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

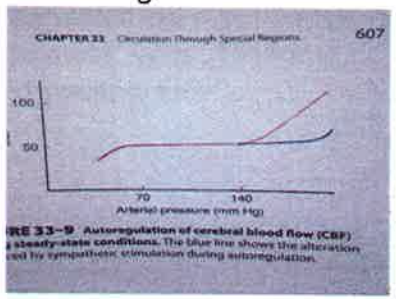
Question b (to pass) - age group and severity of illness + at least 2 symptoms or syndromes associated with each type.

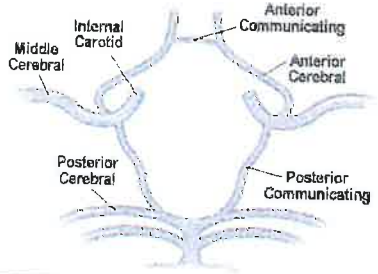
Age + 2 clinical + 1 pathology to pass

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