CVS VIVAs (Pathology)

shakEM

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2015.1.A.3

Question 1 Cardiomyopathy Subject: Path LOA: 2	Name the types of cardiomyopathy. *(Prompt: based on function/pathology)* What are the causes of acquired cardiomyopathy? How do dilated and hypertrophic cardiomyopathy differ? *Prompt: left ventricular structure and function*	**Dilated cardiomyopathy (DCM), Hypertrophic cardiomyopathy (HCM),** Restrictive cardiomyopathy **Infections** (viral, bacterial, fungal, protozoal); **Metabolic** (hyperthyroidism, nutritional) **Infiltrative** (sarcoid, carcinoma) **Immunological** (autoimmune myocarditis) **Drugs/toxins** *(alcohol, chemotherapy)* Ischaemic, hypertensive, valvular. DCM: cardiac dilatation, **poor LV EF** (<40%). **Impaired contractility** (systolic dysfunction) HCM: myocardial hypertrophy, **normal or high LV EF. Impaired compliance** (diastolic dysfunction)	Bold 3/5 bold + and examples Bold for each

2015.1.C.1

Question 3 Aortic Dissection **Subject:** Path LOA: 1	What sequence of changes occur in the vessel wall in aortic dissection? What are the risk factors? What are the types of aortic dissection? Prompt = classification?	**Intimal tear** into media of aorta, **strips along laminar planes**, formation of blood filled channel which may then rupture outwards. Men aged 40-60 with **hypertension** Connective tissue disorders eg Marfans Complication of arterial cannulation Trauma Stanford Type A – proximal ascending + (DeBakey I)/-(DeBakey II) distal, may rupture back through Ao Valve . B is Stanford Type B – beyond subclavian artery (DeBakey III)	Bold (conceptually) **Hypertension + one other** **Concept (prox & distal)**

2015.1.D.2

Question 2 Atherosclerosis **Subject:** Path LOA: 1	1. What are the systemic and local factors that lead to atherosclerosis? 2. Which arteries are most often affected by atherosclerosis? 3. How does an atherosclerotic plaque suddenly cause symptoms?	1. **Hypertension, hyperlipidemia,** toxins from **cigarette smoke,** homocysteine, infectious agents. Inflammatory cytokines (e.g., tumor necrosis factor [TNF]) can also stimulate pro-atherogenic patterns of endothelial cell gene expression. The two most important causes of endothelial dysfunction are ***hemodynamic disturbances and hypercholesterolemia***. **Local flow disturbances (e.g., turbulence at branch points)** leads to increased susceptibility of certain portions of a vessel wall to plaque formation. 2. **Lower abdominal aorta, the coronary arteries, the popliteal arteries, the internal carotid arteries, and the vessels of the circle of Willis.** 3. **Rupture, ulceration, or erosion** of the intimal surface of atheromatous plaques exposes the blood to highly thrombogenic substances and induces **thrombosis.** Such thrombosis can partially or completely occlude the lumen and lead to downstream ischemia **Haemorrhage into a plaque.** Rupture of the overlying fibrous cap, or of the thin-walled vessels in the areas of neovascularization, can cause intra-plaque haemorrhage. **Atheroembolism**: Plaque rupture can discharge atherosclerotic debris into the bloodstream, producing microemboli. **Aneurysm formation**: Atherosclerosis-induced pressure or ischemic atrophy of the underlying media, with loss of elastic tissue, causes weakness resulting in aneurysmal dilation and potential vessel rupture	1. Bold to pass 2. 3 of 5 bold to pass 3. 2 of 4 bold to pass

2014.2.A.2

Question 4 Calcific Aortic Stenosis (pp 561-563) **Subject:** Path LOA: 2	1. What are the predisposing factors for calcific aortic stenosis?	**Age**: normal valve 70-90 yrs, bicuspid 50-70 Bicuspid valve or other congenital abnormality Wear and tear, chronic injury Hyperlipidemia, hypertension, inflammation Other factors associated with atherosclerosis	Bold and one other
	2. What are the clinical consequences of aortic stenosis?	Gradual **obstruction of LV outflow** leads to concentric **LVH** – pressure overload **Ischaemia**/angina Can get systolic and diastolic dysfunction **CHF** and syncope herald decompensation.	3 out of 4 concepts in bold to pass
	3. What are the potential complications of a congenital bicuspid aortic valve?	**Calcification, stenosis**, regurgitation, infective endocarditis, aortic dilatation, dissection	Bold and 2 other

