiates GABAergic inhibition** through hyperpolarization (through Chloride) -Acts throughout brain 3. -Strong amnestic effect -Anticonvulsant -Anxiolytic -Sedative-hypnotic -Antiemetic -Reduced sensitivity to CO2	**2/4 Bold** **Bold** **2 other than anticonvulsant**
Stem: Moving onto Anatomy. You notice eye deviation during the seizure			
Question 3 Eye (model) **Subject:** Anatomy LOA: 1	Name and identify the extrinsic muscles of the eye What is the innervation of each muscle?	**Superior rectus (III oculomotor)** **Inferior rectus (III oculomotor)** **Medial rectus (III oculomotor)** **Lateral rectus (VI abducent)** **Superior oblique (IV troclear)** **Inferior oblique (III oculomotor)** (Superior, medial, inferior and lateral rectus arise from common tendinous ring (surrounding optic canal) Superior oblique arises from the body of the sphenoid, passes forward above the medial rectus and gives way to a slender tendon which passes through the trochlea (pulley). Then turns backwards and laterally and passes under the sup rectus to insert into posterosuperior lateral quadrant of sclera.	Bold

	What movements are generated by these muscles ? *[Bonus Prompt : What muscles make the eye look directly up?]*	Inferior oblique arises from maxilla on the floor of the orbit near ant margin. Passes obliquely backwards and laterally below inferior rectus and curves up deep to lateral rectus to be attached in posteroinferior lateral quadrant) **MR – medial (horiz plane)** **LR- lateral (horiz plane)** **SR –** inserted in front of coronal equator, and line of pull is medial to axis of rotation, therefore **up and in** **IR –** same, **down and in** **IO – up and out** **SO – down and out** IO plus SR – up SO plus IR – down	4/6 bold
Stem: She has a widespread rash and she is not immunised			
Question 4 Photo – mac / pap rash **Clinical Building Block**	Describe and interpret the rash. What could be causing the rash in this scenario?	Diffuse **maculo(papular)** rash Likely **viral** eg measles, rubella	Bold
Stem: Moving onto Pathology. Measles is suspected.			
Question 5 Measles **Subject:** Pathology LOA: 2	What type of virus is Measles? How is it spread? Describe some of the clinical manifestations of Measles infection. [What are the serious manifestations?]	Single stranded **RNA virus,** a member of the **Paramyxovirus** family (Mumps, RSV and Paraflu). There is only one strain of virus – so preventable by vaccine. **Respiratory droplet spread.** Viral pneumonia (60% of deaths) Conjunctivitis and Keratitis – scarring and blindness **Acute Measles Encephalitis** (1:1000) Adults> kids Subacute sclerosing panencephalitis (1:100000) Diarrhoea (enteropathy) Immunosuppression Croup	1 Bold to pass Bold **Encephalitis** and 1 other

	What immune responses occur as a result of Measles infection?	T-cell mediated immunity controls the infection and produces the rash – a hypersensitivity reaction to viral antigens in the skin. (no rash if deficient cell mediated immunity) Antibody mediated immunity to Measles virus protects against reinfection	Bonus

ACEM PRIMARY VIVA C **Thursday Afternoon Session 2** ***Candidate Number:*** **AGREED MARK**

Stem: A 75-year-old man with chronic airways disease presents unconscious after a fall down stairs. Arterial blood gases are done.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 ABG showing acute respiratory acidosis **Clinical Building Block**	Describe and interpret the arterial blood gas	ABG on room air pH 7.25 – acidaemia pCO2 - 65 – elevated – **respiratory acidosis** HCO3 - 33 – **elevated** – metabolic compensation (chronic) pO2 / SaO2 decreased – **hypoxia**	Bold to pass
Stem: Moving onto Physiology.			
Question 2 Alveolar gas equation **Subject:** Phys LOA: 1	What are the causes of hypoxaemia in general? [What are the lung related causes?] [can you give examples?] [How does that pathology cause hypoxaemia?]	**Hypoventilation**: Eg Drugs – Morphine/ barbiturates/ Chest wall damage/ Resp m. paralysis **Diffusion limitation**: Impaired diffusion process of oxygen across the Pulmonary capillary Eg Exercise/ thickened blood gas barrier state, Low O_2 mixture inhaled **Shunt**: Shunt refers to blood entering the arterial system without going through ventilated areas of the lung.Eg: Abnormal vascular connection (AV fistula/ CCHD defecet in R/L sides of heart). **Ventilation – Perfusion inequality** most common. Vent/Perfusion ratio determines gas exchange for any Resp unit. Regional variance exists. Hypoxaemia caused by V/Q mismatch cannot be eliminated with increased ventilation. Eg Pulmonary Embolism	3 of 4 Bold + 2 examples altogether

	What is the Alveolar gas equation? [Prompt: What is the calculation?] How is it used clinically? (Bonus)	Useful formula to measure the relationship between the fall in PO_2 and the rise in PCO_2 that occurs in Hypoventilation. Alveolar arterial difference (A/a gradient) – a useful measure of the V/Q inequality. **$PAO2 = PIO_2 - \frac{PACO_2}{R} + F$** Where: PAO_2 = Alveolar Oxygen partrial pressure PIO_2=Partial pressure Inspired (dry) Oxygen: FIO_2 less 47 mmHg Water Vapour (~ 149mmHg) when FiO2 21% and 760 mmHg. $PACO_2$ = Measured $PaCO_2$ R= Respiratory Quotient is the given CO2 production/ O2 consumption determined by the metabolism at steady state. Typically 0.8. Also called Respiratory Exchange ratio. F is a small correction factor for inert gases (typically 2mm Hg and can be ignored). A/a gradient calculated by subtracting the measured PaO_2 (arterial) from the calculated PAO_2.	Describe formula Define all Bold terms. Bonus
Stem: Moving onto Pharmacology. He has a seizure and is loaded with phenytoin.			
Question 3 Phenytoin – pharmacokinetics **Subject:** Pharm LOA 1	Describe the pharmacokinetics of Phenytoin What is the rationale for using a loading dose of phenytoin?	High oral availability (90%), poor IMI Peak serum concentration 3-12hrs later **Highly plasma protein bound** (90%) Vd 45L/70kg (brain, liver, mm, fat) **Elimination is dose-dependent** (capacity limited / nonlinear / saturable elimination) At low blood concentrations first order kinetics; at higher blood concentrations – **hepatic enzymes saturated – elimination slows** t1/2 variable (12-36hrs) as a result **Metabolised** to inactive metabolites **by the liver** then urinary excretion (< 2% excreted unchanged) Otherwise need 4 half lives to get to **steady state**, so reach **target** concentration more rapidly Dose = VolumeDist x TargetConc	Bold to pass incl. concept of dose-dependent elimination Bold or Concept

Stem: Moving onto anatomy. There is concern for cervical spine injury.			
Question 4 C1/2 (bone) **Subject:** Anatomy LOA: 1	Identify the features of this bone.	**C2 or axis:** Body, **dens**, superior and inferior articular facets, lamina, pedicle, transverse process, transverse foramen, bifid spinous process, vertebral foramen	ID the bone and dens + 4
	Describe the joints between C1 and C2	**2 x Lateral atlanto-axial joints** (facet joints) are synovial joints, between inferior articular facet of atlas and superior articular facet of axis each side. Lax capsule **Median atlanto-axial joint:** synovial joint between anterior arch of C1 and dens – a pivot joint.	bold
	Which ligaments stabilise the joints between C1 and C2?	**Cruciate or cruciform ligament** –made up of STRONG transverse lig across atlas behind dens (bursa between) and WEAKER vertical bands from back of body of axis posterior to dens to basiocciput (bypass atlas) Holds dens in position and prevents pressure from dens on medulla Alar (x2) ligaments from sides of dens to the edge of foramen magnum. Strong and limit rotation (with weak apical lig from apex dens to FM) Tectorial membrane is a continuation of post longitudinal ligament, attached from back of body of axis to ant half of FM. Lies in front of dura	**Must know cruciate + 1 other**

Stem: He has multiple fractures. Moving onto Pathology.			
Question 5 Fracture healing **Subject:** Pathology LOA 1	1. How are fractures classified	• Complete/incomplete • Open/Closed • Comminuted • Displaced • Pathologic • Stress	3/6
	2. Describe the steps in fracture healing	1. **Haematoma** fills fracture gap – provides fibrin mesh framework (hours) 2. **Influx** of inflammatory cells, fibroblasts, new vessels (days) 3. Haematoma organising –> **Procallus** 4. **Ossification –> bony callus** (2-3/52) 5. Callus matures, **remodelling** (6 weeks)	• 4 of 5 steps • Logical sequence
	3. What factors can impede the healing of fractures	• **Inadequate immobilisation** • Marked displacement/soft tissues • Vascular compromise • **Infection** (open fractures/foreign bodies) • Systemic factors (nutrition, osteoporosis, smoking…)	BOLD and 1 other

ACEM PRIMARY VIVA C **Friday Morning Session 3** ***Candidate Number:*** **AGREED MARK:**

Stem: A 36-year-old woman returns from Africa febrile. Starting with Pathology			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: Malaria **Subject:** Path LOA: 1	What organisms cause malaria? Name the types [What sort of organism is that?] Describe the pathogenesis of malaria [What happens next?] [What does it do to the blood?] How does P falciparum present clinically?	Malaria is a **protozoal** infection, intracellular **parasite** *Plasmodium* **P falciparum**, P ovale, P vivax, P malariae Infectious stage (sporozoite) is found in saliva of female anopheles **mosquito.** Sporozoites released into blood and attach and **invade hepatocytes** . **Multiply** rapidly. **Hepatocyte ruptures**, releasing up to 30000 merozoites (NB P vivax and ovale have dormant hepatic stage therefore can relapse). Released merozoites from liver **bind to** surface of **RBC**, grow in vacuole. In RBC become trophozoite (single chromatin), then schizont (multiple chromatin masses) then each chromatin becomes merozoite again. **RBC lyses** and new merozoites infect additional RBCs. Only erythrocytic parasites cause illness **Fever,** severe **anaemia**, ARF, **cerebral symptoms**, pulm oedema, DIC Congestion and enlargement of spleen Infected RBCs clump → ischaemia due to poor perfusion→ manifestations of cerebral malaria (vessels plugged with parasitized RBCs, local venous stasis, local hypoxia and inflammatory infiltrate) ARF (Hb casts in tubules, pigment etc in glom) Stimulates cytokines, TNF, IFN, IL1 → pulm oed, fever, shock	**Parasite or protozoa** **P falciparum** + 1 **Bold and general idea** **Fever + 1**

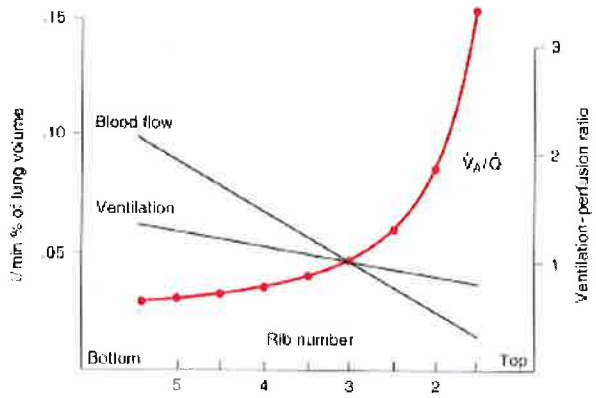
Stem: She is confused. A CT brain was performed.			
Question 2 Brain CT **Subject:** CBB	Describe the abnormality on this CT image?	Axial non-contrast CT brain Hyperdense oval area right cerebellar hemisphere – haemorrhage +/- surrounding oedema	CTcereb8.jpg **Describe abn**
Stem: Moving onto Anatomy.			
Question 3 CNS Ventricular system **Subject:** Anat: LOA: 1	1. Describe the ventricular system of the brain. [What connects the third and fourth ventricles?] 2. Outline CSF flow in the brain. (if not already covered in 1)	**Lateral** Ventricles (ant/ post and inf horns) = largest each opens into 3rd via interventricular foramen **3rd ventricle** = slit like btw diencephalon halves Continues as **cerebral aqueduct** (post-inf) which connects 3rd and 4th **4th ventricle** = pyramid shaped, post part of pons and medulla. Tapers, continuous central canal CSF secreted by **choroid plexuses** in ventricles Drains into subarachnoid space from 4th via median (1) and lateral (2) apertures. It then passes to the **subarachnoid** space – multiple cisterns	Can use same CT scan Must get **4 ventricles and cerebral aqueduct** to pass **PLUS** some additional information (i.e. location in brain, size/shape) **2 Bold**
Stem: Moving onto Pharmacology. She has been taking doxycycline for malaria prophylaxis			
Question 4 Doxycycline Pharmacodynamics **Subject:** Pharm: LOA: 2	1. What is the mechanism of action of doxycycline? [in general?] 2. What are the side effects of doxycycline? [Specifically for doxycycline?]	**Protein synthesis inhibitor -** Binds reversibly to 30s subunit of ribosome (bacteriostatic) In malaria inhibits protein synthesis Active against erythrocytic schizonts of all malaria parasites Used for prophylaxis. Not used as single agent in treatment – slow and not active against liver stages GI – nausea and vomiting Photosensitivity Candidal vaginitis Hepatotoxicity Discolouration teeth & bones (binds to calcium in newly forming bone/teeth - in pregnancy & children <8 years old) Intracranial hypertension	Bold 3 of list to pass including 1 of photosensitivity and teeth

	3. Other than malaria what are the other indications for doxycycline?	Respiratory tract infections STI's (eg Chalmydia, syphilis) Skin infections (eg acne) Rickettsia (eg. Q fever) Vibrio species (eg. Cholera) Antihelminthic Anthrax Gram negatives (rarely)	2 to pass
Stem: Moving onto Physiology. Her respiratory rate falls.			
Question 5 Control of ventilation **Subject:** Phys: LOA: 1	1. What parts of the brain control respiration? 2. What stimuli affect respiration? [Prompt for Chemical or non-chemical control]	1. -**Cerebral cortex** for voluntary control -**Medulla** for automatic control (driven by pacemaker cells in the pre-Bötzinger complex) 2. Chemical control : chemoreceptors -**CO_2** (readily penetrates the BBB and enters CSF and brain interstitial fluid H^+ concentration), promptly hydrated and H+ ions increase - chemoreceptors in **medulla** sensitive to changes in H^+ Increase in H+ conc in CSF stimulates ventilation Some CO_2 regulation via carotid/aortic bodies -**O_2 / H^+** (via **carotid/aortic bodies**) ↓ O_2 results in ↑ glomus cell activity in carotid/aortic bodies Fast response to decreased O2 Dec pH causes response in carotid body only. Nonchemical control -vagal afferents from pulmonary stretch receptors -afferents from pons/hypothalamus/limbic system -afferents from proprioceptors in mm, tendons, jts -afferents from baroreceptors: arterial, atrial, ventricular, pulmonary	Bold Bold + concept for CO2/O2 regulation + 1 non-chemical control

	3. How does hypoventilation affect respiration? [Bonus Question] Prompt: What is the role of H+ ions?	3. BBB is permeable to CO_2; relatively impermeable to HCO_3^- ↑ blood pCO_2 → ↑CSF pCO_2→↑ H^+ in CSF→ stim vent ↑ H^+ in CSF stimulates ventilation ↓ H^+ in CSF inhibits ventilation; causes cerebral vasodilatation → enhance diffusion of pCO_2 into CSF	General concept of bolded

ACEM PRIMARY VIVA C **Friday Afternoon Session 4** ***Candidate Number:*** **AGREED MARK:**

Stem: A 50-year-old woman is given Ceftriaxone for septic shock. Starting with Pharmacology.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Ceftriaxone **Subject:** Pharm LOA: 1	What kind of antibiotic is ceftriaxone? [What group?]	**Third generation cephalosporin.** **Beta lactam antibiotic.**	2 to pass
	What is the mechanism of action of ceftriaxone? [What is its site of action?]	**Bacteriocidal antibiotic.** Only kills growing bacterium. **Inhibits** transpeptidation reaction of **bacterial cell wall synthesis**. Halts peptidoglycan synthesis, leading to inhibition of bacterial growth, and ultimately cell death.	2 bold to pass
	Explain the microbiological spectrum of activity of ceftriaxone [Is there anything it is not active against?]	**Not usually degraded by bacterial beta-lactamases,** therefore broader spectrum of activity. **Expanded gram-negative** cover and crosses the blood brain barrier. Effective against many B-lactamase producing **Haemophilus** and **Neisseria** and penicillin-resistant pneumococcus. **Not active against pseudomonas**	3 of 5 bolded
	What is ceftriaxone's plasma half life? [How is this relevant clinically?]	Half life of 7 to 8 hours, meaning it may be administered once daily at 15 to 50mg/kg	Bonus
Stem: A blood gas is performed			
Question 2 Metabolic acidosis **Clinical Building Block**	Describe and interpret the venous blood gas	pH 7.10 – acidaemia pCO2 - 23 – reduced – **respiratory alkalosis/compensation** HCO3 - 12 – reduced – **metabolic acidosis** Lactate – 4.1 – raised – **Lactic acidosis from septic shock** pO2 – 53 - decreased – Venous Gas sample so inaccurate	**BOLD** to pass

		(40% O2 inspired)	
Stem: Moving onto Physiology. She is hypoxic.			
Question 3 VQ mismatch **Subject:** Phys LOA: 1	What does Ventilation- Perfusion ratio mean? In the upright lung, how does the V-Q ratio change? [Prompt: Can you graph the distribution of ventilation and Blood flow in the upright lung?] [Can you draw a diagram of the lung showing the V-Q Ratios?]	The concentration of oxygen (PO_2) in any Respiratory unit is determined by the ratio of the amount of air getting to the alveolus (ventilation) and blood flow through the Pulmonary capillary (Perfusion). V/Q ratio 0.8 (4.2 litres gas flow/ 5.5L blood flow) **Ventilation increases** slowly from top to bottom of the lung, and **perfusion increases more rapidly**. V/Q perfusion ratio DECREASES down the lung. It is **HIGH at the top** of the lung (where blood flow is minimal) and much **LOWER at the bottom** of the lung.  **Figure 5-8.** Distribution of ventilation and blood flow down the upright lung (compare Figures 2-7 and 4-7). Note that the ventilation-perfusion ratio decreases down the lung.	Definition Bold If used graph must include V/Q relationship
Stem: Moving onto Anatomy. A femoral central line needs to be placed.			

Question 4 Femoral triangle photo – structures and relationships **Subject:** Anat LOA: 1	(McMinn Atlas photo 365A) Name and indicate the boundaries of the femoral triangle Name and demonstrate the contents What does the femoral nerve supply?	**Superior: Inguinal ligament;** **med – medial border of add longus;** **lateral: Sartorius;** **floor iliopsoas & pectineus** **Femoral Nerve, artery and vein** (nodes & lymphatics) Muscle: (L2,3,4 post roots) iliopsoas (hip flexion) & quads (knee extension); Superficial division has 2 cutaneous(intermed & medial cutaneous N thigh) and 2 muscular divisions (sartorius & pectineus). Deep division: muscular (quads: rectus femoris, vastus med, vastus lateralis & vastus intermedius) & cutaneous saphenous (skin medial leg and foot) Proprioception hip joint from nerve to rectus femoris; Nerve to vastus medialis provides proprioception to knee	4/5 All 3 **Quads and sensory thigh**
Stem: Moving onto Pathology. Blood cultures grow a Streptococcus			
Question 5 Streptococcal infection **Subject:** Path LOA: 1	1. What is the microscopic appearance of streptococci? 2. What are some post-infectious syndromes caused by streptococcal infections? 3. List some infections caused by streptococcus	1. **Gram positive cocci in pairs** or chains 2. -**Rheumatic fever** (+/- complications, chorea) -**Immune complex glomerulonephritis** -Erythema nodosum, rash, myoclonus, myalgia, arthritis, neuropsychiatric sequelae, tics 3. Mouth – dental caries – *S.mutans* Skin – erysipelas – *S.pyogenes (grp A strep)* Skin – scarlet fever – *S.pyogenes* ENT – pharyngitis - *S.pyogenes* Lungs – pneumonia – *S.pneumoniae / pneumococcus* CNS - meningitis – *S.agalactiae (grp B strep)* Neonatal sepsis CV – endocarditis – *S.viridans*	1. **Bold** **2. 1 bold +1** **3. Any 4**

ACEM PRIMARY VIVA D **Thursday Morning Session 1** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: A 70yo man presents with vomiting and abdominal pain. He is given metoclopramide. We will start with Pharmacology.			
Question 1 Metoclopramide / antiemetics **Subject:** Pharmacology LOA: 1	a. Describe the mechanisms of action of metoclopramide b. Describe the potential adverse effects of metoclopramide	**Dopaminergic** (D2) **antagonist at chemoreceptor trigger zone/CTZ.** Increases oesophageal motility. Increases LOS pressure. Increase gastric emptying CNS: Restlessness, drowsiness, insomnia, anxiety, agitation – common (20%), esp. elderly Extrapyramidal effects: **acute dystonia**, akathisia, parkinsonian effects, more likely with higher doses Tardive dyskinesia with chronic dosing Prolactinemia – galactorrhea	Bold + 1/3 to pass bold + 2
Stem: Abdominal X-rays are taken. Moving onto the Clinical Building Block.			
Question 2 AXR (bowel obstruction) **Clinical Building Block**	Describe this abdominal x-ray	Erect abdominal x-ray showing markedly **dilated small intestine. Multiple air-fluid levels.** Minimal (empty) large bowel loop indicating proximal large **bowel obstruction.**	Bold to pass
Stem: He has previously had a normal CT abdomen. Moving onto Anatomy.			
Question 3 Normal CT at transpyloric plane **Subject:** Anatomy LOA: 2	a. Name the structures on this CT. b. Describe the arterial blood supply of the small and large intestine	**Liver**, portal vessels, R Kidney (top), **aorta**, IVC (not clearly differentiated), **L kidney**, **spleen**, splenic vein (not tortuous), bowel loops, pancreas, antrum. Vertebra, ribs, paravertebral muscles, intercostal and abdominal wall muscles, fat, skin The small intestine (jejunum and ilium) is supplied by the branches which arise from the **superior mesenteric artery** (ileal and jejunal aa). The large intestine is supplied by **both the superior mesenteric** (ileocolic, middle colic and right colic aa to the ascending and prox 2/3 of transverse colon) and the **inferior mesenteric artery**.(left colic, sigmoid aa & superior rectal aa). The duodenum is supplied by a branch of the coeliac trunk.	4 bold and 2 others Bold to pass

Stem: Moving onto Pathology.			
Question 4 Intestinal obstruction **Subject:** Pathology LOA: 2	a. Describe the common causes of bowel obstruction	Adhesions, hernia, malignancy, volvulus, intussusception, mesenteric infarct, strictures (due to Crohns, radiation, mesenteric ischaemia)	4 of 7
	b. How does a hernia form, and cause a bowel obstruction?	**Weakness/defect in abdominal wall**, protrusion of serosa lined pouch of peritoneum (hernia sac). **Visceral protrusion** (small bowel, large bowel, omentum most often involved.) **Entrapment of hernia sac in a narrow neck** causes pain. Ongoing obstruction → venous stasis, oedema → incarceration and strangulation Common locations (inguinal, femoral, scars, umbilical)	Bold + 2 others
	c. Describe some important clinical sequelae of ongoing bowel obstruction	**Intestinal perforation, intestinal ischaemia** peritonitis, sepsis, abscess, electrolyte disturbance, vomiting and aspiration, death	Bold
Stem: He has not passed urine for 12 hours. Moving onto Physiology.			
Question 5 Control of micturition. **Subject:** Physiology LOA: 2	a. Describe the neurological pathways involved in normal micturition.	**Sacral spinal reflex** mediated by S2, S3 and S4 nerve roots. **Facilitated and inhibited by higher centres;** **subject to voluntary control.** - First urge to void at 150ml. Marked fullness at 400ml - sudden rise in intra-vesical pressure triggers reflex contraction. Micturition reflex: - Stretch receptors in bladder wall. Afferent limb in pelvic nerves. **Parasympathetic efferent fibres** (via same pelvic nerves) mediate contraction of detrusor muscle. - Pudendal nerve (S2, S3 and S4) permits voluntary contraction of perineal muscles/external urethral sphincter, to slow or halt flow. - Sympathetic nerves to bladder play no role in micturition	To Pass: Spinal Reflex Parasympathetic control Higher centre control
	b. Describe the muscles involved in micturition. *Prompt: What is the bladder muscle called?*	1. **Bladder**: smooth muscle arranged in spiral, longitudinal and circular bundles. Circular bundle is called the **detrusor muscle.** Contraction of detrusor is responsible for involuntary emptying. **2. External urethral sphincter –** skeletal muscle sphincter of the membranous urethra. Relaxes during micturition. This is voluntarily controlled. 3. Perineal muscles. Relaxes during micturition. Also voluntarily controlled. 4. In males, urine left in urethra expelled by several contractions of bulbocavernosus muscle. 5. Contraction of abdominal wall muscles aids expulsion of urine. NB: Internal urethral sphincter (smooth muscle bundles passing on either side of urethra) plays no apparent role in micturition	Bold to pass

ACEM PRIMARY VIVA D **Thursday Afternoon Session 2** ***Candidate Number:*** **AGREED MARK**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: A 70yo man presents with left leg cellulitis. He has been treated with Flucloxacillin. Starting with Pharmacology.			
Question 1 Flucloxacillin **Subject:** Pharm LOA: 1	a. What is the mechanism of action of flucloxacillin?	Beta lactam Inhibits bacterial growth by binding to active site of **penicillin binding proteins (PBP), interfering with transpeptidation of bacterial cell wall synthesis** → cell death (bactericidal)	Bold
	b. What microorganisms are susceptible to flucloxacillin?	**Staphylococcal** (including beta lactamase producing) **Streptococci** Not active against MRSA, enterococci, anaerobes, gram negatives	Bold
	c. What are the important side effects of flucloxacillin?	**Allergy/anaphylaxis**, GIT upset (n/v), **Hepatic (cholestasis),** Renal (interstitial nephritis) , Haematological (neutropenia/thrombocytopenia), Serum sickness	Bold + 1
Stem: A knee x-ray is performed. Moving onto Anatomy.			
Question 2 Knee joint (normal x-ray) *Use AP and lateral xrays* **Subject:** Anatomy LOA 1	a. Demonstrate the bony features on this xray	**Femur: medial and lateral condyles** and epicondyles. Adductor tubercle. **Tibia: medial and lateral condyles** that form a relatively flat superior articular surface (tibial plateau). **Intercondylar eminence/tibial spine** formed by medial and lateral intercondylar tubercles. Tibial tuberosity. **Fibula: Head of fibula** (contacting fibular articular facet of tibia = Tibiofibular joint) **Patella**	Bold
	b. Describe the anatomy of the Cruciate Ligaments	**Anterior cruciate: arises from anterior intercondylar area of tibia** just posterior to attachment of medial meniscus. Extends superiorly, posteriorly and laterally to attach to posterior part of medial side of **lateral femoral condyle** (weaker of 2 cruciates and has poor blood supply) **Posterior cruciate: arises from posterior intercondylar area of tibia**. Passes superiorly and anteriorly on medial side of ACL. Attaches to anterior part of lateral surface of **medial femoral condyle** (strongest of 2 cruciates)	Bold
	c. What are the factors that contribute to stability of the Knee Joint?	Relatively mechanically weak joint due to incongruence of its articular surfaces **Large quadriceps femoris muscle** (particularly inferior fibres of vastus medialis and lateralis) **most important** Tibial (medial) and Fibular (lateral) Collateral Ligaments Anterior and Posterior Cruciate Ligaments	Bold

Stem: You perform a joint aspirate of the knee. Moving on to the Clinical Building Block.			
Question 3 Joint aspirate of septic arthritis **Clinical Building block**	Please describe & interpret this aspirate result. *Prompt: What is the differential diagnosis?*	Very **high WCC** (>90,000), predominantly neutrophils - suggest infection more likely than other causes **DDx Septic arthritis** Crystal arthropathies Inflammatory arthropathies	Bold to pass + one extra DDx
Stem: You are concerned about Septic Arthritis. Moving onto Pathology			
Question 4 Septic Arthritis; Staphlyococcal infections Subject: Pathology LOA 1	a. Which organisms may cause septic arthritis? b. What are some predisposing conditions for septic arthritis? c. Name two different species of Staphylococci and give examples of infections they cause?	**Staph**, **Strep,** Gonococcus, H influenza, Gram neg (E coli, Salmonella, Pseudomonas) - Immunosuppression – DM, steroids, other; - Joint trauma/surgery/prosthesis, - Chronic arthritis, IVDU **S. Aureus** – skin (furuncle, boil, carbuncle, impetigo, abscess, wound), pneumonia, osteomyelitis, GI/gastro, TSS S. Epidermidis – opportunistic, eg catheterized, IVDU, prosthetic valves S. Saprophyticus – UTI in young females	Bold to pass 2 to pass S.aureus and 2 examples + 1 other Staph and example to pass
Stem: The patient has an acute kidney injury from his sepsis. Moving onto Physiology.			
Question 5 Renal tubular function **Subject:** Physiology LOA 1	a. In the renal tubules, what are the mechanisms of reabsorption & secretion? b. What are the main mechanisms for Na reabsorption in the renal tubule? *Prompt: In the proximal renal tubule, what other transport proteins are involved in the movement of sodium and chloride across the apical membrane?*	**Co-transporters** (sec active transport), **exchangers**, ion channels, pumps, endocytosis, passive diffusion, facilitated diffusion, active transport **Prox tubule** 60% reab, mostly **Na/H exchanger** Thick AL 30% reab, mostly Na K 2Cl Co-transporter DCT: 7% reab, mostly Na Cl CT Prox: **Na/glucose** CT, Na/phosphate CT, Na/lactate CT, Na/amino acid CT, **Na/H** exchanger, Cl/base exchanger	Bold + 1 other to pass Bold to pass Bold to pass (Note Na/K ATPase is in basolateral membrane, & Na channel is in collecting duct)

ACEM PRIMARY VIVA D **Friday Morning Session 3** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: A 65yo man has fallen off a ladder. He has significant shoulder pain. Starting with Anatomy.			
Question 1 Scapula (bone) **Subject:** Anatomy LOA: 1	a. Identify the main features of this bone b. Demonstrate on this bone the attachments of the scapulohumeral muscles *Prompt if needed: what are the rotator cuff muscles?**	1. **Glenoid cavity, spine, supra & infraspinous fossae, subscapular fossa, acromion & coracoid process** Suprascapular notch, supra & infraglenoid tubercles, deltoid tubercle, inf. angle, med. & lat. border 2. **Deltoid – acromion & spine of scapula** ***Supraspinatus – supraspinous fossa** ***Infraspinatus – infraspinous fossa** *Teres minor – middle part lat. border Teres major – post. surface inf. angle ***Subscapularis – subscapular fossa**	Bold to pass Bold to pass
Stem: Ketamine is given for analgesia. Moving onto Pharmacology.			
Question 2 Ketamine pharmacokinetics and Pharmacodynamics **Subject:** Pharm: LOA: 1	a. Describe the pharmacokinetics of ketamine *Prompt: Why does it wear off quickly?* b. What are the CNS effects of ketamine? c. What are the cardiac and respiratory effects of ketamine?	1. **Highly lipid soluble, hence rapid onset. Effect terminated by redistribution** to inactive tissue sites. Low protein binding (12%). **Metabolised in liver** (N-demethylation by cyt. P450) -> norketamine (1/3 – 1/5 potency of ketamine) -> hydroxylated & conjugated into H2O sol. Inactive metabolites -> **excreted in urine** **Dissociative anaesthetic,** profound analgesic, stimulates symp. n.s., incr. cerebral bl. flow (cerebral v/d, raised ICP), nystagmus, partial amnesia, anticonvulsant **CVS** - Incr. BP, HR & CO, lesser direct myocardial depressant **Resp –** bronchodilation, minimal resp. depression, maintain airway reflexes, bronchorrhoea, laryngospasm (paeds), hypersalivation	Bold to pass Bold + 1 to pass 1 CVS and 2 Resp effects to pass
Stem: Biochemistry is performed. Moving on to the Clinical building block.			
Question 3 Renal impairment **Subject:** CBB	Describe and interpret this biochemistry result Prompt if needed: *"What does the elevated creatinine indicate?"*	All results within reference range aside from **elevated Creatinine.** Indicates **renal impairment**. Potassium and HCO_3 normal, indicating absence of acute kidney injury.	Bold to pass

Stem: Moving onto physiology.			
Question 4 Glomerular filtration **Subject:** Physiology LOA: 1	a. What is the definition of glomerular filtration rate (GFR)? b. What is the GFR in a normal average adult? c. What general factors within the glomerulus affect GFR? *Prompt: Other than hormones...*	1. Overall rate of fluid filtered through the renal corpuscles, into the renal tubules. **125ml/minute.** (Accept **100 to 150 ml/min)** Note it is distinct from renal blood flow (approx. 1250ml/min) or renal plasma flow (approx. 625ml/min). GFR/renal plasma flow is the filtration fraction (which is 10 to 20%). A. **Overall surface area of capillary bed within the glomerulus.** This is determined by glomerular mesangial cells (like smooth muscle cells). Contraction of these cells reduce surface area, and hence GFR. Conversely, relaxation of these cells increases GFR. B. **Permeability of glomerular capillaries.** C. **Hydrostatic pressure within glomerulus.** Increased by afferent arteriolar dilatation, efferent arteriolar constriction, increased renal blood flow. Systemic BP may be directly proportional if it's outside the range of auto-regulation. D. **Hydrostatic pressure within Bowman's capsule.** If increased, eg. ureteric obstruction, will reduce GFR. E. **Oncotic pressure within glomerulus.** If increased, will reduce GFR. F. **Number of functioning renal corpuscles.** Loss of corpuscles reduces GFR. This may result from many causes eg. atrophy, parenchymal disease, acute kidney injury, nephrectomy.	Concept to pass Bold 4 out of 6 bold
Stem: He has an anaphylactic reaction to the analgesia given. Moving onto Pathology.			
Question 5 Type 1 Hypersensitivity **Subject:** Pathology. LOA: 1	a. Outline the immunological mechanisms leading to anaphylaxis. *Prompt: Start with the initial exposure to a substance.* *Prompt- at tissue level, what are the end organ effects of the anaphylactic response?* b. What are the clinical manifestations of systemic anaphylaxis?	Exposure to **antigen** - presentation of antigen to **T helper cells** by dendritic cells - T helper cells differentiate into T_H2 cells - these release cytokines that act on **B cells to produce IgE** - **IgE** binds to **mast cells** - **repeat exposure to the antigen** - binds to and cross-links IgE antibodies on surface of mast cells - release of **vasoactive amines** and **lipid mediators** (immediate reaction) and cytokines (late phase reaction) from mast cells - **action of mediators on end organs** results in clinical manifestations of anaphylaxis: vasodilation, vascular leakage, smooth muscle spasm). Skin, Respiratory (Upper and Lower), GIT, Cardiovascular, Neurological	Antigen, IgE, mast cells + 3 other bold to pass Concept of exposure, antigen processing by cell lines, mast cell priming and release of mediators to pass. Two systems described to pass

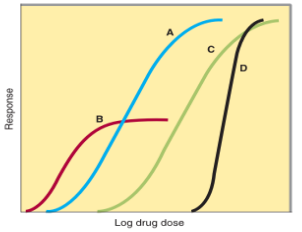
ACEM PRIMARY VIVA D **Friday PM Session 4** ***Candidate Number:*** **AGREED MARK:**

Stem: A 3yo boy presents with a fever and a rash. Starting with a Clinical Building Block.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Rash **Clinical Building Block**	Please describe the photo. *Prompt if needed: what would be the differential diagnosis?*	Macular **widespread** (face, scalp, upper limbs and torso) rash Most marked/**erythematous** on cheeks, confluent in areas ?lip involvement also right forearm lesion?papule ?vesicle ?petechial Well nourished Difficult to comment on hydration ?dry lips **Likely viral exanthema, allergic reaction,** Stevens-Johnson syndrome, meningococcemia, erythema multiforme	Bold to pass + 2 descriptors One of two bold
Stem: His temperature is 38°C. Moving to Physiology.			
Question 2 Thermoregulation **Subject:** Phys LOA: 1	a. How is heat lost from the body? b. How is fever produced in the body? *Prompt: Outline the pathophysiological mechanism of fever.*	**Radiation and conduction** (70%), **vaporisation of sweat** (27%), respiration (2%), urination and defaecation (1%). Endotoxins, inflammation and other **pyrogens** act on monocytes, macrophages and Kupffer cells to **produce cytokines** (eg. interleukins and TNF). Cytokines act on circumventricular organs (eg. OVLT) which activate the pre-optic area of the **hypothalamus**. Local release of prostaglandins **raises the temperature set point**.	Bold to pass 3 of 4 bold points

Stem: He is noted to have a barking cough. Moving on to Anatomy.			
Question 3 Upper airway **Subject:** Anat *(Start with model with L half mandible and tongue removed; then split in half. Take model back before part b))* LOA: 2 Model of airway (model FS5/1)	a. Using the model, demonstrate the main features of the larynx *Prompt with knitting needle if needed* b. What is the motor nerve supply of the larynx? *Prompt: Does the recurrent laryngeal n. supply ALL the intrinsic mm.?*	**20 thyroid cartilage, cricoid cartilage**, arytenoid cartilages, **epiglottic cartilage/epiglottis**, epiglottic vallecula, cuneiform and corniculate cartilages, **23 crico-thyroid membrane, 59 vocal cords** Inferior laryngeal nerve is a continuation of the **recurrent laryngeal nerve** and **supplies all intrinsic muscles except one:** cricothyroid (which is supplied by the superior laryngeal n.) Larynx innervated by superior and inferior laryngeal branches of the vagus nerves (CN X).	Bold to pass Bold to pass
Stem: The child is diagnosed with croup. Moving onto Pathology.			
Question 4 Croup and Acute inflammation **Subject:** Path LOA: 1	a. What is croup? *Prompt: What is the effect on the airway* Prompt: *What viral agents cause croup*? b. Describe the main characteristics of acute inflammation. *Prompt: Describe the general characteristics of acute inflammation.*	1. **Acute laryngotracheobronchitis** in children: inflammatory/spasmodic **narrowing of the airway** produces barking cough, inspiratory stridor. Causes are predominantly viral, esp **Parainfluenza virus.** RSV, adenovirus and influenza are others. 2. Main characteristics of Acute Inflammation: A. Relatively rapid onset. B. Alterations in vascular calibre that **increase blood flow.** C. **Leaky microvasculature**: Structural changes in microvasculature that permit plasma proteins and leucocytes to leave circulation. This leads to oedema. D. **Emigration of leucocytes (esp neutrophils),** their accumulation at site of infection, and activation to eliminate offending agent. E. Duration of hours to days.	Bold Bold

Stem: He is treated with Dexamethasone. Moving on to Pharmacology.			
Question 5 Dexamethasone (Dose/PD) **Subject:** Pharm LOA: 1	a. What is the usual dose of dexamethasone for treatment of croup?	0.15-0.60mg/kg PO, single dose	dose range to pass
	b. How can dexamethasone be administered? *Prompt: Any other routes?*	Oral, IV, IM, topical	3/4 to pass
	c. How does the anti-inflammatory effect of dexamethasone compare to hydrocortisone? *Prompt: How does the duration of action differ?*	Dexamethasone is 30 times more **potent** and **longer acting**	Bold to pass
	d. Describe the anti-inflammatory and immunosuppressive effects of glucocorticoids.	Effects on concentration, distribution and function of peripheral leucocytes Suppression of inflammatory mediators (cytokines, chemokines) Inhibit function of macrophages and antigen presenting cells Inhibit PLA2 -> decrease PG/LT/PAF	2/4 to pass

Stem: You review a 42-year-old man with your intern who has facial pain. We will start with the clinical building block.			
Question 1 Photo of face **Subject**: CBB	a) Describe the image. b) List possible differential diagnoses *Prompt:* any other etiologies (non infectious)?	a) **Swollen and erythematous right side** face near **angle of mandible.** b) Trauma: soft tissue injury, mandible #, dental injury Infection: cellulitis, sialadenitis (parotid, submandibular), lymphadenitis, skin abscess, dental abscess Tumour: lymphoma, LN met, salivary gland	**Bold** to pass 1 infectious cause plus 2 others (1 non infectious)
Stem: Moving onto Anatomy.			
Question 2 Mandible (bone) **Subject:** Anatomy LOA: 1	a) Demonstrate the features of this bone. b) Which nerve passes within this bone and demonstrate the entry and exit points. c) What nerve does the inferior alveolar nerve arise from?	a) **Body, angle, ramus, condyle** (includes head & neck), **coronoid** process, pterygoid fossa, mandibular notch, lingula, mylohyoid groove, submandibular fossa, sublingual fossa, symphysis, mental protuberance, alveolar processes, mental tubercles, digastric fossa, mental spines b) **Inferior alveolar nerve** enters mandibular foramen, (within mandible supplies mandibular teeth), and exits mental foramen as mental nerve (supplies skin + mucous membranes lower lip, skin of chin. c) Mandibular nerve (V3 – 3rd branch of trigeminal n)	**4/5 Bold** plus 1 other **Bold** to pass
Stem: Moving onto Pathology. The most likely diagnosis is acute parotitis.			
Question 3 Inflammation **Subject**: Path LOA: 1	a) Describe the vascular changes in acute inflammation b) What are the mechanisms responsible for increased vascular permeability in inflammation? c) Describe the role of complement in inflammation	a) • **Vasodilatation**: opening of arterioles and capillary beds mediated by histamine and nitric oxide (NO) leading to increased blood flow • **Increased vascular permeability** • **Stasis:** due to plasma protein permeability and increased viscosity b) • Endothelial contraction / retraction: gaps in venules due to histamine, bradykinin and leukotrienes , causing immediate transient response (lasting 15 - 30 mins). Other stimuli (eg UV radiation, burns, some bact toxins) result delayed prolonged leakage (delay 2-12 hrs and may last hrs to days) • Direct vascular endothelial injury (eg. in severe burns, microbial toxin injury), rapid onset but may last days • Leukocyte mediated leakage: in venules and pulm capillaries, long lasting (hrs) • Trancytosis : increased transport of fluid and protein thru endothelial cells, VEGF increases number +/- size transport channels c) >20 proteins (incl C1-9) – once activated, trigger cascade • Recruitment and activation of lymphocytes (C3a, C5a) – inflammation trigger • Formation Membrane Attack Complex (MAC) – causing cell lysis • Phagocytosis (C3b) – Phagocyte recognizes C3b bound to microbe.	**all 3 bold** to pass 2 out of 4 to pass 2 out of 3 to pass

Stem: Moving onto Physiology. He also reports pre-existing blurred vision.			
Question 4 Visual pathways **Subject:** Phys LOA: 1	a) Describe the neural connections of the visual pathways.	a) **Retina, optic nerve, optic chiasm, optic tract**, lateral geniculate body (thalamus), geniculocalcarine tract, primary visual cortex **(occipital lobe). At optic chiasm, nasal fibres decussate to the contralateral side.** Other connections**:** - Optic tract (via superior colliculus) to pretectal midbrain, then to Edinger-Westphal nuclie in oculomotor nerve (pupillary reflexes, eye movement) - Frontal cortex (refined eye movement - vergence, near point response) - Retinal ganglion cells to suprachiasmatic nucleus hypothalamus (endocrine & circadian responses to day/night cycle)	**Bold** to pass
	b) Why is the fovea important for visual acuity?	b) Point where **VA greatest**; fovea is the centre of the macula, a thinned out rod-free portion of the retina where the **cones are densely packed** & each synapses on a single bipolar cell, which, in turn, synapses on a single ganglion cell, providing a direct pathway to brain	One of **bold plus one** other to pass
	c) What ocular factors influence visual acuity?	c) **Optical factors**: state of the image-forming mechanisms eg cataracts, keratitis, astigmatism, myopia, hyperopia **Retinal factors** eg the state of the cones, retinopathies, optic neuritis **Stimulus factors** eg illumination; brightness of the stimulus; contrast between stimulus and background; length time exposed to stimulus)	**3 factors**
Stem: Moving onto Pharmacology. He is given morphine for his pain.			
Question 5 Potency and efficacy **Subject:** Pharm LOA: 1	a) Can you define potency? *Prompt:* *What does ED50 or EC50 refer to?*	a) The **amount of drug required to produce an effect** of certain intensity. Refers to the concentration (EC50) or dose (ED50) of a drug required to produce 50% of that drug's maximal effect. Dependent on affinity of drug for receptor and number of receptors available.	Refers to amount/conc required for a given effect. **Bold** to pass
	b) Can you define efficacy?	b) **Maximal effect a drug can produce** when all receptors are occupied, irrespective of conc required to produce that response (or irrespective of dose). Determined by the drug's mode of interactions with receptors or by characteristics of the receptor-effector system involved.	**Bold** to pass
	c) Show the difference between efficacy and potency by drawing graded dose response curves. *Optional if time allows* Compare the potency of morphine to fentanyl.	c) A and B have similar potency. A&B are more potent than C which is more potent than D for mild to moderate responses/effects. A, C & D have similar efficacy and greater efficacy than B. B is a partial agonist (producing less than full response despite full receptor occupancy) Fentanyl 100x more potent. 0.1mg fentanyl = 10mg morphine	Draw graph, & explain, correct axes  **FIGURE 2–15** Graded dose-response curves for four drugs, illustrating different pharmacologic potencies and different maximal efficacies. (See text.)

