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