TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Haemostasis LOA: 1	**In hemostasis, describe the sequence of events at the site of vascular injury**	• Transient **vasoconstriction** by neurogenic and via local secretion of factors eg endothelin • Endothelial damage exposes ECM, leads to • **Platelet adherence, secretion & activation** leading to the primary haemostatic plug • Tissue factor is exposed, resulting in activation of **coagulation cascade** and **thrombin** generation, converting **fibrinogen to fibrin** leading to secondary haemostasis consolidating the initial platelet plug • **Polymerised fibrin and platelet aggregates** to form permanent plug • Counter regulatory mechanisms limit plug to site of injury	**Must state** • Vasoconstriction • Platelets • Coagulation cascade • Fibrin
	What factors restrict clotting to the site of vascular injury? **Prompt:** What prevents runaway clotting of the vascular tree?	• **Endogenous anticoagulants** o **Antithrombins** eg AT III, inhibit thrombin and IXa, Xa, Xia, XIIa o **Proteins C and S -** inactivate Va, VIIIa o **TFPI** (Tissue factor pathway inhibitor) • **Fibrinolytic cascade activation** o **Plasmin** from plasminogen (via factor XII or plasminogen activators) to break down fibrin & interfere with its polymerisation o **tPA** = the most important plasminogen activator	**Must include** concepts of : • Endogenous anticoagulants • Activation fibrinolysis
Question 2 Fracture healing LOA: 1	**How do fractures heal?** Prompt: What are the timeframes of these stages? **What factors impair fracture healing?**	1 Haematoma formation/fibrin mesh - hrs 2 Inflammatory cell influx - days 3 Fibroblast/ Osteoprogenitor cells-procallus 4 Organised haematoma - 1wk, 5 Woven bone , bony callus - 2-3 wks 6 Callus maturation remodelling - 6 wks Inadequate immobilisation, severe displacement, vascular compromise, infection /FBs, poor nutrition, systemic illnesses	Must have reasonable sequence and approximate times, at least 4 components to sequence **At least 3**

Question 3 Subarachnoid haemorrhage	**Where in the cerebral circulation are saccular (berry) aneurysms commonly located?** ***Prompt:*** At what part of these vessels are they most likely to arise? **What factors increase the likelihood of rupture of these aneurysms?** **What are the pathological sequelae of subarachnoid haemorrhage?**	90% near major arterial **branch points – Anterior** Cerebral A / ACoA (40%); MCA / AChoroidalA (34%); ICA / PCoA (20%); Basilar A / PCoA. Multiple in 20% – 30% cases at autopsy. Increased likelihood with **size** (> 10mm) – 50% risk of rupture per year. May occur at anytime but in about 1/3 associated with **acute increases in ICP** (e.g. straining at stool; orgasm). Acute events (hours to days) – **ischaemic injury (stroke)** from **vasospasm** (especially basal SAH). Late events (healing process) – meningeal fibrosis and **scarring**; may lead to obstruction to **CSF** flow and /or to CSF absorption. Death	Mention of branch points and anterior circulation to pass. **Bold** to pass. **Two of bold** to pass.
Question 4 Endocarditis LOA: 1	**What factors predispose patients to infective endocarditis?** **Which organisms commonly cause infective endocarditis?** **What are the complications of infective endocarditis?** (Prompt to get each group)	**Cardiac factors** – Myxomatous mitral valve, calcific aortic stenosis, bicuspid aortic valve, prosthetic valves, rheumatic heart disease **Host factors** – neutropaenia, immunodeficiency, malignancy, therapeutic immunosuppression, diabetes, alcohol, intravenous drug use, bacteraemia. **Streptococcus viridans**; **Staph aureus**; Staph epidermidis; enterococci; HACEK (Haemophilus, Actinobacillus, Cardiobacterium, Kingella); fungi **Local – erosion / destruction** of underlying cardiac tissue (valve, myocardium); abscess formation. **Systemic – systemic emboli** – infarcts / septic infarcts – brain, kidneys, lung, subcutaneous tissues, retina. Other - glomerulonephritis (immunologically medicated)	Need 4 (2 from each group) **Bold plus one other** to pass **1 local and 1 systemic**
Question 5 ALTE/SIDS LOA: 2	**What is Sudden Infant Death Syndrome?** **What risk factors have been identified?**	**The sudden death of an infant under 1 year of age which remains unexplained after thorough investigation and autopsy.** Parental risks- young mum <20, maternal smoking or drug use, low SES, deficient pre-natal care Infant risks- premature, low BW, male, SIDS in sibling, brainstem anomalies. Environment- prone sleeping, soft bedding and co-sleeping, hyperthermia	Accurate definition (age & unexplained nature) **At least 3** risk factors