Stem: A 70-year-old diabetic man is being treated for sepsis from an ulcer on his foot. We will start with Anatomy.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Foot (bone) **Subject** Anat LOA: 1	a) Identify the bones of the foot b) Demonstrate the attachments of the medial collateral ligament (deltoid ligament) c) Describe the structures running immediately posterior to the medial malleolus	a) Talus, Calcaneus, Cuboid, Navicular, Cuneiforms (medial, intermediate, lateral), Metatarsals, Phalanges b) Posterior tibio-talar (to medial tubercle of talus) Tibio-calcaneal (to calcaneal shelf = sustenaculum tali) Tibio-navicular (to tuberosity of navicular) Anterior tibio talar c) Tibialis post tendon, flex digit long tend, **post tibial art** and vn, posterior tibial nv, flexor hall long tendon	At least **6 out of 7** **2/4** to pass **2 tendons and post tibial art** to pass
Stem: Some blood tests are taken upon arrival			
Question 2 Blood tests **Subject** CBB	a) Describe the abnormalities b) What could cause these abnormalities in this patient?	a) Slightly low/normal sodium (or Extra: corrected Na 137), **Hyperkalaemia,** low bicarb (met acidosis) , **Renal failure** (likely intra-renal with chronic component)**, hyperglycaemia** b) Sepsis, diabetic nephropathy**,** dehydration, drug toxicity, DKA	**Bold** **At least two**
Stem: Moving onto Physiology. His urinalysis shows a pH of 6.0.			
Question 3 Renal - H+ handling **Subject**: Phys LOA: 1	a) Where does the acidification of the urine occur? *(Prompt : where is hydrogen secreted in the kidneys?)* b) How is H+ secreted in each of those areas? c) What is the limiting pH of urine and where is it reached?	a) Proximal and distal tubules, and collecting ducts b) **Proximal tubule – Na-H exchange transporter** (one Na and one HCO_3 reabsorbed for each H secreted) Distal tubule and collecting duct – the secretion of H+ is independent of Na. ATP driven proton pump - stimulated by aldosterone. Also H-K ATPase pump, and anion exchanger 1. c) **The limiting pH is 4.5** (1000x concentration in plasma). It is the maximal H+ gradient that can be achieved in the tubules. It occurs in the **collecting duct**. Possible due to buffers (bicarb, dibasic phosphate and ammonia)	**2 of 3** **Na-H and 1** mechanism in DCT/ CT **Bold**

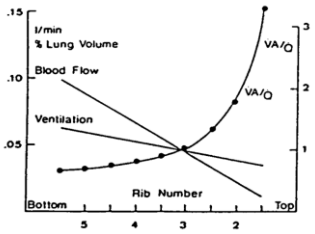
Stem: Moving onto Pharmacology**.** He is treated with Gentamicin.			
Question 4 Gentamicin **Subject:** Pharm LOA: 1	a) What class of antibiotic is Gentamicin? b) What is its mechanism of action? c) Please describe the pharmacokinetics of Gentamicin? d) What are the advantages of single daily dosing regimen for Gentamicin e) What are its adverse effects?	a) **Aminoglycoside** b) It acts by **binding to the 30S ribosomal proteins -** inhibiting protein synthesis in the bacteria. Bactericidal – gram neg. Concentration dependent killing. Post antibiotic effect. c) Route: parenteral (IV or IM), inhalation, topical Distrib: Small Vd because < 10% protein bound Metab: not metabolised. Elim: **renal dependent**. Glomerular filtration. T1/2 = 2-3 hours, typically given once daily d) **↓toxicity time** and **concentration dependent killing –** once daily results in less time above toxic threshold concentration**.** OP therapy, Cost effective e) **Nephrotoxic** **Ototoxic** Prolongs NM blockade	**Bold** **Bold** **Bold plus 2** others **Bold** **Bold**
Stem: Moving onto Pathology. He has confirmed Gram Negative sepsis.			
Question 5 Gram-negative sepsis **Subject:** Path LOA: 1	a) What are the mechanisms of Gram negative sepsis? *Prompt :what other immune components are involved?"* *Prompt : what other blood components are involved?"* b) What are the potential outcomes of septic shock?	a) Combination of **direct microbial injury** and activation of host inflammatory responses e.g. by **endotoxins** (lipid A, O Ag) 1. Inflammatory mediator release TNF, IL (1, 6,8,10), PGs, NO, PAF, reactive O2 species, PAI-1 (Plasminogen activator inhib 1) 2. Activation innate cells of immune system- neutrophils. macrophages and monocytes 3. Humoral interaction to activate complement and coagulation pathways 4. Direct endothelial injury and activation 5. Metabolic abnormalities (insulin resistance and hyperglycaemia, glucocorticoid excess/def) 6. Immune suppression (activation counter-regulatory mechanisms with anti-inflamm mediators, lymphocyte apoptosis, hyperglycaemia inhibits neutrophils) End organ and systemic dysfunction, incl a) Cardiomyopathy b) Hypotension c) ARDS d) DIC e) Renal failure f) MSOF g) Death	**Bold** **2 of 6** **3 of 7**

ACEM PRIMARY VIVA A **Friday Morning** ***Candidate Number:*** **AGREED MARK:**

Stem: A 55-year-old man falls while mountain climbing. A CT brain is performed.			
Question 1 CT Brain **Subject:** CBB	Describe his CT brain.	**Transverse/axial CT brain slice** (level of third ventricle) **Right** acute **extradural haematoma (frontal region) –** lenticular shape No midline shift, raised intracranial pressure	**Bold** to pass
Stem: Moving on to Anatomy. X-rays reveal a fractured humerus.			
Question 2 Humerus (Bone) **Subject**: Anat LOA: 1	a) Describe the main visible features of this bone.	a) **Correct side** Proximal: **Head,** Anatomical & **surgical neck, Greater tuberosity/**Lesser tuberosity (major attachment areas for rotator cuff/deltoid), intertubercular groove (long head biceps), shaft**,** radial groove on shaft (radial nv, profunda brachii art), deltoid tuberosity Distal: **Lateral** (ext origin) **and medial** (flex origin) **epicondyle** (ulna nerve inf), coronoid, radial and olecranon fossa, **capitulum, trochlea**	**Bold plus 2 others** to pass
	b) How does this bone articulate with the scapula?	b) Glenohumeral joint • Ball and socket • Articular surface of humeral head in contact with shallow surface of glenoid cavity (deepened by glenoid labrum).	Must explain humeral head in contact with glenoid cavity
	c) What anatomical features contribute to the stability of the shoulder joint?	c) **Joint capsule** with fusion of the tendons of the scapular/rotator cuff muscles (pull humerus into glenoid) Ligamentous: **Glenohumeral** and **coracohumeral ligaments** **Coracoacromial arch** superiorly created by coracoacromial ligament **Deepening of glenoid cavity by glenoid labrum** **Tendons** of biceps and triceps (long head)	**3/6 bold** to pass
Stem: Moving on to Pharmacology. He is on clopidogrel.			
Question 3 Clopidogrel **Subject:** Pharm LOA: 1	a) What is the mechanism of action of clopidogrel?	a) Anti-platelet effect by **inhibiting ADP pathway** (irreversible blockade ADP receptor on platelet for life of platelet).	**Bold** to pass
	b) Describe the pharmacokinetics of clopidogrel.	b) A **prodrug**, metabolised to a pharmacologically active metabolite and inactive metabolites. **Activated in liver by cyto P450** (including CYP2C19). 80% platelet activity inhibited within 5 hrs oral dose. **Elimination t ½** ~ 0.5 to 1.0 h. **Effects last life of platelet** Following an oral dose: 50% excreted in the urine and 46% in the faeces in next five days. **Loading dose 300 - 600mg** or **75mg daily**	**2/6 Bold** to pass
	c) What are the adverse effects?	c) - **bleeding,** rash**,** (rarely **-** pancytopaenia & TTP) - diarrhoea, abdominal pain, reflux, gastric ulcers - sensation of tingling, numbness	**Bold** plus 2 others

Stem: Moving on to Physiology. His fall occurred at high altitude.			
Question 4 High Altitude **Subject:** Phys LOA: 1	a) What are the initial physiological responses at high altitude?	1. Hyperventilation : decreases CO_2 > O2 2. Alkalosis : limited by movement of bicarbonate from CNS (1-2 days) and renal excretion HCO_3 3. Increased 2,3-DPG – R shift O_2-Hb dissociation curve (early), then left shift at higher altitudes due alkalosis 4. Alveolar hypoxia induces pulm vasoconstriction, then pulmonary HTN 5. Decreased work of breathing	3/5 to pass
	b) What are the longer-term physiologic effects of altitude exposure?	• Polycythaemia (incr EPO) • Incr viscosity of blood • Increased 0_2 carriage • Pulm HTN resulting in RVH • More capillaries • Increased oxidative enzymes. • Increased mitochondria	3 to pass
Stem: He becomes shocked. Moving onto Pathology.			
Question 5 Haemorrhagic Shock **Subject**: Path LOA: 1	a) Define shock	**Tissue hypoperfusion due either** 1 Reduced Cardiac Output, or 2 Reduced effective Blood volume	**Bold** to pass **plus 1 or 2**.
	b) Describe the stages of haemorrhagic shock *Prompt : describe what happens during each stage?*	▪ Non-progressive – reflex compensatory mechanisms maintain vital organ perfusion ▪ Progressive – tissue hypo-perfusion and onset metabolic disturbances (lactic acidosis) ▪ Irreversible – non reversible tissue and cellular injury, MOF	All 3 stages to pass plus some detail for each
	c) Describe initial clinical presentation of shock	Narrowed pulse pressure / ↑CPR time / Tachycardia / Hypotension / Tachypnoea /Cool Clammy Skin / Cyanotic Skin / Oliguria / Altered mental state	3/5 to pass
	d) What other types of shock are there – with an example of each?	Distributive (septic, anaphylactic), Obstructive (PE, PTX, tamponade), Cardiogenic (MI, cardiomyopathy, arrhythmia), Neurogenic (spinal trauma), Dissociative (poisoning), Hypovolaemic (burns, GI losses)	3 to pass

ACEM PRIMARY VIVA A **Thursday Morning** ***Candidate Number:*** **AGREED MARK:**

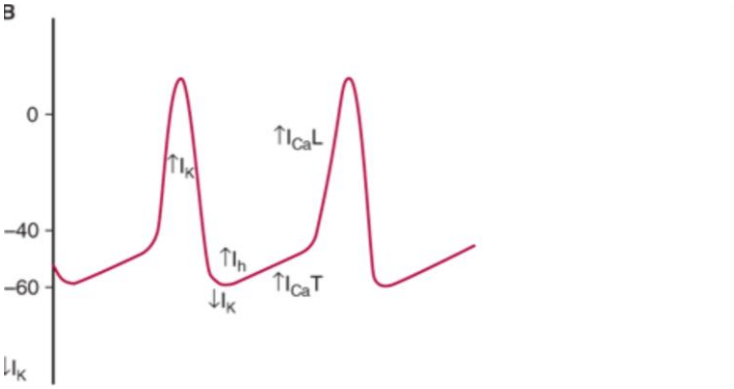
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: A 60 year-old man presents to the Emergency Department with shortness of breath. An arterial blood gas is performed.			
Question 1 ABG **Subject:** CBB ABG	a) Please describe the abnormalities b) What conditions could cause this result in this patient?	a) • Alkalaemia • CO_2 low, thus primary **respiratory alkalosis** • Low PaO_2 and SaO_2 – profound **hypoxaemia** • Raised A-a gradient • Conclusion: Hypoxia leading to hyperventilation and respiratory alkalosis b) • Any NON central causes (infection, asthma, PE, pulm oedema etc)	Bold to pass Two causes to pass
Stem: Moving on to Physiology.			
Question 2 V/Q inequality **Subject**: Physiology LOA: 1	a) Describe the normal relationship between ventilation and perfusion in an upright lung. *Prompt 1: How does gravity affect the ventilation & perfusion in the lung?* b) What conditions can increase V/Q mismatch? c) Which tests can be done in clinical practice to demonstrate a V/Q mismatch? *Prompt 3: Is there a calculation we can perform on the ABG we had earlier?*	a) Pulmonary circulation is affected by gravity. a. **Apex**: Less blood flow, larger alveoli, slightly less ventilation; **ventilation > perfusion**, **high V/Q ratio**. b. Middle: ventilation = perfusion, V/Q = 1 c. **Base**: More blood flow, smaller alveoli, more ventilation; **perfusion > ventilation**, **low V/Q ratio**. b) **Pulmonary embolism** (high V/Q ratio), pulmonary oedema, pneumonia, emphysema (low V/Q ratio). c) **A-a gradient** (also V/Q scan, CTPA)	Candidate may draw graphs in West, Chapter 5. Bold plus concept  b)Bold plus 1 to pass c) Bold to pass
Stem: Moving on to Pathology. You suspect this patient has pulmonary emboli.			
Question 3 Pulmonary embolism **Subject:** Pathology LOA: 1	a) Describe the pathogenesis of thrombotic pulmonary embolism (PE). *Prompt: Where do PEs originate?* *Prompt: Where do they lodge?*	a) PEs originate from **deep vein thrombosis**. (~95% from lower limb). Fragmented thrombi from DVTs are carried through the venous system and into the right side of the heart before lodging in the **pulmonary arterial vasculature:** main pulmonary artery, pulmonary artery bifurcation or smaller branching arteries.	Bold to pass

Robbins and Cottran P 127 to 129	b) What are the symptoms and signs of pulmonary embolism? c) List 2 other types of emboli.	b) Clinical manifestations depend on size and location of the thrombus in the pulmonary vasculature. -Most PEs (60-80%) are small and produce no symptoms nor signs A. Positive Symptoms - Chest pain - Dyspnoea - Collapse/syncope B. Positive Signs - Hypoxaemia/ tachypnoea - Tachycardia - Hypotension - Shock / sudden death - Acute right heart failure c). - Fat (bone marrow) - Air/other gas - Amniotic fluid - Foreign body (eg fragment of catheter)	5 of list to pass 2 to pass
Stem: Moving on to Pharmacology. The patient becomes haemodynamically unstable and he is to be thrombolysed.			
Question 4 Thrombolytics **Subject:** Pharmacology LOA: 1	a) What are the classes of thrombolytic agents? Prompt: What are the 2 classes? b) What is the mechanism of action of tissue plasminogen activator (t-PA)? c) What are the adverse effects of thrombolytic agents?	a) **t-PA** (tissue plasminogen activator e.g. alteplase, tenecteplase, reteplase) and **streptokinase** (a protein synthesized by streptococci). b) t-PA is an enzyme that directly converts **plasminogen to plasmin**. Plasmin is the major fibrinolytic enzyme. c) **Bleeding** – cerebral haemorrhage, gastrointestinal, previous surgery/wounds Allergy (especially streptokinase)	Bold to pass. Candidates may draw diagram (Katzung, 11th Ed, fig 34-2, p280). Bold to pass. Bold to pass.
Stem: Moving on to Anatomy. It is decided to insert an internal jugular central line. Here is a photo of the thoracic inlet and mediastinum.			
Question 5 Thoracic inlet and mediastinum (photo, McMinn's 7th Ed page 206) **Subject**: Anatomy LOA: 2 (root of neck)	a) Please identify its main features (demonstrate and/or identify by number) b) What structures do you need to avoid when placing an internal jugular central line?	a) Thoracic inlet structures (numbered): 2 Cricoid cartilage 3 Ascending cervical artery 4 Brachiocephalic trunk 7 Inferior thyroid veins 8 **Internal jugular vein** 11, 12 **thyroid gland** (isthmus, lateral lobe) 13 Left brachiocephalic vein 14 Left **common carotid** artery 17 Phrenic nerve 18 Right brachiocephalic vein 19 Right **common carotid** artery 20 Recur laryngeal n 21 Right subclavian artery 22 **Right vagus nerve** 23 Scalenus anterior 24 Subclavian vein; 26 Superior vena cava 31 Thymus; 32 Thyrocervical trunk; 33 **Trachea** 35 Upper trunk of brachial plexus b) **Common carotid artery**, apex of lung, vagus nerve, oesophagus	Bold to pass plus 3 others Bold to pass

Stem: A 73 year old man presents in acute urinary retention. We will start with Anatomy			
TOPIC	**QUESTIONS**	**KNOWLEDGE (essential in bold)**	**NOTES**
Question 1 Genitourinary tract (Model Male Pelvis **Subject:** Anatomy LOA: 2	a) Identify the structures on this model.	a) **Bladder,** coccyx, corpus cavernosum, deep dorsal vein of penis, ductus deferens, **prostate,** prostatic urethra, pubic symphysis, rectosigmoid junction, rectovesical pouch, **testis,** epididymis, tunica albuginea, tunica vaginalis, penile urethra	Bold plus 3 others to pass
	b) Describe the parts of the male urethra	b) - intramural part (pre-prostatic) – surrounded by internal urethral sphincter (within bladder neck) **prostatic part** (widest part, receives prostatic and ejaculatory ducts) - **intermediate (membranous) part** – surrounded by external urethral sphincter (narrowest and least distensible part except for external urethral orifice) - **spongy (penile) part** with intrabulbar fossa proximally and navicular fossa distally	Bold
	c) What is the innervation of the urethra? (bonus question)	c) prostatic nerve plexus (arising from inferior hypogastric plexus) to first 3 parts above - dorsal n. of the penis (from pudendal n.) to spongy part	No pass criteria as bonus question
Stem: Moving on to Pathology. His urinary retention is caused by benign prostatic hyperplasia.			
Question 2 Hyperplasia **Subject**: Pathology Robbins 9th edition pages 35-36 LOA: 1	a) What is hyperplasia?	a) **↑number of cells** in organ/tissue → usually ↑mass	Bold
	b) What are the different types of hyperplasia and give examples (Prompt: Types other than BPH)	b) **Physiologic** a. Hormonal: female breast at puberty & preg b. Compensatory: post-partial hepatectomy, skeletal muscle with increased workload **Pathologic** a. Excess hormones: BPH, DUB b. Viral infection – papillomavirus	Bold plus one example in each category to pass
	c) Apart from urinary retention, what are the clinical features of BPH?	c) Frequency, nocturia, difficulty in starting and stopping stream, dribbling, dysuria, ↑risk of infections	Any 2.

Stem: Moving on to Pharmacology. He is on Prazosin.			
Question 3 prazosin/alpha blockers **Subject:** Pharm LOA: 1 Katzung and Trevor 13th ed, chap 11.	a) What is the mechanism of action of prazosin? Prompt: Which receptors does prazosin bind to? Prompt: How does prazosin reduce blood pressure? b) List 3 other effects of prazosin. Prompt: What are the side effects of prazosin?	a) Prazosin selectively **blocks alpha-1 receptors** in arterioles and venules**.** Reduces arterial pressure by dilating both resistance and capacitance vessels. Alpha$_1$-receptor selectivity allows noradrenaline to exert unopposed negative feedback (mediated by presynaptic α_2 receptors) on its own release. b) Postural hypotension /dizziness / syncope - Reflex tachycardia / palpitations - Headache - Lassitude - Reduced prostate smooth muscle tone, thus alleviating prostatic urinary obstruction - Positive serum antinuclear factor - ↓LDL & TGs and ↑HD	Bold 3 to pass
Stem: Here are his blood results.			
Question 4 EUC/renal failure **Subject:** CBB	a) What are the abnormalities and what is your interpretation? b) What are the broad categories of renal failure? Please provide an example of each c) Which is most likely in this man?	a)**AKI (or ARF), hyperkalaemia** b)**Pre-renal, renal, post-renal** c)**Post-renal**	Bold
Stem: Moving on to Physiology of the kidney.			
Question 5 Renal regulation sodium **Subject**: Physiology LOA 1	a) Where does Na reabsorption occur in the nephron? b) How is Na transported from the tubular cell into the interstitium? c) Following high Na intake, what mechanisms act to enhance Na excretion? *Prompt: What mechanisms reduce Na reabsorption?* *Prompt: Can you describe any mechanisms mediated outside the kidney?*	a) **Primarily** (60%) **PCT** by Na-H exchange but also a range of co-transport (gluc, AA, lactate) - 30% thick asc limb of LoH (Na –K-2Cl co-transporter, Na-H exchange) - Nil at thin part of LoH - 7% DCT (NaCl co-transporter) - 3% collecting ducts through Na channels (ENaC) b) **Na/K ATPase active transport.** (3Na/2K) across basolateral membrane predominantly into the lateral intercellular spaces. c) A slight increase in ECF occurs triggering various mechanisms: - stretch receptors in RA and pulm veins → inhibits sympathetic outflow to kidneys → decreased Na reabs - small increase in arterial pressure → pressure natriuresis - suppression AT-II formation - reduced aldosterone secretion secondary to reduced AT-II formation - stimulation of ANP	2 out of 5 described correctly including bold to pass Bold 2 mechanisms

Stem: A 75-year-old man presents following a transient ischaemic attack (TIA). We will start with Anatomy			
TOPIC	**QUESTIONS**	**KNOWLEDGE (essential in bold)**	**NOTES**
Question 1 Carotid artery (McMinn's Photo of Anterior triangle of Neck, 7th edition, page 29) **Subject:** Anatomy LOA: 1	a) What are the boundaries of the anterior triangle of the neck b) Identify the major Neurovascular structures on this photo. c) Discuss the anatomy of the right common carotid artery.	**a) Anterior border of sternocleidomastoid, lower border of the mandible and the midline** **b) Common Carotid artery (8)** **External Carotid artery (11)** **Internal jugular vein (22)** Vagus nerve (63) **c) Right Common Carotid**: **begins at the bifurcation of the brachiocephalic trunk** behind the sternoclavicular joint into common carotid and subclavian arteries. **In the neck it lies within** the medial part of **the carotid sheath**, internal jugular vein lateral to it and the Vagus nerve deeply placed between the two vessels. The **common carotid bifurcates** at the level of the upper border of the lamina of the thyroid cartilage (upper border of C4 vertebrae **into the external and internal carotid arteries**.	All correct Bold to pass 3 out of 5 bold to pass
Stem: An ECG was obtained			
Question 2 ECG – AF **Subject**: CBB	a) Describe and interpret the ECG b) What other types of narrow complex tachycardia are there?	a) Narrow complex tachycardia Rhythm**: irregularly irregular – atrial fibrillation** **Rate 135/min (125-145)** No P waves b) PSVT, Re-entrant pathway (e.g. WPW), atrial flutter, sinus tachycardia	Bold to pass 2 to pass

Stem: Moving on to Physiology			
Question 3 Conducting system **Subject:** Physiology LOA: 1	a) Describe the normal cardiac conduction pathway b) This is a normal ECG complex. What does each wave of the ECG represent? c) Draw and describe the action potential of a cardiac pacemaker cell	**a) SA node** (pacemaker) **Atria** (3 internodal pathways) **AV node; Bundle of His; Right and left bundle branch** **Anterior and posterior fascicles on left** **Purkinje fibres** **Ventricular muscle: l**eft side of IV septum first, spread down septum to apex, Up AV grooves, spread from endocardial to epicardial surfaces **- P wave: Atrial depolarisation** **- QRS complex: Ventricular depolarisation** **- T wave: Ventricular repolarisation** NB: atrial repolarisation is not represented because it is buried in the QRS complex.  arman, Scott Boitano, Heddwen L. Brooks: Ganong's Review of Medical Physiology, 25th Ed. . All rights reserved. 1. **Pre-potential is initially due to a decrease in K^+ efflux**, then completed by Ca^{2+} influx through CaT channels (prepotential). 2. The **action potential is due to influx of Ca^{2+}** via CaL channels. 3. **Repolarisation is due to K^+ efflux**	6 of 8 Bold to pass Prompt: what is happening electrically? Bold Need approx. correct shape of curve and approx. potential values

Stem: Moving on to Pharmacology. Intravenous fluids are commenced.			
Question 4 Intravenous fluids **Subject:** Pharmacology LOA: 1	a) What are the different classes of intravenous fluid, give an example of each. b) How does the electrolyte composition of normal saline differ from Hartmann's? c) What are the complications of crystalloid fluid therapy?	a) 1.Colloid Albumin Dextran Gelatin 2. Crystalloid Isotonic: Normal saline, Hartman's, Plasmalyte Hypertonic Saline: 3% or 7.5% NaCl Hypotonic: 0.45% NaCl, Dextrose (5%, 10%) 3. Blood and blood products b) Normal Saline: Na 154 mmol/L, Cl 154 mmol/L Hartmann's: Na 130mmol/L, Cl 112mmol/L, **K** 5.4mmol/L, **Ca** 1.8 mmol/L, **Lac** 27mmol/L c) Acute pulmonary oedema Hypothermia Dilutional coagulopathy Acidosis Tissue oedema limb and abdominal compartment syndromes Electrolyte abnormalities Extravasation	3 examples and 2 classes Accept 150 to 160 At least 2 of bold plus understanding of lower [Na] 3 to pass

Stem: Moving on to Pathology. His TIA is most likely embolic.			
Question 5 Embolism **Subject**: Pathology LOA: 1	a) What is an embolus?	a) An embolus is a **detached intravascular** solid, liquid or gaseous mass that is **carried by the blood** to a **site distant** from its point of origin.	Bold to pass
	b) Name the different types of emboli Prompt: what can embolise?	b) Thromboembolus Venous: pulmonary Arterial: systemic • Fat embolus • Gas embolus • Amniotic fluid embolus • Air embolus	3 out of 5 to pass
	c) What is systemic thromboembolism?	c) Systemic thromboembolism refers to emboli **in the arterial circulation.**	Bold
	(d) Name the sources of systemic thromboembolism?	d) Most (80%) arise from **intracardiac mural thrombi**, two thirds of which are associated with left ventricular wall infarcts and another quarter with left atrial dilation and fibrillation. The remainder originate from aortic aneurysms, thrombi on ulcerated atherosclerotic plaques or fragmentation of a valvular vegetation, with a small fraction due to paradoxical emboli. 10 to 15% of systemic emboli are of unknown origin.	Bold plus one other
	Bonus question What are the differences in the lodgement of venous and arterial thrombi?	Venous thrombi tend to lodge primarily in one vascular bed (the lung). Arterial thrombi can travel to a wide variety of sites; the point of arrest depends on the source and the relative amount of blood flow that downstream tissues receive. Major sites of arterial embolization are the lower extremities (75%) and the brain (10%), with the intestines, kidneys, spleen and upper extremities involved to a lesser extent.	

ACEM PRIMARY VIVA A **Friday Afternoon 2017.1** ***Candidate Number:*** **AGREED MARK:**

Stem: A 60-year-old woman presents with tachypnoea and chest pain. This is her venous blood gas.			
TOPIC	**QUESTIONS**	**KNOWLEDGE (essential in bold)**	**NOTES**
Question 1 Venous gas **Subject:** Clinical Building block LOA: 1	a) Please describe this blood gas. b) What is the abnormality c) What are possible causes for this abnormality in this patient?	a) **Alkalaemia, Hypocarbia**, Positive base excess b) **Acute respiratory alkalosis** c) Hypoxia induced (Pneumonia, PE, asthma) Increased respiratory drive (CNS, Hypermetabolic states, environmental, drugs)	Bold to pass Bold One from each category
Stem: Moving on to Anatomy. A Chest X-ray shows a large spontaneous pneumothorax. A chest drain is to be inserted.			
Question 2 Normal CXR **Subject**: Anatomy LOA: 1 *Moore 7th ed, page 110 to 120*	a) Demonstrate the parietal pleural reflections on this normal chest x-ray. b) What is the preferred point of insertion of a lateral chest drain? What are the anatomical structures that border this area? c) Which anatomical structures may be injured if it is inserted outside this area.	a) Both sides start at supraclavicular fossa. **Right** - Travels inferomedially behind middle of sternum (anterior median line) to level of 6th costal cartilage, behind xiphoid process. Moves laterally reaching: - midclavicular line at 8th rib - midaxillary line (MAL) at 10th rib - paravertebral line at 12th rib **Left** - Descends in anterior median line to 4th costal cartilage - Then laterally to 6th costal cartilage, creating a notch due to contact with pericardium b) **4th or 5th intercostal space just above the superior border of the rib. Mid-axillary line.** Posterior: Anterior border of the latissimus dorsi Anterior: Lateral border of the pectoralis major muscle Inferior: Line superior to the horizontal level of the nipple Superior: Apex of axilla c) Too far anterior: breast tissue, chest wall muscle Too far posterior: long thoracic nerve Too far inferior: p**erforation of diaphragm and puncture of intra-abdominal organ**	Demonstrate understanding & differences between right and left side Bold 2 of 4 to pass Bold

Stem: Moving onto Pathology. She has bronchiectasis.			
Question 3 Bronchiectasis **Subject:** Pathology LOA: 1	a) What is bronchiectasis? *Prompt: what are the major morphological features?* b) What conditions are associated with the development of bronchiectasis?	a) Bronchiectasis is a disease characterised by **permanent dilation of bronchi** and bronchioles caused by **destruction of the muscle and elastic tissue**, resulting from or associated with chronic necrotizing **infections.** Also scarring and persistent infections b) *Congenital/hereditary* = Cystic fibrosis, immunodeficiency, ciliary dyskinesia, Kartagener's *Post infectious (necrotising pneumonia)* = Staph aureus, Haemophilus; TB, Pseudomonas; adenovirus; HIV, Influenza, fungi, aspergillosis *Bronchial obstruction* = tumour, foreign body, mucous impaction *Other:* Rh Arthritis, SLE, Inflam bowel disease, post transplantation Idiopathic 25 -50%	Bold concepts to pass 4 causes
Stem: Moving onto Physiology. She is tachypnoeic.			
Question 4 Control of ventilation **Subject:** Physiology LOA: 1	a) What parts of the brain control respiration? b) How are chemoreceptors involved in the control of ventilation? c) What other sensors are involved in the control of ventilation?	a) Voluntary - **Cerebral cortex**. Automatic – **Medulla** (Pacemaker cells in pre-Botzinger complex). Pons – pneumotactic centre modifies medulla activity. b) **Chemoreceptors – central and peripheral** Central (ventral surface medulla) - **sensitive to changes in H+.** **CO2** readily penetrates BBB and enters CSF and brain interstitial fluid. Increased CO2 causes increased H+ in CSF, stimulating ventilation. Peripheral (carotid and aortic bodies) – fast response to **decreasing O2**, stimulating bodies. Decreased pH causes carotid response only. Minor response to CO2. c) Pulmonary - Stretch receptors in lungs, muscles, joints, Irritant receptors in airways, J receptors. Irritant receptors in nose and upper airways Baroreceptors (arterial, atrial. Ventricular, pulmonary) – stimulation may cause reflex hypoventilation Pain/temperature – may cause initial apnoea, then hyperventilation Proprioceptors (muscle spindles from intercostals and diaphragm, other muscles/tendons/joints.	Bold to pass Bold and concept for response to CO2/O2. 3 examples

Stem: Moving onto Pharmacology. She is given Ibuprofen to help treat her pain			
Question 5 NSAIDS **Subject**: Pharmacology LOA: 2	a) Describe the pharmacokinetics of ibuprofen. b) Describe the pharmacodynamics of Ibuprofen. c) What are the side effects of NSAIDs?	a) NSAIDs well absorbed, food does not substantially change their bioavailability. Highly protein bound Highly metabolised by liver – Cytochrome P450. b) I**nhibition of prostaglandin biosynthesis.** Additional possible mechanisms of action, including inhibition of chemotaxis, down-regulation of interleukin-1 production, decreased production of free radicals and superoxide, and interference with calcium-mediated intracellular events. **NSAIDs are reversible inhibitors of COX** **Anti-inflammatory, antipyretic and analgesic**. c) Central nervous system: Headaches, tinnitus, and dizziness. CVS: Fluid retention, hypertension, oedema, and rarely, myocardial infarction, and congestive heart failure. **GIT:** Abdominal pain, dyspepsia, nausea, vomiting, ulcers or bleeding. **Renal**: Renal insufficiency, renal failure, hyperkalaemia, and proteinuria. Haematologic: Rare thrombocytopenia, neutropenia, aplastic anaemia. Hepatic: Abnormal liver function tests and rare liver failure. Pulmonary: Asthma. Skin: Rashes, all types, pruritus.	2 to pass Bold to pass At least 4 side effects (must include renal and GIT)

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: A 30 year old man collapsed while hiking. Starting with Physiology.			
Question 1 Counter current mechanism **Subject:** Physiology LOA: 1	How does the Countercurrent mechanism enable the kidney to concentrate urine? Prompt: what processes produce this gradient and where do they occur Candidates may choose to use diagram for demonstartion	Concentrating mechanism depends on maintaining a **gradient of increasing osmolality along medullary pyramids**. **Gradient is produced by Countercurrent multipliers** in the **LOH and maintained by Vasa recta acting as counter current exchangers**. 1) **Water moves out of the thin descending limb** (via aquaporin 1), **2) Active transport of Na and Cl out of thick ascending limb of LOH** 3) Continued inflow of isotonic fluid into the proximal tubule and out of desc tubule ..H2O moves out of collecting duct (into the hypertonic interstitium of the medullary pyramids) under the influence of ADH Vasa recta acts as countercurrent exchangers in the kidney in which NaCI & urea diffuse out of the ascending limb of the vessel & into the descending limb, while water diffuses out of the descending into the ascending limb of the vascular loop. As a result, the solute remains in the medulla pyramid & maintain the interstitial concentration.	Bold to pass. Need to demonstrate understanding.
Stem: Moving on to Pharmacology. The patient is vomiting.			
Question 2 Antiemetics - Ondansetron **Subject:** Pharmacology LOA: 1	a) List anti-emetics with different mechanisms of action	- Serotonin 5HT3 antagonists (e.g. Ondansetron) - Dopamine antagonist: Phenothiazines (Prochlorperazine) and Butyrophenones (droperidol) . Metoclopramide(has peripheral effects) - H1 antihistamines and anticholinergic (e.g. Hyoscine, Diphenhydramine) - Corticosteroids (i.e dexamethasone) - Benzodiazepines (i.e diazepam, lorazepam) - Cannabinoids - Neurokinin Receptor antagonist (e.g. Aprepitant)	Minimum of 3
	b) How is the effect of ondansetron mediated?	b**)** Mostly **peripheral 5HT3/Serotonin receptor blockade** on extrinsic intestinal vagal and spinal afferent nerves. Some effect on Central 5HT3 receptor blockade in vomiting centre and chemoreceptor trigger zone. Anti-emetic action mostly restricted to emesis attributable to vagal stimulation (e.g. postop) and chemotherapy. Less effect for other emetic stimuli (e.g. motion sickness).	Bold to pass
	c) What are possible adverse effects of Ondansetron?	c) Headache, dizziness, constipation, diarrhoea Uncommon – small prolongation of QT.	Minimum of one.

Stem: You suspect he has been bitten by a snake. Blood tests are performed on this patient.			
Question 3 **Subject:** Clinical Building block	These are the blood test results for this patient. Please interpret and provide differential diagnoses.	See separate document of pathology results with Venom induced consumptive **coagulopathy, rhabdomyolysis.** DIC from other cause unlikely given clinical scenario	Recognise coagulopathy and differentials for this. Most likely envenomation.
Stem: His knee is swollen. Moving on to Anatomy.			
Question 4 Knee (x-ray) **Subject:** Anatomy	a) Identify the bony structure shown on this x-ray.	**a) Femur** – condyles (medial and lateral), epicondyles (medial and lateral). Adductor tubercle **Tibia** – condyles (medial and lateral), tibial plateau, intercondylar eminence with intercondylar tubercles (medial and lateral) **Fibular** – head with apex, neck **Patella**	All bold plus 6 others
LOA: 1	b) What factors stabilise the knee joint?	b) Strength and actions of surrounding **muscles and their tendons** – most important **quadriceps femoris**, esp. vastus medialis and lateralis. **Ligaments** connecting femur and tibia – The main ones being the anterior and posterior **Cruciates and** medial (tibial) and lateral (fibular) **collaterals** Other ligaments are; posterior oblique and arcuate ligaments and the patella ligament (Most stable position = erect extended knee. Articular surfaces most congruent. Cruciates and collaterals taut and joint splinted by many tendons).	Must identify muscle groups and all 4 main ligaments
	c) Describe the attachments of the cruciate ligaments.	c) ACL attaches anteriorly and runs up laterally, PCL opposite.ACL arise from anterior intercondylar tibial eminence, passes superiorly and posterolaterally to medial aspect of thelateral femoral condyle. PCL arise from posterior intercondylar area of tibia, passes superiorly and anteromedially to thelateral aspect of the medial femoral condyle	Must identify A/P tibial attachments.

Stem: Moving onto Pathology. He represents one week later with fever and a rash.			
Question 5 Type 3 hypersensitivity **Subject**: Pathology LOA: 1	a) What is the pathogenesis of Type III Hypersensitivity?	(Ig G or IgM) **Antibodies bind antigens & then induce inflammation** directly or by activating complement. The recruited leukocytes produce tissue damage by release of lysosomal enzymes and generation of toxic free radicals 3 phases (systemic diseases) a) Formation of **antigen antibody complexes (immune complexes)** in circulation b) Deposition of **immune complexes** in various tissues c**) inflammatory reaction** at the site of deposition, causing tissue injury	Bold to pass
	b) List some examples of diseases caused by Type III Hypersensitivity.	b) Serum sickness, SLE, polyarteritis nodosa, post strep GN, Acute GN, reactive arthritis, arthus reaction.	2 to pass
	c)What symptoms or signs may patients present with?	c) Arthritis, Skin lesions, Vasculitis, Nephritis, fever	2 to pass

Stem: A 66-year-old man presents with central chest pain. This is his ECG.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 **Subject:** CBB	Describe the ECG. Prompt: What is the most likely rhythm?	Regular **broad/wide complex tachycardia** **rate :150 ,** looks regular concordance (no RS complex) no obvious Fusion and capture beats Dx: **VT**	**Bold** to pass
Stem: Moving onto Physiology			
Question 2 Coronary blood flow **Subject:** Phys LOA: 1	a) What is the coronary blood flow at rest? b) Describe coronary artery blood flow during the cardiac cycle. Prompt: Which part of the myocardium is most vulnerable to reduced coronary artery blood flow? c) What chemical factors may cause coronary vasodilation? d) What receptors govern coronary blood flow?	***250ml/min or 5%*** *of the Cardiac Output.* **Greater flow in diastole compared with systole. LV subendocardium most vulnerable.** RV flow continuous **Hypoxaemia,** local increase in **CO2, H+,** K+, **lactate,** PG, adenosine and adenine nucleotides. Coronary arterioles have **alpha receptors** – **vasoconstriction**, **B receptors** – **vasodilation** Cholinergic receptors - vasodilation.	**Bold** to pass. a) Accept range of 200-300ml/min acceptable. 4-6% b) Both bold c) 2/4 bold d) 1/2 bold

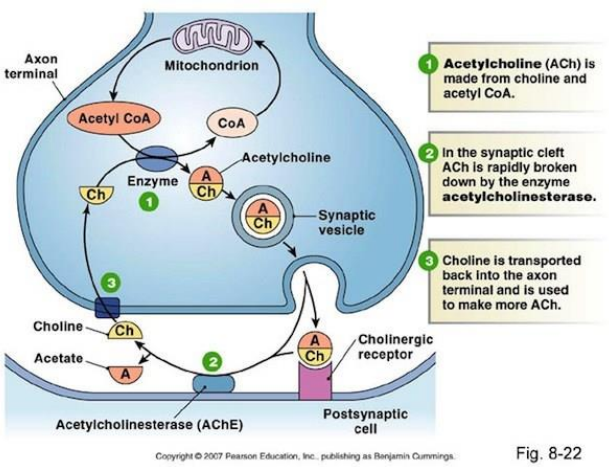
Stem: Moving onto Pharmacology. He is on frusemide			
Question 3 Frusemide **Subject:** Pharm LOA: 1	a) What class of drug is it? b) What are the pharmacokinetics of frusemide?	**Loop diuretic** Absorption: Well absorbed / variable oral bioavailability /10-100%. Onset post oral is 1-3 hour. Post IV is 15-30 mins. Duration post oral is 2-6 hours. Post IV 2 hours. Distribution: highly albumin bound. Metabolism: Liver (small amount). Elimination: Renal.	Bold 3 of 6
	c) What are the adverse effects?	orthostatic hypotension, dehydration Hyponatremia, hypokalemia, hypomagnesemia, metabolic alkalosis - ototoxicity, tinnitus, vertigo - GIT: pancreatitis, jaundice, N&V Raised uric acid- causing gout - thrombocytopenia - hypersensitivity reactions – rash	1 electrolyte abnormality and CVS effect (the other ones are quite rare)
	d) What are the possible drug interactions?	- NSAID, aminoglycosides - anticoagulants - digoxin, lithium, propranolol, probenecid, thiazides, amphotericin B , cisplatin	1 to pass

Stem: A CT-aortogram is performed. Moving onto Pathology			
Question 4 Aortic dissection **Subject:** Path LOA: 1	a) Describe the pathogenesis of an aortic dissection. Prompt; what are the risk factors for aortic dissection	**Hypertension**, aorta of hypertensive patients have medial hypertrophy of vasa vasorum and degenerative changes in the media Connective tissue disease (inherited or acquired) Both of the above cause **weakness in the media** An aortic dissection starts with an **intimal tear** and the blood dissect in the media either distally or proximally leading to a tear in the media	Bold
	b) How are aortic dissections classified?	By site of involvement: Stanford Type A prox, Type B distal OR Debakey I – asc and desc; II asc only, III desc only	Either classification to pass.
	c) What are the potential consequences of aortic dissection? Give examples	- Rupture back into intima or through adventitia - Rupture out or into pericardial, pleural or peritoneal cavities - Cardiac tamponade, aortic insufficiency, MI, distal ischaemia, spinal cord ischaemia - Death	3 examples to pass.

