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

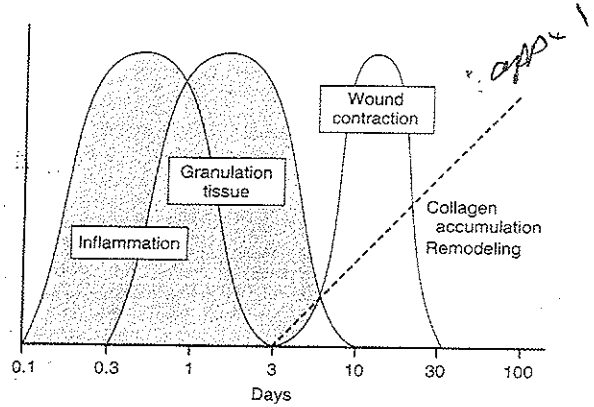
Qn 3 ACEM PRIMARY 2009/2 PATHOLOGY VIVA Thursday 17 am *Candidate Number*..... **AGREED MARK**.....

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