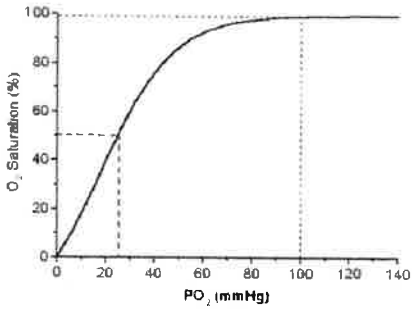
ACEM PRIMARY VIVA B **Thursday Morning Session 1** ***Candidate Number:*** **AGREED MARK:**

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