Stem: Moving onto Anatomy. A CT scan is performed			
Question 5 Aorta **Subject:** Anat LOA: 1	a) Describe the structures on this CT (Axial image from Anatomy prop inventory) b) Describe the course of the thoracic aorta? Name anatomical relations.	**Liver,** portal vessels, **R kidney** (top), **aorta, L kidney**, spleen, splenic vein, bowel loops, **pancreas**, IVC, vertebra, ribs, paravertebral muscles, intercostal and abdominal muscles, fat, skin **Ascending aorta**: **begins at aortic orifice.** **Arch** of the aorta: begins behind 2nd sternocostal jt. Passes supero-posteriorly and to the left anterior to the right pulm art and the carina. The apex of the arch lies to the left of the trachea/oesophagus and descends posterior to the left lung root, ending back at the level of the T4 (2nd sternocostal joint.) **Descending aorta**: Origin is at the left side at level of the T4 vertebra. It courses inferiorly to the level of T12. It approaches the midline as it descends alongside the oesophagus. At the inf border **T12** it **exits through the aortic hiatus** and becomes the abdominal aorta	5 Bold + 2 others Bold

ACEM PRIMARY VIVA B **Friday Morning** ***Candidate Number:*** **AGREED MARK:**

Stem: A 75-year-old man presents with a painful rash. We will start with clinical building block.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Shingles rash **Subject:** CBB Picture	a) Describe this rash? What is the likely diagnosis? b) What complications may occur?	**Herpes zoster** (vesicular lesions, crusting, not crossing midline, involving eyelid) Ocular involvement (Herpes ophthalmicus) Secondary bacterial infection / cellulitis Ramsay Hunt syndrome Disseminated herpes zoster (immunocompromised pt) Post herpetic neuralgia	Bold to pass 2/5 to pass
Stem: Moving onto Pharmacology. The patient is on carbamazepine.			
Question 2 Carbamazepine **Subject:** Pharm LOA 1	a) What receptors do carbamazepine affect? b) What are the most common dose-related adverse effects? c) What important drug interactions does carbamazepine have?	a) **Sodium channel blocker,** adenosine receptors Anti-cholinergic (anti-muscarinic) - nystagmus, diplopia, ataxia (cerebellar) - drowsiness - anti-cholinergic effects - dry mouth, tachycardia, blurred vision, delirium - CVS- hypotension c) - **Induces CYP450/induces hepatic drug metabolizing enzymes** and P-glycoprotein, results in increased clearance of some drugs, reducing their therapeutic blood levels (e.g. warfarin, phenytoin, valproate, lamotrigine, diazepam, phenobarbitone) - Can result in breakthrough seizures - Increases metabolism of OCP reducing its effectiveness	Bold to pass 1 cerebellar sign plus one other Bold plus one example

Stem: Moving onto Physiology. He is tachycardic and has a dry mouth.			
Question 3 Cholinergic neurotransmission **Subject:** Phys LOA 1	a) Please describe the synthesis, release and action of acetylcholine at a nerve synapse. You may draw a diagram	 Copyright © 2007 Pearson Education, Inc., publishing as Benjamin Cummings. Fig. 8-22	**Key sequence to pass**. - Synthesis: acetyl CoA and choline - Release from the synaptic vesicle - Bind to post-synaptic receptor
	b) Once acetylcholine is released into the synaptic cleft, how is its effect terminated?	Diffusion **Acetylcholine esterase** Re-uptake of choline into presynaptic nerve terminal	Bold to pass.
Stem: Moving onto Pathology.			
Question 4 Herpes Zoster **Subject:** Path LOA 2	Describe the pathogenesis of Herpes Zoster.	1 the patient has **had previous exposure** to herpes (chickenpox or subclinical) 2 VZV evades immune defenses & **infects sensory neurons** in and around dorsal root ganglia 3 Able to **remain latent** here for many years 4 Usually a single episode of recurrence in the form of zoster/ shingles 5 **Reactivation** often in the elderly or immuno-compromised **6** Vesicular eruption along **dermatome of one or more sensory nerves** Associated intense burning, itching and pain due to radiculoneuritis. May cause nerve dysfunction (e.g. Ramsay Hunt syndrome)	Bold to pass

Stem: Moving onto Anatomy. His eye movements are examined.			
Question 5 Model Extraocular muscles (F13) **Subject**: Anat LOA 1	a) Identify the extraocular eye muscles on this model.	Superior rectus Inferior rectus Medial rectus Lateral rectus Superior oblique Inferior oblique	All to pass.
	b)Describe their actions	b) Recti • Superior (elevation, adduction, medial rotation) • Inferior (depression, adduction, lateral rotation) • Medial (adduction) • Lateral (abduction) Obliques • Superior (depression, abduction) • Inferior (elevation, abduction)	5 muscles described to pass.
	c) What nerves supply these muscles?	c) Oculomotor (CN III) Nerve to all, except: Abducens (CN VI) Nerve to Lateral Rectus Trochlea (CN IV) Nerve to Superior Oblique	2/3 to pass
	d) How are the actions of these muscles tested clinically? BONUS QUESTION	d) Abduction (Lateral Rectus) • Elevation (Superior rectus) • Depression (Inferior Rectus) Adduction • Elevation (Inferior Oblique) • Depression (Superior Oblique)	Abduction isolates recti and adduction isolates obliques to pass

ACEM PRIMARY VIVA B **Thursday Morning** ***Candidate Number:*** **AGREED MARK:**

Stem: A 68 year old man presents with right sided weakness. A CT brain is performed.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 **Subject:** CBB CT brain Image LOA: 1	a) Describe this image. b) What is the clinical diagnosis c) What signs may be found on examination?	a) **Hypodensity** in **left** parieto-occipital region b) L. MCA territory infarct/stroke c) MCA stroke signs: • contralateral hemiparesis • contralateral hemisensory loss • contralateral homonymous hemianopia • aphasia, if the dominant hemisphere is involved • contralateral neglect, if the non-dominant hemisphere is involved • affects the face and arm more severely than the leg	**BOLD** to Pass **Concept to pass** 3/6 to pass

Stem: Moving on to Anatomy, we will discuss the blood supply to the brain.			
Question 2 **Subject:** Anatomy CT brain Image (use CBB) LOA: 1	Question: a) What are the main arteries contributing to the blood supply of the brain? b) What are the main cerebral arteries? c) Describe which lobes of the brain they supply. BONUS QUESTION IF TIME PERMITS d) (Which vessels make up the posterior circulation?)	a) • **Vertebral arteries** merging to form the basilar artery • **Internal carotid arteries** • Anastomosing via the anterior and posterior communicating arteries • To form the Circle of Willis b+c) Some overlap in lobar supply: • **Anterior cerebral**: frontal, parietal lobes • **Middle cerebral**: frontal, lateral temporal, parietal lobes • **Posterior cerebral**: medial temporal, parietal, occipital lobes d) • Posterior cerebral • Superior cerebellar • (Anterior and posterior) inferior cerebellar • Pontine)	**BOLD** to pass Candidates may elect to draw and label the circle of Willis (McMinn p.67)

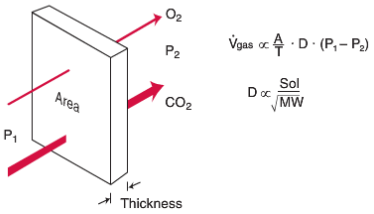
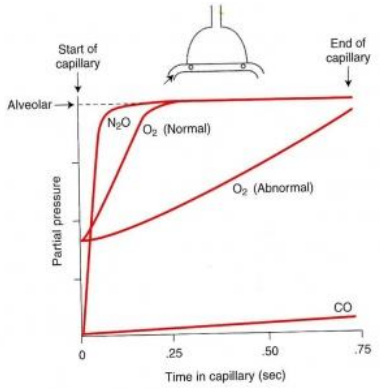
Stem: Moving on to Physiology. He is hypertensive at 220/100.			
Question 3 Brain Metabolism and Energy Sources **Subject:** Physiology LOA: 1 Ganong 25th Edition pp 609-610	1. How is brain perfusion maintained in brain injury?	Aim is to maintain CPP With high ICP need to increase MAP to maintain CPP **CPP = MAP - ICP** Raised MAP results in systemic hypertension and reflex bradycardia with Vagal stimulation	BOLD or explanation of equation to pass
	2. What proportion of the total body Oxygen does the brain consume?	**20%** (despite brain weight 2% of body weight)	10-30%
	3. What energy substrates can be used by the brain?	**Glucose,** glutamate in prolonged starvation amino acids,	Glucose to pass

Stem: Moving on to Pathology.			
Question 4 Cerebral infarction **Subject:** Pathology LOA: 1	(a) What are the causes of ischaemic cerebral infarction? (b) Where are some sources of cerebral thromboemboli?	(a)**Arterial thrombosis** - Cerebral emboli - lacunar infarcts from small vessels - cerebral arteritis - arterial dissection - venous infarction (b)left atrium/ventricle thrombus - valvular vegetations - PFO causing paradoxical emboli - Carotid plaque	Thrombus + one At least 2 sources
Stem: Moving on to Pharmacology. The patient has aspirated. He is treated with antibiotics, one of which is metronidazole.			
Question 5 Metronidazole **Subject**: Pharm LOA: 1	(a)Describe the pharmacokinetics of Metronidazole. (b)What are the adverse effects of metronidazole?	Absorption - Well absorbed orally; Oral/IV/suppository (99% oral bio-availability); Metabolised in liver (can accumulate in hepatic insufficiency) Excreted via kidney; Low protein binding (10-20%); Half-life 7.5 hours - GIT: Nausea, diarrhoea, dry mouth, metallic taste - Neuro: Headache, paraesthesia, dizziness - thrombophlebitis - Disulfiram-like effect, hence avoid alcohol	3 pharmacokinetic parameters (Absorption / Metabolism / Excretion /protein binding/half-life) 2 systems side effects

ACEM PRIMARY VIVA B **Thursday Afternoon** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE	NOTES
Stem: A 75 year old man presents with haemoptysis. He has had a chest x-ray.			
Question 1 **Subject:** CBB LOA:	(a) Please describe his x-ray (b) What is your differential diagnosis?	a) Right middle lobe opacities /midzone/loss of right heart border b) consistent with pneumonia, PE, malignancy, TB	Appropriate description Include pneumonia to pass
Stem: We will start with Pathology.			
Question 2 Robbins – 9th Edition. Page 704-6 **Subject: Path** LOA: 1	a) What are the most common causes of bacterial community acquired pneumonia? b) What factors predispose patients to the development of acute bacterial pneumonia?	a) The most common bacterial causes are: - **Strep pneumoniae** - mycoplasma pneumoniae - Haemophilus influenzae - Moraxella catarrhalis - Staph aureus - Klebsiella pneumoniae - Pseudomonas aeruginosa - Legionella pneumoniae b) 1. Extremes of age. 2. Underlying chronic disease such as COPD, Diabetes mellitus, Congestive cardiac failure, 3. Immunodeficiency: congenital or acquired, Abnormal splenic function: decreased splenic function or asplenia.	Strep plus 2 others 3 examples from 2 groups to pass

Stem: Moving onto Pharmacology. He is commenced on antibiotics, one of which is azithromycin.			
Question 3 **Subject:** Pharm Katzung 13th Edition page 793. LOA:	1. What class of antibiotic is azithromycin 2. What is its mechanism of action? 3. What organisms does azithromycin cover? 4. What is an important cardiac side effect?	1. **Macrolides** 2. **Inhibits protein synthesis** by binding to the ribosomal RNA. It is bactericidal at high concentrations. 3. • Haemophilus influenza • Chlamydia species. • Mycobacterium avium complex • Staph • Strep • Mycoplasma • Legionella 4. Prolonged QT interval.	Bold to pass Bold to pass 3 to pass Bonus question

Stem: Moving onto Physiology. He is dyspnoeic and his oxygen saturation is 90% on high flow oxygen			
Question 4 **Subject:** Physiology West's Respiratory physiology 10th edition. P 29 LOA:	1.. Explain Fick's law of diffusion. *Prompt :Fick's law of diffusion describes the factors influencing the diffusion of gases across the alveolar wall* *Prompt for diffusion constant: What factors determine the diffusion constant in Fick's law?*	1. Gases diffuse across a surface by passive diffusion. Fick's law says that the rate of diffusion is directly proportional to the area of the diffusion membrane, the pressure gradient across the membrane and the diffusion constant. It is inversely proportional to the thickness of the membrane.  	1**. Concept explained to pass.** Or adequately explains the formula – if written Dependent on the substances making up the membrane AND proportional to the solubility of the gas across the membrane - inversely proportional to the square root of the molecular weight of the gas.
	2. What is the difference between a diffusion limited and a perfusion limited gas? *Prompt – you may draw a graph to illustrate your answer*	2. <u>A perfusion limited gas</u> is one where the partial pressure on both sides of the membrane equilibrates rapidly such that no further diffusion into the blood can occur from the alveoli unless the blood perfusion rate increases. In the graph (see picture), there is no gap between the alveolar pp of the gas at the time blood leaves the pulmonary capillary. <u>A diffusion limited gas</u> is one where the partial pressure of the gas does not achieve equilibration in the time that blood spends in the pulmonary capillaries. In the graph, there is a gap between the pp of the gas at the end of the pulmonary capillary perfusion time.	2**. Concept explained to pass**. 

Stem: Moving onto Anatomy. Coarse crackles are heard in his right posterior chest.			
Question 5 **Subject**: Anatomy Moore's anatomy. 7h Edition. Pages 109-110 LOA: 1	a) Using the CXR (CBB), name and indicate the position of the lobes of the lung	a) **Right** and **left upper lobe** fill apices/upper zones Lingular lobe abuts the left heart border. **Left lower lobe abuts the left hemidiaphragm.** **Right lower lobe abuts the right hemidiaphragm.** **Right middle lobe abuts the right heart border.**	**bold location concepts**
	b) Describe the surface anatomy of the parietal pleural reflections	b) The right and left sternal parietal pleural reflections are asymmetrical but the costal and diaphragmatic reflections are symmetrical. The right and left sternal pleural reflection **start at the apices of the right and left lung** They descend inferomedially in parallel to the sternoclavicular joint and pass to the posterior aspect of the sternum in the anterior median line. **At the level of the 2-4 costal cartilage**, they lie parallel to each other. Inferior to this level, they become asymmetrical. On the left side, at the level of **the 4th costal cartilage**, the pleura **deviates** to the **left side** of the sternum and reaches **the 6th costal cartilage level just lateral to the left lateral sternal edge**. The right side passes inferiorly until it reaches the **6th costal cartilage in the anterior median line**. From then on, both sides **passes laterally and posteriorly** with the following markers: At the **level of the 8th costal cartilage and the mid clavicular line.**	4 **of 7 bold concepts**

		At the l**evel of the 10th costal cartilage at the mid axillary line.** At the **level of the 12th costal cartilage at the neck of the 12th rib.** The diaphragmatic pleural reflection is in close contact to the diaphragm.	

ACEM PRIMARY VIVA B **Friday AM** ***Candidate Number:*** **AGREED MARK:**

Stem: A 75-year-old male with metastatic lung cancer presents acutely short of breath. We will start with Physiology.

TOPIC	QUESTIONS	KNOWLEDGE	NOTES
Question 1 Dead space **Subject:** Physiology *West's Respiratory Physiology 10th edition pages 19 - 21*	(a) What is the anatomical dead space?	a) The anatomical dead space refers to the airway volume with **ventilation and no blood flow.** The conducting airways (to division 16) **take no part in gas exchange**. Vol = approx. 150mls.	Bold to pass
	(b) How does it differ from physiological dead space? *Prompt: What happens in normal vs diseased patients?*	b) Anatomical dead space is determined by morphology of the airways and lung. Physiological dead space is the volume of airways and lung that does not eliminate CO2. The two dead spaces of volume are **almost the same in normal subjects**, but the physiological dead space is **increased in many lung disease**s due to inequality of blood flow and ventilation in the lung. (VQ mismatch)	Bold to pass
	Bonus Q: How are these different dead spaces measured?	Measurement: Fowler's = Anatomic Dead Space Bohr = Physiological Dead Space *(bonus)*	

Stem: He is drowsy and has low oxygen saturations on room air. You perform an arterial blood gas.			
Question 2 ABG **Subject:** CBB	a) Describe and interpret this ABG Prompt "move along" if trying to calculate A-a gradient	a) Acidaemic, hypoxic, hypercarbic (respiratory acidosis), **Acute Respiratory acidosis with no metabolic compensation,** type 2 Respiratory Failure	Bold to pass
	b) What are possible causes of these abnormalities in this patient?	b) Respiratory acidosis • CNS depression from drugs, injury, or disease. • Hypoventilation due to pulmonary disease (cancer, effusion, pneumonia, atelectasis) • Hypoventilation due to musculoskeletal or neuromuscular (paraneoplastic?) disease	1 cause

Stem: Moving on to Pharmacology. He is given intravenous antibiotics and has an allergic reaction requiring adrenaline.			
Question 3 Adrenaline **Subject:** Pharm LOA: 1 *Katzung 13th edition page 140-144*	a) Describe the pharmacokinetics of adrenaline?	a) <u>Absorption / Routes of administration:</u> Subcut**, IMI, IV and** nebulised**,** oral = poor**.** <u>Distribution:</u> Crosses placenta, does not cross BBB. 50% protein bound. Onset: seconds. Duration: approx. 2 min. <u>Metabolism</u>: terminated by metabolism in sympathetic nerve terminals and metabolised by **COMT and MAO**, circulating adrenaline metabolised by COMT. Metabolites = VMA/MOPEG <u>Excretion:</u> metabolites via urine.	Should be able to describe bold routes of administration and metabolism to pass.
	b) What are the pharmacodynamic effects of adrenaline?	b) **Equal effect at both Alpha and Beta receptors** (low dose mainly Beta, higher doses increased Alpha) **Alpha = vasoconstriction** **B1 = positive inotropy and chronotropy** **B2** = **smooth muscle relaxation** → bronchodilation and skeletal muscle vasodilatation *(this may cause fall in TPR reflected in fall in diastolic BP sometimes seen)*	Must get Alpha, Beta 1 & 2 effect

Stem: Moving on to Pathology. He has multiple metastases from his lung cancer			
Question 4 Lung Cancer **Subject:** Pathology LOA: 1 *Robbins 9th edition Pg. 712-719*	a) What factors predispose to lung carcinoma?	a) **Tobacco smoking** **Environmental exposures**: radiation, asbestos, air pollution (particulates), occupational inhaled substances (Nickel, Chromates, Arsenic) **Genetic mechanisms**: dominant oncogenes (c-MYC, k-RAS) & loss of tumour suppressor genes (e.g. p53, RB) **Precursor lesions**: squamous dysplasia, carcinoma in situ, atypical adenomatous hyperplasia	Tobacco smoke + 2 concept areas to pass
	b) What are the classic clinical features of lung carcinoma	b) Cough (75%), weight loss (40%), chest pain (40%), dyspnoea (20%).	3 of 4
	c) What paraneoplastic syndromes are associated with lung carcinoma? *Prompt: what hormones can be released by tumours?*	c) SIADH - hyponatraemia; ACTH - Cushings syndrome; hypercalcaemia - parathormone, parathyroid hormone-related peptide or prostaglandin E; hypocalcaemia - calcitonin; gynaecomastia - gonadotropins; carcinoid syndrome -serotonin, bradykinin . Others: Lambert-Eaton Myaesthenic sy, peripheral neuropathy, acanthosis nigricans, clubbing (hypertrophic pulm. osteoarthropathy).	At least 3

Stem: Moving on to Anatomy. He has pain from bony metastases in his shoulder.			
Question 5 Rotator Cuff **MODEL ;** Articulated Shoulder joint **Subject**: Anatomy LOA: 1 Pg. 700-705 Moore's Anatomy 7th Edition	a) Identify the major bony features of the scapula using this model.	a) • **Subscapular fossa** • **Glenoid cavity** • **coracoid process,** • **acromion** • facet for clavicle • **Supraspinous fossa** • Suprascapular notch • Spine of scapula • **Infraspinous fossa** • neck of scapula • Deltoid tubercle of spine	Bold to pass
	b) Demonstrate the proximal attachments of the four rotator cuff muscles on the scapula	b) Supraspinatus – supraspinous fossa Infraspinatus – infraspinous fossa Teres minor – middle part lateral border of scapula Subscapularis – subscapular fossa	Must know all four
	c) What are the actions of these individual rotator cuff muscles?	c) Supraspinatus – abduction Infraspinatus – lateral rotation Teres minor – lateral rotation Subscapularis – medially rotates arm	3 of 4 minimum

ACEM PRIMARY VIVA B **Friday Afternoon** ***Candidate Number:*** **AGREED MARK:**

Stem: A 90 year-old man with a urinary catheter presents with sepsis. Previous cultures have grown E.coli. We will start with Pathology.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 E.coli **Subject:** Pathology LOA: 1 *Robins 9th Ed; Pg 350; Pg 665; Chapter 14:*	a) Which bacterial class does Escherichia coli belong to?	a) E.coli is a **gram negative rod** which is a facultative **anaerobe**. It is a normal GI pathogen.	Bold
	b) What is the difference between an endotoxin and an exotoxin?	b) Endotoxins are lipopolysaccharides (LPS) in the outer membrane of the **cell wall of Gram-negative bacteria which cause injury via the host immune response** Exotoxins are proteins that are **secreted by the bacterium and cause direct injury**	Bold concepts
	c) List some types of infections that can be **commonly** caused by E.coli?	c) Urinary tract infections; prostatitis; epididymo-orchitis; infectious enterocolitis; cholecystitis; bacterial peritonitis	3 to pass from this list

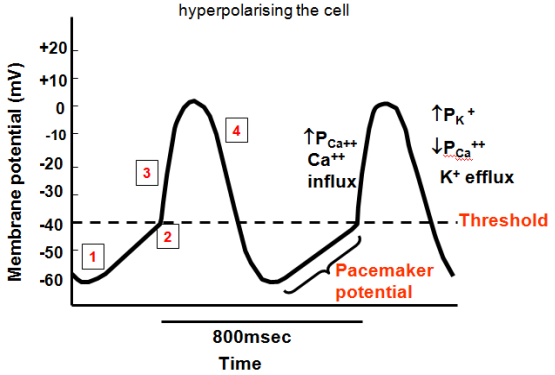
Stem: Moving onto Physiology. He has decreased urine output.			
Question 2 GFR **Subject:** Physiology LOA: 1 Ganong's review of Medical Physiology. 25th edition. Chapter 37 Renal function and micturition. Glomerular filtration.	a) What is the definition of the glomerular filtration rate? b) What is the normal Glomerular filtration rate? c) List some factors that affect the GFR. Prompt " physiological factors" "are there any particular cells in the kidney that are involved in regulating GFR?" d) What substances act on mesangial cells to change GFR?	a) Amount of fluid (plasma filtrate) filtered by the glomerulus per unit time b) Normal GFR – **125ml/min** (180L/24Hrs) in normal adult. 10% lower in females. c) -Size of capillary bed. Regulated by **mesangial cells** (contractile cells) located in the glomerulus (between the basal lamina and the endothelium). -Permeability of glomerular capillaries (50 x skeletal muscle capillaries) -Hydrostatic and osmotic pressure gradients Oncotic pressure (plasma protein concentration) Glomerular capillary hydrostatic pressure -Systemic blood pressure -Afferent arterial pressure (renal artery blood flow - kept stable by autoregulation 90-210mmHg) -Afferent or efferent arteriolar constriction -Hydrostatic pressure in Bowman's capsule -Intrarenal interstitial pressure (ureteral obstruction, renal oedema) -Age d) Increased – ANP, dopamine, PGE2, cAMP Decreased – NA, vasopressin, AGII, Histamine, PGF2, endothelins, TXA2, leukotrienes	Concept to pass +/- 10% to pass 110-140ml/min Bold to pass. Must mention mesangial cells and 3 other factors. List at least one of each increased/decreased

Stem: Moving onto Pharmacology. His medications include Ramipril.			
Question 3 ACE inhibitors **Subject:** Pharmacology LOA: 2 *Katzung 13th, 184-5.*	a) Discuss the pharmacodynamics of Ramipril. *Prompt: how does this class of drug work?* *Prompt: do they have other non-antihypertensive benefit (eg. in DM)?*	a) Inhibit the peptidyl dipeptidase (angiotensin converting) enzyme that hydrolyzes **angiotensin I to angiotensin II** **Stops inactivation of bradykinin**, a potent vasodilator, which works at least in part by stimulating release of nitric oxide and prostacyclin. Inhibits the renin-angiotensin system and and stimulates the kallikrein-kinin system. Diminishes proteinuria and stabilizes renal function (even in the absence of lowering of blood pressure) - particularly valuable in diabetes - now recommended in diabetes even in the absence of hypertension.	Bold to pass
	b) How is Ramipril eliminated? Why is this important?	b) **It is eliminated primarily by the kidneys.** Doses of these drugs should be reduced in patients with renal insufficiency.	Bold to pass
	c) What are some adverse and toxic effect of ACE Inhibitors?	c) Severe hypotension can occur after initial doses (esp. fluid deplete pts). **ARF** – esp. with bilateral renal artery stenosis or solitary kidney. Dry cough and wheeze. **Hyperkalemia** – esp. with K^+ sparing diuretics, DM and CRF. **Angioedema** Contraindicated in pregnancy. In high doses with CRF - neutropenia and proteinuria Altered taste Allergic skin reactions Drug fever (in up to 10% of pts) Effect may be reduced with concomitant NSAIDs	2 bold/total 4 to pass

Stem: He is noted to be tender over his back. A thoracic spine x-ray is arranged.			
Question 4 Lateral T-spine X-ray **Subject**: CBB	a) Describe this x-ray. What is the abnormality? b) Name possible causes for this finding. c) Which complications would you look for?	a) Thoracic spine (AP and lateral) **T12 crush fracture (** > 50% loss of vertebral height) b) Trauma, osteoporosis, pathological c) Looking for neurological compromise (weakness, sensory loss, bowel or bladder dysfunction)	Bold At least 2 causes 2 signs to pass
Stem: Moving onto Anatomy.			
Question 5 Thoracic vertebra (bone) **Subject:** Anatomy LOA: 1 *Moore's 7th edition Fig 1.4 & 1.5 p.77*	a) Identify this bone and demonstrate its bony features b) What movements occur at the thoracic vertebra? c) List the ligaments responsible for the stability of the spine	a) **Thoracic vertebrae** **Body, Pedicle, Transverse process** **Articular facets** – Superior and inferior **Costal facets –** Superior/Inferior costal facets (head of rib); Transverse costal facet (tubercle of rib) **Spinous process, Lamina** **Vertebral foramen** and space for intervertebral foramina b) **Rotation,** some lateral flexion, very limited flexion and extension c) Anterior longitudinal, posterior longitudinal, Supraspinous, Ligamentum Flavum	Thoracic vertebra plus 5 Bold 3 to pass

ACEM PRIMARY VIVA C Thursday Morning ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: A 65-year-old woman presents with chest pain and shortness of breath.			
Question 1 ECG **Subject:** CBB	Describe her ECG Prompt: What is your interpretation of the ECG?	**Sinus rhythm**, rightward axis, **ST elevation Leads V2-V5** and aVL. **ST depression II, III, aVF** (Q waves absent) **Anterolateral STEMI** with reciprocal inferior changes.	Bold to pass
Stem: She is a Type 1 diabetic. Moving on to Pharmacology			
Question 2 Insulin **Subject:** Pharm LOA: 1	a) What is the mechanism of action of insulin? b) What different formulations of insulins are there (prompt - please describe in terms of duration of action and name an example from each group) c) How are their differing properties used to optimize glycaemic control d) Give another Emergency Department use for insulin other than the regulation of blood glucose	a) **Promotes the uptake of glucose from blood into tissues,** esp. fat, liver, and skeletal muscle and promotes glycogen synthesis (Insulin receptors found on cell membranes) **b) Rapid and short acting.** Clear solution, neutral pH, contain Zn, rapid onset, short duration of action. examples: insulin neutral, insulin lispro, insulin glulisine **Intermediate acting** Turbid solution, neutral pH, protamine in phosphate buffer (NPH) to prolong action examples: insulin isophane, insulin aspart protamine **Long acting** Clear solution, soluble, slow onset, prolonged action. Daily administration mimics basal insulin secretion examples: insulin glargine, insulin detemir **c) Combination of insulins with different durations of actions** aim to replace basal insulin requirement (50%) and meal requirement (50%). d) Management of hyperkalaemia, Ca^{++} channel blocker overdose (+/- Beta-blocker)	Bold to pass Bold to pass plus 2 of 3 correct insulins Concept to pass One alternate use to pass
Stem: Moving on to Anatomy			
Question 3 Heart (model) **Subject:** Anat LOA: 2	a) Demonstrate on this model the arterial supply of the heart. (Prompt - can you name the main branches of the coronary arteries?) b) Occlusion of which vessel would result in an anterolateral STEMI? c) Describe the venous drainage of the heart? Prompt; What is the final common venous tributary that empties into the heart called?	**a) Right coronary artery** - gives off SA nodal branch (60%), right marginal branch, AV nodal branch, posterior interventricular 2/3, inter ventricular septal. **Left coronary artery** - gives off **circumflex artery** which branches to give the SA nodal artery in 40%, left marginal artery, posterior interventricular (15%). Left coronary artery - gives off **LAD** which supplies anterior 2/3 septum, lateral diagonal. b) Proximal LAD (before first diagonal branch) c) **Coronary sinus (6),** Great cardiac veins (accompanies LAD then LCX), Middle cardiac veins (accompanies PIV), Small cardiac veins (accompanies R marginal), Left posterior ventricular, Left marginal, Anterior cardiac (start ant. surface RV, drain straight into R atrium), Oblique veins on left atrium, Venae cordis minimae (drain direct into chambers)	Bold to pass LAD Coronary sinus plus one other to pass

Stem: The patient is in heart failure. Moving on to Pathology.			
Question 4 Fluid and oedema **Subject:** Path LOA: 1	a) What factors govern the movement of fluid between the vascular and interstitial spaces?	**1. Hydrostatic pressure** **2. Colloid osmotic pressure** **3. Normal capillary walls** - most protein remains intravascular, fluid leaks out. Fluid out of vessel at arteriolar end. Most fluid returned to vessel at venular end. Small amount of fluid returns via lymphatics.	2 bold plus concept
	b) What are the major mechanisms of oedema formation? Give examples of each.	**1 Increased hydrostatic pressure** (i) Local venous; venous obstruction, compression, thrombosis (ii) Local arteriolar; dilation, heat, neurohumeral dysregulation (iii) Systemic; CCF, constrictive pericarditis, impaired venous return 2. **Reduced plasma oncotic pressure** (mainly protein loss e.g. nephrotic syndrome or poor production e.g. cirrhosis, malnutrition, gut loss). **3. Inflammation -** acute or chronic inflammation, angiogenesis **4. Lymphatic obstruction** - Inflammatory, neoplastic, post-surgical, post irradiation, **5. Sodium retention with water** - renal insufficiency, activation of renin-angiotensin system, renal hypoperfusion.	3 out of 5 bold, must include hydrostatic and COP. 5 conditions covering three groups
	c) What are the clinical features of heart failure?	1. Lung - dyspnoea, orthopnoea, PND, APO, pleural effusions. 2. Cardiac - 3rd heart sound, displaced apex beat, AF, murmurs, JVP elevation. 3. Renal - fluid retention, pedal oedema, AKI. 4. Brain - confusion secondary to hypoxia. 5. Hepatic - congestion, ascites, cirrhosis late	3 of 5 organ system symptoms to pass
Stem: Moving on to Physiology			
Question 5 Cardiac pacemaker **Subject**: Physiology LOA: 1	a) Draw the action potential in a **cardiac pacemaker** cell and explain the ionic fluxes.	1. 'Funny' sodium channels (I_f channels) are open ($\uparrow P_{Na^+}$); and closing K^+ channels. 2. Transient Ca^{2+} (T-type) channels open, pushing the membrane potential to threshold. 3. Long-lasting Ca^{2+} (L-type) channels open, giving rise to the action potential. 4. Opening of K^+ channels, ($\uparrow P_{K^+}$), and closing of Ca^{2+} (L-type) channels, hyperpolarising the cell  i) Pre potential due to increased influx of Na via 'funny channels' (open in response to hyperpolarisation), **decrease of K efflux**, then **completed by influx of Ca** through T channels ii) **Action potential due to influx of calcium** via L channels iii) **Repolarisation due to efflux of K,** no plateau	Correctly drawn action potential curve and 2 out of 3 bold sections to pass
	b) What is the effect of sympathetic and parasympathetic stimulation on the prepotential?	**i)** Noradrenaline binds to Beta 1 receptors and raises cAMP, resulting in opening of L channels and Ca influx. **Increased slope of prepotential** and increased firing rate**.** **ii)** Acetylcholine binds to M2 receptor and decreases cAMP resulting in both slowing of calcium channel opening and opening of special K channels (counter decay of K efflux) leading to greater fall in prepotential. Leads to **decreased slope of prepotential** and firing rate.	Bold to pass

ACEM PRIMARY VIVA C **Thursday Afternoon** ***Candidate Number:*** **AGREED MARK:**

Stem: A 23-year old woman with a history of intravenous drug use presents with severe dyspnoea.			
TOPIC	QUESTIONS	KNOWLEDGE (essential in bold)	NOTES
Question 1 CXR **Subject**: CBB	a) Please describe the major abnormalities on her chest x-ray b) What could be the causes of these findings?	Lung parenchyma - **diffuse opacities throughout** Causes: aspiration, ARDS, infection (pneumonia/pneumonitis), interstitial oedema	Bold 2 causes
Stem: She requires urgent intubation. Moving on to Pharmacology.			
Question 2 Rocuronium **Subject:** Pharm LOA: 1	a) What type of drug is Rocuronium? b) What are the pharmacokinetics of Rocuronium? c) How does Suxamethonium differ from Rocuronium?	**Nondepolarizing** muscle relaxant. Steroid derivative Absorption: IV BA = 1 **Dose: 1.2 mg/kg (accepted range 0.9-1.2mg/Kg)** **Onset: 45-60sec** **Duration: 20 – 75 minutes** **Metabolism: liver** **Elimination: Liver 75-90%, and kidney** **Duration of action is much shorter,** Sux 5-10 min Different side effects and contraindications Sux is a depolarizing muscle relaxant with phase I and Phase II. Phase I is augmented by pseudocholinesterase inhibitors Sux is metabolized by plasma pseudocholinesterase Roc has an antidote – sugammadex	Bold 3 Bold Bold plus 2
Stem: The patient has Acute Respiratory Distress Syndrome. Moving on to Pathology.			
Question 3 ARDS **Subject:** Path LOA: 1	a) Describe the pathogenesis of ARDS b) What conditions are associated with the development of ARDS?	Initial **injury to alveolar capillary membrane** (endothelium); **acute inflammatory response** (neutrophil mediated); results in **increased vascular permeability** and alveolar flooding; **fibrin deposition;** formation of **hyaline membranes;** and widespread **surfactant abnormalities** (damage to Type II pneumocytes); eventually – organisation with scarring **Infection** (sepsis, diffuse pulmonary infection, gastric aspiration) **Physical / Injury** (trauma – head, pulmonary, fractures, near drowning, burns, radiation) **Inhaled irritants** (O2 toxicity, smoke, irritant gases and chemicals) **Chemical injury** (Heroin, barbiturate, acetylsalicylic acid, Paraquat) **Haematological conditions** (multiple transfusions, DIC) **Other** (pancreatitis, uraemia, cardiopulmonary bypass, hypersensitivity – organic solvents, drugs)	3 Bold 1 example from 3 groups

Stem: You proceed to intubate the patient. Moving on to Anatomy.			
Question 4 Larynx (model) **Subject:** Anat LOA: 1	a) Identify the key structures on this model. b) What is the nerve supply of the muscles of the larynx? c) Demonstrate the landmarks for a cricothyroidotomy? d) Which cartilage in the larynx is fully circumferential?	Tongue, **vallecula**, **epiglottis**, **cricoid**, vocal cords, trachea, thyroid cartilage, hyoid bone **Recurrent laryngeal nerve** (derived from vagus) supplies all the muscles except for the cricothyroid muscle – supplied by the external laryngeal n. Thyroid cartilage, cricoid cartilage, **cricothyroid membrane** Cricoid cartilage	Bold plus 2 others Bold Bold
Stem: Once intubated, she is difficult to ventilate. Moving on to Physiology.			
Question 5 Lung compliance **Subject**: Phys LOA: 1	a) What is pulmonary compliance? b) What factors decrease or increase pulmonary compliance? c) What are the physiological effects of surfactant of the lung?	Compliance = **volume change/pressure change (ΔV/ΔP)**, maximal in mid inspiration, lower at extremes, approx. 200ml/cm H20 **Decreased:** alveolar oedema, pulmonary fibrosis, pulmonary venous hypertension, unventilated lung, **Increased:** age, emphysema i. Increased lung compliance ii. Reduced work of breathing iii. Improved stability of alveoli iv. Keeps alveoli dry	Bold with concept 3 examples 1 example 2 of 4

ACEM PRIMARY VIVA C **Friday Morning** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: A 40-year-old man presents with haematemesis. His pulse is 120 / minute and blood pressure is 90/60 mmHg. We will start with Physiology.			
Question 1 Cardiovascular response to moderate haemorrhage **Subject:** Phys LOA 1	a) Describe the factors affecting Cardiac Output b) What are the physiological responses to losing 1L of blood in an adult? Prompt: Are there any other non-cardiovascular responses Prompt: Are there any late compensatory responses?	**CO=SVxHR** SV related to contractility, preload and afterload, HR controlled by intrinsic rate, autonomic, exogenous factors, heat, thyroid Acutely: -↓venous return, **reduced stimulation of baroreceptors**, catecholamine release, tachycardia, vasoconstriction 12 to 72 hours: - ↓ renal blood flow – activation of **renin angiotensin system** fluid shifts 3 to 4 days: - hepatic synthesis of proteins increasing PP 10 days+: - **increased RBC production** by ↑EPO	Bold + 2 mechanisms from each SV and HR Bold and 2 Bold with some explanation Bold
Stem: Liver Function tests were performed.			
Question 2 LFTS (acute hepatitis) **Subject:** CCB	a) Please describe these results b) What are possible causes for this blood picture?	**Acute hepatitis** (elevated bilirubin, ALP, GGT, transaminases, INR; hypoglycaemia) **Impaired synthetic function** (low albumin, abnormal Coags) Alcohol, viral (A, B(+/-D), C, E, EBV, CMV) toxins (paracetamol, isoniazid, methyl-dopa, methotrexate, mushrooms), others e.g. a-1-AT deficiency, Wilson's disease, AI diseases	**Bold** with justification e.g. transaminitis (mild ALP elevation also) Alcohol plus 1 toxin/drug and 2 viruses
Stem: He has a history of heavy alcohol use. Moving on to Pathology.			
Question 3 Alcoholic Liver Disease **Subject:** Pathology LOA 1	a) Describe the pathological features of the liver in alcoholic liver disease? PROMPT: please describe the morphological features b) Which of these features are reversible? c) What are the possible sequelae of cirrhosis? Prompt: Complications?	**1. Hepatic steatosis-** fatty change, perivenular fibrosis **2. Hepatitis:** liver cell necrosis, inflammation, Mallory bodies, fatty change, fibrosis **3. Cirrhosis:** extensive fibrosis, hyperplastic nodules **4. Hepatocellular carcinoma** **Steatosis and Hepatitis are reversible**. Cirrhosis irreversible. **Portal Hypertension**, GIT Bleeding, Hepatic Failure, Coagulopathy, Hepatocellular Ca, Hepatorenal Syndrome, Hepatopulmonary Syndrome, Encephalopathy, Infection	Pass Bold to pass Bold plus 3

Stem: This is a picture of the liver from below and behind. Moving on to Anatomy.			
Question 4 Liver **Subject:** Anatomy LOA 1	a) Identify the main structures	**Lobes – Right** (24), **left** (14), caudate (2), quadrate (21) Vascular – IVC (13), Hepatic art (11), **Portal vein** (20) Biliary – common hepatic duct (5), **gallbladder** (9) Ligaments – coronary (12), L triangular (15), R triangular (25), ligamentum teres (17), Diaphragm (6)	Identify right and left lobes of liver, portal vein and gall bladder
	b) Describe the anatomy of the biliary tree. *Prompt: Draw the biliary tree*	L & R **hepatic ducts** run into **common hepatic duct** Joined by **cystic duct** from **gallbladder** to become **common bile duct** which runs into **duodenum**	4 out of 6 to pass
Stem: His treatment includes the administration of Octreotide. Moving on to Pharmacology.			
Question 5 Octreotide **Subject**: Pharm LOA 2	a) What is the mechanism of action of Octreotide?	Somatostatin analog, **reduces** splanchnic & **portal blood flow** by poorly understood mechanism & hence variceal pressures. Inhibits endocrine & paracrine factor secretion including insulin, glucagon, gastrin, GH, TSH,	Bold plus general concept
	b) What are the routes of administration for octreotide?	IV, IM, SC	Any 2 to pass
	c) What are its adverse effects?	Anaphylaxis, Local irritation during injection (redness, burning) GIT symptoms (nausea & vomiting, decreased intestinal motility, bowel obstruction, cholelithiasis) Hypo/hyper glycaemia Cardiac – sinus bradycardia, conduction disturbances	Any 2 to pass

ACEM PRIMARY VIVA C **Thursday Morning** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Stem: A 25-year-old man presented with a painful elbow after a fall during football. An X-ray was taken.			
Question 1 Elbow x-ray **Subject:** CBB	Please describe the X- ray. *Prompt: What is the abnormality and outline the bony features?* What other important adjacent structures are at risk from this injury? *Prompt: Where do they lie in relation to the elbow?*	**Posterior dislocation of right elbow**. **Radial head**, coronoid process of ulna, **articular surfaces of humerus** (trochlea or capitulum/capitellum). **Empty olecranon fossa**. Bony fragment in olecranon fossa Nil other obvious injury. Median nerve and brachial artery (anterior) Ulnar nerve (posteriomedial)	Bold to pass 2 of 3, and indicating correct location of one on XRay to pass
Stem: Moving on to Pharmacology. He was given Ketamine to reduce the injury.			
Question 2 Ketamine **Subject:** Pharm LOA: 1	Describe the pharmacodynamics of ketamine. What are the systemic effects of Ketamine? What are the adverse effects?	**NMDA receptor antagonist**. Inhibits reuptake of catecholamine and serotonin. Potent short acting sedative, amnestic, analgesic and anaesthetic agent CNS: dissociative **anaesthesia**. (Cataleptic state) Profound **analgesia**. Cerebral vasodilator and increases cerebral blood flow and cerebral metabolic rate (Increases ICP – not clinically significant). May have anticonvulsant properties CVS: **haemodynamically stable**, increases HR, BP and cardiac output, cardiac workload and myocardial oxygen consumption. Respiratory: **Intact airway reflexes**. Min. respiratory depression. Causes lacrimation and salivation that may cause laryngospasm in children. Bronchodilator. Ocular: nystagmus CNS - emergence phenomenon - dysphoria, hallucinations, seizures GI – vomiting Resp – Laryngospasm, increased salivation	**Bold to pass** **Bold** + 1 other One adverse effect

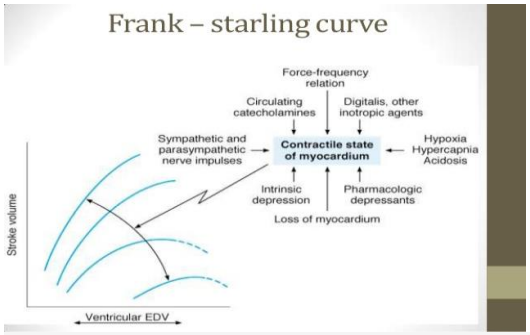
Stem: Moving on to Anatomy. Following reduction, he complains of numbness in his fingers			
Question 3 Ulnar nerve **Subject:** Anatomy (Discussion) LOA: 1	Describe the course of the ulnar nerve around the elbow.	**Passes through the elbow posterior to the medial epicondyle of the humerus**.	**Bold**
	What clinical findings would you expect if the ulnar nerve is injured at the elbow?	Sensory, loss of sensation- • **Medial half of the palm** - Palmar cutaneous branch • **Medial one and a half fingers, and the associated dorsal hand area** - Dorsal cutaneous branch • **Palmar surface of the medial one and a half fingers** - Superficial branch	sensory
	Prompt: What motor findings would you expect *What does ulnar nerve supply?*	Motor, unable to- • **FLEX and ADDUCT hand at wrist** - Flexor carpi ulnaris • **FLEX Distal interphalangeal joints of 4th and 5th digits** - Flexor digitorium profundus III and IV • **FLEX and ABDUCT 5th MCPJ** - Hypothenar muscles: Abductor digiti minimi, Opponens digiti minimi, Flexor digiti minimi • **ADDUCT thumb** - Adductor pollicis, half of flexor pollicis brevis • **ABDUCT and ADDUCT 4th and 5th fingers** - Interosseous muscles, 3rd and 4th lumbricals	2 of 5 motor
	How would you differentiate an ulnar nerve lesion at the elbow from one at the wrist? (Bonus question)	More pronounced claw hand if lesion is more distal as FCU and FDP preserved.	Concept to pass

Stem: Moving on to Physiology. As part of your assessment of the nerve injury, his upper limb reflexes are assessed			
Question 4 Reflexes **Subject:** Physiology LOA: 1	Describe the components of the stretch reflex. How is it different from the withdrawal reflex? *Prompt – describe a polysynaptic reflex*	Sensor (muscle spindle), **afferent** nerve, integrator (**monosynapse** on motor neurone), **efferent** nerve, effector (intrafusal fibres). Withdrawal reflex is a Polysynaptic reflex. Also has afferent and efferent limbs, but sensory organ is **nociceptor (painful stimulus**). Central integrator consists of **polysynaptic connections in the spinal cord** i.e. one or more interneurons and interposed between afferent and efferent neurons. Efferent limbs are motor nerves to effector muscles on the ipsilateral and contralateral sides. **Flexion and withdrawal of the ipsilateral limb** and extension of the contralateral limb.	Bold to pass Bold to pass

Stem: Moving on to Pathology. His nerve injury does not resolve and many months later he has marked wasting of his hypothenar muscles.			
Question 5 Atrophy **Subject**: Pathology Robbins 9th Ed, pages 36-37 LOA: 1	(a) What is atrophy?	**Decrease in the size of an organ or tissue resulting from a decrease in cell** size and number. Can be physiological or pathological.	Bold to pass
	(b) What are the causes of atrophy?	**Decreased workload** (eg.# immobilized in plaster) **Denervation** **Diminished blood supply** (eg.due to arterial occlusion) **Inadequate nutrition** (eg. protein-calorie deficit - marasmus-> use of adipose stores + muscle for energy) **Loss of endocrine stimulation** (eg. endometrial atrophy after menopause). **Ageing** **Pressure**	4 of 7 bold
	(c)What are the mechanisms of atrophy?	**Decreased protein synthesis** **Increased protein degradation**. May be accompanied by increased autophagy (self-eating) - where a starved cell eats its own components in an attempt to find nutrients and survive.	One bold to pass

ACEM PRIMARY VIVA C **Thursday Afternoon** ***Candidate Number:*** **AGREED MARK:**

Stem: An 85 year old man presents with heart failure. He is on verapamil. We will start with Pharmacology.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 **Subject: Pharm** Verapamil	Describe the mechanism of action of verapamil.	**Blocks** voltage gated L-type **calcium channels** (α1 subunit), reduced frequency of opening when depolarized resulting in decreased transmembrane calcium current and calcium in-flux.	**Bold** + concept of blocking Ca influx
LOA: 1	Describe the effects of verapamil on the heart and blood vessels.	**Reduced contractility/CO**, oxygen demand, Reduced impulse generation/**AVN conduction block**. **Vascular smooth muscle relaxation** (less than dihydropyridines) or reduced coronary artery spasm.	2 of 3 bold
	What are the adverse effects of verapamil?	CVS; **bradycardia**/**AV block**, cardiac arrest, **heart failure**, **hypotension** Minor: flushing, dizziness, nausea, constipation, peripheral oedema.	2 of 3 bold

Stem: Moving on to Physiology.			
Question 2 **Subject:** **Physiology** Cardiac Output LOA: 1	What two factors determine cardiac output? Can you draw a graph to show the Frank Starling law as it relates to cardiac muscle? What factors shift the Frank Starling curve?	**CO = HR x SV** **SV** is related to preload and afterload of the heart and the intrinsic contractility of the myocardial cells. **HR** – sympathetic versus parasympathetic stimulation.  The dashed lines indicate portions of the ventricular function curves where maximum contractility has been exceeded; Circulating catecholamines Inotropes (caffeine, theophylline, digitalis) Sympathetic input *All shift the curve up and to the left.* Acidosis/Hypercarbia/Hypoxia Vagal/parasympathetic stimulation Pharmacological depressants (quinidine, procainamide & barbiturates) Intrinsic depression (with heart failure) *All shift the curve down and to the right.* (The causes of this depression are not fully understood but may reflect down-regulation of β-adrenergic receptors and associated signaling pathways and impaired calcium liberation from the sarcoplasmic reticulum).	**Bold** Correctly draws and labels curve and able to discuss reason for dotted lines. **Two positive** and **two negative** factors.