2013.2.C.2

Question 4 **PATHOLOGY** **Healing post MI** LOA: 1	1. What are the consequences and complications of a myocardial infarction	1. Contractile dysfunction/CCF, Arrhythmias, Myocardial rupture, Pericarditis, R vent infarction & RHF, infarct extension, Infarct expansion, Mural thrombus (=>embolism), Ventricular aneurysm, Papillary muscle dysfunction, Progressive late HF, Remodelling, death	6
Robbins pp 551-553, 102-106	2. What are the main cardiac rupture syndromes	2. Free wall -> tamponade (most common of 3 occurs at 1-10 days) Septum -> VSD and L->R shunt Papillary muscle dysfunction -> severe Mitral Regurg	1 of 3
	3. What changes occur in ventricular remodelling	**3. Hypertrophy** and **dilatation**, increased oxygen demand -> **ischaemia** & **depressed cardiac function**, **scar formation** -> **stiffening** and hypertrophy.	3
	4. What systemic factors affect infarct healing?	4. Nutritional: **protein, Vit C** Metabolic: **diabetes** Circulatory: **arterial** or **venous** Hormonal: **glucocorticoids**	3

2013.1.2

Question 3 IHD LOA: 1	1. In myocardial infarction, what sequence of events leads to acute coronary artery occlusion? Prompt- pathological events	**1. Sudden change in atheromatous plaque** haemorrhage, erosion, ulceration, rupture, fissure **Platelet** adherence, activation & aggregation leading to microthromi Vasospasm from plt released mediators Activation of coagulation pathway causing **thrombus** Vessel occlusion	**Bold to pass**
	2. Describe the time course of myocardial injury after acute coronary artery occlusion. Prompt- What happens to the myocardial tissue over time?	**2. Reversible** • cessation of aerobic metabolism seconds • decreased ATP production • lactic acid production (noxious metabolites) • loss of contractility, acute heart failure 1 min • ultrastructural changes – myofibrillar relaxation, glycogen depletion, cell & mitochondrial swelling few minutes • ATP depletion up to 40 min **Irreversible** • myocyte injury – defects in sarcolemmal membrane and cell leakage 20 - 40min • initially subendocardial then transmural myocyte death • microvascular injury 1 hour • coagulation necrosis > 2 hours (more protracted if collaterals)	**Bold to pass with minutes to hours concept**

2012.2.1

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

2012.2.2

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.

2012.2.4

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

2012.1.1

Question 4 Endocarditis LOA: 1	**What factors predispose patients to infective endocarditis?**	**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.	Need 4 (2 from each group)
	Which organisms commonly cause infective endocarditis?	**Streptococcus viridans**; **Staph aureus**; Staph epidermidis; enterococci; HACEK (Haemophilus, Actinobacillus, Cardiobacterium, Kingella); fungi	**Bold plus one other** to pass
	What are the complications of infective endocarditis? (Prompt to get each group)	**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)	**1 local and 1 systemic**

2011.1.1

Question 3. **Hypertension**	1. **What factors are thought to contribute to essential hypertension?** □	Multiple genetic polymorphisms and interacting environmental factors: **Genetic factors** - familial, multi-gene foci interactions - single gene disorders altering Na reabsorption (rare) **Vasoconstrictive influences** - vasoconstriction/structural change in vessel wall -> increase in peripheral resistance -> primary hypertension **Environmental factors** - stress, obesity, smoking, physical inactivity, high salt intake	**2 of 3 bold**, with detail
	2. What are the long term consequences of essential hypertension?	Major risk factor for atherosclerosis • Coronary artery disease • Cerebrovascular disease) • Aortic dissection • Renal failure • Cardiac hypertrophy • Cardiac failure • Multi infarct dementia • Retinal changes	**4 of 7** consequences□
	3. Describe the clinical features of malignant hypertension?	Clinical syndrome characterised by • severe hypertension with SBP > 200, DBP > 120 • renal failure • encephalopathy • CVS abnormalities • retinal haemorrhages +/- papilloedema • often superimposed on previous benign hypertension • < 5% of hypertensive patients • rapidly rising BP • untreated -> death in 1-2 years□	**Must mention 3 organ systems.**