ACEM PRIMARY 2012/1 Pathology VIVA March 29 Thursday Afternoon Session 2 ***Candidate Number……..*** **AGREED MARK……**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Thrombosis LOA: 1	**What factors predispose to thrombus formation?** (Prompt: Give an example of a clinical situation where each factor occurs) **Expanding on hypercoagulable states, what are the broad categories and give examples of each type?**	Virchow's triad - • **Endothelial injury** • **Alteration in blood flow** • **Hypercoagulability** • **Primary (Genetic)** Mutations- Factor V Leiden, Prothrombin Increased - factors VIII, IX, XI, or fibrinogen Deficiencies- AT3, Protein C, S • **Secondary (Acquired)** Prolonged bed rest, immobilisation, MI, AF, Tissue injury, prosthetic valves, cancer, DIC, HITS, Anti phospholipid Antibody Cardiomyopathy, nephrotic syndrome, pregnancy, post partum, OCP, sickle, smoking Note often multifactorial	**Bold 3** Plus **1 example for each** **Bold + 2 examples** **Bold + 3 examples**
Question 2 Septic shock LOA: 1	**How do microbes initiate septic shock?** **What are the effects of the mediators on the coagulation pathway?** **What are the consequent effects on tissues?**	1. Interaction with innate cells of immune system- neutrophils. macrophages and monocytes 2. Humoral interaction to activate complement and coagulation path 3. Direct endothelial action 4. End result is mediator release TNF,IL 6,8,10, NO,PAF, PAI-1 Microvascular thrombosis, decreased fibrinolysis, DIC Tissue ischaemia, multi organ failure	**at least 3 to pass** **2/3 to pass** **Either**
Question 3 Jaundice LOA: 1	**Outline the normal metabolism and elimination of bilirubin?**	Bilirubin production from heme (breakdown of senescent **erythrocytes**) 2. Binds to serum **albumin** and delivered to liver. 3. **Hepatocellular uptake**. 4. **Glucuronidation** – bilirubin glucuronides excreted into bile. 5. Gut **deconjugation** – colourless urobilinogens. These and pigment residues excreted in faeces. ~20% urobilinogens **reabsorbed** in ileum and colon and returned to liver. Small amount of reabsorbed urobilinogen excreted in urine	**three of bold** to pass

	What are the common causes of jaundice? (Prompt for bold)	Disorders that affect the production and metabolism of bilirubin: 1. Predominantly unconjugated: ↑**production** (haemolyisis; resorption of blood from internal haemorrhage; ineffective erythropoiesis); ↓hepatocyte uptake (drug interference with membrane carrier systems; Gilbert syndrome – some cases); impaired bilirubin conjugation (physiological jaundice of newborn - ↓UGTA1 activity; breast milk jaundice - β-glucuronidases; genetic deficiency of UGTA1 (Crigler-Najjar); Gilbert syndrome (autosomal recessive ↓UGTA1 activity); **hepatitis** (diffuse hepatocellular disease eg viral; drugs; cirrhosis). 2. Predominantly conjugated: **impaired bile flow;** deficiency of canalicular membrane transporters (Dubin-Johnson syndrome; Rotor syndrome)	**Bold to pass**
Question 4 ARDS LOA: 2	**Describe the pathogenesis of ARDS** **What conditions are associated with the development of ARDS?**	Initial **injury to alveolar capillary membrane** (endothelium); **acute inflammatory response** (neutrophil mediated); results in **increased vascular permeability** and alveolar flooding; fibrin deposition; formation of hyaline membranes; and widespread **surfactant abnormalities** (damage to Type II pneumocytes); eventually – organisation with scarring **Infection** (sepsis, diffuse pulmonary infection, gastric aspiration) **Physical / Injury** (trauma – head, pulmonary, fractures, near drowning, burns, radiation) **Inhaled irritants** (O2 toxicity, smoke, irritant gases and chemicals) **Chemical injury** (Heroin, barbituate, acetylsalicylic acid, paraquat) **Haematological conditions** (multiple transfusions, DIC) **Other** (pancreatitis, uraemia, cardiopulmonary bypass, hypersensitivity – organic solvents, drugs)	**3 of 4 bold** **Need 3 groups** (with **example** from each); **must include infection**
Question 5 Anaemia LOA: 2	**What are the causes of intravascular haemolysis?** **What are the manifestations of intravascular haemolysis?** (Prompt: In the blood? In the urine?)	-mechanical injury to cells (valves, microthrombi, other physical trauma) - complement fixation (eg transfusion reaction) -toxic injury (eg clostridia), - parasites (eg malaria) Anaemia, haemoglobinuria, haemoglobinaemia, jaundice, haemosiderinuria	**3 causes** **3 manifestations**

ACEM PRIMARY 2012/1 Pathology VIVA March 30 Friday Morning Session 3 *Candidate Number……..* AGREED MARK……