Stem: The patient has right leg pain. This is a photo of his lower limbs.			
Question 3 **CCB** Photo of legs Clinical Building Block	(a) Please describe the appearance of the right leg? (b) What are potential causes for these changes?	Cyanotic, Mottled, slightly swollen right leg/foot, shiny skin. Wasting. Arterial occlusion, venous occlusion**,** small vessel occlusion, microemboli (trash foot) stasis/occlusion/insufficiency. Cellulitis	Any two to pass Any two to pass

Stem: Moving on to Anatomy. As part of your assessment you examine the distal pulses in the lower limb.			
Question 4 **Subject: Anat** Image of foot showing arteries. Arterial Supply of the Foot LOA: 1	Where do you palpate the distal pulses?	PT – Between the medial malleolus and the achilles tendon DP - Mid arch between the 1st and 2nd metatarsal or lies midway between malleoli or between EHL and EDL/EBL	Both
	Identify the dorsalis pedis artery and the surrounding landmarks on this image.	Able to point out **Dorsalis Pedis artery (3),** EHL (7), EDL (5)/EBL (6), metatarsals	DP and one adjacent structure to pass
	Describe the venous drainage of the foot.	Superficial and deep veins. Deep: paired veins which accompany all arteries internal to the deep fascia. Superficial: subcutaneous and not accompanied by arteries. Perforating veins provide one-way shunting of blood from superficial to deep veins. **Dorsal venous network/arch** of digital and metatarsal veins drain into the dorsal venous arch of the foot. Plantar venous network forms the medial marginal vein which becomes the **great saphenous vein** or lateral marginal vein which becomes the small saphenous vein.	**Bold** to pass

Stem: Moving on to Pathology. Venous thrombosis is considered to be the most likely cause for his leg swelling.			
Question 5 **Subject**: **Path** **Thrombosis** LOA: 1	1. What pathological mechanisms may contribute to venous thrombus formation in a vessel? 2. What are some of the different risk factors for venous thrombosis? *Prompt: You have named one genetic risk factor, can you name another one?* *Prompt: You have named one relating to X (e.g. stasis), can you name others with different mechanisms?*	**Endothelial injury (damage to vessel)**, **alteration in blood flow** (stasis, turbulence), **hypercoagulability** of blood. <u>Primary (genetic)</u> • Mutations – Factor V Leiden, prothrombin gene • Increased levels – factors VIII, IX, XI, fibrinogen • Deficiencies – AT3, protein C, S • Fibrinolysis defects, homozygous homocysteinuria • Non O blood group <u>Secondary (acquired)</u> • Stasis – (e.g. prolonged bed rest, immobilization, long distance travel) • Tissue injury (e.g. MI, surgery/burns/fractures) • AF • Cancer • Prosthetic cardiac valves/devices • Indwelling vascular devices (e.g. PICC, CVC etc) • External vessel compression (e.g. pregnancy (>20 weeks) May Thurner syndrome, etc) • Platelet abnormalities (e.g. DIC, HITS, Thrombocytosis) • Cardiomyopathy, nephrotic syndrome, • Hyperoestrogenic states (pregnancy, post-partum, OCP etc.) • Sickle cell anaemia • Smoking • Antiphospholipid syndrome • Hyperviscosity states (PCRV, leukaemia, hyperproteinaemia)	All bolded to pass. 2 examples of genetic causes and 2 non genetic causes with *different mechanisms* (i.e. from a different line) to pass.
	3. What are possible outcomes of a venous thrombus in a vessel?	**Propagation** (eg resulting occlusion), **embolisation**, **dissolution, organization, recanalisation.**	3 of 5 to pass.

ACEM PRIMARY VIVA C **Friday AM** ***Candidate Number:*** **AGREED MARK:**

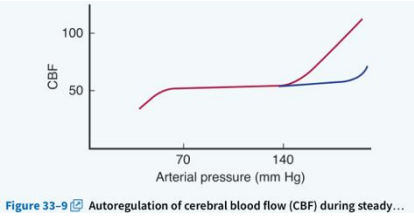
Stem: A 35-year-old man presents with severe pain in his knee following a twisting injury at football. He is given analgesia at triage. Starting with Pharmacology			
TOPIC	**QUESTIONS**	KNOWLEDGE **(essential in bold)**	**NOTES**
Question 1 Drug clearance **Subject:** Pharmacology LOA: 1 *Katzung 13th* *pp 42-46*	What is drug clearance?	Measure of the ability of the body to eliminate a drug. Rate of elimination in relation to the concentration OR Vol of plasma cleared of a drug per unit time.	Reasonable definition concept of rate over time
	What factors affect clearance?	**Concentration** – dose/ bioavailability **Elimination** – specific organ function /blood flow /protein binding Major sites of elimination are kidneys and liver – therefore factors that affect these organs function and blood flow will have most effect	**One factor for each** element
	What is the difference between capacity limited and flow dependent drug elimination? (prompt – what are the differences in elimination kinetics?)	**Capacity limited – is saturable (zero order)** e.g. aspirin, phenytoin, ethanol (so clearance varies depending on drug concentration). **Flow dependent – is non-saturable (1st order) –** most of drug **is** cleared on 1st pass of blood through an organ, so elimination depends on the rate of drug delivery to the organ - and hence on blood flow. Plasma protein binding and blood cell partitioning may also play a small role. e.g. Amitriptyline / imipramine / Labetalol / Lig/ Morphine / Verapamil	**Bold**

Stem: Moving on to Pathology. The knee swells over several hours following the injury			
Question 2 Oedema Robbins 9th edition pages 113-115 **Subject:** Path LOA: 1	a) What is oedema b) What are some of the causes of oedema *(prompt, what are the non-inflammatory causes)* c) What is the difference in the composition of the fluid, between inflammatory and non-inflammatory oedema	**Increased interstitial fluid** **Inflammatory** (acute / chronic):- infection, tissue necrosis, foreign body, immune, traumatic **Non-inflammatory**: - increased hydrostatic pressure (eg cardiac failure, DVT), hypoproteinaemia (chronic liver disease, nephrotic syndrome), lymphatic obstruction, sodium retention **Inflammatory**: - exudate**, high protein** conc, **Non-inflammatory**: - transudate, **low protein** conc (effectively an ultrafiltrate of plasma)	**Bold** **2 examples from each to pass** Must know
Stem: A knee x-ray is performed.			
Question 3 Knee x-ray **Subject:** CBB LOA:1	Describe this X ray *Prompt – is there anything else on the lateral view?*	Tibial plateau fracture, depressed lateral condyle, lipohaemarthrosis (on lateral view)	Must identify lateral tibial condyle fracture and joint effusion

Stem: Moving on to Anatomy			
Question 4 Model knee ligaments **Subject:** Anatomy Moore 7th Pp 636-642 LOA: 1	Identify the ligaments and tendons of the knee on this model	Ligaments: **Med (tibial) collateral, Lat. (fibular) collateral, Ant cruciate, Post cruciate**, posterior meniscofemoral, lat meniscofemoral (not on model) Tendons: Quadriceps, patellar	**4 bolded** + one correctly named tendon to pass
	What are the functions of the cruciate ligaments	ACL: Prevents posterior displacement of femur on tibia. Prevents hyperextension of the knee. Limits posterior rolling of the femoral condyles on tibial plateau in flexion	**1 of 3**
		PCL: Limits anterior rolling of the femur on the tibial plateau in extension. Prevents anterior displacement of the femur on tibia. Helps prevent hyperflexion of the knee.	**1 of 3**
	Describe the medial meniscus and its attachments	Crescentic. Fibrocartilage. Thickens towards joint margins. Ant horn attached to anterior intercondylar area of tibia anterior to ACL. Post horn attached to post intercondylar area of tibia anterior to PCL. Firmly attached to TCL (deep MCL or MCL) Less mobile than the lat meniscus	**3 of 7** must include 1 attachment

Stem: Moving on to Physiology. He has painful quadriceps muscle cramping in the injured limb.			
Question 5 Resting membrane potential **Subject**: Phys LOA: 1 *Ganong 25th Pp90, 130, 131, 14*	Define the resting membrane potential of a nerve	**Potential difference across the membrane** at rest with inside negative relative to outside In nerves, it is – 70 mV	**Bold**
	How is this resting membrane potential maintained?	The gradients are actively maintained by **NA^+/K^+ ATPase** NA^+/K^+ ATPase actively pumps Na out and K into the cell using ATPase for energy. Na then passively flows back into the cell via channels down concentration gradient, and K passively flows out of cell via K channels down concentration gradient. BUT at rest, there are more open K channels than Na channels, so the permeability to K is greater (passively).	**Bold** + concept Na out and K in Passive flow in opposite direction
	Describe the sequence of events that occur at the motor end plate following discharge of a motor neuron	Activation of voltage gated Ca2+ channels in presynaptic membrane Calcium influx into the cell Exocytosis of preformed ACh into synaptic cleft Diffusion of ACh across synaptic cleft Binds to post synaptic nicotinic receptor Increase Na+ and K+ conductance in end plate membrane (muscle) Generation of end plate potential. Generation of action potential in muscle fibres. Spread of depolarisation along T tubules. Ca++ released from Sarcoplasmic Reticulum (diffusion to thick and thin filaments). Binding of Ca++ to trop C uncovering myosin-binding sites on actin. Actin-myosin binding and sliding of thin on thick filaments producing movement	**6 of 12** steps
	Describe what occurs to the ACh released at the motor end plate?	ACh removed from synaptic cleft by Acetylcholinesterase Choline re-uptake Acetate to liver and metabolised	

ACEM PRIMARY VIVA C **Friday Afternoon** ***Candidate Number:*** **AGREED MARK:**

Stem: A 75-year-old man presents following a collapse. His GCS is 6/15. We will start with Physiology.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Cerebral blood flow **Subject:** Physiology LOA: 1 *Ganong,25th edition. Chapter 33 Circulation through special regions. Cerebral blood flow and its regulation.*	(a) What factors affect cerebral blood flow?	1. **Intracranial pressure** 2. **Mean arterial pressure** 3. **Mean venous pressure** at brain level 4. Local factors: pH, pCO2 – constriction and dilatation of cerebral arterioles 5. Blood viscosity	**2 of 3 Bold**
	(b) What is meant by the term autoregulation of cerebral blood flow? (You may draw a diagram).	The process by which CBF is maintained at a constant level (approx. 750ml/min) despite variation of arterial pressure (MAP 65 – 140mmHg)	Understanding of concept.  Figure 33–9 Autoregulation of cerebral blood flow (CBF) during steady…
	(c) What is the Monro-Kellie doctrine? *(prompt: what is the relationship between volume of blood, volume of CSF, and brain tissue)*	**Volume** of blood, CSF and brain tissue **must be relatively constant.** When ICP rises, cerebral vessels are compressed resulting in reduced cerebral blood flow. Rise in venous pressure also causes decreased cerebral blood flow by decreasing effective perfusion pressure and compressing cerebral vessels.	Understanding of concept.

Stem: It is planned to intubate him. Moving on to Pharmacology.			
Question 2 Drugs in the elderly **Subject:** Pharmacology LOA: 2 *Katzung 13th, 1025-1027*	(a) What physiological changes in the elderly may influence the pharmacokinetic properties of drugs	**Distribution:** Body water - decreased Lean body mass - decreased Body fat - increased Serum albumin – decreased **Metabolism:** Liver metabolism changes inconsistent. Hepatic blood flow - decreased **Elimination:** Kidney weight (% of young adult) - decreased Reduced creatinine clearance with age. **Polypharmacy and multiple drug interactions/toxicity**	Mention at least 3 physiological changes
	(b) What changes would you make to the administration of intravenous morphine in this patient compared to a younger one and why?	Reduce the initial dose, wait longer before administering a second IV dose, increase interval between repeat doses The elderly are often markedly more sensitive to the respiratory effects of opioid analgesics because of age-related changes in respiratory function. Increased distribution time to the CNS due to reduced cardiac output. The elimination half-life of morphine will be increased	At least 1 dose adjustment with correct explanation

Stem: A CT Brain is performed on this patient			
Question 3 CT Brain – intracerebral haemorrhage **Subject:** CBB	(a) Describe this CT Brain *Prompt can you see blood anywhere else?*	Large left basal ganglia **intraparenchymal haemorrhage** with **intraventricular extension** and mass effect: There is compression of the left frontal and parietal lobes, compression of the thalamus, 1 cm midline shift to the right, enlargement of the posterior horn of the left lateral ventricle	**2 Bold** and one other description of mass effect to pass
	(b) What potential clinical complications can occur as a result of this?	Decreasing GCS, focal neurological deficits, compromised airway, seizures, impending 'coning' (dilated pupil, bradycardia, hypertension), death If the patient survives, there will be severe neurological deficit and disability	At least 2 complications
Stem: Moving on to Anatomy. A Femoral central line is inserted.			
Question 4 Femoral triangle (Mc Minns Atlas Photo 365A) **Subject:** Anatomy LOA: 1	(a)Identify the boundaries of the femoral triangle on this image	**Superior: Inguinal ligament (11)** **Medial: Medial border of add longus (1)** **Lateral: Sartorius (23)** **Floor: Iliopsoas (not seen) and pectineus (19)**	**3/4** Boundaries with structure
	(b)Name and demonstrate the contents	**Femoral nerve (5), artery (4) and vein (6)** (medial - lateral) Deep inguinal lymph nodes (29), lymphatics (15)	**All 3 bold**
	(c)Which spinal roots is the femoral nerve derived from?	**L2 L3 L4**	**Bold**
	(d)What anterior thigh muscles does the femoral nerve innervate?	Sartorius, articularis genu, and quadriceps including rectus femoris, vastus medialis, vastus lateralis and vastus intermedius	**4/6**

Stem: Moving on to Pathology. The patient has an intracerebral haemorrhage.			
Question 5 Intracerebral haemorrhage **Subject**: Pathology LOA: 2 Robbins and Cotran's Pathologic Basis of Disease; 9th Edition; Chapter 28: The Central Nervous System; Page 1268	(a) What are the main pathophysiological causes of spontaneous intracerebral haemorrhage?	**Hypertension and cerebral amyloid** are the main causes. Other causes include systemic coagulation disorders, neoplasms, vasculitis, aneurysms, and vascular malformations.	**1 of 2 bold** and two others to pass
	(b) Which areas of the brain do hypertensive intracerebral haemorrhages most commonly occur?	Hypertensive intracerebral haemorrhage may originate in the putamen (50% to 60% of cases), thalamus, pons, cerebellar hemispheres (rarely) Accept basal ganglia, brainstem	At least two to pass.
	(c) Describe the pathophysiology of cerebral amyloid angiopathy? (Bonus question)	There is deposition of amyloidogenic peptides in the walls of medium- and small-calibre meningeal and cortical vessels. This deposition can result in weakening of the vessel wall and risk of haemorrhage	

Stem: A 30-year-old male presents with a severe exacerbation of Asthma. We will start with Pathology			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Asthma **Subject:** Path LOA: 1	a) What is the pathological definition of asthma	a) Disorder of the conducting airways usually caused by an immunological reaction, marked by episodic **bronchoconstriction** due to airway sensitivity to a variety of stimuli, **inflammation** of the bronchial walls, and increased **mucus** secretion.	Bold to pass
	b) Name the main inflammatory cells involved	b) A wide range of inflammatory cells are involved – (lymphocytes, eosinophils, mast cells, macrophages, neutrophils)	Name 2
	c) How is asthma categorized pathologically?	c) Types of asthma: • **Atopic** – Most common. IgE (type 1) hypersensitivity reaction – T_H2 mediated. It is characterised by an immediate (bronchoconstriction) and late-phase (inflammation) reactions. TH2 cytokines, IL-4, IL-5 and IL-13 are important mediators (IL-17 & IL-9 in some). • **Non-Atopic** – No evidence of allergen sensitization (& negative skin test). Family history is rare. • **Drug induced (eg aspirin)** • **Occupational (eg epoxy fumes)**	Must list at least 2 types including atopic and trigger mechanism (Robbins, 9th ed. 679-682)

	d) Name some common triggers	d) Triggers:
		• **Atopic**
		  ○ Triggered by environmental factors (dust, pollens, food, etc.) in synergy with other pro-inflammatory cofactors such as respiratory viral infections. Positive family history and skin test for allergens.
		• **Non-Atopic** (Triggers are less clear)
		  ○ Viral respiratory infections (rhinovirus), parainfluenza, RSV)
		  ○ Inhaled air pollutants –smoking, sulfur dioxide, ozone, nitrogen dioxide
		  ○ Exercise induced
		  ○ Exposure to cold
			2 triggers

Stem: Moving to Physiology			
Question 2 Airway resistance **Subject**: Phys LOA: 1	a) Describe factors affecting airway resistance **PROMPT**: What is Poiseulle's law? **BONUS**: Define dynamic compression of airways and its effects on flow	• In laminar flow, resistance is proportional to **the length** of the tube and **viscosity**, and inversely proportional to fourth power of the radius of the tube. (**Poiseuille's Law**: R = 8 x Length x Viscosity/n x **radius to 4th power**) • Turbulent flow is most likely to occur at high Reynolds numbers, that is, when inertial forces dominate over viscous forces. (Reynolds No. = density x Diameter x Velocity /Viscosity) • Highest in the medium-sized bronchi; low in the very small airways • Airway resistance decreases as lung volume rises because the airways are then pulled open by radial traction • Bronchial smooth muscle is controlled by the autonomic nervous system; stimulation of β-adrenergic receptors causes bronchodilation. Reduced alveolar PCO2 causes increased resistance. Intrapleural pressure > alveolar pressure causing airway compression. Dynamic compression of airways limits airflow during forced expiration.	Must understand Poiseuille's law (bolded) List at least 2 other factors. (West, 9th ed. 108-119) Concept
Stem: Moving to Pharmacology. He requires intubation. Ketamine is used as the induction agent.			

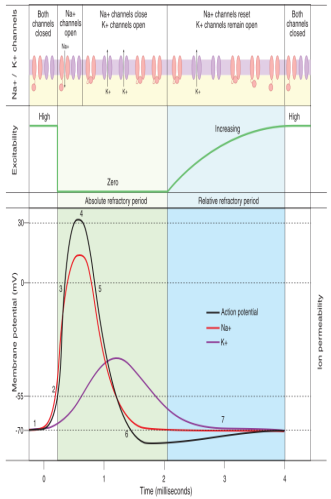
Question 3
Ketamine
 	
Subject: Pharm
 	
LOA: 1
	a) What is the mechanism of action of ketamine?
	
	b) Besides the anaesthetic effect what are the other indications of ketamine?
	
	c) In what conditions might you avoid using ketamine?
	
	BONUS:
	What are the organ system effects of ketamine?
		• Ketamine mechanism of action is complex, but the major effect is probably produced through the **inhibition of the NMDA (N-methyl- D aspartate) receptor complex**
		or
		• **blockade** of the membrane effects of the excitatory neurotransmitter glutamic acid at the **NMDA (N-methyl- D aspartate) receptor complex**
		
		• Analgesia, Bronchodilator effect in asthma, Acute behavioural disturbance, Procedural sedation
		
		• Allergy, RICP, RIOP, Recent or current URTI, Shock
		
		• Organ system effects
		  ○ CNS - Cerebral vasodilation and increase blood flow
		  ○ CVS - Increase in BP,HR,COP (centrally mediated sympathetic stimulation)
		  ○ Resp - Relaxation of bronchial smooth muscle
		  ○ Other - Increase salivation (secretion), lacrimation, nystagmus, myoclonus
			Bold
			
			2 to pass
			
			at least one to pass (excluding allergy)

Stem: Post intubation he is difficult to ventilate and has decreased air entry over his right side. This is the clinical building block.			
Question 4 CXR **Subject:** CBB	Describe his CXR.	AP film. Large/complete right **pneumothorax** with significant midline shift to left. ETT shifted to left but in correct place above carina. Left lung mid/lower zone and costophrenic angle obscured by left shifted heart with possible collapse. All suggest pneumothorax under radiological **tension.** Cardiac monitoring leads noted.	Bold
Stem: Moving to Anatomy. You insert a second intravenous (IV) cannula into his right cubital fossa.			
Question 5 Cubital fossa (Mc Minns, 7th Ed Page 151) **Subject**: Anat LOA: 1	a) Describe the boundaries of the cubital fossa (NO IMAGE)	• Superiorly - imaginary line between epicondyles of humerus • Medially - lateral border pronator teres • Laterally - medial border brachioradialis • Floor - brachialis (and supinator) • Roof - deep fascia reinforced by bicipital aponeurosis, subcutaneous tissue and skin	3/5 correctly described
	b) In this photo identify the contents of the cubital fossa	**Radial nerve** – superficial terminal branch (16), Biceps/tendon (3), **Brachial artery (4)** dividing into Radial artery (13), and Ulnar artery (17), **Median Nerve (12)**, Brachialis (5)	Bold
	c) Describe the superficial lymphatics of the upper limb	• Originate from lymphatic plexuses in hand and **ascend mostly with superficial cephalic and basilic veins** • Some accompanying the basilic veins enter the cubital LNs • Efferent vessels from here drain to **axillary LNs** • Lymphatics accompanying the cephalic veins enter the axillary LNs with some entering deltopectoral LNs earlier	Bold

Stem: A 65-year-old alcoholic woman presents following a fall. She is short of breath. We will start with Pathology.

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Cardiomyopathy **Subject:** Path LOA: 1	a) What is the definition of cardiomyopathy?	• Heterogenous group of diseases of the myocardium associated with **mechanical and/or electrical dysfunction** that usually (but not invariably) exhibit inappropriate **ventricular hypertrophy or dilation** • Primary cardiomyopathies can be congenital or acquired • Secondary cardiomyopathies have myocardial involvement as a component of a systemic or multisystem disorder	Bold
	b) What are the types of cardiomyopathy? Give a cause of each. **PROMPT**: Structural changes?	**Hypertrophic**. 75% genetic cause. Autosomal dominant HCM. **Dilated –** alcohol, myocarditis, idiopathic, peripartum, genetic **Restrictive**. Infiltrative: Amyloidosis. Sarcoidosis. Non-infiltrative: Idiopathic, scleroderma	Bold, with one example for each
	c) What type of cardiomyopathy is alcoholic cardiomyopathy?	Dilated	

Stem: Moving onto Anatomy. She has upper limb weakness. Here is a photo of the right brachial plexus.			
Question 2 Brachial plexus (Mc Minns, Pg 142, 7th Ed) **Subject:** Anat LOA: 1	a) Identify the components of the brachial plexus b) What are the nerve roots that make up the posterior cord of the Brachial Plexus? c) What are the terminal branches of the posterior cord and what do they supply?	Axillary nerve (1), Lateral cord (6), Lat root median n (8), Medial cord (12), Med cut n of arm (13), Medial cut n of forearm (14), Medial root median n (16), Musculocutaneous (18), Median n (17), Posterior cord (20), Ulnar n (26) **C5,6,7,8 and T1** **Axillary nerve** (C5, 6) • Joint - glenohumeral (shoulder) • Muscles – deltoid and teres minor • Skin over inferior aspect of deltoid **Radial nerve** (C5-T1) • All muscles in the posterior compartment of arm and forearm • Skin over postero, inferolateral arm, posterior forearm and dorsum of hand lat to ring finger Other branches are • Upper scapular n C5 supplies subscapularis • Lower subscapular n C6. Supplies inferior part of subscapularis and teres major muscle • Thoracodorsal n C6,7,8 supplying Lat dorsi	Requires 6 in total Bold to pass Both nerves and one example of supply for each
Stem: She has sustained a laceration to her arm. This is the clinical building block.			
Question 3 Clinical image **Subject**: CBB	Describe this photo.	• **Laceration on posterior (dorsal) aspect of right forearm extending to the medial (ulnar) aspect** • Length – any reasonable estimation accepted • **Depth** – extends through subcutaneous fat into muscle • No evidence of active bleeding	Bold to pass

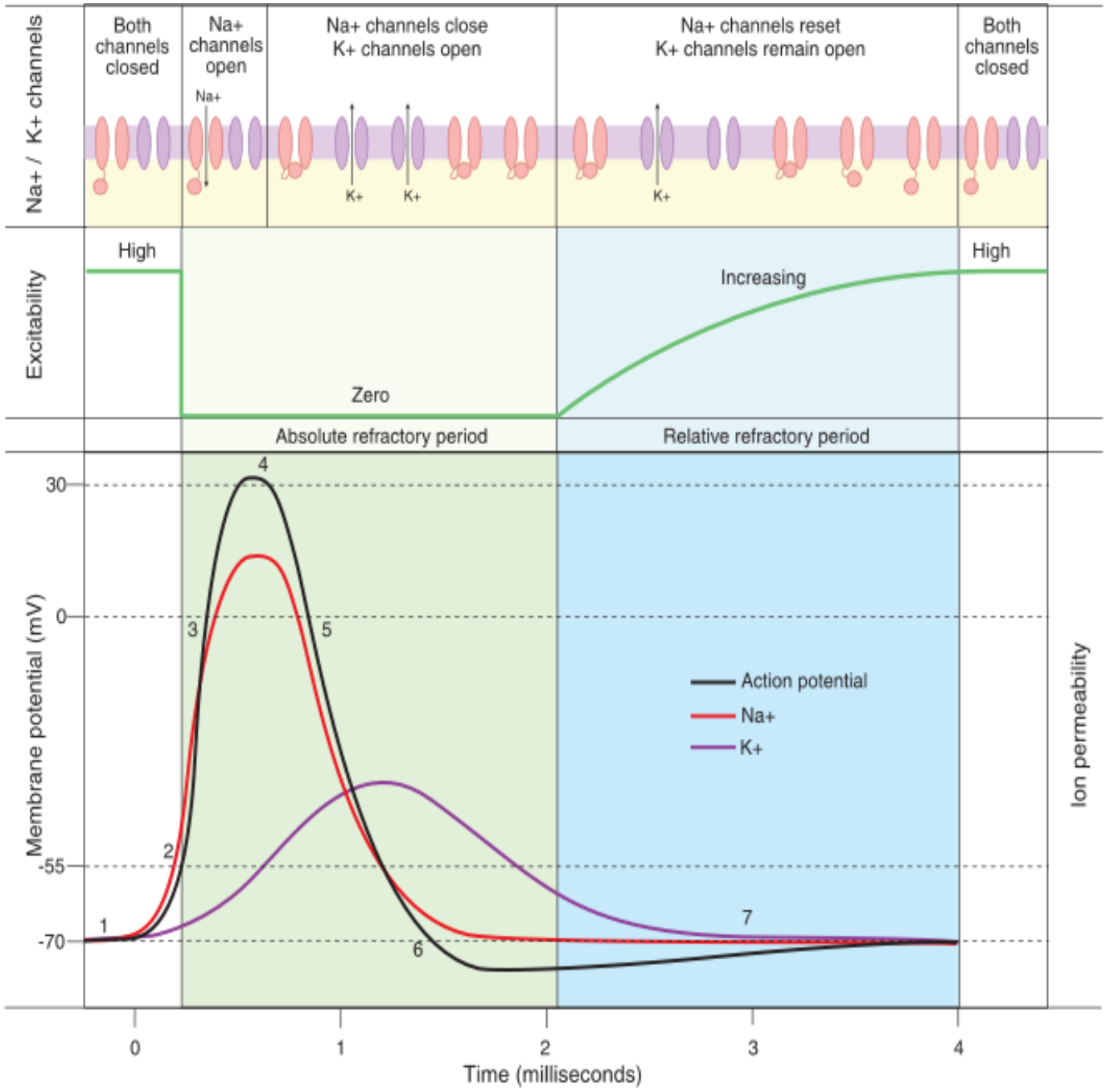
Stem: Moving onto Physiology. She has a nerve injury.			
Question 4 Nerve action potential and excitation **Subject:** Phys: LOA: 1	a) Please draw a nerve action potential and indicate the sequence of events that occur	It depends on the change in conductance of Na and K ions. 1. When a **depolarising stimulus** occurs, the voltage-gated **Na channels** become active, Na enters the cell 2. When the **threshold potential** is reached the voltage-gated Na channels overwhelms the K channels. 3. Entry of Na causes opening of more voltage-gated Na channels and further depolarisation (positive feedback loop) resulting in the upstroke of AP 4. The membrane potential moves close to the equilibrium potential for Na (+60mV). 5. The voltage gated Na channels then enter an inactivated state for a few milliseconds before returning to the resting state 6. Reversal of membrane potential limiting further Na influx and **opening of voltage-gated K channels** results in **repolarisation** and end of AP 7. Slow return of K channels results in hyperpolarization 8. Returns to resting membrane potential	Bold to pass 
Stem: Moving onto Pharmacology.			
Question 5 Ethanol **Subject:** Pharm LOA: 2	a) What are the pharmacodynamic effects of ethanol?	CNS: sedation, disinhibition, impaired judgement, impaired motor skills, ataxia, slurred speech -> coma, respiratory depression CVS: depressed contractility Smooth Muscle: vasodilator (-> hypothermia)	3 x CNS plus 1 other
	b) What are the pharmacokinetics of ethanol?	Absorption – rapid from GI tract peak levels within 30 min Distribution – rapid Vd ~TBW 0.5-0.7L/kg **Metabolism** - liver – **ZERO order** and mainly by alcohol dehydrogenase Excreted - lungs, urine	Bold plus 2
	c) What does zero –order kinetics mean?	That independent of the drug concentration the elimination of the drug occurs at a constant state	Understand concept

Stem: A 50-year-old man who is on dialysis is being treated for right hip osteomyelitis. We will start with Pathology.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Osteomyelitis **Subject:** Path LOA: 1	a) Describe the pathogenesis of acute osteomyelitis	Haematogenous spread of organism to bone Extension from a contiguous site Local bone injury and direct organism entry	2/3
	b) What organisms cause osteomyelitis?	**Staphylococcus aureus** >80% of pyogenic ones Others: Escherichia coli, Klebsiella pneumoniae, Pseudomonas aeruginosa from IVDU and GU; Haemophilus influenzae, Group B streptococcus – in neonates	Bold + 1 other organism to pass
	c) What pathological changes occur to the bone?	• **Acute inflammation -** neutrophilic • **Abscess –** sub-periosteal / surrounding soft tissue • **Necrosis –** dead bone - sequestrum • Involucrum (fibrous tissue and reactive bone deposition) forms around devitalized infected bone	Bold to pass

	d) What are the possible sequelae of osteomyelitis?	• **Resolution**
		• **Chronic – up to 25%**
		  ○ Acute flare-ups
		  ○ Pathological fracture
		  ○ Endocarditis
		  ○ Severe sepsis
		  ○ SCC in draining sinus tracts
		  ○ Sarcoma in infected bone
			Bold + 1 complication of chronic osteomyelitis to pass

Stem: Moving onto Anatomy. An X-ray of his right hip was ordered.			
Question 2 Hip (x-ray) **Subject:** Anatomy LOA: 1	a) Identify the proximal landmarks of the femur on this x-ray	**Head, neck, greater & lesser trochanters, shaft**	Bold to pass
	b) Describe the blood supply to the head of the femur	Medial and lateral **circumflex** femoral arteries • Usually branches of deep artery of thigh (profunda femoris) • These branch to form retinacular arteries (medial > lateral), feed under posterior unattached capsule (medial) or through iliofemoral ligament (lateral) • **Distal to proximal** Artery to head of femur – branch of obturator artery (less important)	Bold to pass
	c) What does the femoral nerve supply?	• **Anterior thigh muscles (quadriceps)** • Pectineus, Sartorius, iliacus • Articular branches to hip and knee joints • Cutaneous branches to anteromedial thigh • Terminal cutaneous branch is saphenous nerve to anteromedial knee, leg, foot	Bold plus one sensory
Stem: Moving onto Pharmacology. He is being treated with Vancomycin.			
Question 3 Vancomycin **Subject:** Pharm LOA: 2	a) What is the mechanism of action of vancomycin?	**Inhibits cell wall synthesis** by binding to peptidoglycan pentopeptide. This inhibits transglycosylase preventing crosslinking and weakening cell wall/membrane **Bactericidal**	Bold to pass
	b) What are the target organisms for vancomycin?	**Gram +ve** (**Staph** incl MRSA, **Enterococci**), G +ve anaerobes (**C. difficile**)	Any 2 to pass
	c) What clinical condition requires dose adjustment?	Renal impairment, Morbid obesity	Either
Stem: A 12 lead ECG was performed. This is the clinical building block.			
Question 4 ECG (Hyperkalaemia) **Subject:** CCB	Describe and interpret this ECG	Sinus rhythm / sinus tachycardia (rate 100-110 bpm), normal axis, PR normal, normal QRS width, peaked T waves (esp V_2-V_6 and inferior leads) Poor R wave progression Suggestive of hyperkalaemia	Structured approach or recognition of abnormalities

Stem: Moving onto Physiology. His potassium is elevated at 7mmol/L.			
Question 5 Renal handling of K+ **Subject**: Phys LOA:1	a) How does the kidney handle potassium? **PROMPT**: What happens to potassium in different parts of the nephron?	**Filtered** in **glomerulus** (~600meq/24hrs) R**eabsorbed** in **proximal tubules** & thick ascending limb of loop of Henle (~560meq/24hrs : >90%) • active transport via Na-K-2Cl co-transporter **Secreted / excreted** by **distal tubules/collecting ducts** (~502meq/24hrs) • amount proportionate to flow rate through distal tubules (rapid flow rates reduces intratubular K^+ concentration, thus facilitating secretion) • under influence of aldosterone (induces K^+ secretion)	Bold to pass (need to have process and site)
	b) What other major ions are involved in potassium transport in the nephron?	Na^+, H^+	Both
	c) How do hydrogen ions influence potassium transport in the nephron?	Coupled to H^+ secretion [if H+ secretion increased, then K^+ excretion decreased as K+ is reabsorbed in exchange for H^+ (H,K-ATPase) in collecting duct cells]	Concept


Na+ / K+ channels
Both channels closed
Na+ channels open
Na+ channels close K+ channels open
Na+ channels reset K+ channels remain open
Both channels closed
Na+
K+
K+
K+
Excitability
High
Zero
Increasing
High
Absolute refractory period
Relative refractory period
Membrane potential (mV)
30
0
-55
-70
1
2
3
4
5
6
7
Action potential
Na+
K+
Ion permeability
0
1
2
3
4
Time (milliseconds)

ACEM PRIMARY VIVA D Thursday Morning ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE	NOTES
Stem: A 25-year-old female presents with malaise, fever and this rash.			
Question 1 Rash (picture) **Subject** CBB	(a) Describe the rash. (b) What are the possible causes for her rash?	Red, maculopapular rash with areas of coalescence. Non-vesicular, non-pustular, some pigmented lesions. (a)Infective: viral exanthem, measles, rubella, erysipelas, scalded skin syndrome, TSS (b)Allergic dermatitis, atopic dermatitis (c)Drug reaction	Concept 2 infective plus one other
Stem: Moving onto Pathology. You suspect Toxic Shock Syndrome.			
Question 2 Staph Aureus **Subject:** Pathology LOA: 1	(a) Describe the virulence factors of Staph aureus *Prompt: How does Staph aureus cause disease*	**Surface proteins:** involved in adherence- (express receptors for fibrinogen (and others) to bind to host endothelial cells, and artificial materials), and evade host immune response **Secreted enzymes**: degrade proteins (promoting invasion and destruction) e.g. lipase degrades skin lipid associated with ability to produce abscess **Secreted toxins**: that damage host cells: - Alpha toxin- membrane depolarisation/ damage - Beta toxin- sphingomyelinase - Exfoliative A and B toxin - Gamma toxin and leukocidin - Superantigens –TSS and food poisoning	Toxins with example plus 1 other bold.
	(b) What are the risk factors for Toxic Shock Syndrome?	Use of tampons, Post op wound infection, Post partum, Nasal Packs, Staph or strep skin infection	Any 2
	(c) What are the clinical features of Toxic Shock Syndrome?	Hypotension (shock), acute renal failure, coagulopathy, respiratory failure, soft tissue necrosis at site of infection, a generalized erythematous rash,	Any 3

Stem: Moving onto Pharmacology. She has taken excess amounts of Paracetamol for her pain.			
Question 3 Paracetamol **Subject:** Pharm LOA: 1	(a) Describe the pharmacokinetics of oral paracetamol. *Prompt: How is it metabolised?*	Absorption: Well absorbed from the GIT. Peak plasma levels 30-60 minutes. It is slightly protein bound. Distribution: T ½ of 2-3 hours. This is increased in liver disease to >6 hours Metabolism: **Hepatic: >95% undergoes glucuronidation and sulfation.** **5% undergoes metabolism via CYP 450 mechanisms** (Phase 1 reaction – hydroxylation) to form NAPQI. NAPQI is toxic, but usually detoxified by glutathione.	Bold to pass
	(b) What is the toxic dose of paracetamol?	150 - 200mg/kg in adult	
	(c) How does paracetamol cause toxicity?	In ODs → glucuronidation and sulfation pathways are saturated and paracetamol is broken down by a Phase 1 reaction. More NAPQI is formed → this consumes glutathione (GSH). Once GSH is depleted, NAPQI then becomes hepatotoxic.	Concept to pass
Stem: Moving onto Anatomy. A pelvic examination is performed.			
Question 4 Pelvis (Female Model MS1) **Subject:** Anatomy LOA: 1	(a) Using this model, identify the major anatomical structures.	Pubic symphysis, bladder, vagina, uterus, rectum, sacrum, external anal sphincter, ovary, fallopian tube, Broad ligaments, internal and external iliac vessels	5 to pass
	(b) Describe the course of the iliac arteries.	**Common iliac origin from aorta L3** Follows medial border of psoas to pelvic brim Divides at level of L5-S1 **Internal iliac artery** enters pelvis **External iliac artery** follows iliopsoas ends at the inguinal ligament and becomes **femoral artery** at **mid inguinal point**	Bold to pass
	(c) What is the blood supply of the uterus? *Prompt: What is the origin of the uterine artery?*	**Uterine artery** from the anterior division of the **internal iliac artery**. Crosses above the ureter on its course to the uterus. Commonly anastomoses with the vaginal and ovarian arteries.	Bold

Stem: Moving onto Physiology. She becomes hypotensive.			
Question 5 Shock **Subject**: Physiology LOA: 1	(a) Describe the receptors that respond to the fall in blood pressure. *Prompt: How do these receptors respond to this change?*	**Baroreceptors (**carotid sinus, aortic arch, atria) Reduced stretch -increased sympathetic stimulation - tachycardia and generalized vasoconstriction (sparing of brain and heart). With increased shock, paradoxical bradycardia (unmasking of vagal depressor reflex) then tachycardia again with further shock. **Chemoreceptors (**carotid body, aorta) Stimulated by reduced blood flow and acidosis. Stimulates vasomotor areas in medulla with increase vasoconstriction.	Receptors plus concepts
	(b) Describe the non-cardiovascular compensatory responses to shock. *Prompt: How do the kidneys compensate for shock?*	**Renal response**: Efferent arterioles constricted more than afferent. Renal plasma flow decreased more than GFR - filtration fraction (GFR/plasma flow). **Na+ retention**: Retained nitrogenous products of metabolism (uremia) **Angiotensin II:** plasma renin causing AG II, AG II maintains BP and causes stimulation thirst center in brain. **Vasopressin:** Retain Na+ and H2O. **Aldosterone**: Stimulated by circulating AG II and ACTH. Helps retain Na+ and H2O. **Adrenal stimulation:** Adrenal medulla secretion catecholamines **Increased circulation NA:** Increased discharge sympathetic NA nerves	Renal plus 2

ACEM PRIMARY VIVA D Thursday Afternoon ***Candidate Number:*** **AGREED MARK:**

Stem: A 52 year old man presents following a fall. He is cachectic and has multiple bruises. Liver function tests are performed.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Blood tests (LFTs) **Subject**: CBB	(a) Describe the abnormalities on this investigation?	**Elevated bilirubin, ALP, GGT, transaminases** Consistent with mixed picture (Normal lipase, normal albumin and coags – suggests normal synthetic function)	Bold to pass
	(b) What could be causing these abnormalities?	**Biliary obstruction** - intraluminal (stone), luminal (malignancy/stricture), extraluminal (malignancy); medications, autoimmune **parenchymal liver** – alcohol, ischemia, infection, toxins	3 causes

Stem: Moving on to Physiology			
Question 2 Bilirubin **Subject:** Physiology LOA: 1	(a) How is bilirubin produced in the body?	By breakdown of haemoglobin (heme is converted to biliverdin and then on to bilirubin)	Concept to pass
	(b) How is bilirubin metabolised?	• Bound to albumin in the circulation • Dissociates in the liver and free bilirubin enters liver cells (Liver: Organic Anion Transport Polypeptide- OATP) • **Conjugation in liver cells** (UDP glucuronyl transferase located in smooth endoplasmic reticulum acts on the bilirubin to form bilirubin-diglucuronide (BiliG) which is H2O soluble) • BiliG is actively transported to biliary canaliculi, bile ducts and then to intestine. (small amounts of BiliG and free Bilirubin leak into the circulation.) • **Intestinal phase:** Intestinal bacteria acts on the BiliG to form unconjugated bilirubin and urobilinogen. These are excreted via the gut. • **Enterohepatic circulation**: unconjugated Bilirubin and urobilinogen can re-enter the portal circulation. • Urobilinogen may enter the general circulation to be excreted by the kidneys.	Bold to pass
	c) Describe the composition of bile.	• 97% water • bile pigments (conjugated bilirubin + biliverdin) • bile salts (cholic acid, chenodeoxycholic acid, deoxycholic acid, lithocholic acid) • inorganic salts others: cholesterol, fatty acids, lecithin, fat	3 to pass

Stem: Moving on to Anatomy**.** He has a tender right hand and wrist.			
Question 3 Hand Model **Subject:** Anatomy LOA: 1	a) On this model identify ligaments of the wrist. *Prompt: What are the joints in the wrist?*	Ulnar collateral ligament (32), Interosseous membrane (21), radial collateral ligament (36), dorsal radiocarpal ligament (33), dorsal radioulnar ligament (34), intercarpal ligaments (37), Palmar carpometacarpal ligaments (1), palmar radiocarpal ligament (5), palmar radioulnar ligament (7)	4 to pass
	b) What movements occur at the wrist and which muscles produce them?	**Flexion:** FCR, FCU plus thumb/finger flexors, PL, APL **Extension:** ECRL, ECRB, ECU plus thumb/finger extensors **Abduction:** APL, FCR, ECRL, ECRB (limited to 15 degrees due to radial styloid) **Adduction:** ECU, FCU Combined movements = **circumduction**	4 movements with one muscle for each to pass
Stem: Moving onto Pathology.			
Question 4 Jaundice **Subject:** Pathology LOA: 1	a) What are the causes of jaundice? *Prompt: are there different types of jaundice? (hyperbilirubinaemia)*	**Predom unconjugated** 1. ↑ production of bili: haemolysis, resorption of haemorrhage, thalassaemia 2.↓hepatic intake: drug interference with membrane carrier systems, Gilbert sy 3. impaired conj: phys J of newborn, breast milk J, Crigler-Najjar sy, Gilbert sy, hepatitis (viral, drugs, auto-immune, cirrhosis) **Predom conjugated** 1.Impaired bile flow (cholangiopathy, biliary stricture, malignancy, choledocholithiasis) 2.Deficiency canalicular membrane transporters (Dubin Johnson sy, Rotor sy)	Bold plus 1 cause each
	b) Apart from jaundice, what are the clinical features of liver failure?	Icterus, pruritis, fetor hepaticus, palmar erythema, spider angiomata, hypogonadism, gynaecomastia, encephalopathy(asterixis), coagulopathy, hepatorenal syndrome, hepatopulmonary syndrome, portal HT(varices, ascites, caput medusae)	5 features.

