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