2010.1.1

Question 4: Aortic dissection	a) Describe the pathogenesis of an aortic dissection.	a)**Medial** weakness (commonly from **hypertension**), medial hypertrophy vasa vasorum, **intimal tear**, blood flow dissects the media -> medial haematoma. Cystic medial degeneration Risk factors - HT, CT disease eg Marfan's, Ehlers-Danlos, iatrogenic, pregnancy,	**Bold to pass**
	b) How are aortic dissections classified?	**By site of involvement, proximal (A) and distal (B),** DeBakey I, II, III I – ascending and descending II – ascending only III – descending only (better prognosis)	bold
	c) What are the potential consequences of the disease?	Rupture back into intima or out through adventitia Most common cause of death is rupture into pericardial, pleural or peritoneal cavities Other outcomes include cardiac tamponade, aortic insufficiency, MI, extension into any of the branches of the aorta causing obstruction +/- ischaemia, transverse myelitis	At least 3

2009.1

Question 5: **Hypertrophic cardiomyopathy**	**What are the characteristics of hypertrophic cardiomyopathy?**	1. **Myocardial hypertrophy without ventricular dilatation** 2. **Asymmetrical septal thickening (septum >> free wall)** 3. Impaired diastolic filling and LV outflow obstruction in 25% of cases	Need bolded Prompt: What are the structural effects on the myocardium?
	What are the complications of HCM?	1. Heart Failure 2. Sudden death, ventricular arrhythmias 3. Atrial fibrillation, mural thrombus / embolisation 4. Stroke 5. Infective endocarditis mitral valve	Need 3/5

2008.2

4. Calcific Aortic stenosis	1. What are the causes of Aortic valve stenosis?	Postinflammatory scarring (Rheumatic fever) Senile calcific Ao Stenosis Calcification of congenitally deformed valve	2/3 to pass
	2. What is calcific aortic stenosis?	Ao Stenosis most common valvular abnormality **Wear and tear** => calcification on normal or cong bicuspid valves Clinical attention in 6-7th decade in bicusid valves, 8-9th decade in prev. normal valves Heaped up **calcified masses within cusps**=> protrude through to outflow tracts. Functional valve area decreased.	Highlighted
	3. What are the consequences of calcific aortic stenosis?	**LV outflow obstruction**=> increased pressure gradient over valve. (severe when valve area 0.5-1cm^2) CO maintained by concentric **LVH**. Hypertrophied myocardium ischaemic. Impaired systolic and diastolic function. Decompensation => angina, CCF, syncope	Highlighted

2008.2

4. Pericarditis	1. What are the causes of acute pericarditis?	**Infectious**; **viral**, pyogenic bacteria Immune mediated(presumed); Rheumatic fever, SLE, Scleroderma, post cardiotomy. Post MI (Dressler's), Drug hypersensitivity reaction. Other; AMI, uraemia, post cardiac surgery, neoplastic, trauma, radiation	Need viral and three others
	2. What types of pericardial fluid exudate occur?	1. Serous; usually non-infectious inflammation, RF, SLE, uraemia, tumours 2. Fibrinous/serofibrinous; (most common) post MI, Dressler's, trauma, post surgery but also as in 1. 3. Purulent/suppurative; almost always bacterial invasion from local infection, lymphatic or blood seeding, or at operation 4. Haemorrhagic 5. Caseous	2/5 to pass
	3. Describe the clinical features of pericarditis	Pericardial rub (may be absent if large effusion). Pain, fever (chills and rigors if suppurative), signs of cardiac failure,	Rub, pain,fever required

2008.2

4. Pathogenesis of atherosclerosis	1. Outline the steps involved in the pathogenesis of atherosclerosis.	Response to injury hypothesis: **1. Endothelial injury and dysfunction** 2. Lipoprotein (mainly LDL) accumulation and oxidation in vessel wall **3. Monocyte adhesion and migration into intima and transformation into foam cells and macrophages** 4. Platelet adhesion **5. Smooth muscle cell migration from media into intima** 6. Subsequent smooth muscle cell proliferation in intima **7. Enhanced lipid accumulation within intimal cells (macrophages and smooth muscle cells)**	Must have highlighted
	2. List the potential causes of endothelial injury?	**1. Hyperlipidaemia,** **2. Hypertension,** **3. Smoking** **4. Haemodynamic factors (disturbed flow patterns)** 5. Homocysteine, 6. Toxins, 7. Viruses, 8. Immune reactions	3 of highlighted and 1 other to pass