Stem: Moving on to Pharmacology. Ethanol abuse is the most likely cause of his symptoms.			
Question 5 Ethanol **Subject:** Pharmacology LOA: 1 Katzung 13th edition pp 384-386	a) Describe the pharmacokinetics of ethanol. *Prompt: How is it metabolised?* b) What does zero-order kinetics mean? c) What other drugs have zero order kinetic metabolism.	Absorption: rapid from GIT (peak level in 30mins) Distribution: rapid. Vol of Distribution: TBW (0.5-0.7 L/kg) Metabolism: **Predominantly liver**. Mainly by **alcohol dehydrogenase** and less by microsomal ethanol oxidising system (MEOS). **Zero-order**. Excretion: Lungs, urine (small amounts) Elimination occurs at a constant rate independent of drug concentration Phenytoin, theophylline, warfarin, salicylate, heparin, paracetamol	Bold to pass concept one

ACEM PRIMARY VIVA D **Friday Morning** ***Candidate Number:*** **AGREED MARK:**

Stem: A 51-year-old male has presented with abdominal pain and vomiting. He is tachycardic and hypotensive. We will start with Pathology			
TOPIC	QUESTIONS	KNOWLEDGE	NOTES
Question 1 Shock **Subject:** Path LOA: 1 Robbins 9th edition P131-134	(a) What is the definition of shock?	Reduction in cardiac output or the effective circulating blood volume; the result is hypotension followed by **impaired tissue perfusion** and **cellular hypoxia.**	Bold to pass
	(b) What are the major categories of shock? Please give examples.	Cardiogenic e.g. AMI, cardiotoxins, arrhythmia Hypovolaemic e.g. haemorrhage, burns, GI losses Septic/ systemic inflammation e.g. sepsis, pancreatitis, trauma (independent of haemorrhage) Distributive e.g. anaphylactic, adrenal crisis, Neurogenic e.g. spinal injury, spinal anaesthetic Obstructive e.g. tension pneumothorax, cardiac tamponade, PE	3 categories with 1 example of each

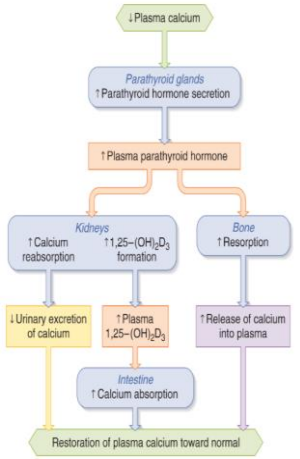
Stem: Moving on to Anatomy. On examination he has a tender lump in his groin.			
Question 2 Inguinal canal **Subject:** Anat LOA: 1 *Moore 7th edition P202-213 inc. table B2.1*	(a) Describe the anatomy of the inguinal canal *Prompt – what are the boundaries?*	Oblique, inferomedial passage, 4cm long, superior to medial ½ of inguinal ligament. **Deep (int) ring superior to middle of ing lig**, lateral to inf epigastric a. **Superficial (external) ring superolateral to pubic tubercle** (split in external oblique aponeurosis) Ant wall – ext oblique aponeurosis Post wall - transversalis fascia (+conjoint tendon) Roof - transversalis fascia, internal oblique and transversus abdominis Floor – inguinal ligament	Locations of deep and superficial rings Basic concepts of walls, roof and floor
	(b) What is the difference between a direct and an indirect inguinal hernia?	**Direct (acquired)** Weakness of anterior abdominal wall, traverses medial 1/3 of canal, exits superficial ring lateral to cord, rarely enters scrotum **Indirect (congenital)** Traverses entire canal within patent processus vaginalis, goes from internal to external ring, inside the cord, commonly into scrotum/labium majus	Basic concepts of direct and indirect hernias and their different directions
	(c) What underlying structures may be involved in this lump?	**Small bowel,** large bowel, mesentery / omentum, other abdominal viscera(bladder, ovaries, appendix)	Bold plus 1
Stem: Abdominal X-rays are performed.			
Question 3 Erect and Supine AXR **Subject:** CBB	a) Please describe these images b) What is the diagnosis?	**Multiple** centrally distributed **distended** bowel loops Multiple **air/fluid levels** Hernia not seen **Bowel obstruction** (level not required)	Bold concepts Diagnosis

Stem: Moving on to Physiology. He has reduced urinary output.			
Question 4 Renal blood flow **Subject:** Phys LOA: 1	(a) What is the normal renal blood flow?	**1.2 – 1.3 l/min (~25% of C.O.)**	Either bold
	(b) Describe the factors which determine renal blood flow.	**Perfusion pressure** (systemic MAP) **Renal arterial effects** (local constriction from NA and Ang II, dilation from ACh, PGs, dopamine) **Renal nerves** (symp/constrict/decr RBF) **Autoregulation** (myogenic, NO, Ang II), BONUS Regional differences cortex to medulla	3 of 4 bold
	(c) How does hypotension activate the renin-angiotensin system? *Prompt: what stimulates renin release?*	Hypotension leads to **reduced perfusion pressure of the afferent glomerular arteriole**, stimulating release of **renin** by the JG cells	Bold concept
Stem: Moving on to Pharmacology. A central venous line is inserted using local anaesthetic.			
Question 5 Local Anaesthetics **Subject**: Pharm LOA: 1	(a) What is the maximum safe dose of lignocaine for local anaesthesia?	Plain – **3mg/kg** (to maximum 300mg) With Adrenaline - **5mg/kg** (to max 500mg)	(3-5mg/kg) (5-7mg/kg)
	(b) What factors affect absorption of lignocaine after local infiltration?	Dose, site of injection, drug-tissue binding, tissue blood flow, vasoconstrictors	3/5 factors
	(c) What are the toxic effects of lignocaine?	**CNS:** EARLY/MILD: circumoral /tongue numbness, metallic taste, paraesthesia, sedation MODERATE: nystagmus, muscle twitching, N&V, tinnitus SEVERE seizures, sedation **CVS:** cardiovascular collapse, hypotension, bradycardia, rarely, arrhythmia, worsen CCF or conduction blocks **GIT**: anorexia N&V (through CNS effects) **Haem:** methaemoglobinaemia **Allergy:** rare with amides	CNS + 2 examples CVS + 2 examples

ACEM PRIMARY VIVA D Friday Afternoon ***Candidate Number:*** **AGREED MARK:**

Stem: A 50-year-old woman presents in an agitated state after self-harming. Diazepam is prescribed. We will start with Pharmacology.			
TOPIC	QUESTIONS	KNOWLEDGE	NOTES
Question 1 Benzodiazepines **Subject:** Pharmacology LOA: 1 *Katzung 13th, 374-7*	a) What is the mechanism of action of benzodiazepines? *Prompt: What receptor is involved?*	Binds to molecular components of **$GABA_A$ receptor** in neuronal membranes in **CNS** (ɣ subunit of the pentamer). **This receptor is a chloride ion channel.** The BDs do not substitute for GABA (major inhibitory neurotransmitter) but appear to enhance GABA's effects without directly activating $GABA_A$ receptors or opening the chloride channels. This causes an increase in the frequency of channel-opening events.	**Bold** plus concept
	b) What are the organ level effects of Diazepam?	**Sedation** – calming effect, **anxiolysis**; low dose effect- psychomotor & cognitive depression, amnesia **Hypnosis**; **Anaesthesia** – at higher doses; **Anticonvulsant** effect; **Muscle relaxation** **Respiratory depression & Cardiovascular depression** – at higher doses and when hypovolaemic/CCF/chronic heart dis.	3 of bold to pass
	c) What are the clinical uses of Diazepam in the ED?	Anticonvulsant, sedation of agitated patient, Etoh/benzo withdrawal, various toxidromes	2 to pass (can take into account part b answer)
Stem: She has injured her hand. This is the clinical building block.			
Question 2 Photo of hand **Subject:** CBB *Moore's 7th edition page 775, figure 6.77*	a) Please describe the findings on this photo.	Deep horizontal laceration across distal palmar surface. Exposed fat, tendon, muscle and bone (cartilage). Reduced flexion of 2nd, third, fourth digits, pallor.	Reasonable description
	b) What clinical examination findings would you seek to assess the extent of her hand injury?	Digital nerve – loss of distal sensation Digital artery – bleeding, loss of distal perfusion FDP – unable to flex DIP FDS – unable to flex PIP	Nerve plus artery plus tendon

Stem: Moving on to Anatomy.			
Question 3 Palmar space (Model NS13) **Subject:** Anatomy LOA: 1 Moore's 7th edition p. 785, Table 6.16, Fig 6.86	a) Using this model, demonstrate the structures which may have been injured. b) Describe the sensory and motor nerve supply of the hand. *Prompt motor and sensory.*	Flexor tendons (FDP, FDS), MCP joint and head, common palmar digital nerves and arteries **<u>Ulnar nerve</u>** - ***Motor functions***: Innervates the majority of the intrinsic muscles of the hand (hypothenar, interosseous, adductor pollicis, deep head of flexor pollicis brevis, ulnar lumbricals for digits 4 & 5) ***Sensory functions*:** Innervates skin of 5th digit and ulnar half of fourth digit and adjacent hand. **<u>Median nerve</u>** - ***Motor functions*:** Supplies innervation to the thenar muscles (except adductor pollicis and deep head of flexor pollicis brevis) and radial two lumbricals in the hand. ***Sensory functions*:** Provides sensation to skin of palmar and distal dorsal aspects of radial three and a half digits. **<u>Radial nerve</u>** – Sensory functions: Dorsum of the radial side of the hand excluding distal fingers.	Correctly identify 3 to pass Must mention median and ulnar nerves with basic concept of sensory distribution and the majority of the motor supply.
Stem: Moving on to Pathology. Her wound is oozing.			
Question 4 Haemostasis **Subject:** Pathology LOA: 1 *Robbins – Pg. 119; Chapter 4: Haemodynamic disorders* *Chapter 12: The Heart; 9th Edition;*	a) Describe the process of primary haemostasis. ***Prompt:*** *how is the primary haemostatic plug formed* b) How is the coagulation cascade activated following injury? Prompt: what happens after it is activated? c) BONUS: What does prothrombin time measure?	Primary haemostasis = formation of platelet plug • Endothelial damage exposes ECM (collagen, vWF) • Platelet activation • adhere (via Gp1b to vWF) • shape change (flat to round) • secretion (ADP, Tx A2, Ca) + negative charge phospholipid) • Platelet aggregation (platelet Gp11b-111a receptors via fibrinogen) Vascular damage and exposure of **tissue factor** converts factor VII to VIIa. This in turn causes a series of amplifying enzymatic reactions that leads to the deposition of a **fibrin clot (secondary haemostasis)**. (Factor X is converted to factor Xa, which in turn converts prothrombin (factor II) to thrombin. Which converts fibrinogen to fibrin (fibrin network)) Assesses the extrinsic and common coagulation pathways.	Platelets plus 3of 6 Bold plus concept At least one

Stem: Moving on to Physiology. Her calcium level was checked.			
Question 5 Calcium haemostasis **Subject**: Physiology LOA: 2 *Ganong. 25th edition.* *Chapter 21 Hormonal control of Calcium and phosphate metabolism and the physiology of bone*	(a) How does the body regulate Plasma Calcium?	**1,25 – dihydrocholecalciferol** (DHCC) (from Vit D) increases Ca absorption from GIT and kidneys. **PTH** mobilises Ca from bone, increases Ca reabsorption in kidneys, increases 1,25 DHCC formation in kidneys. **Calcitonin** (from thyroid) inhibits bone resorption, increases Ca excretion in urine.	Mention Vit D and PTH PLUS correct direction of effect on Ca to pass
	(b) How is the synthesis of 1,25 – dihydrocholecalciferol (DHCC) (Vit D) regulated?	1,25 –DHCC formed in kidneys by 1alpha-hydroxylase. **Low Ca increases PTH** which stimulates 1alpha-hydroxylase **and increases 1,25 DHCC formation** Low PO4 directly stimulates 1alpha-hydroxylase **High Ca**/high PO4 **inhibits 1,25-DHCC** (increases inactive 24,25-DHCC instead).	b. concept in bold to pass 

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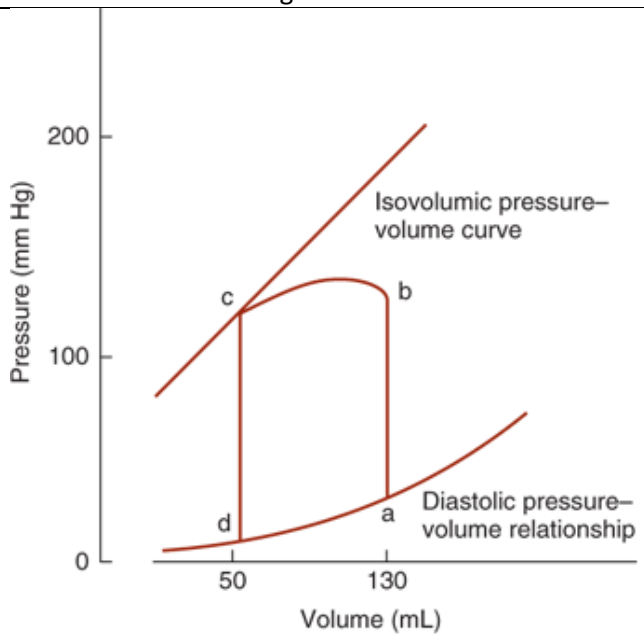
TOPIC 1	ECG	SUBJECT:	CBB	LOA:	Choose an item.
STEM	A 50-year-old man presents to the Emergency Department with chest pain. We will start with the Clinical Building Block.				
Question A	**Please interpret his ECG.**				
Knowledge:	**Atrial Fibrillation** with rapid ventricular response rate **Narrow complex**, Rhythm**:** irregularly irregular **Rate** 135/min (125-145), No P waves, No ischaemic changes				
Notes:	**Recognise AF, narrow complex, HR / tachycardia to pass**				
Question B	**What are the causes of this rhythm?**				
Knowledge:	Common • IHD / Valvular heart disease /HT / Cardiomyopathies / Thyrotoxicosis Congenital aberrant pathways /Sepsis Less Common • Drugs e.g. alcohol, caffeine / PE / Myocarditis / Rheumatic heart disease				
Notes:	**3 to pass**				

TOPIC 2	Digoxin	SUBJECT:	Pharmacology	LOA:	1
STEM	Moving on to Pharmacology. The patient is on digoxin for this rhythm				
Question A	**Describe the pharmacodynamics of digoxin.** *Prompt: What are the cardiac effects of digoxin?*				
Knowledge:	**Inhibitor of Na^+/K^+ ATP-ase.** i) Increases intracellular Na^+ and decreases intracellular K^+. ii) increased intracellular Na+ leads to reduced Na/Ca^{2+} exchanger activity which leads to increase in intracellular calcium. iii) increased Intracellular Ca^{2+} causes an increase in contractility (inotropy). Inhibition of the Na^+/K^+-ATPase in vascular smooth muscle causes depolarization, which causes smooth muscle contraction and vasoconstriction. iv) **Electrical effects as a concept** -Direct: shortening of AP- shortened atrial and ventricular refractoriness -Increased automaticity of the heart muscle: bigeminy, followed by VT and then VF. v) Parasympathetic and sympathetic effects -At lower doses, parasympathetic effects: early signs of toxicity = bradycardia and AV block. -At higher doses of toxicity, increase sympathetic effect which may further sensitize the myocardium to automaticity.				
Notes:	**2 bold to pass**				
Question B	**What are the non-cardiac symptoms and signs of digoxin toxicity?**				
Knowledge:	i) GIT: anorexia/nausea/vomiting/diarrhoea. ii) CNS: disorientation, hallucinations and yellow/green vision (or some variation of) / CTZ (chemoreceptor trigger zones)				
Notes:	**1 CNS to pass (disorientation or visual disturbance or CTZ)**				
Question C	**What factors may predispose a patient to digoxin toxicity?**				
Knowledge:	(i) **<u>Electrolyte imbalance</u>** -Hypokalaemia (K+ inhibits digoxin binding to the Na/K ATP-ase). -Hypercalcaemia (potentiates digoxin toxicity by increasing the intracellular Ca^{2+} stores, producing automaticity) -Hypomagnesemia (ii) **Drugs** that increase digoxin effect Amiodarone (by increasing plasma digoxin concentrate), diltiazem, verapamil, quinidine, macrolide antibiotics (azithromycin, erythromycin and clarithromycin), K+ depleting drugs (including diuretics), spironolactone (iii)Organ disease -**Renal failure (important because of kinetics)** -Hypothyroidism				
Notes:	**2 bold topics (with an example of each) to pass**				

ACEM PRIMARY VIVA A	Thursday	*Candidate Number:*		AGREED MARK:	

TOPIC 3	Acute Coronary Syndromes / Ischaemic Heart Disease	SUBJECT:	Pathology	LOA:	1
STEM	Moving on to Pathology. The patient has ongoing chest pain.				
Question A	**What is an acute coronary syndrome?**				
Knowledge:	ACS is a clinical manifestation of ischaemic heart disease, and can present as unstable angina, acute MI (either STEMI or NSTEMI) or sudden cardiac death.				
Notes:	**Reasonable definition to pass**				
Question B	**What are the pathological processes that underlie acute coronary syndrome?**				
Knowledge:	ACS typically initiated by an unpredictable and abrupt conversion of a stable atherosclerotic plaque to an unstable and potentially life-threatening atherothrombotic lesion through rupture, superficial erosion, ulceration, fissuring, or deep hemorrhage. In most instances, plaque changes—typically associated with intra-lesional inflammation—precipitate the formation of a superimposed thrombus that partially or completely occludes the artery.				
Notes:	**Explanation of stable progresses to unstable plaque**				
Question C	**Following acute myocardial infarction, what complications might a patient have?**				
Knowledge:	1. Contractile dysfunction causing hypotension Cardiogenic shock in 10-15% 2. Arrhythmias e.g. sinus bradycardia, AF, HB, tachycardia, VT, VF 3. Myocardial rupture: ventricular free wall rupture (2-7 days pots MI), septum rupture, papillary muscle rupture 4. Ventricular aneurysm 5. Pericarditis (2-3 days post MI)/ Dressler's syndrome 6. Infarct expansion 7. Papillary muscle dysfunction 8. Progressive heart failure				
Notes:	**At least 3 complications to pass**				

ACEM PRIMARY VIVA A	Thursday	*Candidate Number:*		AGREED MARK:	

TOPIC 4	Cardiac cycle	SUBJECT:	Physiology	LOA:	1
STEM	Moving on to Physiology. The patient becomes hypotensive.				
Question A	**Please draw and label the pressure volume curve of the left ventricle.**				
Knowledge:	a to b: isovolumetric contraction b to c: ventricular systole c to d: isovolumetric relaxation d to a: ventricular filling				
Notes:	 Source: Kim E. Barrett, Susan M. Barman, Scott Boitano, Heddwen L. Brooks: Ganong's Review of Medical Physiology, 25th Ed. www.accessmedicine.com Copyright © McGraw-Hill Education. All rights reserved. **Draw graph with axis, curves and pressures to pass**				
Question B	**Starting with systole describe the pressure and volume changes in the left ventricle .** *Prompt: What happens to the valves?*				
Knowledge:	(a to b) Start of systole, mitral valve closes at a. Pressure rises sharply from a to b. **Isovolumetric contraction** until LVP> aortic pressure (80mmHg) then aortic valve opens (at b). ESV 50ml (b to c) Pressure rises to a plateau and volume falls from b to c during ventricular ejection (c to d) Momentum of ejected blood is overcome by arterial pressure, then the aortic valve closes at c. Pressure falls from c to d. Isovolumetric relaxation as the ventricular pressure drops rapidly until below atrial pressure. At d, MV opens. (d to a) Diastole commences. The mitral valve opens to start ventricular filling. EDV 130ml, SV 70-90ml				
Notes:	**Bold and concepts including valves to pass**				

ACEM PRIMARY VIVA A	Thursday	*Candidate Number:*		AGREED MARK:	

TOPIC 5	Heart (Model)	SUBJECT:	Anatomy	LOA:	1
STEM	Moving on to Anatomy				
Question A	**Please identify the great vessels and branches which enter and exit the heart of the on this model.** *Prompt: what's this?*				
Knowledge:	**SVC**- Right Brachiocephalic vein, Left Brachiocephalic vein **IVC** **Ascending aorta**- Brachiocephalic trunk, left common carotid artery, left subclavian artery, **Pulmonary Trunk** and pulmonary arteries Pulmonary veins				
Notes:	**Bold to pass**				
Question B	**(NB - Open the heart model). Please identify the chambers and valves of the heart?**				
Knowledge:	**RA, LA, RV, LV** **Tricuspid Valve, aortic valve, pulmonary valve, mitral valve,**				
Notes:	**All 4 chambers and valves to pass**				
Question C	**Describe the structures of the conducting system of the heart.**				
Knowledge:	SA node: Anterior-lateral near the junction of the SVC and RA AV node: Posterior- inferior region of the interatrial septum, near the opening of the coronary sinus AV bundle of HIS: Through the fibrous skeleton of the heart, along the membranous part of the interventricular septum Divides into Right and Left bundles which pass on each side of the muscular IV septum				
Notes:	**SA node, AV node and rough location to pass**				
Question D	**Describe the arterial supply to the cardiac conduction system** *Prompt: What's the usual arterial supply to the cardiac conduction system?*				
Knowledge:	SA node- RCA 60%, circumflex 40% AV node and bundle- RCA, AV nodal artery Right and Left Bundles and Purkinje fibres- LAD				
Notes:	**SA and AV nodes typically supplied by RCA to pass**				

ACEM PRIMARY VIVA A	Friday	*Candidate Number:*		AGREED MARK:	

TOPIC 1	VBG (Acute kidney injury)	SUBJECT:	CBB	LOA:	n/a
STEM	An elderly patient presents with abdominal and back pain. Blood tests are performed. We will start with the Clinical Building Block.				
Question A	**Please interpret this VBG**				
Knowledge:	High anion gap **metabolic** (lactic) **acidosis** with incomplete respiratory compensation and acute kidney injury**.** Low pH = acidaemia Low pCO2 + bicarb = metabolic Expected pCO2 = 31 Winters (HCO3 x 1.5) + 8 Partial resp comp. Anion Gap = 31 HAGMA with high lactate Hyper K + high Cr = AKI Raised glucose = stress response				
Notes:	**Metabolic + Acidosis to pass**				
Question B	**What are the potential clinical causes for this VBG in this patient?**				
Knowledge:	Ruptured AAA, ischaemic gut, pancreatitis, perforated viscous, sepsis, dissection. Candidates may mention CATMUDPILES (CO, CN, Alcoholic ketoacidosis, toluene, methanol, uraemia, DKA, Paraldehyde, phenformin, iron, isoniazid, lactic acidosis, ethylene glycol, salicylates, starvation ketoacidosis)– Examiners to decide on how to rule on this				
Notes:	**Bold = 2 intra-abdominal causes to pass**				

TOPIC 2	Posterior Abdo Wall Vessels	SUBJECT:	Anatomy	LOA:	2
STEM	Moving on to Anatomy. You are concerned about this patient. A CT scan is performed.				
Question A	**Identify structures that can be seen in this axial slice of his abdominal CT scan.**				
Knowledge:	Liver, Intestines, Pancreas, Spleen, Kidneys, Descending Aorta, Vertebral Body, Rectus Muscle, Diaphragm, Inferior Vena Cava				
Notes:	**6 out of 10 to pass including one vascular structure**				
Question B	**Describe the branches of the abdominal aorta?**				
Knowledge:	**Coeliac, Superior Mesenteric, Inferior Mesenteric**, Suprarenal, **Renal**, Gonadal (urogenital/endocrine); Subcostal, Inferior Phrenic, Lumbar, Common Iliac arteries				
Notes:	**At least 2 bold + any 2 others**				

TOPIC 3	Aneurysms	SUBJECT:	Pathology	LOA:	1
STEM	Moving on to Pathology.				
Question A	**What are the risk factors for abdominal aortic aneurysm?** *Prompt: Clinical or pathological risk factors*				
Knowledge:	Male, smoking, age > 60, FHx, CTD (eg Marfans, Ehlers-Danlos), vasculitis, **hypertension**, diabetes, **atherosclerosis**, trauma, congenital, infection, inflammation				
Notes:	**2x Bold + 2 others to pass**				
Question B	**Describe the pathogenesis of an aneurysm?**				
Knowledge:	Aneurysms can occur when the structure or function of the connective tissue within the vascular wall is compromised. Atherosclerotic plaque in intima Compressed media with degeneration and weakness of the wall and cystic medial degeneration. Local inflammation (proteolytic enzymes with collagen degradation - role of matrix metalloprotinases) Loss of vascular smooth muscle cells Inappropriate synthesis of non-elastic ECM				
Notes:	**Concept to pass**				
Question C	**What are clinical consequences of an aneurysm?**				

Knowledge:	Pain(less) mass **Rupture** - risk increases with diameter > 5cm (modest increase > 4 cm) Retroperitoneal or intraperitoneal Obstruction: branch obstruction e.g. mesenteric, vertebral, renal Embolism: plaque or thrombus Impingement/compression of adjacent structures e.g. ureter Infection / mycotic
Notes:	**Bold + 2 others to pass**

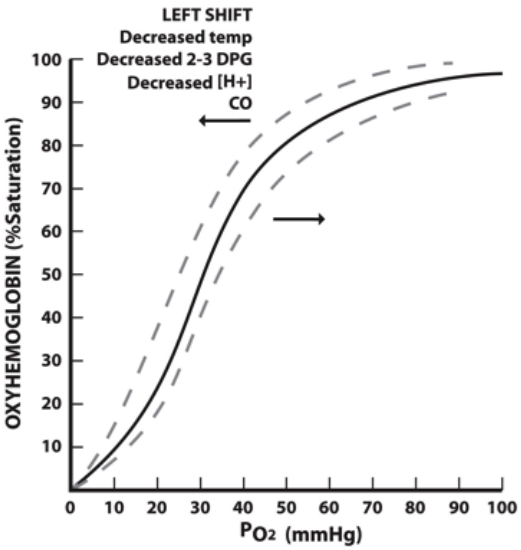
TOPIC 4	Disordered Renal Function	**SUBJECT:**	Physiology	**LOA:**	1
STEM	Moving on to Physiology. The patient has impaired renal function.				
Question A	**What are the physiological consequences of this?**				
Knowledge:	**Proteinuria** (predominantly albuminuria) - Due to increased permeability of glomerular capillaries. **Uraemia** - Accumulation of breakdown products of protein metabolism resulting in symptoms of uraemia. **Acidosis** - Failure to excrete acid products of digestion/metabolism with urine maximally acidified. Total amount H+ secreted reduced due to impaired renal tubular production of NH4+. -Exception – Renal tubular acidosis (impaired ability to acidify urine) Hyperkalaemia – H+/K+ exchange **Abnormal Na+ handling (retain excess amounts Na+). 3 mechanisms:** -Acute GN – amount of Na+ filtered markedly decreased -Nephrotic syndrome – incl. aldosterone causes salt retention. Low plasma protein means fluid shifts from plasma into interstitium. Resulting low plasma volume triggers Renin-angiotensin system. -Volume overload				
Notes:	**3 out of 4 bold to pass**				
Question B	**Why does the kidney lose the ability to concentrate and dilute urine in a patient with impaired renal function?**				
Knowledge:	In advanced kidney disease osmolality fixed at plasma level indicating that the ability to concentrate or dilute urine has been lost This is due to - disruption of the countercurrent mechanism - loss of functioning nephrons NB positive feedback in that the increased filtration on the remaining nephrons eventually damages more nephrons from fibrosis				
Notes:	**Concept of disruption of the countercurrent mechanism and loss of functioning nephrons to pass**				

TOPIC 5	Metoprolol	**SUBJECT:**	Pharmacology	**LOA:**	1
STEM	Moving on to Pharmacology. The patient is on Metoprolol				
Question A	**Describe the pharmacokinetics of metoprolol**				
Knowledge:	Oral or IV. Well absorbed orally but Bioavailability 50% due to 1st pass effect. Vd – large (>200L) Half-life - 3 – 4 hrs Metabolised in liver				
Notes:	**2 pharmacokinetic parameters to pass**				
Question B	**What are the cardiovascular effects of metoprolol?**				
Knowledge:	**Negative inotropic and chronotropic effects.** Slow A-V node conduction with increased PR on ECG Decrease BP by a mechanism not fully understood but probably includes suppression of renin release and CNS effects.				
Notes:	**Bold to pass**				
Question C	**How does metoprolol differ from propranolol in its receptor action?**				

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Knowledge:	**Metoprolol is β_1 specific and propranolol is not** (equipotent at β_1 and β_2). β_1 equipotent β_2 50-100 fold less potent At higher doses is less specific
Notes:	**Bold to pass**

TOPIC 1	CXR (Pneumonia)	**SUBJECT:**	CBB	**LOA:**	n/a
STEM	A 72-year old woman presents with shortness of breath and fever. We will start with the Clinical Building Block.				
Question A	**Please describe and interpret her CXR.**				
Knowledge:	Erect CXR, PA view, slightly rotated, trachea deviated to right **Right mid-zone opacity /consolidation,** likely **pneumonia** in the superior aspects of the RLL.				
Notes:	Bold to pass				
Question B	On a PA CXR how do you differentiate between a right lower and right middle lobe pneumonia?				
	R middle lobe has loss of R heart border				
Question C	**What factors predispose patients to the development of pneumonia?**				
Knowledge:	Extreme age Underlying chronic disease**:** DM, COPD, CCF Immunodeficiency**:** congenital or acquired, abnormal splenic function, decreased splenic function or asplenia. Recent viral respiratory tract infection eg influenza Smoker Neurological: poor swallow (CVA, Parkinsons etc)				
Notes:	3 to pass				

TOPIC 2	Oxygen haemoglobin dissociation curve	**SUBJECT:**	Physiology	**LOA:**	1
STEM	Moving on to Physiology. Her oxygen saturation is 90% on room air.				
Question A	**Draw and describe the oxygen haemoglobin dissociation curve.**				
Knowledge:	 PaO_2 27mmHg = SaO_2 50% PaO_2 30mmHg = SaO_2 60% PaO_2 40mmHg = SaO_2 75% PaO_2 56mmHg = SaO_2 90% PaO_2 80mmHg = SaO_2 87% PaO_2 90mmHg = SaO_2 97%				
Notes:	Correct shape and labels and have 2 points of saturation **Prompt: could you label 2 points of saturation on the curve**				

Question B	**What are the implications of the shape of the curve?**
Knowledge:	UPPER: Flat upper part means if pO_2 in alveolar gas falls, loading of O_2 is little affected LOWER: steep lower part means peripheral tissue can draw large amount of O_2 for only small drop in capillary pO_2
Notes:	Clear concept to pass
Question C	**What factors shift the curve?**
Knowledge:	Right: increased temperature, increased pCO_2, increased H^+(decreased pH), and increased 2-3 DPG Left: Reverse of above
Notes:	3 factors to pass

TOPIC 3	Pneumonia	**SUBJECT:**	Pathology	**LOA:**	1
STEM	Moving on to Pathology				
Question A	**What are the pathological patterns of bacterial pneumonia?**				
Knowledge:	**Bronchopneumonia: patchy** consolidation of lung**,** areas of acute suppurative inflammation, may be patchy through one lobe but is more often multilobar and frequently bilateral and basal because of tendency of secretions to gravitate into the lower lobes. **Lobar Pneumonia**: fibrinosuppurative consolidation of a large portion of a lobe or an entire lobe. Patterns overlap, patchy involvement may become confluent producing lobar consolidation**.**				
Notes:	Bold to pass Prompt: What patterns of pneumonia can be seen in a CXR?				
Question B	**Describe the stages of the inflammatory response seen in lobar pneumonia**				
Knowledge:	**Four stages of inflammatory response**: 1) **Congestion:** vascular engorgement, intra-alveolar fluid with few neutrophils and often the presence of numerous bacteria. Lung appears heavy, boggy and red. 2) **Red hepatisation**: massive confluent exudation with neutrophils, red cells and fibrin filling the alveolar spaces, lobe appears red, firm and airless with a liver like consistency. 3) **Gray hepatisations**: progressive disintegration of red cells and the persistence of a fibrinosuppurative exudate, lobe appears greyish, brown and dry surface. 4) **Resolution**: exudate within the alveolar spaces undergoes progressive enzymatic digestion to produce granular, semifluid debris that is resorbed, ingested by macrophages, expectorated or organised by fibroblast growing into it.				
Notes:	3 stages to pass Prompt: What are the classic morphological changes seen in lobar pneumonia?				

Question C	**What are the complications of pneumonia?**				
Knowledge:	1) **Abscess formation**: tissue destruction and necrosis causing abscess formation particularly common with type 3 pneumococci or Klebsiella infections. 2) **Empyema:** spread of infection to the pleural cavity, causing the intrapleural fibrinosuppurative reaction. 3) **Bacteraemic dissemination**: dissemination to heart valves, pericardium, brain, kidneys, spleen or joints causing metastatic abscesses, endocarditis, meningitis or suppuratives arthritis. 4) Local extension : pleuritis 5) Parapneumonic effusion 6) Respiratory failure / ARDS 7) Bronchopleural fistula 8) Pulmonary fibrosis 9) Sepsis 10) Death				
Notes:	2 of bolded response plus 1 other to pass				
TOPIC 4	Rib bones- 1st rib	**SUBJECT:**	Anatomy	**LOA:**	1
STEM	**Moving on to Anatomy.**				
Question A	**Identify this bone and demonstrate its features.**				
Knowledge:	**First rib** Broadest, flattest, shortest rib. Lies nearly horizontal, with wide body. Left side. Determined by identifying superior surface. Features: Head, Neck, Tubercle, Shaft Single facet in its head for articulation with T1 vertebra only Superior surface, from neck to tip: - groove for subclavian artery and lower trunk of brachial plexus (posterior to artery) - scalene tubercle and ridge, attachment of scalenus anterior - groove for subclavian vein - flat outer surface is attachment of first digitation of serratus anterior - attachments to costoclavicular ligament (inner) and subclavius (outer) Tip articulates with costal cartilage **Prompt: Which side is this?**				
Notes:	To pass: - First rib - Correct side - Groove for subclavian artery - PLUS any 2 features				
Question B	**Describe the neurovascular relations of this bone?**				
Knowledge:	**Nerves: C8 above and T1 nerve root below the neck;** these unite to form lower trunk of brachial plexus, sitting above surface behind subclavian artery Sympathetic trunk (cervicothoracic ganglion) in contact with anterior border of neck In groove for subclavian artery is lower trunk of brachial plexus, behind the artery **Vessels: Subclavian artery** runs in groove, behind scalene tubercle, touching outer border of rib. **Subclavian vein** runs anterior to scalene tubercle, in its own groove. **First intercostal neurovascular bundle beneath the undersurface**, covered by parietal pleura				
Notes:	To pass: 1 neurological and 1 subclavian vessel				

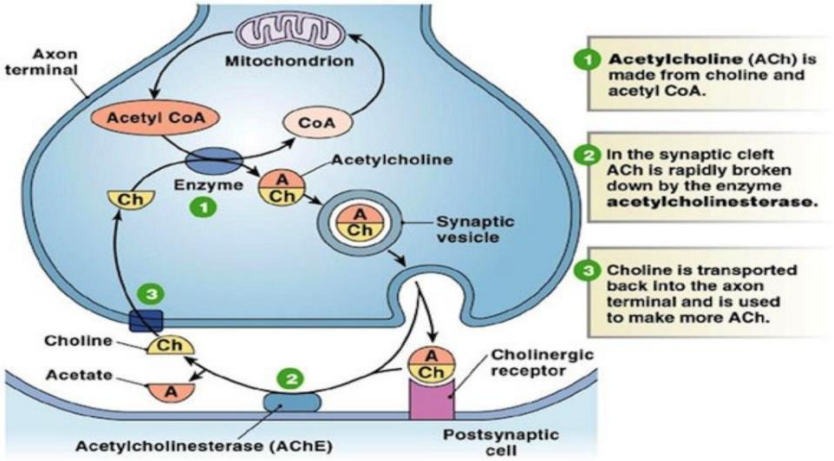
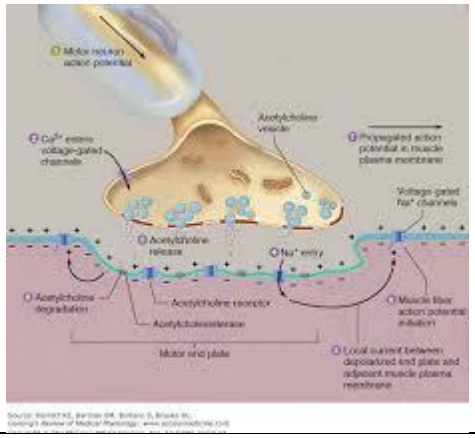
TOPIC 5	Paracetamol	**SUBJECT:**	Pharmacology	**LOA:**	1
STEM	Moving on to Pharmacology. Paracetamol is given for her fever.				
Question A	**Describe the pharmacokinetics of paracetamol.**				
Knowledge:	Rapid absorption Bioavailability 70-90%. Peak concentration after 30-60 minutes Slightly protein bound Metabolism: Hepatic, >95% undergoes glucuronidation and sulfation, 5% undergoes metabolism via CYP 450 mechanism (phase 1 reaction – hydroxylation) to form NAPQI, NAPQI is toxic but usually detoxified by glutathione. First order kinetics, Half life is 2-3 hours				
Notes:	3 to pass				
Question B	**Describe the mechanism by which paracetamol causes toxicity?**				
Knowledge:	**Zero order kinetics** Paracetamol is conjugated with glucuronide and sulphate (by transferase enzymes) – this **pathway becomes saturated** in overdose, allowing increasing paracetamol to be metabolized by the smaller CYP 2E1 pathway to NAPQI (N-acetyl-p-benzoquinone imine) NAPQI is detoxified by **glutathione** which becomes depleted resulting in high levels of **toxic metabolite (NAPQI)**				
Notes:	Bold to pass				
Question C	**How does N-Acetylcysteine work in the treatment of paracetamol overdose?**				
Knowledge:	Sulfhydryl group donor – restores hepatic reduced glutathione levels**.** Or acts as an alternative substrate for conjugation with the toxic metabolite.				
Notes:	Concept to pass				

ACEM PRIMARY VIVA B	Friday	*Candidate Number:*		AGREED MARK:	

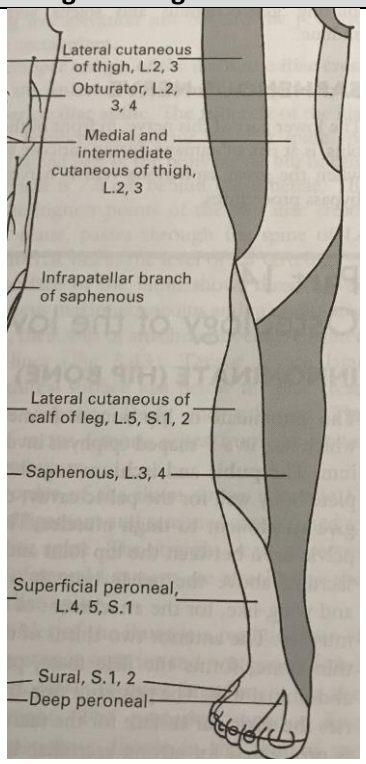
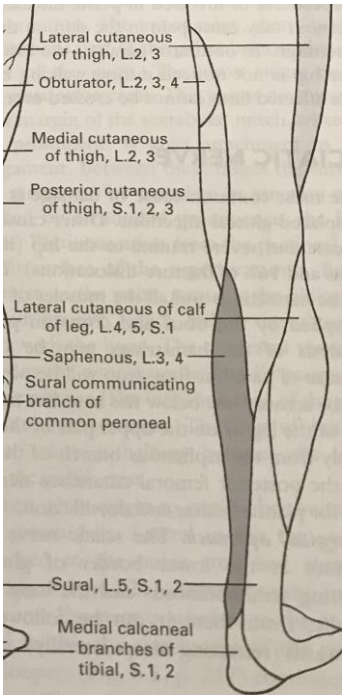
TOPIC 1	X-ray - Lumbar spine	SUBJECT:	CBB	LOA:	n/a
STEM	A 60-year-old woman presents to ED with back pain after a fall. An x-ray of her lumbar spine is performed. We will start with the Clinical Building block				
Question A	**Describe the X-ray.**				
Knowledge:	Lateral Lumbar X-ray (T12-L5) **L1 body Crush fracture**, wedged in appearance Posterior elements appear intact No retropulsed fragments				
Notes:	Crush fracture of L1				
Question B	**What findings on examination would you look for?**				
Knowledge:	Focal tenderness over spine Lower limb neuro exam looking for changes in tone, absent reflexes, loss of power, absent sensation Abnormal gait Cauda equina syndrome – anal tone (incontinence), urine retention Other injuries				
Notes:	3 to pass including 1 neuro				

TOPIC 2	Acute inflammation	SUBJECT:	Pathology	LOA:	1
STEM	Moving on to Pathology.				
Question A	**Please describe the major components of acute inflammation.**				
Knowledge:	1. **Small vessel dilatation** - leading to increased blood flow 2. **Increased vascular permeability** - enabling plasma proteins and leukocytes to leave the circulation 3. **Leukocyte emigration**. Emigration of the leukocytes from the microcirculation, their accumulation in the focus of injury, and their activation to eliminate the offending agent.				
Notes:	2 out of 3 Bold to pass				
Question B	**What are the mechanisms responsible for increased vascular permeability?**				
Knowledge:	1. **Contraction of endothelial cells** – resulting in increased inter-endothelial spaces (most common) 2. **Direct endothelial injury** – resulting in endothelial cell necrosis and detachment (burns, toxins, neutrophils themselves) 3. **Transcytosis** – increased transport of fluids and proteins through the endothelial cell (may be stimulated by VEGF; contribution to acute inflammation is uncertain.				
Notes:	2 out of 3 to pass				
Question C	**i) What is chemotaxis of leukocytes?** **ii) What are the mediators that aid chemotaxis?**				
Knowledge:	1. Chemotaxis: **locomotion/movement of white cells** along a chemical gradient. After exiting circulation, leukocytes move in the tissues by chemotaxis toward the site of injury. 2. Chemo-attractants include: a. Exogenous – most commonly **bacterial products**/proteins/peptides b. Endogenous – **Cytokines** (e.g. IL-8), **complement** (e.g. C5a), **arachidonic acid** metabolites (e.g. LTB4).				
Notes:	Describe chemotaxis. 2 mediators				

ACEM PRIMARY VIVA B	Friday	*Candidate Number:*		AGREED MARK:	

TOPIC 3	Neuromuscular Junction	SUBJECT:	Physiology	LOA:	1
STEM	You are concerned she may have some limb weakness. Moving on to Physiology.				
Question A	**Describe the synthesis of acetylcholine at the neuromuscular junction?**				
Knowledge:	 1.Acetylcholine is synthesised in the pre-synaptic terminal and stored in synaptic vesicles along with ATP and proteoglycan until required for synaptic neuronal transmission 2.Reaction: Acetyl CoenzymeA + Choline which is catalysed by the enzyme Choline Acetyltransferase Prompt: You may draw a diagram				
Notes:	Concept to pass				
Question B	**Describe the sequence of events that leads to the release of acetylcholine at the neuromuscular junction**				
Knowledge:	 1. Impulse arrives at the motor neuron ending which causes **calcium voltage gated channels** to open 2. **Influx of calcium** triggers release of Acetylcholine into the synaptic cleft				
Notes	Concept to pass				
Question C	**What happens to acetylcholine after release into the synaptic cleft?**				
	1. When vescicle releases ACh into the synaptic cleft, it is rapidly broken down into acetate and choline by the enzyme Acetylcholinesterase 2. Choline is actively transported back into the presynaptic terminal to be reused 3.ACh binds to Nicotinic receptors on the motor end plate leading to Na entry and a subsequent depolarising end plate potential Prompt: you may draw a diagram				
Notes:	Concept to pass				

ACEM PRIMARY VIVA B	Friday	*Candidate Number:*		AGREED MARK:	

TOPIC 4	Myotomes	SUBJECT:	Anatomy	LOA:	1
STEM	Moving on to Anatomy. On examination, the patient has decreased lower limb reflexes and sensation.				
Question A	**What are the myotomes of the lower limb**				
Knowledge:	• Hip flexion L2/3, extension L4/5 • Knee extension L3/4 flexion L5 /S1 • Ankle flexion L4/5 extension S 1/2 • Inversion L4 eversion L5/S 1 • Big toe L5/S1 extension S1/2				
Notes:	6 out of 10 to pass				
Question B	**Using this diagram show me the cutaneous nerve supply of the leg**				
Knowledge:	  **Lateral thigh:** Lateral cutaneous nerve of thigh **Anterior and medial thigh:** Anterior cutaneous branch of femoral nerve; intermediate and medial femoral cut nerves; obturator nerve **Posterior thigh:** Posterior cutaneous nerve of thigh **Lateral leg from knee to mid-calf:** Lateral cutaneous nerve of calf and leg **Anterolateral leg from mid-calf**: superficial peroneal (fibular) nerve **Posterolateral leg from mid-calf:** sural nerve **Medial leg:** Saphenous nerve **Dorsum of foot, except 1st web space:** Superficial peroneal (fibular) nerve **Dorsum of 1st web space:** Deep peroneal (fibular) nerve **Heel:** Medial calcaneal branch of tibial nerve **Sole of foot except heel:** medial and lateral plantar branches of tibial nerve				
Notes:	5 to pass				

ACEM PRIMARY VIVA B	Friday	*Candidate Number:*		AGREED MARK:	

TOPIC 5	Tricyclic anti-depressants	SUBJECT:	Pharmacology	LOA:	1
STEM	The patient was taking amitriptyline for pain.				
Question A	What is the mechanism of action of tricyclic anti-depressants.				
Knowledge:	1. **Inhibition of serotonin** (5-HT) and **noradrenaline/norepinephrine** (NE) **reuptake**. This increases the amount of 5-HT and NE in certain parts of the brain (cortex and limbus) – "Monoamine hypothesis for depression" – and spinal cord (ascending corticospinal tract – neuropathic pain). 2. Also blocks a. Na+ channels b. K+ channels c. Muscarinic (M1) receptors – anti-cholinergic d. Histaminic (H1 receptors) e. Alpha-1 adrenergic receptors (peripheral post-synaptic) (From toxicology handbook)				
Notes:	Bold to pass and one other				
Question B	What clinical manifestations would be seen in an overdose of tricyclic anti-depressants?				
Knowledge:	**1. Cardiac** a. Tachycardia b. Hypotension (alpha blockade, impaired contractility) c. ECG – PR prolonged, widened QRS (Na blockade), prolonged QT (K blockade), VT, VF **2. CNS** a. Drowsiness b. Delirium (due to anti-cholinergic effects) c. Seizures d. Coma **3. Anti-cholinergic** a. Agitation b. Delirium c. Mydriasis d. Dry, warm, flushed skin e. Urinary retention f. Ileus				
Notes:	One example of each system Prompt : If only mentions one system prompt for others				

2018.1 Primary Examination Clinical (Viva) Topic Overview

THURSDAY 22 MARCH 2018

	Anatomy	Pathology	Physiology	Pharmacology	CBB
Viva A	Heart (model)	Acute Coronary Syndromes / Ischaemic Heart Disease	Cardiac Cycle	Digoxin	ECG
Viva B	Ankle X-ray	Anaphylaxis	Work of Breathing	Penicillin	Joint Aspirate
Viva C	Anterior Triangle of Neck (photo)	Meningitis	Osmolality	Local Anaesthetics	Biochemistry Panel
Viva D	Humerus (bone)	Fracture Healing	Modulation of Pain	Fentanyl	Supracondylar Fracture (X-ray)

FRIDAY 23 MARCH 2018

	Anatomy	Pathology	Physiology	Pharmacology	CBB
Viva A	Posterior Abdominal Wall Vessels	Aneurysms	Disordered Renal Function	Metoprolol	Venous Blood Gas (metabolic acidosis and acute kidney injury)
Viva B	Base of Skull (bone)	Neoplasia	Lung Volumes	Phenytoin	CT Brain
Viva C	Knee Joint (model)	Oedema	Liver Metabolism	Spironolactone	Liver Function Tests (hepatitis)
Viva D	Vascular Supply of Arm	Chronic Renal Failure	Renal Tubular Function	Ceftriaxone (beta-lactam antibiotics)	ECG (hyperkalaemia)