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Embolism LOA: 1	**What is an embolus?** **What types of emboli do you know of?** **What are the features of fat embolism syndrome?** **Prompt** – What systems may be affected in fat embolism syndrome?	A **detached intravascular** solid/liquid/gas **mass** that is carried by the blood stream from its site of origin to a **distant site**. • Pulmonary • Arterial thromboemboli • Fat emboli • Air emboli • Amniotic fluid • **Associated with long bone fractures,** rarely soft tissue injury/burns • Only 10% symptomatic • **Pulmonary insufficiency**- SOB, ↑RR,↑HR • **Neurologic symptoms**- irritability, restlessness, delirium, coma • **Anaemia**- due to RBC aggregation/haemolysis • **Thrombocytopaenia**- platelet adhesion/aggregation, leads to petechial rash	**Bold to pass** **3 examples to pass** **3/5 bold to pass**
Question 2 Wound Healing LOA: 1	**Describe the process of healing of an incised skin wound?** (Prompt: include the timing of these processes.) **What factors influence wound healing?**	**a) Formation of a blood clot – immediate** **b) Neutrophil migration at wound margins – within 24 hours** **c) Formation of granulation tissue (fibroblasts and vascular endothelial tissue). Blood vessels are leaky and proteins and fluid pass into the extravascular space leading to oedema– 24-72 hours** d) Cell proliferation and Collagen deposition – neutrophils are replaced by macrophages between 48 and 96 hours e) Scar formation – leucocytic infiltrate, oedema and increased vascularity disappear; increased accumulation of collagen – second week f) Wound Contraction – formation of myofibroblasts at the wound edges that contract. g) Connective tissue remodelling h) Recovery of Tensile strength – 10% at 1 week to a peak of 70-80% at 3 months a) Local (infection / mechanical eg motion of wound / FB / size, location, type eg incised vs blunt trauma) b) Systemic (nutrition / metabolic status / circulatory status / hormones)	**Bold 3 and 2 others = 5** **To pass: 2 local & 2 systemic**
Question 3 Cor	**What is cor pulmonale?**	Right sided heart failure that is **not secondary to left sided heart failure (pure RHF).** It can be acute (eg massive PE) or chronic (eg chronic lung disease).	**Bold** to pass

pulmonale			
	What are the common causes of cor pulmonale?	Diseases of pulmonary parenchyma (COPD; fibrosis; bronchiectasis). Diseases of pulmonary vessels (Primary pulmonary hypertension; recurrent PE; extensive pulmonary arteritis eg Wegener's granulomatosis). Disorders affecting chest movement (marked obesity; kyphoscoliosis; neuromuscular). Disorders causing pulmonary arterial constriction (hypoxaemia; metabolic acidosis; chronic sleep apnoea; altitude sickness). Common feature of all these is **pulmonary hypertension.**	**Bold** plus 3 other to pass
	What are the major morphological features of cor pulmonale? (Prompt: what are the organ features?)	Pulmonary congestion is minimal whereas engorgement of the systemic & portal venous systems may be pronounced. Heart: right ventricular hypertrophy and dilatation; leftward bulging of septum. Liver / portal system: congestive hepatomegaly; centrilobular necrosis; congestive splenomegaly Pleura, pericardial and peritoneal spaces: effusions; ascites. Subcutaneous tissues: oedema (dependent and peripheral portions of body; anasarca)	At least **three** to pass.
Question 4 UTI	**What organisms cause acute pyelonephritis?** Prompt: what are the most common?	**G-ve bacilli** (>85%), endogenous organisms E Coli, proteus, klebsiella, enterobacter, strep faecalis Other: staph, fungi, (viruses in immunocompromised and renal transplant patients)	**G-ve & 3 organisms pass**
	What steps are involved in ascending infection?	**5 steps:** 1. colonisation distal urethra 2. entry into bladder 3 . urinary tract obstruction / stasis of urine 4. vesicoureteric reflux 5. intrarenal reflux	Need to explain the steps clearly
	What are the features of chronic pyelonephritis?	**Chronic** = chronic reflux or obstruction causes pelvocalyceal damage. Recurrent infections lead to recurrent bouts of renal inflammation and **scarring**	**Bold** & concept
Question 5 Chronic Pancreatitis	**What are the morphological features of chronic pancreatitis?**	Parenchymal fibrosis, reduced number and size of acini with relative sparing of islets of Langerhans. Variable dilation +- blockage of pancreatic ducts. Destruction of exocrine parenchyma and in later stages destruction of endocrine parenchyma. Calcification.	**Any 3.**
	What are the clinical consequences?	Irreversible impairment of pancreatic function including: Diabetes; Steatorrhea; Malabsorption chronic attack not immediately life threatening but long term outlook poor(50% 20-25 mortality) Disease may be silent. Amylase, lipase may not raise in chronic attack	**Any 3**

		Pseudocyst	