2018.2 Primary Examination Clinical (Viva) Topic Overview

THURSDAY 13 SEPTEMBER 2018

	Anatomy	Pathology	Physiology	Pharmacology	CBB
Viva A	Head & Neck (Eye Model)	Cardiomyopathy	Cardiovascular	Atropine	Complete Heart Block (ECG)
Viva B	Rib Bone (First Rib)	Pneumonia	Oxygen Haemoglobin Dissociation Curve	Paracetamol	Pneumonia (CXR)
Viva C	Forearm (Superficial Muscles)	Diabetes	Renal Handling of Glucose	Antibiotics (Vancomycin)	Venous Blood Gas
Viva D	Lower Limb (Pelvis/Hip Joint)	Skin Tear - Wound Healing	Osteoporosis/ Calcium Metabolism	Opioids/Drugs in Elderly	Fractured Hip (Pelvis X-ray)

FRIDAY 14 SEPTEMBER 2018

	Anatomy	Pathology	Physiology	Pharmacology	CBB
Viva A	Brain (Ventricles)	Subarachnoid Haemorrhage	Cerebral Autoregulation	Nimodipine	CT Brain
Viva B	Myotomes	Acute Inflammation	Neuromuscular Junction	Tricyclic Anti-depressants	Lumbar Spine (X-ray)
Viva C	Compartments of the Hand (Hand Model)	Haemostasis	Control of Ventilation	ADT/Tetanus/TIG /Immunisations	Palmar Laceration (Photo)
Viva D	Retroperitoneum	Renal (Renal Stones)	Glomerular Filtration Rate	Antibiotics (Gentamicin)	Renal Failure (Blood Tests)

TOPIC 1	ECG (Hyperkalaemia)	**SUBJECT:**	CBB	**LOA:**	Choose an item.
STEM	A 60-year-old woman presents feeling light headed, dizzy and nauseated. We will start with the Clinical Building block.				
Question A	**Describe her ECG?**				
Knowledge:	• rate 60/min • regular • P wave not visible • Wide QRS complexes • Peaked T waves				
Notes:	Two to pass				
Question B	**What is the likely cause of these ECG changes?**				
Knowledge:	• Hyperkalaemia				
Notes:	Hyperkalaemia to pass				
Question C	**The potassium has come back as 7.8 mmol/L. What immediate treatment would you consider and why?**				
Knowledge:	1. Stabilising the myocardium: Calcium Chloride/Gluconate • 2. Reduce serum K by redistribution into the intracellular space: Insulin/Dextrose, Sodium bicarbonate, Beta agonist / salbutamol • 3. Eliminate potassium from the body: Resonium, Normal Saline, Frusemide, Dialysis PROMPT: What other treatment could be considered?				
Notes:	Requires 1 or 2 to pass with justification				

TOPIC 2	ACE Inhibitors	**SUBJECT:**	Pharmacology	**LOA:**	LOA 2
STEM	Moving on to Pharmacology. The patient is on Ramipril.				
Question A	**What is the mechanism of action of Ramipril**?				
Knowledge:	ACE Inhibitors result in a reduction in systemic BP due to the following mechanism: • Inhibits Angiotensin Converting Enzyme from hydrolysing Angiotensin I to Angiotensin II. • Angiotensin II is a vasoconstrictor hence its reduction results in a decrease in vascular tone (main effect) • Angiotensin II leads to aldosterone secretion. Hence its reduction leads to reduced Na and H_2O retention, leading to a reduction in BP • Angiotensin II metabolises bradykinin to its inactive form. Hence its reduction results in an increase in bradykinin, leading to vasodilation and hence reduction in BP				
Notes:	Needs 2 out of 4 concepts to pass				
Question B	**What are the adverse effects of Ramipril?**				
Knowledge:	Dizziness, hypotension, headache, weakness, loss of taste, diarrhoea, rash, fever, joint pain, cough, wheezing, angioedema, mild hyperkalaemia, acute renal failure, teratogenic.				
Notes:	Hypotension and 2 others				

Question C	**What adverse drug interactions may occur with ACE inhibitors?**
Knowledge:	• Diuretics/antihypertensives → Hypotension • General Anaesthetics→Hypotension • Lithium→ Lithium toxicity • NSAIDs → Hyperkalaemia and reduced effects of ACE inhibitor • Potassium sparing diuretics/potassium supplement: Hyperkalaemia **PROMPT:** Can you give some examples of drug interactions with ACE inhibitors and the adverse effect that results?
Notes:	Two to pass
Question D	**What advantages do angiotensin receptor blockers have over ACE inhibitors?**
Knowledge:	• No effect on bradykinin, so reduced incidence of cough, angioedema • More complete inhibition of actions of angiotensin II PROMPT: What is the mechanism to reduce the incidence of cough?
Notes:	Concept to pass

TOPIC 3	Femoral Triangle	**SUBJECT:**	Anatomy	**LOA:**	1
STEM	Moving on to Anatomy. You are unable to secure a peripheral line and decide to insert a femoral central line				
Question A	**What are the boundaries of the femoral triangle?**				
Knowledge: (Model of femoral triangle)	• Superior: Inguinal ligament (11) • Medial: lateral border of adductor longus (1) • Lateral: Sartorius (23) • Floor: Iliopsoas and pectineus (19)				
Notes:	Two to pass				
Question B	**Identify the contents of femoral triangle on this image?**				
Knowledge:	Lateral to medial: 1) Femoral nerve (5) and its branches, 2)Femoral artery (4) and several of its branches 3,)Femoral Vein (6) and its proximal tributaries 4) Deep inguinal lymph node (14), lymphatics (15)				
Notes:	Three to pass				
Question C	**What are the surface markings when trying to locate the femoral vein?**				
Knowledge:	Using the anatomical landmark for the femoral artery: Femoral artery is found below the inguinal ligament; midway between ASIS and pubic tubercle, Femoral vein is just medial to artery.				
Notes:	Adequate description				
Question D	**Describe the course of the Femoral vein in the femoral triangle?**				
Knowledge:	• Starts as popliteal vein • Medial to femoral artery, lateral to canal • Ends as external iliac vein • Draining into it: profunda femorus, great saphenous				
Notes:	Two to pass				

TOPIC 4	Renal Handling of potassium	**SUBJECT:**	Physiology	**LOA:**	1
STEM	Moving on to Physiology.				
Question A	**How does the kidney handle potassium?**				
Knowledge:	**Filtered in Glomeruli** (600meq /24 hours**)** **Reabsorbed in proximal tubules** and thick ascending limb of loop of Henle (560meq/24 hours: >90%) o Active transport via Na-K-2Cl Co-transporter **Secreted/Excreted by distal tubules**/ collecting ducts (502meq/24 hours) o amount proportionate to flow rate through distal tubules (rapid flow rate reduces intertubular potassium concentration, thus facilitating secretion) o under influence of aldosterone, increases potassium secretion into the urine.				
Notes:	Bold concepts to pass				
Question B	**Explain K+ transport in the collecting duct?**				
Knowledge:	H-K ATPase in the cells of the collecting ducts reabsorbs K+ in exchange for H+. Hence if H+ secretion is increased, K+ excretion is decreased				
Notes:	Concept to pass				
Question C	**How does aldosterone increase potassium secretion into the urine?**				
Knowledge:	Aldosterone secretion is triggered by high serum K. Aldosterone acts at the DCT and cortical collecting ducts. <u>Action of aldosterone:</u> • Stimulates to Na/K ATP pump at the basolateral surface of the principal cells in the collecting tubule. 2 K enters the principal cells in exchange for 3 Na into the bloodstream at the basolateral surface • Causes K channels to form at the apical surface of the principal cells. Higher intracellular K concentration means the K enters the tubular lumen • Causes Na channels to form at the apical surface of the principal cells. Na enters the principal cells from the tubular lumen and gets into the bloodstream via the Na K ATP ase pump				
Notes:	Where action occurs and 1 action to pass				

TOPIC 5	Renal failure	**SUBJECT:**	Pathology	**LOA:**	2
STEM	Moving on to Pathology. Her blood results show acute renal failure thought to be due to acute tubular injury.				
Question A	**Define acute tubular injury.**				
Knowledge:	Clinically by acute deterioration of renal function. There is often morphological evidence of renal tubular injury.				
Notes:	Concept to pass				
Question B	**What pathological processes can cause an acute tubular injury? Please give an example of each.**				
Knowledge:	• Ischaemia due to decreased or interrupted blood flow: microscopic polyangiitis, malignant hypertension, microangiopathies and systemic conditions associated with thrombosis (e.g., HUS, TTP, DIC), decreased effective circulating blood volume, renal artery stenosis • Direct toxic injury to the tubules (e.g., by drugs, radiocontrast dyes, myoglobin, haemoglobin, radiation) • Acute tubulointerstitial nephritis: hypersensitivity reaction to drugs, infections, metabolic diseases, chronic urinary tract obstruction, transplant rejection, sjogren syndrome, vascular disease. • Urinary obstructions by tumour, prostatic hypertrophy, stones or blood clots.				
Notes:	Two causes with an example of each.				
Question C	**Describe the clinical course of an Acute tubular injury?**				
Knowledge:	Highly variable clinical course • Initiation phase: duration about 36 hours, slight decline in renal function, rise in Urea. • Maintenance Phase: sustained decreases in urine output 40 to 400ml/day, salt and water overload, rising urea, hyperkalaemia, metabolic acidosis and other manifestations of uraemia • Recovery phase: steady increase in urine output may reach up to 3L/day, large amount of sodium, potassium and potassium are lost in urine. Eventually, renal tubular function is restored, and concentrating ability improves.				
Notes:	2 out of 3 phases with basic description/concept necessary to pass				

Australasian College for Emergency Medicine

Primary Viva Examination

2019.1 Topic Overview

Thursday 2 May 2019

	Anatomy	Pathology	Physiology	Pharmacology	CBB
Viva A	Thorax / Lung	Cardiogenic Shock	CVS - Cardiac Contractility	Ketamine	CXR
Viva B	Facial Muscles and Nerves	Sepsis	Pain - Modulation/ Neurotransmitters	Antibiotics - Metronidazole	Blood Tests
Viva C	Femoral Triangle	Renal Failure	Renal Handling of Potassium	ACE Inhibitors	ECG (Hyperkalaemia)
Viva D	Humerus and Radial Nerve	Neoplasia	Lung Compliance	Opiates/ Oxycodone	Pathological Fracture

Friday 3 May 2019

	Anatomy	Pathology	Physiology	Pharmacology	CBB
Viva A	Pelvis Bone	DIC	Shock	Vitamin K	Pelvis X-ray
Viva B	Left Lung Root and Mediastinum	Aortic Dissection	Pressure Volume Curve	Glyceryl Trinitrate (GTN)	CXR
Viva C	Shoulder Joint (Model)	Osteomyelitis	GFR	Cephalosporins	Blood Results
Viva D	CT Brain	CVA	Oxygen Delivery	Midazolam	ECG Atrial Fibrillation

ACEM PRIMARY VIVA	Friday VIVA D	*Candidate Number:*	

TOPIC 1	Salbutamol	SUBJECT:	Pharmacology	LOA:	1
STEM	A patient presents to the ED with shortness of breath and wheeze. Salbutamol is administered. We will start with Pharmacology				
Question A	**What is the mechanism of action for Salbutamol?**				
Knowledge:	• Binds to **β2**-adrenergic receptor (abundant on airway smooth muscle cells). • Stimulates *adenylyl cyclase* and increases the formation of *intracellular cAMP* thereby: relaxing airway smooth muscle (bronchodilation) inhibiting release of bronchoconstricting mediators from mast cells. They may also inhibit microvascular leakage and increase muco-ciliary transport.				
Notes:	To pass – correct receptor and smooth muscle relaxation/**bronchodilation** (prompt for bronchodilation if mention smooth muscle relaxation)				
Question B	**What are the side effects of salbutamol?**				
Knowledge:	**Muscle tremor** due to stimulation of β_2 receptors in skeletal muscle is the most common **Tachycardia/palpitations/arrhythmias i.e. SVT** are due to reflex cardiac stimulation secondary to peripheral vasodilation, from direct stimulation of atrial β_2 receptors **Hypokalaemia** due to β_2 receptor stimulation of potassium entry into skeletal muscle **Ventilation-perfusion V/Q mismatch** due to pulmonary vasodilation in blood vessels previously constricted by hypoxia results in the shunting of blood to poorly ventilated areas and a fall in arterial oxygen tension. **A Metabolic effect** (increase in free fatty acid, insulin, glucose, pyruvate, and lactate) are usually seen only after large systemic doses. Lactic acidosis occasionally seen. -GI effects: **vomiting**				
Notes:	1 from each group, minimum of 3 to pass				
Question C	**What other drugs may be used to treat an acute exacerbation of asthma?**				
Knowledge:	1. Anti-muscarinics /anticholinergics e.g. Ipratropium bromide 2. Steroids e.g. Prednisolone, Hydrocortisone 3. $MgSO_4$ 4. Methylxanthines e.g. aminophylline 5. Adrenaline (life threatening cases) 6. Others- oxygen, ketamine, some volatile anaesthetic agents				
Notes:	Any 3 to pass				

TOPIC 2	Arterial blood gas (FiO2 1.0)	SUBJECT:	CBB	LOA:	Choose an item.
STEM	Moving on to the Clinical building block. An arterial blood gas is performed.				
Question A	**Describe and interpret the blood gas**				
Knowledge:	Acidaemia High pCO_2 Normal HCO_3 Hypoxia **Acute respiratory acidosis** consistent with respiratory failure Consistent with tiring patient requiring escalation of management (pharmacotherapy/assisted ventilation)				
Notes	Bold to pass				
Question B	**Describe the common causes of respiratory acidosis?**				
Knowledge:	Central respiratory depression- drugs (e.g. opiates, sedatives, anaesthetics), CVA, raised ICP, seizure Neuromuscular disorders Lung or chest wall injury/abnormality e.g. infection, trauma Airway obstruction – COPD, asthma *Prompt: that's one group, any other causes?*				
Notes:	Any 3 causes to pass				

ACEM PRIMARY VIVA	Friday VIVA D	*Candidate Number:*	

TOPIC 3	Respiratory, CO_2 transport	SUBJECT:	Physiology	LOA:	1
STEM	Moving on to Physiology.				
Question A	**How is carbon dioxide transported in the blood?**				
Knowledge:	**Three forms:** 1. **Dissolved** (5 to 10%): carbon dioxide twenty times more soluble than O_2, therefore dissolved CO_2 plays a significant role in its carriage – **10%** of gas evolved into the lung from blood is in dissolved form 2. **Bicarbonate** (90%) formed by sequence**:** $\mathbf{CO2 + H20 \leftrightharpoons H2CO3 \leftrightharpoons H + HCO3}$ (first step aided by carbonic anhydrase) 3. **Carbamino compounds** (5%): combination of CO_2 with terminal amine groups in blood proteins. Most importantly is the globin in haemoglobin. Reduced Hb binds more CO_2 as carbaminohaemoglobin than HbO_2. *Percentages are for arterial blood				
Notes:	Must get Bicarb as majority and 1 other				
Question B	**What is the chloride shift?**				
Knowledge:	The ionic dissociation of carbonic acid results in HCO_3^- and H^+ formation within the red cell. The HCO_3^- diffuses out but H^+ cannot as the cell membrane is relatively impermeable to cations. To maintain electrical neutrality, Cl^- ions move into the cell from the plasma; the hamburger effect				
Notes:	Correct description to pass				
Question C	**What is the Haldane effect in the peripheral circulation?**				
Knowledge:	The fact that the de-oxygenation of the blood increases its ability to carry CO_2 (the reduced Hb in peripheral blood help with the loading of CO_2); R shift of CA buffer equation;				
Notes:	Concept to pass				

TOPIC 4	Asthma	SUBJECT:	Pathology	LOA:	1
STEM	Moving on to Pathology				
Question A	**What is the definition of Asthma?**				
Knowledge:	Asthma is a chronic disorder of the conducting airways, usually caused by an immunological reaction, which is marked by episodic **bronchoconstriction** due to increased airway sensitivity to a variety of stimuli; **inflammation** of the bronchial walls; and **increased mucus secretion**. *Prompt: What are the primary features of asthma?*				
Notes	Must state bronchoconstriction and inflammation and increased mucous secretion				
Question B	**Describe the pathogenesis of acute atopic asthma**				
Knowledge:	Classic example of IgE mediated (Type 1) hypersensitivity Atopic triggers: environmental allergens e.g. dust, pollens, dander, food On re-exposure to antigen (Ag), the Ag-induced cross-linking of IgE bound to Fc receptors on mast cells. These cells release preformed mediators that directly and via neuronal reflexes induce bronchospasm, increased vascular permeability, mucus production, and recruitment of leukocytes. *Prompt: please provide more detail?*				
Notes:	Must mention IgE mediated & mast cell degranulation				
Question C	**What are the potential trigger agents for non-atopic asthma?** *Prompt: what are the different subtypes of asthma?*				
Knowledge:	Non-atopic: respiratory viruses, air pollutants, exercise induced, cold induced Drug induced e.g. aspirin Occupational triggers = fumes (epoxy resins, plastics), organic and chemical dusts (wood, cotton, platinum), gases (toluene), or other chemicals (formaldehyde, penicillin products). Asthmatic bronchitis in smokers				
Notes	Any Two (2) to pass				

ACEM PRIMARY VIVA	Friday VIVA D	*Candidate Number:*	

TOPIC 5	Thorax (Bones; 1st rib and normal rib)	SUBJECT:	Anatomy	LOA:	1
STEM	Moving on to Anatomy.				
Question A	**What are the features of this left/right rib?**				
Knowledge:	**Head:** wedge shaped, has two facets separated by crest of the head, one fact for articulation with the numerically corresponding vertebrae and one facet for the vertebra superior to it. **Neck:** connects head of the rib with the body at the level of the tubercle **Tubercle:** located at the junction of the neck and body, smooth articular part articulates with corresponding transverse process of the vertebra. Rough non-articular part provides attachment for costotransverse ligament. **Body:** thin, flat and curved most markedly at the costal angle where the rib turns anterolaterally. Costal groove parallels inferior border of the rib.				
Notes:	3 of 4 bold to pass (*See pic at the end of Viva)				
Question B	**What are the features of this L/R first rib?**				
Knowledge:	1. **Broadest, shortest** and most **sharply curved** of the seven true ribs. 2. **Single facet** on its head for articulation with T1 vertebrae only 3. **Two transverse grooves** on superior surface for subclavian vessels 4. These grooves separated by **scalene tubercle** (attachment for anterior scalene muscles) *Prompt: what's unique about it?*				
Notes:	Two of four to pass				
Question C	**Which muscles are responsible for respiration?**				
Knowledge:	**Diaphragm** is primary muscle of respiration, responsible for most of inspiration (expiration passive) **Costal muscles** move the ribs during forced respiration. Function primarily to support intercostal spaces, resisting negative and positive intra-thoracic pressures. Externals-inspiration; internals-help forced expiration **Accessory muscles**: scalene, sternocleidomastoid, pectoralis major and minor, serratus anterior **Abdominal wall muscles**: forced expiration *Prompt: which is the primary muscle?*				
Notes:	Two of three to pass. Diaphragm primary muscle over others.				

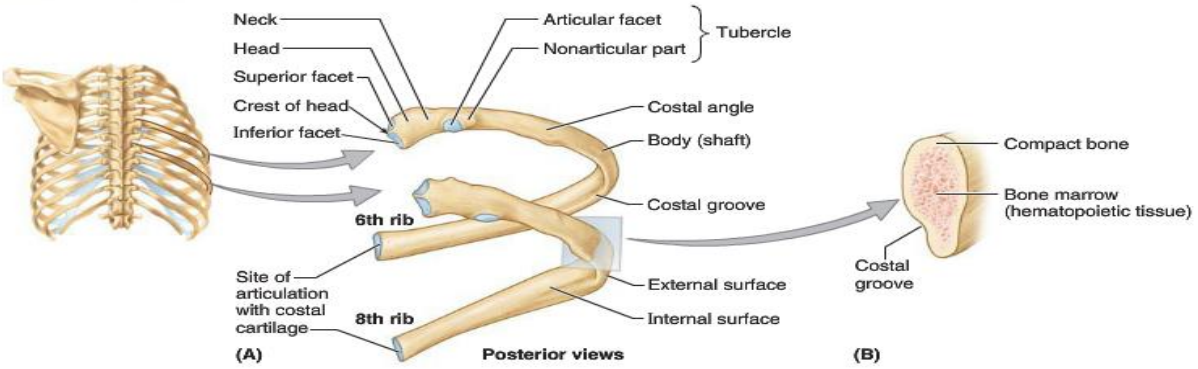
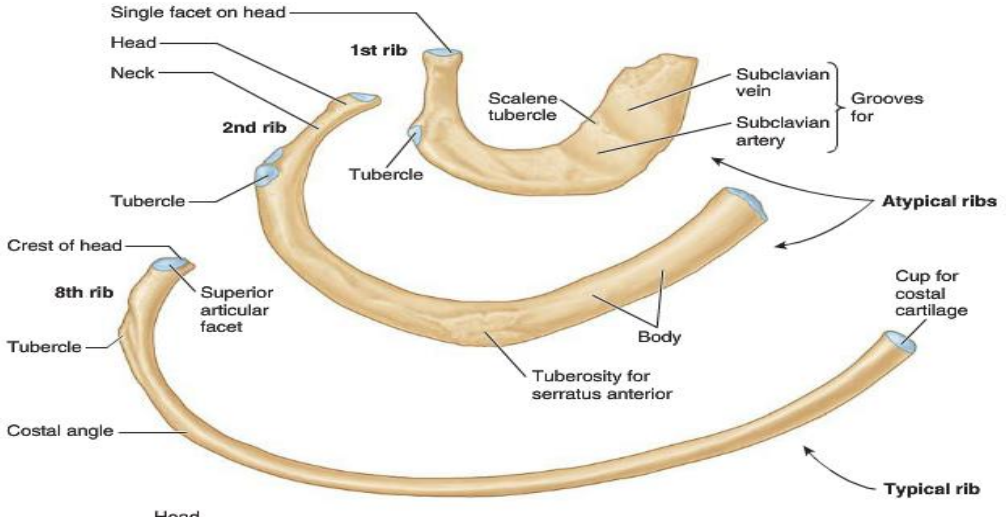


FIGURE 4.2. Typical ribs. A. The 3rd–9th ribs have common characteristics. Each rib has a head, neck, tubercle, and body (shaft). **B.** Cross-section of the midbody of a rib.



Australasian College for Emergency Medicine

Primary Viva Examination

2019.2 Topic Overview

Thursday 31 October 2019

	Anatomy	Pathology	Physiology	Pharmacology	CBB
Viva A	Hand Model	Inflammation / Wound Healing	Respiratory – Gas diffusion / Transport	CNS (Nitrous)	Hand Wound (photo)
Viva B	Femur (bone)	Coagulation Cascade	Cardiac Output	Ropivacaine	Hip X-ray
Viva C	Abdominal CT	Cholecystitis	Bilirubin	Ampicillin	Liver Function Tests
Viva D	Internal Jugular Vein	Cardiomyopathy	Cardiac Action Potential	Adenosine	ECG (SVT)

Friday 1 November 2019

	Anatomy	Pathology	Physiology	Pharmacology	CBB
Viva A	CT Brain Lobes / Ventricles	Oedema	Neuromuscular Junction	Levetiracetam	CT Brain
Viva B	Foot (photo)	Septic Arthritis	Renal Tonicity / ADH	Vancomycin	Diabetic Foot Ulcer (photo)
Viva C	Forearm (model)	Acute Coronary Syndrome	Coronary Blood Flow	Anti-platelet Agents	ECG (STEMI)
Viva D	Thorax (bones)	Asthma	Respiratory / CO_2 Transport	Salbutamol	Arterial Blood Gas

ACEM PRIMARY VIVA	VIVA A	*Candidate Number:*	

Topic 1	Atrial Fibrillation	SUBJECT:	CBB	LOA:	n/a
STEM	An 80 year old female presents after having an unwitnessed fall. An ECG is performed. We will start with the Clinical Building Block (CBB).				
Question A	**Please describe and interpret the ECG.**				
Knowledge:	Irregularly irregular rhythm, absence of p-waves Atrial fibrillation with a ventricular response of 100- 110 bpm. T wave inversion in inferior leads and minor ST depression in lateral leads V4+5 ? rate related ischaemia Coarse fibrillatory waves in V1 Normal axis				
Notes:	Atrial fibrillation to be at standard				
Question B	**What are the possible causes for the findings on this ECG in this lady?**				
Knowledge:	Sepsis – urinary, respiratory Cardiac ischemia/heart failure - cardiomyopathy/valvular pathology/ hypertension Electrolyte deficiency – potassium/magnesium Drug toxicity / alcohol Pulmonary embolus Endocrine abnormalities - thyrotoxicosis				
Notes:	3 to be at standard				
Question C	**What clinical features would indicate that this patient is unstable due to the rhythm disturbance?**				
Knowledge:	Altered mental state Angina / persistent chest pain Hypotension Hypoxia/crepitations on chest auscultation suggestive of LVF				
Notes:	2 to be at standard				

TOPIC 2	Femoral triangle (McMinn's 7th ED Page 320, 8th edition Page 328)	SUBJECT:	Anatomy	LOA:	1
STEM	Moving on to Anatomy. She is hypotensive. A femoral central line is to be inserted.				
Question A	**Please identify the boundaries of the femoral triangle on this photo.**				
Knowledge:	Superior = inguinal ligament (13) Medial = lateral border of adductor longus (1) Lateral = medial border of sartorius (19) Floor = iliopsoas (11) and pectineus (15)				
Notes:	3 boundaries which are correctly identified on the image to be at standard				
Question B	**Please identify the contents of the femoral triangle on this photo.**				
Knowledge:	Femoral Nerve (7) Femoral Artery (6) Femoral Vein (8)				
Notes:	2 to be at standard				
Question C	**What are the origin and the terminal branches of the femoral artery?**				
Knowledge:	Origin of femoral artery = **external iliac artery** Terminal branches of femoral artery = **profunda femoris** deep to adductor longus and **superficial femoral artery**				
Notes:	The origin and one terminal branch to be at standard				

TOPIC 3	Gentamicin	SUBJECT:	Pharmacology	LOA:	1
STEM	Moving on to Pharmacology. You diagnose urinary sepsis and administer Gentamicin.				
Question A	**Describe the mechanism of action of Gentamicin.**				
Knowledge:	Irreversible inhibitor of protein synthesis (bactericidal). Concentration dependent killing. Passive diffusion via porin channels then active into cytoplasm via O_2 dependent process. 2 mechanisms: 1) Bind 30S ribosome and inhibit protein synthesis 2) Disrupt polysomal structure, resulting in non-functional monosomes				
Notes:	Bind 30S ribosome and inhibit protein synthesis to be at standard				
Question B	**Describe the pharmacokinetics of gentamicin.**				
Knowledge:	-Poor oral absorption -Well absorbed IM, usually given IV. -Peak serum concentration within 30-90 mins -Highly polar, water soluble and thus does not enter cells well. Variable penetration of tissue spaces – Most tissues low except renal cortex. CSF-20 % plasma levels, bile-30 % plasma level, pleural/ synovial 50-90 %. -Dose 5-7 mg/kg ; single daily dose -Not metabolised, cleared by the kidney. Dosage adjustment needed for renal impairment. -Half-life:2-3 hours. -Post antibiotic effect – activity lasts longer than detectable serum levels. 40-60 % removed by HD.				
Notes:	Renal clearance plus any 2 to be at standard				
Question C	**How do bacteria develop resistance against gentamicin?**				
Knowledge:	Transferase that inactivates drug, carried by plasmid Impaired cell entry (cell wall) Altering ribosomal receptor protein				
Notes:	One mechanism to be at standard				

TOPIC 4	Pressure Volume Loop of Left Ventricle	SUBJECT:	Physiology	LOA:	1
STEM	She is hypotensive. Moving on to Physiology.				
Question A	**Describe the pressure and volume changes in the left ventricle during systole**				
Knowledge:	**Isovolumetric contraction** -End diastolic volume of 130mL in the LV -Intraventricular pressures rise sharply, until the pressure in the left ventricle exceeds the pressure in the aorta (80 mmHg) **Ejection / outflow phase** -The aortic valve opens. -Ventricular ejection begins. -Peak pressures in the left ventricle is 120mmHg -Stroke volume 70-90mL -Strength of ventricular contraction diminishes to point where intraventricular pressure < aortic pressure → closure of aortic valve **Isovolumetric relaxation** -Relaxation of ventricular muscle → rapid fall in intraventricular pressure -End-systolic ventricular volume of 50mL				
Notes:	1 point from each phase with adequate description.				
Question B	**What are the causes of reduced end diastolic volume (preload)?**				
Knowledge:	**Decrease venous return** - ↓Blood volume - Venous pooling (↓ skeletal muscle pump) - ↓Venous tone - Gravity / upright posture **Decrease ventricular filling** - ↑Intrathoracic / intrapericardial pressure (tamponade) - ↓Ventricular compliance (diastolic dysfunction) - Tachycardia (↓filling time) - Loss of atrial contraction (e.g. AF) - Valve stenosis (mitral / tricuspid)				
Notes:	3 examples with 1 from each category				

ACEM PRIMARY VIVA	VIVA A	*Candidate Number:*	

TOPIC 5	Gram negative sepsis	SUBJECT:	Pathology	LOA:	1
STEM	Moving on to Pathology				
Question A	**Which common organisms are the likely cause of urosepsis in this lady?**				
Knowledge:	Gram Negative Bacilli (80 %): E. coli, Proteus, Klebsiella, Enterobacter, Strep. faecalis Gram Positive: Enterococcus faecalis, Staphylococci				
Notes:	E coli plus 2 others				
Question B	**What is the pathogenesis of septic shock?**				
Knowledge:	1. Inflammatory and counter- inflammatory responses: microbial products (PAMPs) trigger production of inflammatory mediators (TNF, IL-1, IFN- alpha), PAF, PG, cytokines, chemokines and complement cascade 2. Endothelial activation and injury: cause widespread vascular leakage and tissue oedema 3. Induction of procoagulant state: decrease in production of anti-coagulant factors, dampening of fibrinolysis 4. Metabolic abnormalities: insulin resistance, hyperglycaemia, cellular hypoxia causes lactic acidosis 5. Organ dysfunction: diminished myocardial contractility, ARDS, multiorgan failure				
Notes:	2 to be at standard				
Question C	**What factors affect the outcomes of septic shock?**				
Knowledge:	1. Extent and virulence of infection 2. Immune status of the host 3. Presence of other co-morbidities 4. Pattern and level of mediator production				
Notes:	2 factors to be at standard				

ACEM PRIMARY VIVA	VIVA A	*Candidate Number:*	

TOPIC 1	Blood tests – Renal failure	SUBJECT:	CBB	LOA:	n/a
STEM	A 63 year old female presents feeling unwell with flank pain. Blood tests are performed. We will start with the Clinical building block.				
Question A	**Please describe and interpret the abnormalities on her biochemistry panel.**				
Knowledge:	Acute Renal failure (elevated Ur and Cr; intrarenal with chronic component) Hyperkalaemia Hyperglycaemia Low HCO_3^- Blood tests – Renal failure				
Notes:	Three of bold points to be at standard				
Question B	**What are the broad categories of renal failure giving an example of each?**				
Knowledge:	Pre-renal: hypovolemia Renal: toxins, medications, infection Post Renal: obstructive uropathy				
Notes:	Any 2 categories with one example to be at standard				
Question C	**"What are the possible causes of renal impairment in this patent?"**				
Knowledge:	Sepsis, Diabetic nephropathy, dehydration, drug toxicity, DKA, renal calculus and pyelonephritis				
Notes:	Renal impairment + 2 others				

TOPIC 2	Anatomy (Abdomen) – photo McMinn 8th edition page 272 Kidneys and suprarenal glands	SUBJECT:	Anatomy	LOA:	1
STEM	Moving on to Anatomy.				
Question A	**Identify the anatomical structures of the renal tract on this photo.**				
Knowledge:	Kidneys – Right (42), Left (18) Ureters- Right (50), Left (25)				
Notes:	3 different structures to be at standard				
Question B	**Identify the main intraabdominal branches of the Aorta.**				
Knowledge:	• Coeliac trunk (T12) – (2) • SMA (L1) – (52) • Renal (L1) – Right (44), Left (20) • Gonadal (L2) – Right (48), Left (23) • IMA (L3) – (3) • Common iliac (L4) - Right (30), Left (6) • Lumbar arteries at multiple levels (not visible in this photo)				
Notes:	4 to be at standard				
Question C	**Describe the arterial supply of the kidneys.**				
Knowledge:	<u>Arteries:</u> • Aorta • Renal arteries arise ~ L1/2 • Right renal artery is longer • Divides into 5 segmental arteries (each independent)				
Notes:	2 anatomical features of the course of the arteries to be at standard				

ACEM PRIMARY VIVA	VIVA A	*Candidate Number:*	

TOPIC 3	Pharmacology: ACE inhibitors	SUBJECT:	Pharmacology	LOA:	1
STEM	Moving on to Pharmacology. The patient is on Enalapril.				
Question A	**What is the mechanism of action of Enalapril?**				
Knowledge:	Competitive **block conversion of angiotensin I to II** → • **Decreased vascular tone** from prevention of vasoconstrictive effects of Ang II (main effect) • **Inhibition of aldosterone** secretion caused by Ang II leading to reduced Na & H2O resorption →decreased BP				
Notes:	2 of 3 Bold to be at standard				
Question B	**What are the adverse effects of ACE inhibitors?**				
Knowledge:	**Hypotension**, dizziness (light headedness) Headache, weakness Loss of taste Nausea, diarrhoea Cough Hyperkalaemia due to decrease in aldosterone secretion AKI Angioedema				
Notes:	Hypotension + 2 others to be at standard				
Question C	**What other drugs can interact with ACE Inhibitors and how?**				
Knowledge:	Diuretics →Hypotension and fluid loss Other nephrotoxins → AKI Anaesthetics (Any) → Hypotension Lithium → Hyperkalaemia & reduced effect of ACE Inhibitors K^+ sparing diuretics / K^+ supplements →Hyperkalaemia NSAIDs → makes ACE I non effective				
Notes:	2 to be at standard – prompt: "***How does the drug interact?"***				

TOPIC 4	Physiology: Renal handling of Potassium	SUBJECT:	Physiology	LOA:	1
STEM	Moving on to Physiology.				
Question A	How does the kidney handle potassium?				
Knowledge:	• Freely filtered in glomeruli. (600meq/24 hours) • **Majority (>90%) is reabsorbed** in **proximal tubules** and thick ascending limb of the loop of Henle (560meq/24 hours). Mainly due to passive resorption proportional to flow in proximal tubule and active transport via Na^+-K^+-$2Cl^-$ cotransporter thick ascending limb of the Loop of Henle. • **Secretion occurs in distal tubules** and collecting ducts and is approximately equal to potassium intake. The amount secreted is proportionate to flow rate through distal tubules (rapid flow rate prevents tubular potassium concentration rising and impairing passive secretion) • excretion is under influence of aldosterone in collecting ducts. Aldosterone increases potassium excretion via Na^+/K^+ ATPase. • small amount K^+ exchanged for H^+ in Collecting Duct				
Notes:	Concepts of reabsorption and secretion to be at standard Bold to pass				
Question B	How does aldosterone affect Potassium excretion?				
Knowledge:	• Aldosterone secretion is triggered by high serum K^+ • Aldosterone acts at **collecting tubules** to **increase Na^+ reabsorption and secrete K^+ and H^+.** • Effect is on principal cells in the collecting tubules: • **Na-K ATPase pump** in basolateral surface of principal cells. 3 Na^+ absorbed into bloodstream in exchange for 2K^+ into the principal cells • K^+ channels in apical cell membrane, higher intracellular K^+ concentration drives K^+ into tubular lumen and out into urine. • Na^+ channels in apical membrane. Na^+ enters from tubular lumen and then pumped into bloodstream via Na-K ATP pump.				
Notes:	**Bold to pass** The effects on the collecting tubules on Potassium and Sodium movement to be at standard				
Question C	How does a metabolic acidosis affect Potassium secretion?				
Knowledge	In a metabolic acidosis state H^+ excretion is increased and K^+ is reabsorbed in exchange, due to the action of H^+ on H^+-K^+ ATPase in collecting duct cells				
Notes	Concept of increased H+ excretion and increased K+ reabsorption in exchange to be at standard.				

ACEM PRIMARY VIVA	VIVA A	*Candidate Number:*	

TOPIC 5	Pathology- Renal failure / Cellular death	SUBJECT:	Pathology	LOA:	1
STEM	Moving on to Pathology.				
Question A	**What is acute tubular necrosis?**				
Knowledge:	• Acute renal failure with morphologic evidence of injury to tubules. • Often necrosis of epithelial cells. • Usually reversible				
Notes:	Acute renal failure with morphologic evidence of injury to tubules to be at standard				
Question B	**What are two patterns of Acute Tubular Necrosis with an example of each.**				
Knowledge:	**Ischaemic** acute tubular necrosis due to inadequate blood flow from local or systemic causes: • Local: e.g. renal artery stenosis, microangiopathies, systemic conditions associated with thrombosis (e.g., HUS, TTP, DIC) • Systemic ○ hypoperfusion from e.g. sepsis, trauma, pancreatitis **Nephrotoxic** acute tubular necrosis • e.g. endogenous toxins: myoglobin, haemoglobin, light chains, exogenous toxins: drugs (gentamicin), contrast dye, heavy metals (Hg), organic solvents (carbon tetrachloride)				
Notes:	Must mention both with 1 example of each to be at standard				
Question C	**Describe the classic clinical course of acute tubular necrosis**				
Knowledge:	**1. Initiation phase:** lasts 36 hours, transient decrease in blood flow and GFR. Decrease in urine output, increase in urea. 2. **Maintenance phase**: sustained oliguria. Rising urea, hyperkalaemia, metabolic acidosis, salt and water overload. 3. **Recovery phase**: begins with increase in urine output, large volumes lost as unable to concentrate urine. Hypokalaemia. Eventually tubular function recovers and urea and creatinine improve. Prognosis depends on extent and duration of injury.				
Notes	2 of the 3 phases to be at standard				

ACEM PRIMARY VIVA A	Thursday	*Candidate Number:*	

TOPIC 1	Brain	SUBJECT:	Anatomy	LOA:	1
STEM	A 42-year-old female presents with fever, headache and neck pain. A CT Brain is performed. We will start with Anatomy.				
Question A	**Identify the structures on this CT scan**				
Knowledge:	**Lobes: Frontal, Temporal or Parietal, Occipital** **Ventricles:** Lateral **–** anterior and posterior horns, third ventricle**,** aqueduct Deep Grey Matter – caudate, lentiform nucleus (globus pallidus and putamen), internal capsule, thalamus				
Notes:	5 structures to be at standard				
Question B	**Name the anatomical layers and spaces covering the brain**				
Knowledge:	Skin, skull, epidural space, dura mater, subdural space, arachnoid mater, subarachnoid space, pia mater, brain				
Notes:	3 structures and their correct anatomical location to be at standard				
Question C	**Describe the circulation of CSF circulation in the brain.**				
Knowledge:	Formed in choroid plexus in lateral, 3rd and 4th ventricles. Lateral ventricles → 3rd ventricle via interventricular foramina 3rd ventricle → 4th ventricle via cerebral aqueduct 4th ventricle → subarachnoid space/ spinal cord via central canal of spinal cord, Foraminae of Luschka and Magendie				
Notes:	Formation and 2 steps to be at standard				

TOPIC 2	CSF	SUBJECT:	CBB	LOA:	n/a
STEM	We will move on to the Clinical Building block. A lumbar puncture was performed.				
Question A	**Please describe and interpret the CSF result**				
Knowledge:	Turbid, low glucose, high pressure, high WCC and high protein Gram stain positive Consistent with **bacterial meningitis**				
Notes:	Bold to be at standard				
Question B	**What are the clinical features of raised intracranial pressure?**				
Knowledge:	Altered mental state Headache (early morning) Vomiting Papilloedema Dilated pupil Bradycardia Hypertension				
Notes:	3 to be at standard				
Question C	**Which CSF findings differentiate between bacterial and viral meningitis?**				
Knowledge:	Viral: 1. Normal glucose 2. Mildly elevated or normal protein 3. Clear in colour 4. Mainly lymphocytes rather than PMN 5. Negative Gram stain Bacterial: 1. Low glucose 2. Elevated protein 3. Turbid in colour 4. Mainly polymorphs 5. Positive Gram stain				
Notes:	2 differences to be at standard				

TOPIC 3	Meningitis	SUBJECT:	Pathology	LOA:	1
STEM	We will move on to Pathology				
Question A	**What are the different types of meningitis?**				
Knowledge:	Bacterial Viral Others- rickettsial, chemical/chemo, autoimmune TB, syphilis, carcinomatous/lymphomatous, fungi				
Notes:	Bacterial plus 1 other type to be at standard				
Question B	**What are the common organisms that cause bacterial meningitis in different age groups?**				
Knowledge:	Neonates – E.Coli, pseudomonas, Listeria, Group B Strep, Staph A Children – Strep pneumoniae, Haemophilus influenzae B, Neisseria meningitidis Adolescents and young adults – Neisseria meningitidis, Strep pneumoniae Older adults – Listeria, Strep pneumoniae, Neisseria meningitidis				
Notes:	3 groups plus 2 example of each to be at standard. Prompt : can you give another example				
Question C	**What are the complications of meningitis?**				
Knowledge:	Acute: • DIC • Septic shock • Loss of limbs • Seizures • Raised ICP • CVA • Hyponatraemia • Death Chronic: • Hearing loss • Learning difficulties / concentration problems				
Notes:	3 to be at standard				

TOPIC 4	Temperature control	SUBJECT:	Physiology	LOA:	1
STEM	We will move on to Physiology. The patent is febrile.				
Question A	**Describe the pathophysiological mechanisms of fever**				
Knowledge:	• Toxins (e.g. bacterial endotoxins) act on monocytes, macrophages and Kupffer cells to release cytokines that act as endogenous pyrogens • Inflammatory endogenous pyrogens – IL1, 6, IFN and TNF-alpha can be independently produced to cause a fever • Cytokines can also be produced in CNS and act directly on thermoregulation centres • Activates the preoptic area of hypothalamus causing release of Prostaglandins which resets the homeostatic set point resulting in a fever.				
Notes:	Description of two mechanisms including at least one of toxins and inflammatory pyrogens to be at standard				
Question B	**How does the body lose heat?**				
Knowledge:	Radiation and conduction – vasodilation (70%) Vaporisation of sweat (27%) Respiration (2%) Urine and defaecation (1%)				
Notes:	3 to be at standard				
Question C	**What are the body's adaptive responses to cold environments?**				
Knowledge:	Muscular – shivering, increased voluntary activity, curling up. Vascular – vasoconstriction Increased Norad/Adrenaline secretion Hunger Horripilation				
Notes:	3 to be at standard				

ACEM PRIMARY VIVA A	Thursday	*Candidate Number:*	

TOPIC 5	Anti-epileptic	SUBJECT:	Pharmacology	LOA:	1
STEM	We will move on to Pharmacology. She has a seizure and is loaded with phenytoin.				
Question A	**What is the mechanism of action of phenytoin?**				
Knowledge:	Na^+ channel blockade/ reduced neuronal Na^+ conductance and prolongation of inactivated state of Na^+ channel. Reduces Ca^{2+} influx into cells and decreases glutamate release, enhances GABA release. Inhibit generation of rapidly repetitive Action Potentials				
Notes:	Na channel blockade to be at standard				
Question B	**Describe the elimination pharmacokinetics of Phenytoin and how it affects toxicity.**				
Knowledge:	Phenytoin has **a dose dependent elimination.** It has **first order kinetics at low serum concentration,** but elimination becomes **zero-order as serum concentrations rises** with prolonged elimination and greater chances of toxicity with recurrent dosing.				
Notes:	Understanding of first and zero order kinetics to be at standard				
Question C	**What are the adverse effects of phenytoin?**				
Knowledge:	Dose related neurotoxic effects: ataxia, drowsiness, dizziness, blurred vision, hallucinations, slurred speech and confusion. Idiosyncratic: hirsutism, gingival hypertrophy, acne, facial coarsening, peripheral neuropathy Rapid IV associated with hypotension and arrhythmias				
Notes:	1 neuro and 1 cardiac to be at standard				

ACEM PRIMARY VIVA C	Friday	*Candidate Number:*	

TOPIC 1	ABG showing hypercapnia	SUBJECT:	CBB	LOA:	n/a
STEM	A 65 year old female is brought to the emergency department with acute shortness of breath. An arterial blood gas is performed. We will start with the Clinical Building Block.				
Question A	**Describe the abnormalities in this ABG.**				
Knowledge:	• Acidaemia/acidosis pH 7.2 • Hypercapnia thus **respiratory acidosis** • Low bicarbonate and negative base excess and high lactate therefore **metabolic acidosis** • Hyperoxia				
Notes:	Mixed respiratory and metabolic acidosis to be at standard				
Question B	**What are possible causes of this blood gas result in this patient?**				
Knowledge:	• Sepsis • pneumonia • CCF/APO • Infective exacerbation of COPD, any acute resp illness • Salicylate toxicity				
Notes:	Any 2 to be at standard				

TOPIC 2	Control of Respiration	SUBJECT:	Physiology	LOA:	1
STEM	Moving on to Physiology. The patient is tachypnoeic.				
Question A	**Which areas of the brain control respiration?**				
Knowledge:	• Medulla: Medullary Respiratory Centre (in particular the Pre-Botzinger Complex) generates intrinsic rhythm • Pons (apneustic centre and pneumotaxic centre) may modulate the activity of the medulla • Cerebral cortex – voluntary control • Limbic system and hypothalamus change respiration in response to emotions				
Notes:	Two areas to be at standard				
Question B	**Explain the central response to rising carbon dioxide.**				

Knowledge:	PCO_2 is tightly controlled and is the most important stimulus to ventilation
	• Main response is mediated by central chemoreceptors in medulla
	  ○ CO_2 diffuses freely across the blood brain barrier then liberates H^+ in the CSF
	  ○ central chemoreceptors stimulate respiration in response to increasing H^+ in the CSF
	• Peripheral chemoreceptors (in carotid bodies and aortic bodies) also increase ventilation in response to rising PCO_2.
	  ○ the response is smaller in magnitude but faster compared to central chemoreceptors
	• The response is magnified if PO_2 is low and reduced by sleep, age, sedative drugs, chronic CO_2 retention				

Notes:	Prompt: Which receptors mediate the response to hypercarbia? Identification of one chemoreceptor response with an explanation to be at standard.				
Question C	**Which other sensors are involved in control of respiration?**				
Knowledge:	• Pulmonary stretch receptors in airway smooth muscle • Irritant receptors between airway epithelial cells; also, in nose and upper airway • J receptors in alveolar walls • Bronchial C receptors • Joint and muscle receptors • Gamma system in muscle spindles • Arterial baroreceptors • Pain and temperature afferents				
Notes:	3 to be at standard				

TOPIC 3	Ceftriaxone	SUBJECT:	Pharmacology	LOA:	1
STEM	Moving on to Pharmacology. The patient is commenced on Ceftriaxone for pneumonia.				
Question A	**Describe the mechanism of action of Ceftriaxone?**				
Knowledge:	• Binds to bacterial cell wall peptidases leading to inhibition of bacterial growth and death of the bacterium. • Bacteriocidal antibiotic				
Notes:	Description of effect on cell wall to be at standard				
Question B	**Describe the pharmacokinetics of Ceftriaxone**				
Knowledge:	• Administration – IV and IM, <1 % bioavailability orally • Excretion: 35-70% excreted unchanged in urine / remainder excreted in bile, with some hepatic/gut metabolism • Half-life - 6 – 9 hours • Distribution – penetrates tissues and body fluids well, including CSF • Volume of Distribution – 5.8 – 13.5L • Negligible protein binding • Duration of action – 24 hours				
Notes:	3/6 to be at standard				
Question C	**What is the spectrum of activity of Ceftriaxone?**				
Knowledge:	• Broad spectrum third generation cephalosporin with good tissue penetration and effective against a broad range of Gram positive and Gram negative organisms including Haemophilus and Neisseria. Not effective against anaerobes, pseudomonas, listeria or MRSA				
Notes:	Gram positive and Gram negative (name each group or one example from each group) and at least one non-susceptible organism to be at standard. PROMPTS: Can you give examples of susceptible organisms? What organisms are NOT susceptible to ceftriaxone?				

TOPIC 4	Anterior aspect of neck	SUBJECT:	Anatomy	LOA:	1
STEM	Moving on to Anatomy. The patient becomes unstable and requires a central line.				
Question A	**Please identify the major venous structures on this photo.**				
Knowledge:	• **L: internal jugular vein (1) joins with subclavian vein (4) to form brachiocephalic vein (5)** • **R: subclavian vein (11), brachiocephalic vein (9)** (int jug vein (12) is cut) • Internal thoracic veins (6), thymic vein (7), inferior thyroid vein (3)				
Notes:	Three of left internal jugular vein (1), left subclavian vein (4), left brachiocephalic vein (5), right subclavian (11) and right brachiocephalic vein (9) to be at standard				
Question B	**What are the contents of the carotid sheath?**				
Knowledge:	• **Common carotid artery** • **Internal jugular vein** • **Vagus nerve** • Cervical lymph nodes, carotid sinus nerve, sympathetic nerve fibres				
Notes:	3 structures including at least 2 of bold structures to be at standard				
Question C	Identify the Left Common Carotid Artery and describe its course.				
Knowledge:	• L common carotid (2) arises from arch of aorta • Ascend into carotid triangle, within carotid sheath, CCA medial to IJV • Divide into internal and external carotid arteries at the level of the superior border of thyroid cartilage (within carotid triangle)				
Notes:	Prompts: Where does the left common carotid artery start and finish? 2 points to be at standard				

ACEM PRIMARY VIVA C	Friday	*Candidate Number:*	

TOPIC 5	Pneumonia	SUBJECT:	Pathology	LOA:	1
STEM	Moving onto Pathology. The chest X-ray shows consolidation.				
Question A	**Describe the two patterns of anatomic distribution for bacterial pneumonia.**				
Knowledge:	• Bronchopneumonia – patchy consolidation of the lung • Lobar pneumonia – consolidation of a large portion of lobe or an entire lobe				
Notes:	Explains the difference between them to be at standard				
Question B	**What are the clinical features of an atypical pneumonia?**				

Knowledge:	• Extrapulmonary symptoms:
	  ○ Fever
	  ○ malaise
	  ○ headache
	  ○ arthralgia
	• Pulmonary symptoms:
	  ○ Dry cough
	  ○ shortness of breath
	  ○ fever
	  ○ chest pain				

Notes:	3 features to be at standard including at least one pulmonary and one extrapulmonary symptom				
Question C	**Which risk factors predispose to the development of bacterial pneumonia?**				

Knowledge:	• Extremes of age
	• Chronic diseases (congestive heart failure, COPD, diabetes)
	• Immune deficiency
	  ○ Congenital or acquired
	  ○ Decreased or absent splenic function (sickle cell disease or post-splenectomy)				

Notes:	Two to be at standard				
Question D	**Which serious complications may develop as a consequence of pneumonia?**				
Knowledge:	• Abscess formation • Empyema • Bacteraemic dissemination – e.g. sepsis, endocarditis, meningitis, pericarditis, suppurative arthritis • Death • Respiratory failure				
	2 to be at standard				

ACEM PRIMARY VIVA C	Thursday	*Candidate Number:*	

TOPIC 1	Skin Rash	SUBJECT:	CBB	LOA:	
STEM	**A 2 year old presents to the ED with a diffuse rash. We will start with Clinical Building Block.**				
Question A	**Please describe the photo.**				
Knowledge:	• Widespread maculopapular rash on both trunk and upper limbs. • No vesicles. • Individual papules coalesce to found macular/nodular patches • Raised urticarial areas, more prominent on the extensor surfaces.				
Notes:	Maculopapular rash and one other feature to be at standard				
Question B	**What are the possible differential diagnoses in this child?**				
Knowledge:	• Allergic reaction – food, medications (antibiotic, anticonvulsant, NSAID), common allergens • Contact dermatitis • Viral e.g., EBV, rubella, roseola, parvovirus (erythema infectiosum) • Erythema multiforme -→ Steven Johnson's • Staph skin infections • Heat rash • Insect bite or stings				
Notes:	3 to be at standard				
Question C	**What further clinical assessment should be performed?**				
Knowledge:	• Vital signs – HR, BP, oxygen saturation levels, temperature, level of alertness, hypoxia, hypotension, tachycardia and fever • Airway and breathing – stridor and wheeze, oropharyngeal swelling • Perfusion – Capillary refill • Mucosal involvement – eyes, mouth • Joint swelling				
Notes	Vital signs plus one other system to be at standard				

TOPIC 2	Baroreceptors	SUBJECT:	Physiology	LOA:	1
STEM	**Moving onto Physiology. His blood pressure is 70/50 mmHg on assessment.**				
Question A	**What are baroreceptors and where are they located?**				
Knowledge:	• Stretch receptors • Located at Carotid sinus and aortic arch and within the vascular adventitia. • They are also present in both atria, at the entrance of SVC and IVC and within the pulmonary circulation.				
Notes:	Stretch receptor and 1 correct location to be at standard				
Question B	**Describe the mechanism of action of baroreceptors.**				
Knowledge:	• Sensitive to changes in pulse pressure • Stimulated by distension of structures in which they are located. Their neuronal discharge increases when the pressure in these structures rises. • The increase in baroreceptor discharges inhibits discharges of sympathetic nerves and excites the vagal (parasympathetic) innervation of the heart**.** • Results in vasodilation, venodilation, fall in BP, bradycardia and decreased CO • Allows rapid adjustments in BP in response to abrupt changes in posture, blood volume, CO or peripheral resistance				
Notes:	Need changes in pressure + parasympathetic vs sympathetic control concept to be at standard				
Question C	**Describe the response of baroreceptors in this child.**				
Knowledge:	The arterial baroreceptors are less stimulated as they are less stretched. The reduction of baroreceptor firing leads to increase in sympathetic discharge, increased HR, vasoconstriction and reduced vagal drive.				
Notes:	Correct description and recognition of hypotension in this child to be at standard. Prompt: what is the response of baroreceptors in hypotension?				

ACEM PRIMARY VIVA C	Thursday	*Candidate Number:*	

TOPIC 3	Adrenaline	SUBJECT:	Pharmacology	LOA:	1
STEM	**Moving onto Pharmacology. The patient requires Adrenaline for his hypotension.**				
Question A	**Describe the pharmacokinetics of Adrenaline**				
Knowledge:	Absorption: Poorly absorbed orally. Routes of administration: S/c, IM, IV, nebulised. Distribution: 50% protein bound. Does not cross BBB but crosses placenta Onset in seconds. Duration: 2 minutes Metabolism: Terminated in sympathetic nerve terminals and metabolised by COMT & MAO with metabolites of VMA/ MOPEG Excreted in urine.				
Notes:	3/6 to be at standard				
Question B	**What are the pharmacodynamic effects of Adrenaline?**				
Knowledge:	• Agonist effects for both Alpha and Beta receptor to the same degree. • Low dose mainly beta, higher dose more alpha • Alpha: vasoconstriction • β1: Positive inotropy and chronotropy • β2: Smooth muscle relaxation → bronchodilatation				
Notes:	Alpha and one beta effect to be at standard				
Question C	**Describe the effects of Adrenaline on other organs besides the heart.**				
Knowledge:	• Respiratory: Bronchodilation • Neuro: Pupillary dilatation, Decrease IOP • GI - relaxation of gastric smooth muscle • GU - uterine smooth muscle relaxation, bladder relaxation, bladder sphincter contraction • Salivary glands - dry mouth • Liver - Increased glycogenolysis • Metabolic acidosis • Lipolysis - increased FA and glycerol in circulation				
Notes:	3 effects (from different systems) to be at standard				

TOPIC 4	Humerus	SUBJECT:	Anatomy	LOA:	1
STEM	**Moving onto Anatomy. After two failed IV attempts an interosseous cannula was inserted into the patient's right humerus.**				
Question A	**Please identify the numbered bony features depicted on this photo of a humerus.** *McMinns page 120, Right humerus upper end – from A - front and B – behind*				
Knowledge:	1. Anatomical neck 2. Deltoid tuberosity 3. Greater tubercle 4. Groove for radial nerve 5. Head 6. Intertubercular groove 7. Lateral lip of intertubercular groove 8. Lesser tubercle 9. Medial lip of intertubercular groove 10. Surgical neck				
Notes:	6 of 10 to be at standard.				
Question B	**Name the muscles of the arm, describe their action and name the peripheral nerve which innervates them.**				
Knowledge:	**Anterior Compartment:** • **Biceps Brachii** – *Musculocutaneous Nerve (C5, **C6,** C7)*. Supinates forearm, flexes forearm when supine, short head resists dislocation of shoulder • **Coracobrachialis** - *Musculocutaneous Nerve (C5, **C6,** C7).* Helps flex and adduct arm, resists dislocation of shoulder. • **Brachialis** - *Musculocutaneous Nerve (C5, C6) and radial nerve (C5, C7*) – flexes forearm in all positions. **Posterior Compartment:** • **Triceps brachii** - *radial nerve (C5, C7, C8)*. Chief extensor of forearm, long head resists dislocation of humerus, especially important during adduction.				
Notes:	Three muscles to be at standard with the correct innervation (name of supplying nerve) and muscle action for them.				

TOPIC 5	Hypersensitivity Reactions	SUBJECT:	Pathology	LOA:	1
STEM	**Moving onto Pathology. The rash is most likely due to an allergic reaction.**				
Question A	**Outline the sequence of events in type 1 hypersensitivity.**				
Knowledge:	• Antigen exposure. • Dendritic cells present Ag to T helper (Th) cells • T Helper cells differentiate into Th2 Cells • Release of cytokines that causes B cells to produce IgE that is combined with mast cells for presentation. • On repeated exposure -Ag binds to and cross link with the IgE on the mast cells leading to activation of the mast cells to release: • Vasoactive mediators e.g. histamine, proteases, chemotactic factors. (immediate) • Lipid mediators: Leukotrienes, Complement, Prostaglandins, PAF (immediate) • Cytokines: TNF (late phase)				
Notes:	Initial and repeated antigen exposure, IgE and mast cells degranulation to be at standard				
Question B	**What are the pathological changes that occur at the tissue level?**				
Knowledge:	• Vasodilatation • Increase vascular permeability • Smooth muscle spasm (bronchospasm) • Cellular infiltration • Epithelial damage				
Notes:	3 / 5 to be at standard				
Question C	**What are the organ effects of an anaphylactic response?**				
Knowledge:	• Overwhelming activation of type 1 hypersensitivity reaction Hypotension and hypoperfusion of organs • Skin reactions: rashes and in some cases blisters • Mucosal involvement: angio-oedema • Respiratory tract: broncho infiltrates, bronchial smooth muscle contraction, Upper airway oedema leading to airway obstruction				
Notes:	3 to be at standard (from different systems)				



A from the front

B from behind



ACEM PRIMARY VIVA D	Friday	*Candidate Number:*	

TOPIC 1	Enterocolitis / Gastroenteritis	SUBJECT:	Pathology	LOA:	2
STEM	A 4 year old male presents with 5 days of vomiting and diarrhoea. We will start with **PATHOLOGY.**				
Question A	**What are the infectious causes of gastroenteritis and give examples**				
Knowledge:	• **Viral** (Norovirus, Rotavirus, Adenovirus) • **Bacterial** (Cholera, Campylobacter, Shigella, Salmonella, Enteric typhoid fever, Yersinia, E. coli, C. difficile) • **Parasitic** (Ascaris, Strongyloides, Necator, Ancylostoma, Trichuris, Enterobius, Schistosomiasis, Intestinal Cestodes, Entamoeba, Giardia, Cryptosporidium) • **Mycobacterial**				
Notes:	Two (2) categories and 1 organism from each category to be at standard. *Robbin's 10E p 789-798*				
Question B	**What are non-infectious causes of enterocolitis**				
Knowledge:	• Cystic Fibrosis • Coeliac Disease • Autoimmune enteropathy • Environmental Enteric Dysfunction • (IBD, IBS, ischaemic gut, diverticulitis) • Lactase (Disaccharidase) Deficiency/ lactose intolerance				
Notes:	2 to be at standard. *Robbin's 10E Ch17. p782-786*				
Question C	**Describe the classification of major categories of diarrhoea**				
Knowledge:	• **Secretory** (isotonic stool and persists during fasting) • **Osmotic** (excessive osmotic force exerted by unabsorbed luminal solutes, diarrhoea abates with fasting) • **Malabsorptive** (generalised failure of nutrient absorption associated with steatorrhea and relieved by fasting) • **Exudative** (secondary to inflammatory disease characterised by purulent, often bloody stools which persist with fasting)				
Notes:	Two (2) out of four (4) with appropriate explanation to be at standard. *Robbin's 10E Ch17. p783*				

TOPIC 2	Blood Gas Analysis	SUBJECT:	CBB	LOA:	
STEM	Moving on to the **CLINICAL BUILDING BLOCK.** A VBG is performed because the urinary ketones are elevated (3+)				
Question A	**Describe and interpret this VBG.** *Prompt: If candidate start to do calculations for AG, examiner will tell them that the AG is 34. If calculations for predicted CO2 started, then tell them it is 20*				
Knowledge:	• **Primary metabolic acidosis** / High anion gap metabolic acidosis AG is 34 • **Respiratory compensation, predicted CO2 20** • **Severe hyperglycaemia** • Anion Gap = >20 • Mild hypochloraemia • Corrected Na (142) and K (4.1) and uncorrected values within normal reference range • Delta-ratio is 1.25 consistent with pure HAGMA Most likely diagnosis is **DKA**.				
Notes:	Primary metabolic acidosis with respiratory compensation and glucose abnormalities to be at standard.				
Question B	**What are the priorities of treatment for DKA?**				
Knowledge:	Careful rehydration/fluid replacement /IV fluids Insulin – infusion or subcutaneous depending on severity Electrolyte monitoring/management – managing K+, Na+ Seek and treat precipitant of DKA				
Notes:	Two (2) of four (4) to be at standard				

ACEM PRIMARY VIVA D	Friday	*Candidate Number:*	

TOPIC 3	Regulation of Glucose / Metabolism	SUBJECT:	Physiology	LOA:	1
STEM	We will now move onto **PHYSIOLOGY**				
Question A	***What factors affect insulin secretion?*** *Prompt: What factors stimulate insulin release? What factors inhibit insulin release?*				
Knowledge:	**Stimulators** • Glucose, Amino acids, Intestinal hormones (GIP, GLP-1, Gastrin, Secretin, CCK), B-keto acids, Acetylcholine, Glucagon, Cyclic AMP, B-agonists, Theophylline, Sulfonylureas **Inhibitors** • Somatostatin, Insulin, Diazoxide, Thiazide diuretics, B-blockers, alpha-agonists (adrenaline, noradrenaline), K depletion, Phenytoin, 2-Deoxyglucose, Mannoheptulose, Galanin, Alloxan				
Notes:	Two (2) from each category to be at standard. *Ganong's Review of Medical Physiology 26E, Ch. 24 – Endocrine Functions of the Pancreas & Regulation of Carbohydrate metabolism, Table 24-4*				
Question B	**Describe the principal actions of insulin.** *Prompt: How does endogenous insulin effect amino acid, lipid and glucose regulation?*				
Knowledge:	**Rapid (seconds)** • Increased transport of glucose into insulin-sensitive cells, • Increased transport of amino acids into insulin-sensitive cells • Increased transport of K^+ into insulin-sensitive cells **Intermediate (minutes)** • Stimulation of protein synthesis • Inhibition of protein degradation • Activation of glycolytic enzymes and glycogen synthase • Inhibition of phosphorylase and gluconeogenic enzymes **Delayed (hours)** Increase in mRNAs for lipogenic and other enzymes				
Notes:	Two (2) actions to be at standard with actions from two (2) different phases. Ganong's Review of Medical Physiology 26E, Ch. 24 – Endocrine Functions of the Pancreas & Regulation of Carbohydrate metabolism, Table 24-1				
Question C	**What are the effects of insulin on carbohydrate regulation and metabolism in different tissues.**				
Knowledge:	• **<u>Adipose</u>**: Increases glucose entry, increases fatty acid synthesis, Increases glycerol phosphate synthesis, Increases triglyceride deposition, Activates lipoprotein lipase, Inhibits hormone-sensitive lipase, Increases K^+ uptake • **<u>Muscle</u>:** Increases glucose entry, increases glycogen synthesis, Increases amino acid uptake, Increases protein synthesis in ribosomes, Decreases protein catabolism, Decreases release of glucogenic amino acids, Increases ketone uptake, Increases K^+ uptake • **<u>Liver</u>:** Decreases ketogenesis, increases protein synthesis, Increases lipid synthesis, Decreases glucose output (decreases gluconeogenesis, increases glycogen synthesis, increases glycolysis) • **<u>General</u>:** Increases cell growth				
Notes:	Two (2) effects in two (2) different tissues. *Ganong's Review of Medical Physiology 26E, Ch. 24 – Endocrine Functions of the Pancreas & Regulation of Carbohydrate metabolism, Table 24-2*				

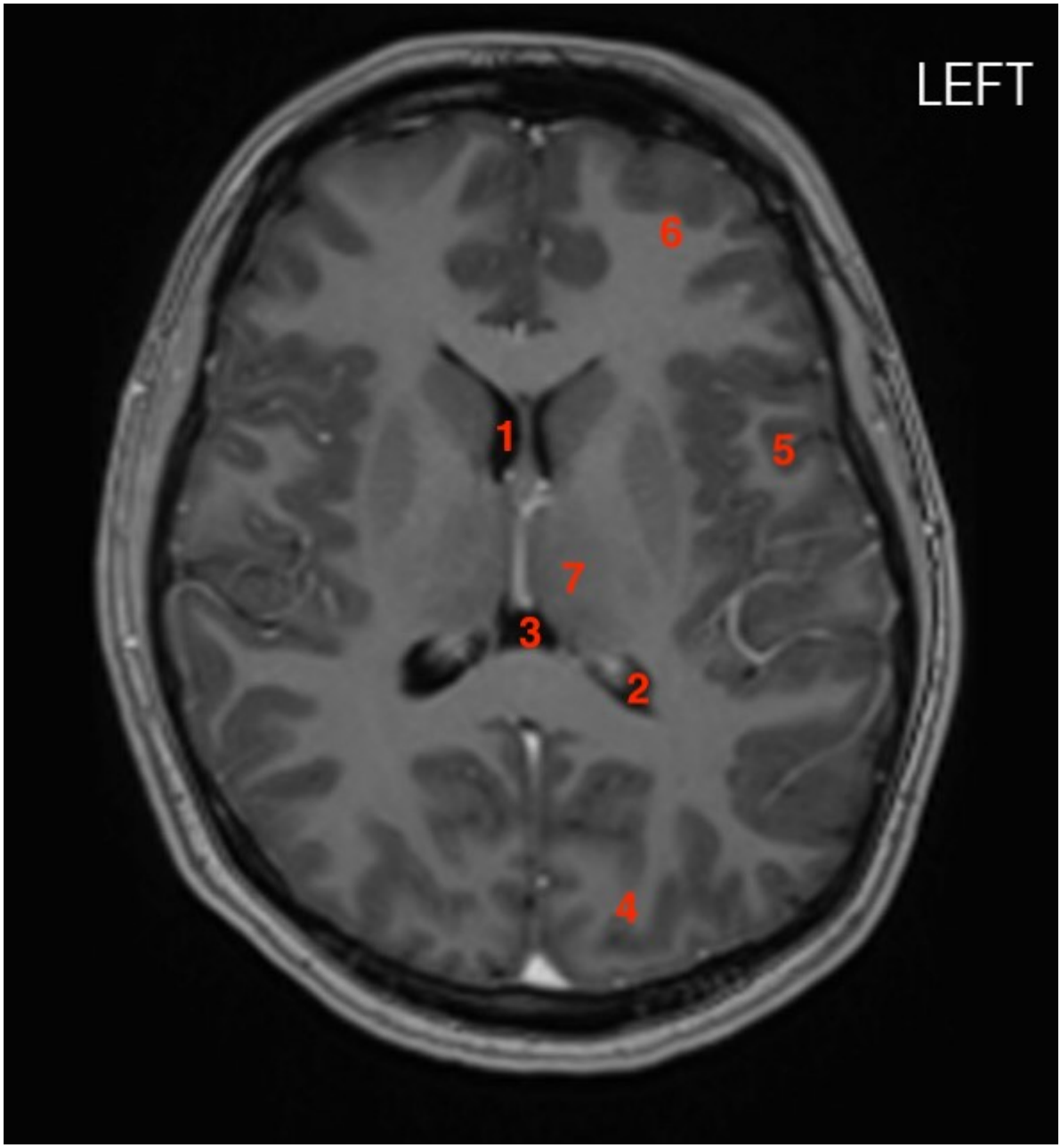
TOPIC 4	Insulin therapy	SUBJECT:	Pharmacology	LOA:	1
STEM	Moving to **Pharmacology.** He is commenced on insulin.				
Question A	**Describe, with an example, the types of exogenous insulins which can be administered**.				
Knowledge:	• **Ultra-short/short acting** – Insulin lispro (Humalog), Insulin aspart (Novorapid), Insulin glulisine (Apidra), Neutral insulin (Actrapid) • **Long acting** – Isophane insulin (Humulin, Protaphane), Insulin detemir (Levemir), Insulin glargine (Lantus Optisulin, Toujeo) • **Mixed** – Neutral with isophane insulin (Humulin, Mixtard 30/70 or 50/50), Insulin aspart with protamine (Novomix-30), Insulin lispro and Lispro protamine (Humalog), Insulin aspart with degludec (Ryzodeg)				
Notes:	Two (2) out of three (3) with one (1) example from each category to be at standard. *Katzung's Basic and Clinical Pharmacology 15E 'Pancreatic Hormones and Glucose Lowering Drugs', Table 41.6*				

Question B	**Describe the different regimens for insulin administration.** If candidate focuses on DKA, then prompt "other options once DKA resolved"
Knowledge:	• **Basal-bolus regimen** using subcutaneous injections of ultra / short acting insulin before each meal, and long-acting insulin once / twice daily • **Continuous subcutaneous infusion** of short / ultra-short acting insulin via pump with bolus doses activated by patient • **Split mixed regimens** using subcutaneous injections of short / ultra-short acting (30%) insulin combined with long-acting (70%) insulin once / twice daily • **IV insulin +/- IV dextrose for DKA**
Notes:	Two (2) to be at standard. *Katzung's Basic and Clinical Pharmacology 15E 'Pancreatic Hormones and Glucose Lowering Drugs'*
Question C	**What are adverse effects of exogenous insulin?**
Knowledge:	• **Hypoglycaemia** • Weight gain • Allergic reactions • Local reactions (erythema, itching, lipodystrophy, lipoatrophy) • Hypokalaemia • Immune insulin resistance
Notes:	Hypoglycaemia and one (1) other to be at standard. *Katzung's Basic and Clinical Pharmacology 15E 'Pancreatic Hormones and Glucose Lowering Drugs'*

TOPIC 5	CSF circulation	**SUBJECT:**	Anatomy	**LOA:**	1
STEM	Several hours later, the child's level of consciousness is reduced. A MRI of his brain is obtained. We will finish with **ANATOMY.**				
Question A	**Give the number and name the structures on this NORMAL MRI.**				
Knowledge:	1. Right anterior horn of lateral ventricle 2. Left posterior horn of lateral ventricle 3. Third ventricle 4. Occipital Lobe 5. Temporal Lobe 6. Frontal Lobe **7.** Thalamus				
Notes:	4/7 structures to be at standard				
Question B	**Describe the formation, circulation and resorption of cerebrospinal fluid**				
Knowledge:	• Secreted mainly by choroidal epithelial cells of choroid plexuses in lateral, 3rd and 4th ventricles. Rate of. 400-700 ml/day • **Lateral ventricles > Interventricular foramina > 3rd ventricle > Cerebral aqueduct > 4th ventricle** • Some CSF exits foramen of Luschka / lateral aperture and foramen of Magendie / median aperture > Subarachnoid space around spinal cord and cerebellum • Most CSF flows into interpeduncular and quadrigeminal cisterns • CSF is absorbed into the venous system through arachnoid granulations to venous sinus.				
Notes:	Point 2 to be at standard (at least 3 of the named structures) and something from point 1. *Moore's Clinically Orientated Anatomy 9E, Ch 10, p896*				
Question C	**What are the functions of cerebrospinal fluid?**				
Knowledge:	• Protects the brain by providing a cushion against blows to the head. • Provides buoyancy that prevents weight of brain from compressing CN roots and blood vessels against internal surface of cranium • Nutrient • Immune • Homeostasis function (CNS nutrition, removal of waste poducts)				
Notes:	2/ 5 to be at standard. *Moore's Clinically Orientated Anatomy 9E, Ch 10, p896*				

Venous Blood Gas

	Value	Reference Range
pH	7.20	7.35-7.45
PCO_2	20 mmHg	35-45
HCO_3	8 mmol/L	24-28
Glu	25 mmol/L	4-8
Na^+	135 mmol/L	135-145
K^+	5 mmol/L	3.5-5.5
Cl^-	93 mmol/L	95-106


LEFT
6
1
5
7
3
2
4

ACEM PRIMARY VIVA D	Thursday	*Candidate Number:*	

TOPIC 1	Shock	SUBJECT:	Pathology	LOA:	1
STEM	A 70 year old female presents with chest pain and hypotension. We will start with **PATHOLOGY.**				
Question A	**Define SHOCK.**				
Knowledge:	State of global circulatory failure that impairs tissue perfusion and leads to cellular hypoxia.				
Notes:	*Reference: Robbins Chapter 4, 10th Ed.*				
Question B	**Describe the principal mechanisms for three major types of shock and provide a clinical example for each.**				
Knowledge:	1. **Cardiogenic:** Low cardiac output due to myocardial pump failure. e.g. AMI, arrhythmia, myocarditis, cardiomyopathy. Also included obstructive causes like cardiac tamponade and PE 2. **Hypovolaemic:** low cardiac output due to low blood or plasma volume. e.g. haemorrhage, massive fluid losses from GIT or skin/burns 3. **Distributive: Shock associated with systemic inflammation/SEPTIC** activation of cytokine cascades, peripheral vasodilatation and pooling of blood, endothelial activation/injury; leucocyte-induced damage, DIC. e.g. overwhelming microbial infections, superantigens, burns, trauma, pancreatitis, neurogenic, anaphylaxis. 4. **Obstructive shock:** cardiac tamponade, pulmonary embolism.				
Notes:	Cardiogenic shock plus 2 others with correct example to be at standard. *Reference: Table 4.3 – Three Major Types of Shock, Robbins and Cotran 10th Ed, Chapter 4* *Note textbook answer classifies obstructive shock as subtype of cardiogenic. We should probably accept both. (if an example is mentioned, e.g. Septic shock, prompt: "What type of shock is this?"*				
Question C	**What are the stages of shock?**				
Knowledge:	1. <u>Initial non-progressive stage</u> Reflex compensatory mechanism maintaining vital end organ perfusion. Changes include vasoconstriction, tachycardia, renal fluid retention, shunting of blood away from the skin. 2. <u>Progressive state</u> Tissue hypoperfusion continues, worsening acidosis / metabolic derangement. Widespread tissue hypoxia. Intervention at this stage prevents vital organs developing irreversible damage. 3. <u>Irreversible stage</u> Tissue injury so severe survival is not possible even with correction of haemodynamic defects. Vital organs fail and can result in death.				
Notes:	2 stages to be at standard. *Reference: Robbins Chapter 2, Box: Key concept- cell injury and necrosis and Chapter 4, pg 137.*				

TOPIC 2	Clinical Building Block	SUBJECT:	CBB	LOA:	n/a
STEM	An ECG is performed. Moving onto the **CLINICAL BUILDING BLOCK.**				
Question A	**Describe and interpret the ECG.**				
Knowledge:	ST elevation in leads I, AVL, V2- V6 with 'tomb stoning' features. Reciprocal changes with ST depression inferior leads, mainly leads III and aVF. Interpretation: Acute anterior STEMI (We will accept Anterolateral, Anteroseptal or any variation of Anterior STEMI, but not Inferior STEMI or RV STEMI).				
Notes:	STE and interpretation to be at standard.				
Question B	**List two important medications that should be prescribed in this patient**.				
Knowledge:	• Aspirin • Clopidogrel or ticagrelor (any suitable second antiplatelet, e.g. Prasugrel) • Anticoagulant: heparin or enoxaparin • Analgesia: morphine or fentanyl • Vasodilator: GTN				
Notes:	2 to be at standard.				
Question C	**What immediate complications can occur?**				
Knowledge:	1. Arrhythmia: Ventricular tachycardia, complete heart block 2. Cardiac arrest/ death: Ventricular fibrillation or asystole 3. Cardiogenic shock 4. Acute pulmonary oedema				
Notes:	2 to be at standard.				

ACEM PRIMARY VIVA D	Thursday	*Candidate Number:*	

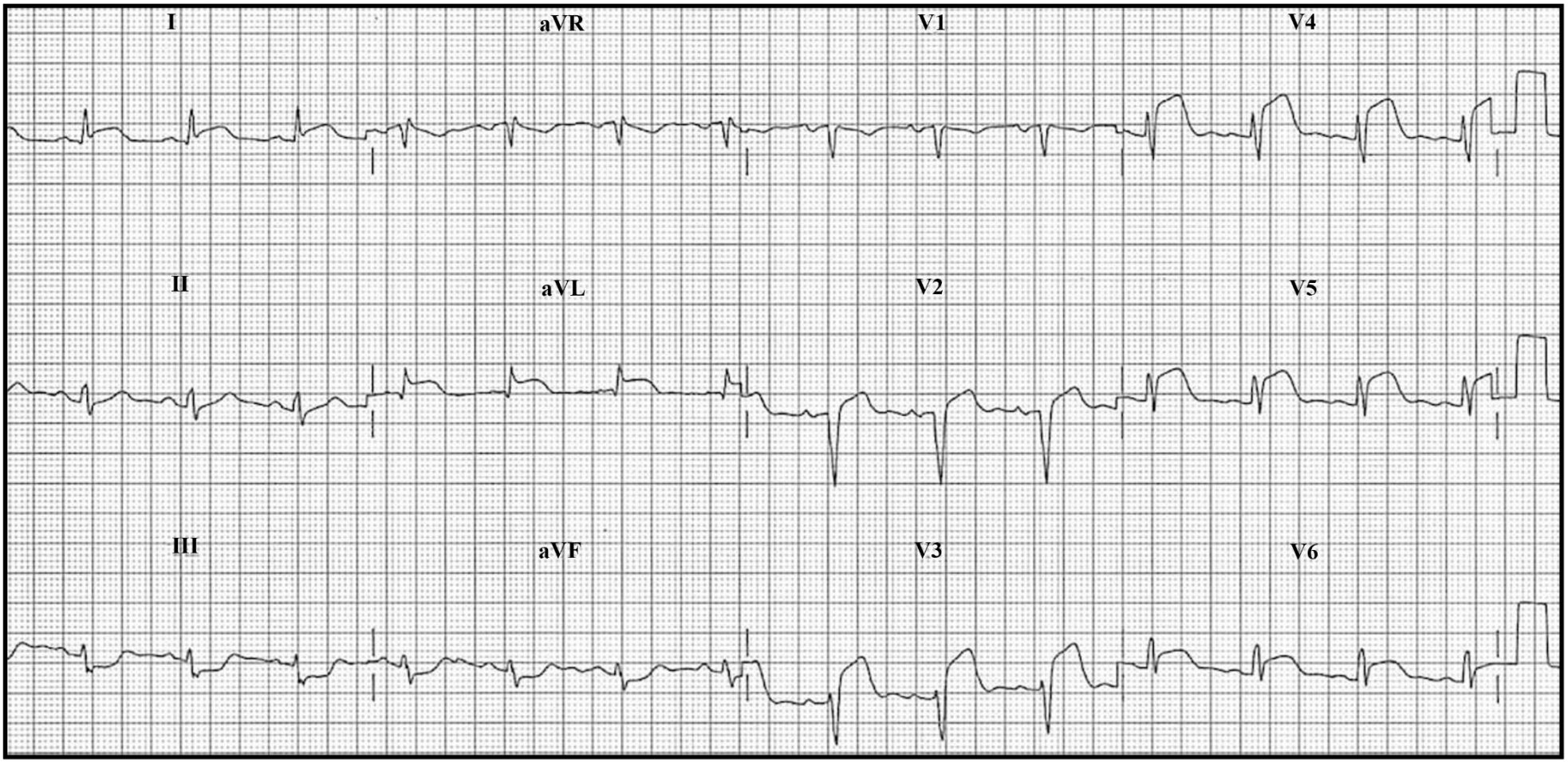
TOPIC 3	Cardiac Cycle	SUBJECT:	Physiology	LOA:	1
STEM	Moving onto **PHYSIOLOGY.**				
Question A	**Describe the phases of the cardiac cycle and the associated left ventricular pressure changes.**				
Knowledge:	**Atrial systole**: Contraction of atria, blood flows into R and L ventricles via open AV valves (mitral and tricuspid). **Isovolumetric ventricular contraction:** AV valves close, pulmonary and aortic remain closed. Ventricles are contracting without a change in volume causing a **sharp rise in pressure**. **Ventricular ejection**: Aortic valve opens when pressure in LV exceeds the aortic pressure, 80mmHg. The stroke volume is ejected, **LV peaks at 120mmHg** then drops back down. **Isovolumetric relaxation**: End of systole and beginning of diastole. Aortic valve closes once LV pressure below 80mmHg. AV valve remains closed. Rapid pressure drop, no change in volume. **Ventricular filling:** AV valves open once the **ventricular pressures fall below atrial pressures**. Blood flows into ventricles from atria.				
Notes:	Atrial and Ventricular systole to be at standard with correct left ventricular pressures. *Reference: Ganong 26 Ed, Figure 30.2 and 30.3.*				
Question B	**When does the aortic valve open and shut?** (if not mentioned above)				
Knowledge:	Aortic valve opens once LV pressure exceeds aortic pressure at 80mmHg marking the end of isovolumetric contraction. Closes once LV pressure drops below 80mmHg again during isovolumetric relaxation.				
Notes:	To be at standard: Closed during isovolumetric contraction. Open during ventricular ejection. Closed during isovolumetric relaxation. See attached diagrams for further reference. (please include) *Reference: Ganong 26 Ed, Figure 30.2 and 30.3*				

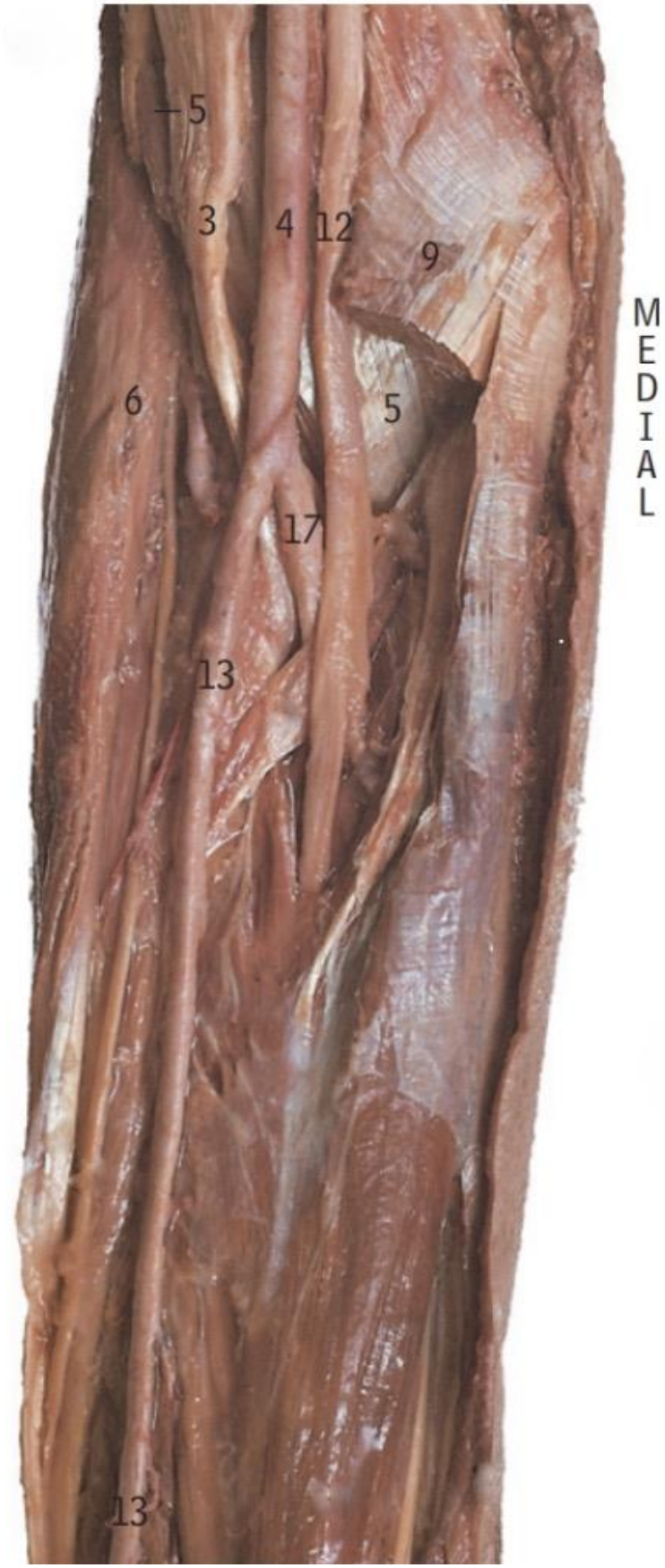
TOPIC 4	Beta Blockers	SUBJECT:	Pharmacology	LOA:	1
STEM	The patient usually takes metoprolol. Moving on to **PHARMACOLOGY.**				
Question A	**What is the mechanism of action for beta blockers in general?**				
Knowledge:	• Bind to beta receptors in the heart, lung and endocrine tissue. • Beta receptor activation by circulating catecholamines is antagonized by beta blockers • Selective bind Beta 1 receptors only, non-selective bind Beta 1 and 2 receptors. • Most are pure antagonists.				
Notes:	Antagonise beta receptors (tissue sites not required) and mention selective, non-selective to be at standard. Prompt: **"Can you expand on the different types of beta receptors that are affected?"** Reference: Katzung 13Ed, Chapter 10				
Question B	**Describe the pharmacokinetics of metoprolol.**				
Knowledge:	• **Absorption:** rapid and complete, given orally (onset 1-2 hours) and intravenously (onset 20mins when infused over 10mins) • **Bioavailability:** 50% • **Distribution:** 3.2 to 5.6L/kg; crosses the blood brain barrier • **Protein binding:** 10-12% to albumin • **Metabolism:** Extensively hepatic via CYP2D6, significant first pass effect • **Half-life elimination:** 3-4 hours in adults • **Excretion:** Urine 95%				
Notes:	4 of 7 to be at standard.				
Question C	**What are the side effects of beta blockers?**				
Knowledge:	• Cardiac: hypotension, bradycardia, heart block, depressed myocardial contractility, heart failure, dizziness • Neurological - fatigue, depression, sexual dysfunction, nightmares • Vascular - peripheral vasoconstriction, Raynaud's, claudication • Respiratory - bronchospasm • Endocrine - masks signs of hypoglycaemia (e.g. tachycardia, pupil dilatation)				
Notes:	Cardiac plus 2 others to be at standard. Prompt: "Any other non-cardiac side effects?"				

ACEM PRIMARY VIVA D	Thursday	*Candidate Number:*	

TOPIC 5	Cubital Fossa	SUBJECT:	Anatomy	LOA:	1
STEM	You place an IVC in the patient's cubital fossa to administer drugs and fluids. Moving on to **ANATOMY.**				
Question A	**Describe the boundaries of the cubital fossa.**				
Knowledge:	Triangular fossa. Base: line between the medial and lateral epicondyles of the humerus Lateral side: medial border brachioradialis Medial side: lateral border pronator teres Floor: brachialis and supinator				
Notes:	3 out of 4 correct to be at standard. *Reference: Moore 8th Ed, p212.*				
Question B	**Name the structures of the cubital fossa in this picture.**				
Knowledge:	3 - biceps/biceps tendon 4 - brachial artery 5 - brachialis 6 - brachioradialis 9 - pronator teres CUT 12 - median nerve 13 - radial artery 17 - ulnar artery				
Notes:	5 to be at standard. *Reference: McMinns p160 cubital fossa dissection – right cubital fossa diagram B.*				
Question C	**Name the branches of the brachial artery that contribute to the peri-articular anastomosis surrounding the elbow?**				
Knowledge:	• Profunda Brachii • Superior ulna collateral artery • Inferior ulna collateral artery				
Notes:	1 to be at standard. *Reference: figure 3.51, Moore's 8th Edition, page 206*				

2023.1 Viva D – CBB (Thursday)




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MEDIAL

ACEM PRIMARY VIVA B	Thursday	*Candidate Number:*	

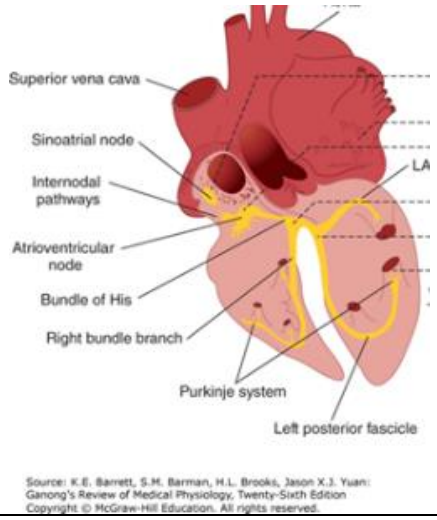
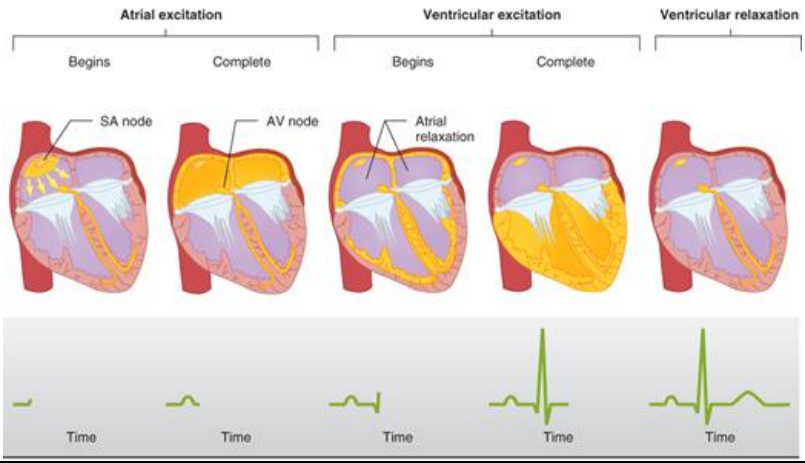
TOPIC 1	Sodium Valproate	SUBJECT:	Pharmacology	LOA:	1
STEM	**A 70 year old male presents following a seizure. We will start with Pharmacology**				
Question A	**Describe the pharmacokinetics of sodium valproate**.				
Knowledge:	Can be administered IV or orally Well absorbed orally with bioavailability of > 80%. Peak blood levels within 2hrs Highly protein bound and low volume of distribution 0.15L/Kg Extensively metabolised in the liver and excreted as glucuronide conjugate in urine (30-50% of the dose) Long half-life 9-18hrs				
Notes:	4 to be at standard *Ref: Katzung 15th edition online – chapter 24*				
Question B	**What are the adverse effects of sodium valproate?**				
Knowledge:	Most common = GI: nausea, vomiting, abdominal pain, heart burn, asymptomatic elevation of LFTs CNS: fine tremor, ataxia, sedation, fatal encephalopathy if there is also genetic abnormality of urea metabolism) Less common: weight gain, increased appetite, hair loss, rash Rare but serious: Idiosyncratic hepatic failure (risk highest < 2yrs old), idiosyncratic thrombocytopenia Teratogenic if given in 1st trimester e.g. neural tube defects, cardiovascular/facial/digital abnormalities hypernatraemia				
Notes:	4 to be at standard including one GI and one CNS *Ref: Katzung 15th edition online (ch 24); Goodman and Gilman 14th edition online (ch 20)*				
Question C	**Sodium valproate exhibits capacity limited protein binding kinetics. What is this?**				
Knowledge:	Sodium Valproate highly bound to plasma proteins (90%) at lower concentrations (75mg/L This mechanism is saturated at higher concentrations (150mg/L) leading to an increase in free drug (70% protein bound) This will result in apparent increased clearance of drug at higher doses and reduction in half life: variable clearance Thus dosage is preferred as a sustained release preparation				
Notes:	Description to be at standard *Ref: Katzung 15th edition online -chapter 24 & 3*				

TOPIC 2	Antecubital fossa	SUBJECT:	Anatomy	LOA:	1
STEM	**Moving onto Anatomy. You insert a cannula in the cubital fossa.**				
Question A	**On this picture please identify the medial and lateral borders of the cubital fossa.**				
Knowledge:	4 = pronator teres = medial border 6 = brachioradialis = lateral border				
Notes:	Both to be at standard				
Question B	**Please identify the contents of the cubital fossa.**				
Knowledge:	1 = brachial vein 2 = Median nerve 3 = Brachial artery – bifurcates into radial and ulnar arteries at the apex and has accompanying veins 7 = Biceps tendon (Radial nerve Is also in cubital fossa but not visible here) (5= Bicipital aponeurosis)				
Notes:	3 to be at standard				
Question C	**Describe the superficial lymphatics of the upper limb.**				
Knowledge:	Originate from lymphatic plexuses in hand and ascend mostly with superficial cephalic and basilic veins Some accompanying the basilic veins enter the cubital lymph nodes Efferent vessels from here drain to axillary lymph nodes Lymphatics accompanying the cephalic veins enter the axillary lymph nodes with some entering deltopectoral lymph nodes earlier				
Notes:	Accompanying cephalic and basilic veins to be at standard *Ref: Moore's clinically oriented anatomy 8th ed pg 162*				

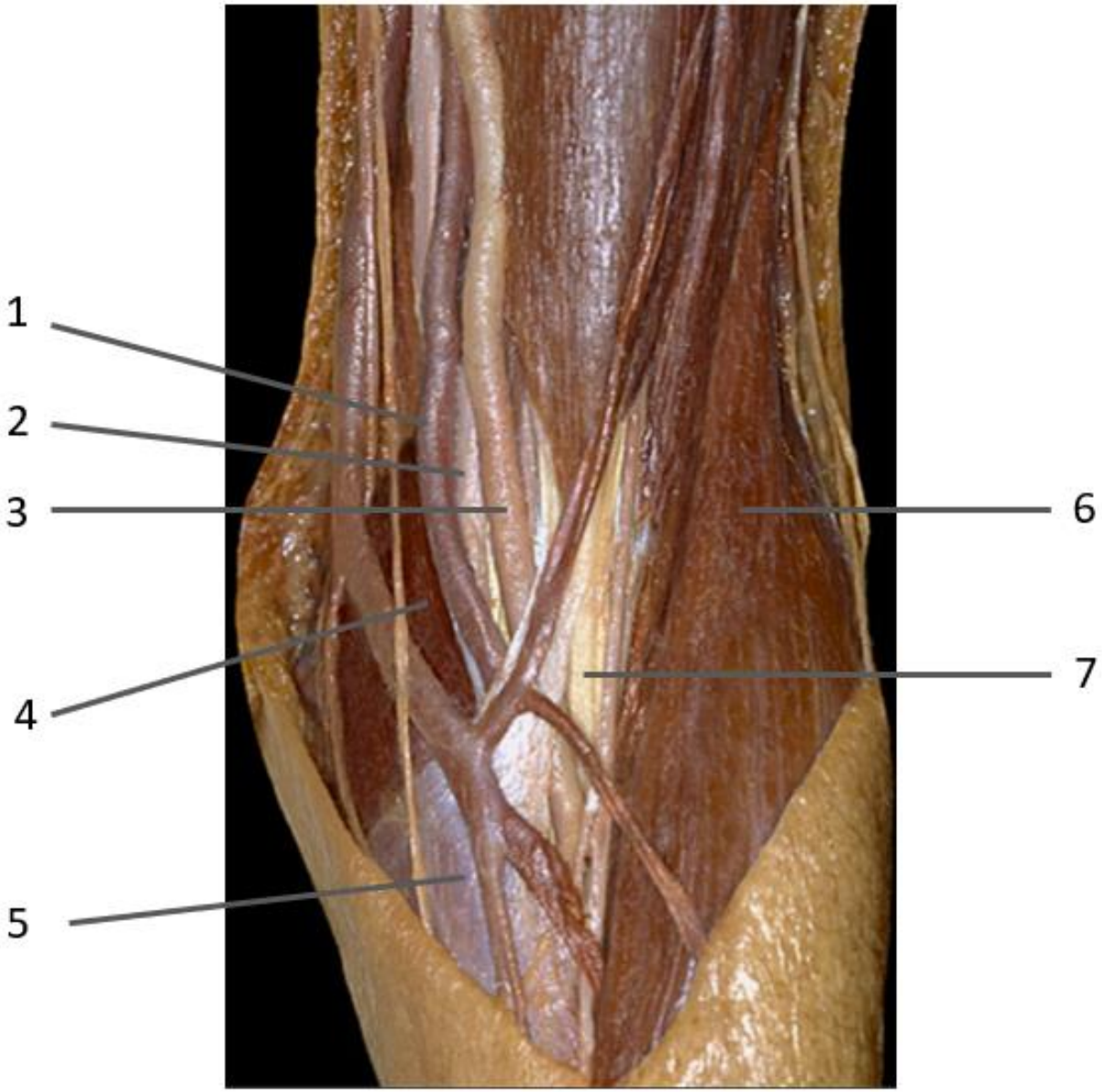
TOPIC 3	Fracture healing	SUBJECT:	Pathology	LOA:	1
STEM	**He sustained a fracture from the seizure. Moving onto pathology.**				
Question A	**Name and describe the different types of fractures.**				
Knowledge:	Simple: the overlying skin is intact. Compound: the bone communicates with the skin surface. Comminuted: the bone is fragmented. Displaced: the ends of the bone at the fracture site are not aligned. Stress: a slowly developing fracture that follows increased physical activity in which the bone is subjected to repetitive loads. Greenstick: extending only partially through the bone, common in children Pathologic: involving bone weakened by an underlying disease process e.g. tumour.				
Notes:	4 to be a standard *Reference: Robbins and Cotran Pathologic Basis of Disease 10th edition: Chapter 26 pp1183-1184*				
Question B	**Describe the steps and timeframe in fracture healing.**				
Knowledge:	1. Haematoma fills the fracture site and provides a fibrin mesh – hours 2. Influx of inflammatory cells, ingrowth of fibroblasts and capillary buds – days 3. Organising hematoma and procallus formation: inflammatory cells release growth factors which activate osteoprogenitor cells; osteoclasts and osteoblasts are activated and form a soft tissue callus – 0-2 weeks 4. Cartilage and bony callus formation: osteoblasts deposit trabeculae of woven bone chondroblasts make cartilage which undergoes endochondral ossification to form bony callus – 2-3 week 5. Resorption and remodelling: as callus matures the portions that are not stressed are resorbed/remodelled				
Notes:	3 of the 5 steps with appropriate timeframe to be at standard				
Question C	**What factors affect fracture healing?** **Prompt if required: Do you know any local factors? Do you know any systemic factors?**				
Knowledge:	Local: Inadequate immobilisation, significant displacement, infection (open, foreign body) Systemic: Diabetes, vascular insufficiency, systemic infection, malnutrition, osteoporosis, osteomalacia, smoking, corticosteroid use, age				
Notes:	2 from local and 2 from systemic to be at standard				

TOPIC 4	Complete Heart Block	SUBJECT:	CBB	LOA:	1
STEM	**Moving onto the Clinical Building Block. An ECG was performed.**				
Question A	**Describe and interpret this ECG.**				
Knowledge:	Complete AV dissociation Atrial rate 60-80 bpm Slow ventricular escape rhythm 30 bpm RBBB Left axis deviation Nil ST changes T Wave inversion V1-3 Complete/ 3rd degree AV block				
Notes:	AV dissociation or CHB to be at standard *Reference: Life in the Fast Lane*				
Question B	**What are the causes of complete heart block?**				
Knowledge:	Ischaemia especially inferior or anterior myocardial infarction Cardiomyopathy AV nodal blocking drugs – calcium channel blockers, beta blockers, digoxin Electrolyte abnormalities e.g. hyperkalaemia Idiopathic degeneration of conducting pathway (Lenegre or Lev's disease) Fibrotic disorders/sarcoidosis/amyloidosis/autoimmune disease Post cardiac surgery, ablation or PCI				
Notes:	3 to be at standard				

ACEM PRIMARY VIVA B	Thursday	*Candidate Number:*	

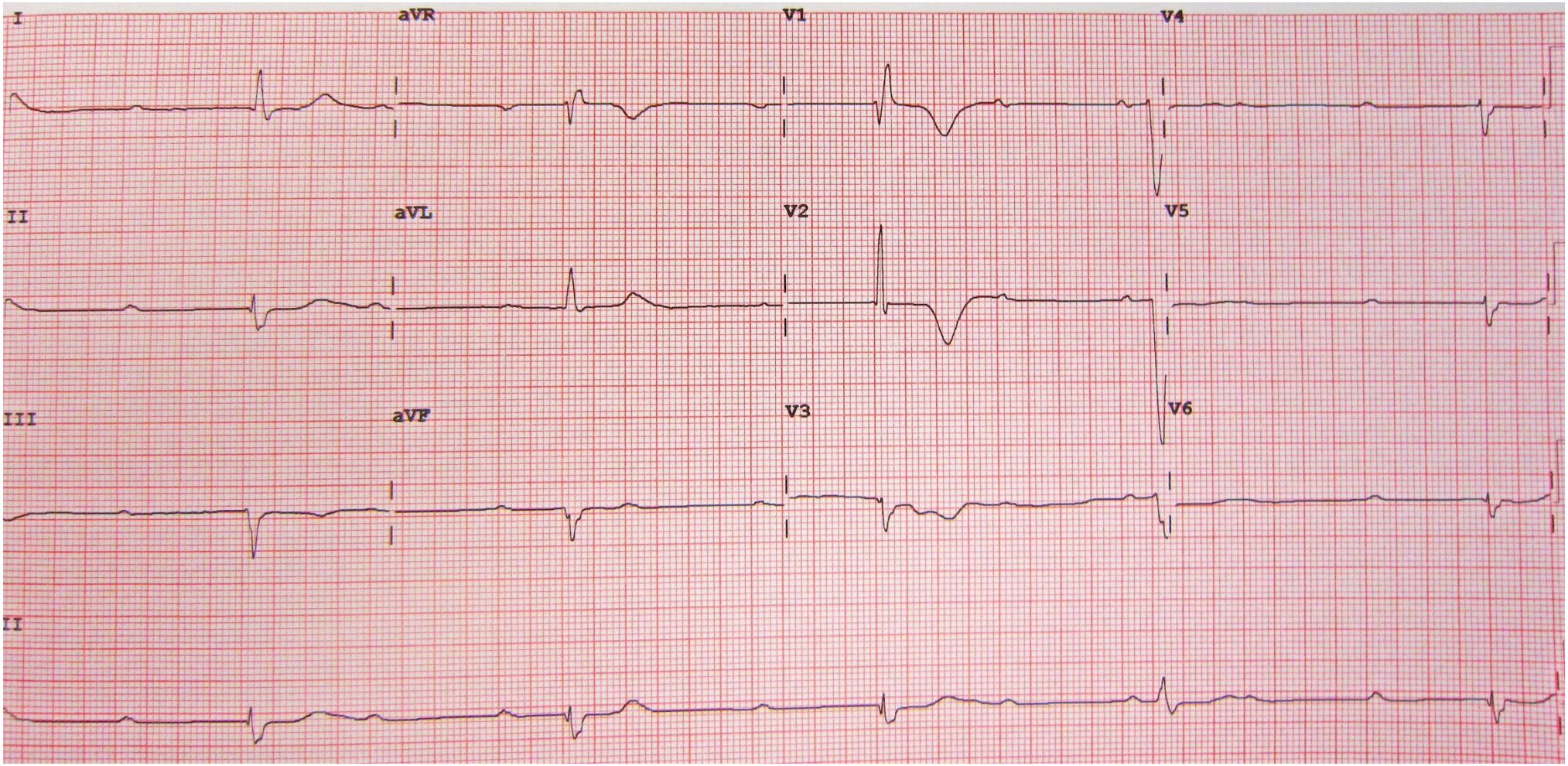
TOPIC 5	Cardiovascular	SUBJECT:	Physiology	LOA:	1
STEM	**Moving onto Physiology**				
Question A	**Please describe the conducting system of the heart.**				
Knowledge:	SA node is located at the junction of the SVC and right atrium. AV node is in the right posterior interatrial septum. Three bundles of atrial fibres connect the SA node to the AV node. AV node is continuous with the bundle of His, which gives off a left bundle branch at the top of the interventricular septum and continues as the right bundle branch. Left bundle branch divides into an anterior fascicle and a posterior fascicle. Branches and fascicles run subendocardially down either side of the septum and come into contact with the Purkinje system. Purkinje fibers spread to all parts of the ventricular myocardium.  Source: K.E. Barrett, S.M. Barman, H.L. Brooks, Jason X.J. Yuan: Ganong's Review of Medical Physiology, Twenty-Sixth Edition Copyright © McGraw-Hill Education. All rights reserved.				
Notes:	SA, AV , RBB and LBB to be at standard *Reference: Ganong Review of Medical Physiology 26th Edition Chapter 29*				
Question B	**Describe the phases of the cardiac cycle that produce the waves and segments in a normal ECG. Prompt if required: how does that relate to the ECG?**				
Knowledge:	P wave : Atrial depolarisation PR interval : AV conduction QRS : Ventricular depolarisation ST segment: plateau portion of ventricular depolarisation QT : ventricular action potential T wave: Ventricular repolarisation  				
Notes:	3 to be at standard				
Question C	**How do membrane changes in infarcted myocytes cause ST segment elevation?**				
Knowledge:	Abnormally rapid repolarisation due to accelerated opening of K+ channels (seconds to few minutes) Decreased resting membrane potential due to loss of intracellular K+ Slow depolarisation of the affected cells compared to surrounding normal cells				
Notes:	1 description to be at standard				

MEDIAL



LATERAL

2023.2 Viva B – CBB (Thursday)



ACEM PRIMARY VIVA B	Thursday	*Candidate Number:*	

TOPIC 1	Fracture dislocation of ankle	SUBJECT:	CBB	LOA:	n/a
STEM	**A 14 year old male presents with an ankle injury. Starting with the Clinical Building Block.**				
Question A	**Describe and interpret this X-ray.**				
Knowledge:	1. Posterior dislocation of the talus relative to the tibia 2. Displaced oblique fracture of the distal fibula 3. Posterior malleolar fracture with moderate displacement 4. Lateral subluxation of talus 5. Tri-malleolar fracture 6. Soft tissue changes e.g. oedema Interpretation: Fracture dislocation of left ankle				
Notes:	Fracture dislocation to be at standard. Higher performing candidate will give % dislocation. Accept Tri malleolar and dislocation.				
Question B	**What clinical findings would you look for on examination?**				
Knowledge:	1. Skin: integrity, skin tenting, open or closed fracture 2. Neuro function: sensation of the foot, movement of toes 3. Vascular: dorsalis pedis/posterior tibial pulse, cap refill time of the toes 4. Palpate: the entire tib/fib and the joint above (knee) for other deformity 5. Assess for other injuries e.g. head/ neck injuries				
Notes:	2 to be at standard. Must mention either skin/neuro/vascular as one.				
Question C	**What are the immediate clinical issues to address with this injury?**				
Knowledge:	1. Pain 2. Bleeding 3. Skin necrosis especially if tenting of skin 4. Infection if open fracture 5. Compartment syndrome 6. Neurovascular injury				
Notes:	Neurovascular injury plus 2 to be at standard. Credit can be given if Neurovascular mentioned in Question B.				

TOPIC 2	Paracetamol	SUBJECT:	Pharmacology	LOA:	1
STEM	**He was given paracetamol for pain. Moving onto Pharmacology.**				
Question A	**Describe the pharmacokinetics of paracetamol**.				
Knowledge:	Usually administered orally. Can also be given IV/PR. Peak blood levels are reached 30-60min following oral administration. Poorly bound to plasma proteins. Mostly metabolised by hepatic microsomal enzymes to inactive glucuronides and sulphates which make up 95% of excreted metabolites. 5% is metabolised via P450 dependant GSH conjugation pathway (dominant pathway in toxicity). <5% is excreted unchanged. Half-life is 2-3 hrs (can be prolonged in liver failure or toxicity). ***Prompt: If only say metabolised by the liver ask if they can give any further detail.***				
Notes:	Description of hepatic metabolism plus 2 other pharmacokinetic properties to be at standard. *Ref: Katzung 16e: Chapter 36*				
Question B	**How does paracetamol cause toxicity in overdose?**				
Knowledge:	In overdose the usual glucuronidation and sulfation pathways become saturated. P450 pathway becomes important. Metabolised to the toxic reactive metabolite NAPQI (N-acetylbenzoiminoquinone). NAPQI is detoxified by glutathione (GSH) but is depleted in toxicity. NPAQI reacts with nucleophilic cellular proteins. Causes direct cellular injury, Generation of reactive oxygen species further enhances toxicity.				
Notes:	P450 pathway and NAPQI to be at standard. Concept that mentions these two points in the pathway. *Ref: Basic and Clinical Pharmacology Katzung 16e: Chapter 4 (Fig 4-5)*				
Question C	**What are the features of paracetamol toxicity?**				
Knowledge:	GI: Nausea, Vomiting, diarrhoea, abdominal pain. CNS: dizziness, disorientation, coma (high doses). Metabolic: HAGMA. Liver injury can lead to liver failure (centrilobular necrosis). Renal failure (Tubular necrosis).				
Notes:	GI symptoms, Liver injury to be at standard. If Liver damage already mentioned in Question B credit can be given. *Ref: Basic and Clinical Pharmacology, Katzung, 16e: Chapter 36*				

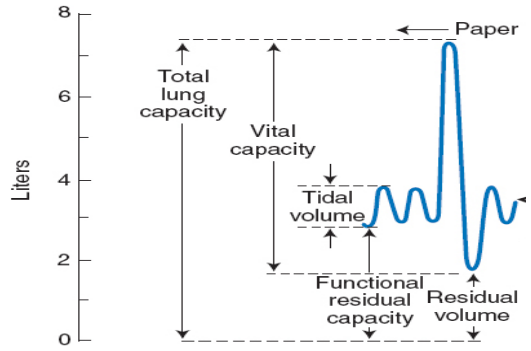
ACEM PRIMARY VIVA B	Thursday	*Candidate Number:*	

TOPIC 3	Ankle joint	SUBJECT:	Anatomy	LOA:	1
STEM	**You examine the rest of his leg. Moving onto Anatomy.**				
Question A	**Name the numbered structures on this tibia bone.**				
Knowledge:	1. Anterior border/shaft 2. Soleal line 3. Tubercles of intercondylar eminence 4. Tibial tuberosity 5. Interosseous border 6. Lateral condyle 7. Posterior surface 8. Medial border 9. Medial condyle				
Notes:	4 numbered structures to be at standard. *Ref: McMinns Clinical Anatomy, 8th edition: Tibia pg 310*				
Question B	**Describe the ligaments of the ankle joint with their attachments.**				
Knowledge:	Medial Collateral ligament (Deltoid ligament) – large strong fan shaped ligament, attaches proximally to medial malleolus. Stabilises joint during eversion and prevents subluxation. Has 4 continuous parts • Anterior and posterior tibiotalar – attaches distally to talus. • Tibio-navicular – attaches distally to navicular. • Tibio-calcaneal – distally to calcaneum. Lateral Collateral ligament – 3 separate parts • Anterior talofibular ligament – flat weak band runs anteromedially from lateral malleolus to neck of talus. • Posterior talofibular ligament – thick strong band that runs horizontally medially and slightly posteriorly from malleolar fossa of fibula to lateral tubercle of talus. • Calcaneofibular ligament – round cord that passes posteroinferiorly from lateral malleolus to lateral surface of calcaneus. ***Prompt: If start to discuss ligaments above the joint, redirect to ligaments below.***				
Notes:	Both medial and lateral ligament and one attachment/ part of each to be at standard. *Ref: Clinically Oriented Anatomy, Moore et al, 9th edition: ligaments of ankle joint Fig 7.101 & 7.102*				
Question C	**In which position of the foot do most ankle injuries occur and why?**				
Knowledge:	Plantar flexion – as the narrow end of trochlea of talus lies loosely between the malleoli, making the ankle most unstable in this position.				
Notes:	Plantar flexion to be at standard. *Ref: Clinically Oriented Anatomy, Moore et al, 9th edition: Articular surfaces of ankle joint and movements of ankle joint*				

TOPIC 4	Acute Inflammation	SUBJECT:	Pathology	LOA:	1
STEM	**You notice the ankle is red and swollen. Moving onto Pathology.**				
Question A	**Describe the reactions of blood vessels in acute inflammation.**				
Knowledge:	1. Vasodilation of small vessels leading to increased blood flow 2. Increased vascular permeability due to endothelial cell contraction and endothelial injury, enabling fluid and proteins to leave the circulation Both mechanisms maximise the movement of plasma proteins and leukocytes out of the circulation and into the site of infection or injury.				
Notes:	Vasodilation or increased vascular permeability to be at standard *Ref: Robbins and Cotran Pathologic Basis of Disease, Kumar et al, 10th edition: chapter 3, p 75*				
Question B	**Describe chemotaxis in acute inflammation.**				
Knowledge:	Leukocytes are drawn to the site of injury by a chemical gradient. Chemoattractants can be exogenous (e.g. bacterial products) or endogenous (e.g. cytokines, complement). May mention others. Chemoattractants bind to membrane (G protein coupled) receptors on leucocytes. This results in a second messenger response increasing polymerised Actin which then reorganises their cytoskeleton to migrate towards the noxious stimulus. ***Prompt: If mention Chemoattractants but no binding, ask how do they work?***				
Notes:	Description of concept of chemical gradient and chemo attractants. Description of binding for higher performing candidate. *Ref: Robbins and Cotran Pathologic Basis of Disease, Kumar et al, 10th edition: Chapter 3, p 79*				

ACEM PRIMARY VIVA B	Thursday	*Candidate Number:*	

Question C	**Describe the different possible outcomes of acute inflammation?**
Knowledge:	1. **Complete resolution*:*** removal of cellular debris and microbes by macrophages and resorption of oedema by lymphatics and regeneration of damaged tissue 2. **Fibrosis/ scarring: healing by connective tissue replacement.** After substantial tissue loss esp in tissues incapable of regeneration or when abundant fibrin exudate in tissue or serous cavities(pleura/peritoneum) 3. **Progression to chronic inflammation**: acute inflammatory response cannot be resolved due to persistence of injurious agent or interference with normal healing
Notes:	2 outcomes to be at standard *Ref: Robbins and Cotran Pathologic Basis of Disease, Kumar et al, 10th edition: Chapter 3, p95*

TOPIC 5	Lung Volumes	**SUBJECT:**	Physiology	**LOA:**	1
STEM	**During the procedural sedation he becomes hypoxic. Moving onto Physiology**				
Question A	**Please describe individual volumes in relation to total lung capacity in an adult male.**				
Knowledge:	 1 = total lung capacity: approx. 7L 2 = vital capacity: volume of a maximal expiration after a full inspiration, ~5000ml 3 = tidal volume: volume of normal breaths, ~ 500mL, 4 = functional residual capacity: volume in lungs after normal expiration, ~ 2400mL, 5 = residual volume: volume left in the lung after maximal expiration, ~1200mL,				
Notes:	3 lung volumes to be at standard. Only approximate volumes required. *Ref: West Respiratory Physiology 11e: chapter 2, Fig 2.2*				
Question B	**How does physiological dead space differ from anatomical dead space?**				
Knowledge:	Physiological dead space: parts of lung with ventilation but no perfusion, volume of the lung that does not eliminate CO_2. Anatomical dead space: conducting zones of the lung ~150mls, increases with large inspirations because of the traction or pull exerted on the bronchi by the surrounding lung parenchyma. Also depends on the size and posture of the person. Both physiological and anatomical dead spaces are almost the same in normal subjects, but physiological dead space is increased in many lung diseases due to inequality of blood flow and ventilation of lung (V/Q mismatch).				
Notes:	Definition of both to be at standard. (Concept only important) *Ref: West Respiratory Physiology 11e: chapter 2, p22-23*				
Question C	**Why is the volume of the physiologic dead space important?**				
Knowledge:	***Prompt: What are the clinical effects of increased physiological dead space? (Give this early)*** The larger the physiologic dead space, the greater the total ventilation an individual must generate to ensure an adequate amount of air enters the alveoli to participate in gas exchange. In patients with either acute or chronic lung disease, the physiologic dead space may be considerably larger because of inequality of blood flow and ventilation – leads to increased WOB.				
Notes:	Increased generation of ventilation to be at standard or increased work of breathing. *Ref: West Physiology 11e, Chapter 2, p22-23*				


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ACEM PRIMARY VIVA D	Friday	*Candidate Number:*	

TOPIC 1	Osteoporosis	SUBJECT:	Pathology	LOA:	2
STEM	**A 68 year old female presents with elbow pain post a fall. We will start with Pathology.**				
Question A	**What is the definition of osteoporosis?**				
Knowledge:	• Osteopenia (decreased bone mass) that is severe enough to significantly increase the risk of fracture. • Radiographically – a bone mass that is at least 2.5 standard deviations below mean peak bone mass in young adults. • May be localised to a certain bone or region, as in disuse osteoporosis of a limb, or may involve the entire skeleton as a manifestation of a metabolic bone disease.				
Notes:	Correct definition (bullet point 1 or 2) to be at standard. *Ref: Robbins and Cotran Pathologic Basis of Disease, Kumar et al, 10th edition: Chapter 26: Bones, Joints and soft tissue tumours*				
Question B	**What conditions contribute or predispose to osteoporosis?**				
Knowledge:	<u>Most commonly:</u> • Age-related (Senile) – diminished proliferative and biosynthetic capacity, reduced response to growth factors • Hormonal (post-menopausal) – secondary to oestrogen deficiency <u>Other causes:</u> • Reduced physical activity – mechanical forces stimulate bone remodelling; load is especially important. • Calcium nutritional state – insufficient calcium in diet of adolescents and reduced peak bone mass and the risk of osteoporosis. • Other endocrine – hyper/hypothyroidism, hyperparathyroidism, diabetes, Addison's • Gastrointestinal – malabsorption, malnutrition, vitamin C/D deficiencies, hepatic disease • Drugs – alcohol, anticoagulants, anticonvulsants, chemotherapy, steroids				
Notes:	Age or hormonal plus 2 others to be at standard (or a total of 3). *Ref: Robbins and Cotran Pathologic Basis of Disease, Kumar et al, 10th edition: Chapter 26: Bones, Joints and soft tissue tumours*				
Question C	**Name some clinical complications of osteoporosis**				
Knowledge:	• Fractures – vertebra(e) pelvis, femoral neck, wrist, • Complications of fractures: pain, immobilisation/poor mobility, pneumonia, pulmonary embolus • Vertebrae: loss of height, deformities (lumbar lordosis, kyphoscoliosis)				
Notes:	At least 2 complications to be at standard. *Ref: Robbins and Cotran Pathologic Basis of Disease, Kumar et al, 10th edition: Chapter 26: Bones, Joints and soft tissue tumours*				

ACEM PRIMARY VIVA D	Friday	*Candidate Number:*	

TOPIC 2	Renal Hydrogen Ion handling	SUBJECT:	Physiology	LOA:	1
STEM	**Moving on to Physiology. A routine urinalysis shows a urinary pH of 6.0.**				
Question A	**Where is hydrogen secreted in the kidneys?**				
Knowledge:	Proximal and distal tubules, and the collecting ducts.				
Notes:	All 3 to be at standard. *Ref: Ganong's Review of Medical Physiology, Barrett et al, 26th edition, Chapter 39: Acidification of the Urine & Bicarbonate Excretion*				
Question B	**Describe how hydrogen ions are secreted into the urine in each of those areas.**				
Knowledge:	<u>In the proximal tubule</u> • Via the Na^+-H^+ exchanger: Na^+ enters the cell along a concentration gradient, and H^+ is excreted into the lumen. • Secreted H^+ combines with filtered HCO_3^- to form H_2CO_3, which is converted by carbonic anhydrase (CA) on the apical membrane to H_2O and CO_2, which both enter the tubule. • Thus 1 Na^+ and 1 HCO_3^- enters the cell for every H^+ excreted. <u>In the distal tubule and collecting ducts</u> • Most H^+ is secreted by an ATP-driven proton pump. o Aldosterone acts on this pump to increase H^+ secretion. • Also, via a H^+-K^+ ATPase anion exchanger 1.				
Notes:	Via the Na^+-H^+ exchanger and one method of secretion in DT/CD to be at standard *Ref: Ganong's Review of Medical Physiology, Barrett et al, 26th edition, Chapter 39: Acidification of the Urine & Bicarbonate Excretion*				
Question C	**Describe the role of buffers in the kidney**				
Knowledge:	• Three buffers of importance – bicarbonate, dibasic phosphate, and ammonia • Aim to return pH to normal by increasing H+ secretion • These remove free H^+, permitting more acid to be secreted. • Allows the H^+ concentration in urine in the collecting ducts to be 1000 times the concentration in plasma – down to pH 4.5 (limiting pH)				
Notes:	2 points to be at standard. *Ref: Ganong's Review of Medical Physiology, Barrett et al, 26th edition, Chapter 39: Acidification of the Urine & Bicarbonate Excretion*				

TOPIC 3	Dislocated Elbow	SUBJECT:	CBB	LOA:	n/a
STEM	**Moving on to the Clinical Building Block. An X-ray is taken of her elbow.**				
Question A	**Please describe the X-ray**				
Knowledge:	Abnormality • Posterior dislocation of right ulna and radius • Bony fragment in joint • Nil obvious anterior/ posterior fat pad				
Notes:	Posterior dislocation of ulna and radius to be at standard				
Question B	**What structures are at risk in this injury?**				
Knowledge:	• Ligaments – ulnar collateral ligament often torn • Bone – fractures of the coronoid process, olecranon process, or head of radius • Nerve – injury to the ulnar nerve, median nerve • Arteries - brachial				
Notes:	Ulnar nerve injury plus one other to be at standard.				
Question C	**Describe the sensory changes you would expect if the ulnar nerve is injured at the elbow**				
Knowledge:	Paraesthesia of • Medial half of the palm (palmar cutaneous branch) • Medial one and a half fingers (superficial and dorsal branches) • Medial aspect of dorsum of hand (dorsal branch)				
Notes:	Medial one and a half fingers to be at standard.				

ACEM PRIMARY VIVA D	Friday	*Candidate Number:*	

TOPIC 4	Ketamine	SUBJECT:	Pharmacology	LOA:	1
STEM	**Moving on to Pharmacology. Ketamine is used during procedural sedation.**				
Question A	**Describe the pharmacokinetics of ketamine**				
Knowledge:	• Highly lipid soluble, rapid onset • Can by given IV/ IM/IN • Effect terminated by redistribution to inactive tissue sites. • Metabolised primarily in the liver (*N*-demethylation by cP450 system) o Primary active metabolite is norketamine (1/3rd-1/5th potency of ketamine) o -> hydroxylated and conjugated into water soluble inactive metabolites that are excreted in the urine • Low protein binding				
Notes:	3 factors to be at standard. *Ref: Basic and Clinical Pharmacology Katzung 16e: Chapter 25: General anaesthetics*				
Question B	**What is the mechanism of action of ketamine?**				
Knowledge:	Major effect is likely produced through inhibition of the NMDA receptor complex.				
Notes:	Inhibition of the NMDA receptor complex to be at standard. *Ref: Basic and Clinical Pharmacology Katzung 16e: Chapter 25: General anaesthetics*				
Question C	**What are the systemic effects of ketamine?**				
Knowledge:	• CNS – dissociative anaesthesia, analgesia, cerebral vasodilator, anticonvulsant, myoclonus, nystagmus • CVS – Sympathetic NS stimulation (increased BP, HR, CO), to lesser extent a direct myocardial depressant • Resp – bronchodilator, transient apnoea if given in large doses rapidly. Laryngospasm may occur due to increased respiratory secretions.				
Notes:	1 CNS and 1 from another system to be at standard. *Ref: Basic and Clinical Pharmacology Katzung 16e: Chapter 25: General anaesthetics*				

TOPIC 5	Elbow joint	SUBJECT:	Anatomy	LOA:	1
STEM	**Moving on to Anatomy.**				
Question A	**Name the numbered structures on this dissection.**				
Knowledge:	1 Abductor digiti minimi 2 Abductor pollicis longus 5 Extensor carpi radialis brevis 6 Extensor carpi radialis longus 7 Extensor carpi ulnaris 8 Extensor digiti minimi 9 Extensor digitorum 10 Extensor indicis 11 Extensor pollicis brevis 12 Extensor pollicis longus 13 Extensor retinaculum				
Notes:	5 to be at standard. *Ref: McMinns Clinical Anatomy, 8th edition, Chapter 3: Upper limb, pg 162*				
Question B	**What muscles are involved in supination and pronation?**				
Knowledge:	<u>Supination</u> • Supinator (when resistance is absent) • Biceps brachii (when power is required because of resistance) • Some assistance from EPL and ECRL <u>Pronation</u> • Pronator quadratus (primarily) • Pronator teres (secondarily) • Some assistance from FCR, palmaris longus, brachioradialis (when forearm is in mid-pronation position)				
Notes:	Supinator and pronator quadratus to be at standard. *Ref: Clinically Oriented Anatomy, Moore et al, 9th edition: Ch 3: upper limb, p 282*				
Question C	**Which nerves are involved with supination and pronation?**				
Knowledge:	• Supination – musculocutaneous and radial nerves • Pronation – median nerve				
Notes:	1 nerve from each to be at standard. *Ref: Clinically Oriented Anatomy, Moore et al, 9th edition: Ch 3: upper limb, p280 - 282*				


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ACEM PRIMARY VIVA B	Friday	*Candidate Number:*	

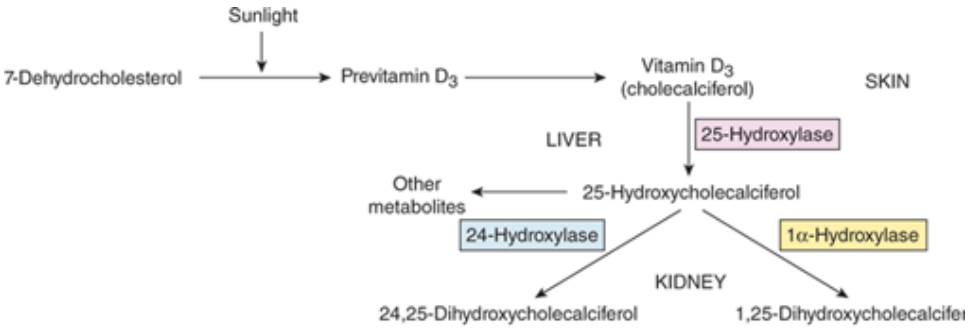
TOPIC 1	Colles fracture - X-ray	SUBJECT:	CBB	LOA:	n/a
STEM	**A 70 year old patient presents with left wrist pain following a fall on an outstretched hand. We will start with the Clinical Building Block.**				
Question A	**Describe and interpret this patient's wrist X-ray.**				
Knowledge:	AP: • Comminuted intra-articular fracture of left distal radial metaphysis. • Minimally displaced • Radial shortening. • No ulnar styloid, carpal or metacarpal bone involvement fractures. • Ring on finger 4 Lateral: • Approximately 40-degrees of dorsal angulation. Diagnosis: • Colles fracture.				
Notes:	3 descriptors to be at standard.				
Question B	**What features would prompt you to consider reducing this fracture in the emergency department?**				
Knowledge:	• >5mm radial shortening • Initial displacement >10mm • Dorsal angulation > 5^{o} from vertical (normal is about 10^{0} volar angulation) • Neurological compromise • Vascular/skin compromise • Delayed operative management				
Notes:	3 to be at standard (exact angle/length not required). *Ref: R.J. Dunn, M. Borland, D. O'Brien. The Emergency Medicine Manual. Venom Publishing: temm.com.au Upper limb injuries, distal forearm injuries, Colles type fracture.*				
Question C	**What are the short and long term complications of this fracture?**				
Knowledge:	Short term: • Open fracture/infection (most commonly volar skin tear) • Median nerve injury/compression • Compartment syndrome – rare 0.25%, almost always volar compartment • Vascular compromise e.g. radial/ulnar artery injury Long term: • Malunion/ delayed union / non-union • Joint stiffness/pain • Radioulnar/radiocarpal instability • Complex regional pain syndrome • Extensor pollicis longus tendon rupture • Secondary osteoarthritis/long term disability from loss of position				
Notes:	4 (any) to be at standard. *Ref: R.J. Dunn, M. Borland, D. O'Brien. The Emergency Medicine Manual. Venom Publishing, P. Cameron, M. Little, B. Mitra, C. Deasy (eds.). Textbook of Adult Emergency Medicine Ch4.4, P158.*				

ACEM PRIMARY VIVA B	Friday	*Candidate Number:*	

TOPIC 2	Antiemetic – Ondansetron	SUBJECT:	Pharmacology	LOA:	1
STEM	**Moving on to Pharmacology. The patient is given ondansetron for their nausea.**				
Question A	**What is the mechanism of action of ondansetron?**				
Knowledge:	• Selective 5-HT_3-receptor antagonists with potent antiemetic properties • Mediated in part through central 5-HT_3-receptor blockade in the vomiting centre and chemoreceptor trigger zone. • Main antiemetic effects are through blockade of peripheral 5-HT_3 receptors on extrinsic intestinal vagal and spinal afferent nerves. • Antiemetic action of these agents is restricted to emesis attributable to vagal stimulation (e.g. postoperative) and chemotherapy; other emetic stimuli such as motion sickness are poorly controlled.				
Notes:	Antagonists at 5-HT_3 receptors to be at standard. *Ref: Basic and Clinical Pharmacology Katzung 16e: Ch 62 Gastrointestinal Drugs.*				
Question B	**What are the side effects of ondansetron?**				
Knowledge:	• Headache, Dizziness • Constipation • QT prolongation • Serotonin syndrome has been reported in patients taking 5-HT_3-receptor antagonists in combination with other serotonergic drugs.				
Notes:	2 to be at standard. *Ref: Basic and Clinical Pharmacology Katzung 16e: Ch 62 Gastrointestinal Drugs.*				
Question C	**In which patients would you consider modifying ondansetron's dosing?**				
Knowledge:	• For patients with severe hepatic insufficiency, dose reduction may be required. • Known/pre-existing QT prolongation, taking other QT prolonging meds • Children				
Notes:	2 to be at standard. *Ref: Basic and Clinical Pharmacology Katzung 16e: Ch 62 Gastrointestinal Drugs.*				

TOPIC 3	Hand – Thenar Muscles.	SUBJECT:	Anatomy	LOA:	1
STEM	**Moving on to Anatomy.**				
Question A	**Please identify the numbered *muscular* structures visible in this dissection.**				
Knowledge:	1. Abductor pollicis brevis 2. Abductor digiti minimi 3. Adductor pollicis 4. Flexor carpi radialis 5. Flexor carpi ulnaris 6. Flexor digiti minimi brevis 7. Flexor pollicis brevis 8. Palmaris brevis 9. Lumbrical				
Notes:	4 to be at standard. *Ref: McMinn's Clinical Atlas of Human Anatomy, Abrahams et al, 8e, Chapter 3: Upper Limb, p166 B*				
Question B	**Describe the actions of the thenar muscles in the hand.**				
Knowledge:	• Opponens pollicis = Opposes thumb. • Abductor pollicis brevis = Abducts thumb; helps oppose thumb • Flexor pollicis brevis = Flexes thumb • Adductor pollicis = Adducts thumb toward lateral border of palm.				
Notes:	2 thenar muscles to be at standard. *Ref: Clinically Oriented Anatomy, Moore et al, 9th edition: Ch 6: upper limb, p 776.*				
Question C	**Which nerves supply the thenar muscles?**				
Knowledge:	• Recurrent br. of median n. (C8, T1) = Opponens pollicis, Abductor pollicis brevis, Flexor pollicis brevis (superficial head). • Deep br. of ulnar n. (C8, T1) = Flexor pollicis brevis (deep head), Adductor pollicis.				
Notes:	Prompt: can you be specific regarding the **branches** of the nerves you identified? 1 nerve and 1 correct muscle innervated to be at standard. *Ref: Clinically Oriented Anatomy, Moore et al, 9th edition: Ch 6: upper limb, pg 776.*				

TOPIC 4	Basic Pathology – Fibrosis and Scar Formation	SUBJECT:	Pathology	LOA:	2
STEM	**Moving on to Pathology.**				
Question A	**What are the phases of scar formation?**				
Knowledge:	Repair by connective tissue deposition consists of sequential processes that follow tissue injury: 1. Inflammation 2. Cell proliferation (epithelial cells, endothelial cells and fibroblasts) 3. Formation of granulation tissue 4. Deposition of connective tissue				
Notes:	2 correct phases to be at standard. *Ref: Robbins and Cotran Pathologic Basis of Disease, Kumar et al, 10th edition: Chapter 3: Inflammation and repair. p105*				
Question B	**What is the role of macrophages in wound healing?**				
Knowledge:	• Play a central role in repair by: o Clearing offending agents and dead tissue o Providing growth factors for proliferation of various cells o Secreting cytokines that stimulate fibroblast proliferation and connective tissue synthesis and deposition • Classically activated macrophages (inflammation and microbe/dead tissue removal) gradually replaced by alternatively activated (M2) macrophages (terminate inflammation and induce repair)				
Notes:	2 actions to be at standard. *Ref: Robbins and Cotran Pathologic Basis of Disease, Kumar et al, 10th edition: Chapter 3: Inflammation and repair. p106*				
Question C	**What factors influence tissue repair?**				
Knowledge:	<u>Wound factors:</u> • Infection • Poor perfusion • Foreign bodies • Type and extent of tissue injury • Character of tissue • Location of injury • Mechanical factors <u>Patient factors:</u> • Diabetes • Nutritional status • Glucocorticoids				
Notes:	2 wound factors and 1 patient factor to be at standard. *Ref: Robbins and Cotran Pathologic Basis of Disease, Kumar et al, 10th edition: Chapter 3: Inflammation and repair. p108.*				

TOPIC 5	Endocrine – Calcium homeostasis	SUBJECT:	Physiology	LOA:	1
STEM	**Moving on to Physiology. The patient is known to have osteoporosis.**				
Question A	**Where is calcium stored in the body?**				
Knowledge:	• Bone (99%) • Diffusible (ionised or complexed to HCO_3, citrate) • Non-diffusible (bound to albumin, globulin)				
Notes:	Bone and 1 other to be at standard. *Ref: Ganong's Review of Medical Physiology, Barrett et al, 26th edition, Ch 21 p370.*				
Question B	**How is the plasma concentration of calcium regulated?**				
Knowledge:	Increase: • PTH: Increases plasma Ca^{2+} by mobilising Ca^{2+} from bone, increasing Ca^{2+} reabsorption in kidney (may be offset by increased filtration) and increasing formation of 1-25 dihydroxycholecalciferol in the kidneys. • 1-25 dihydroxycholecalciferol: Increases Ca^{2+} absorption from intestine and kidneys. • Growth hormone: Increases Ca^{2+} excretion in urine and absorption in intestine. Net effect increases plasma concentration. Decrease: • Calcitonin: Secreted by thyroid to lower plasma Ca^{2+} by inhibiting bone resorption and increasing excretion from kidney. • Glucocorticoids: Decrease plasma Ca^{2+} by inhibiting osteoclast formation and activity acutely, (decrease bone formation and increase bone resorption over long periods) • Oestrogen: Inhibit stimulatory effects of cytokines on osteoclasts				
Notes:	**Prompt if needed: What is the site of action of "x" and what is the effect of "x" at that site?** 1 increase and 1 decrease factor and correct action to be at standard. *Ref: Ganong's Review of Medical Physiology, Barrett et al, 26th edition, Ch 21 p377*				
Question C	**How is 1,25 dihydroxycholecalciferol synthesised?**				
Knowledge:	 • Sunlight converts 7-dehydrocholesterol to Pre vit D3 • Vit D3 is converted to 25- hydroxycholecalciferol by 25- hydroxylase in the liver • 25- hydroxycholecalciferol is converted to 1, 25 dihydroxycholecalciferol in the kidney				
Notes:	2 correct steps to be at standard. *Ref: Ganong's Review of Medical Physiology, Barrett et al, 26th edition, Ch 21 p372.*				

2025.1 Viva B – CBB (Friday)






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