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Abdominal Aortic Aneurysms 2013-2-A

Pathology: Abdominal Aortic Aneurysms	1. What are the risk factors for development of abdominal aortic aneurysms?	Male; Smoking; Age > 60; Family History; Connective tissue disease (eg. Ehlers Danlos); Vasculitis; Hypertension, Diabetes; Atherosclerosis	5 to pass
	2. Describe the pathogenesis of AAA formation	**Atherosclerotic plaque** in intima compresses media with **degeneration and weakness of wall** and cystic medial degradation Local inflammation **Proteolytic enzymes** with collagen degradation -role of matrix metalloproteinases (MMP). Loss of vascular smooth muscle cells. Inappropriate Synthesis of non-elastic ECM	2 of 3 bold to pass
	3. What are the clinical consequences of an AAA?	**Rupture**: increase with diameter (higher if >5cm) & can be retroperitoneal OR intra peritoneal with rapid fatal haemorrhage Obstruction: ischaemia from branch vessel obstruction eg. mesenteric, vertebral, renal Embolism: plaque or thrombus Impingement or compression of adjacent structure (eg. ureter) Painless mass	Bold and 2 others.

Abdominal Aortic Aneurysms 2010-2

Question 4.3 Abdominal Aortic Aneurysm	1. Describe the pathogenesis of an aneurysm	1. **Structure or function of the vascular wall connective tissue is compromised** 1.1. **Poor intrinsic quality of the vascular wall connective tissue eg Marfan syndrome**, Ehlers-Danlos 1.2. **Collagen degradation vs synthesis by local inflammation** (proteolytic enzymes) **eg atherosclerotic plaque, vasculitis,** 1.3. **Loss of vascular smooth muscle cells** or the inappropriate synthesis of noncollagenous or nonelastic ECM (cystic medial degeneration)	1. 2/3 bold, 2 examples
	2. What are the clinical consequences of an AAA?	2. 2.1. **Rupture** into the peritoneal cavity or retroperitoneal tissues with massive, potentially fatal haemorrhage 2.2. **Obstruction of a branch vessel** resulting in ischemic injury, eg. iliac, renal, mesenteric, or vertebral arteries 2.3. **Embolism** from atheroma or mural thrombus 2.4. **Impingement** on an adjacent structure, e.g. ureter, vertebrae 2.5. Nothing (if < 4cm and no embolic complic's)	2. 3 out of 5
	3. What is the risk of rupture of an AAA?	3. Related to size - 3.1 4 cm or less in diameter nil 3.2 between 4 and 5 cm 1% per year 3.3 between 5 and 6 cm 11% per year 3.4 greater than 6 cm in diameter 25% per year	3. Low < 5cm, much higher > 5cm

Abdominal Aortic Aneurysms 2004-2

TOPIC: Abdominal aortic aneurysm **NUMBER: 4**

OPENING QUESTION	What are the morphological features of an abdominal aortic aneurysm?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	An aneurysm is a localised dilatation of the abdominal aorta. It is usually between the renal arterial and the bifurcation of the aorta into iliac vessels. The aneurysm often contains atheromatous ulcers covered with mural thrombi, with thinning and destruction of the media.		
SECOND QUESTION (if needed)	List the common causes of abdominal aortic aneurysm		
POINTS REQUIRED	- Atherosclerosis - Congenital (cystic medial degeneration) - Mycotic - Syphilis - Trauma - Immunological - (Salmonella)	Need 2 causes to pass	
THIRD QUESTION	What are the complications of an abdominal aortic aneurysm?	Need 3 to pass	
	- Rupture - Occlusion of branch – iliac, mesenteric, vert. - Embolism of atheroma/thrombi - Impingement adj. Structure (ureter, erosion vert.) - Presentation abdominal mass - Rupture Risk (2% <4cm) (5-10% each year >5cm) - Operative mortality (unruptured 5%) (ruptured >50%)		

Aortic Dissection 2017-1-B

Stem: A CT-aortogram is performed. Moving onto Pathology			
Question 4 Aortic dissection **Subject:** Path LOA: 1	a) Describe the pathogenesis of an aortic dissection. Prompt; what are the risk factors for aortic dissection	**Hypertension**, aorta of hypertensive patients have medial hypertrophy of vasa vasorum and degenerative changes in the media Connective tissue disease (inherited or acquired) Both of the above cause **weakness in the media** An aortic dissection starts with an **intimal tear** and the blood dissect in the media either distally or proximally leading to a tear in the media	Bold
	b) How are aortic dissections classified?	By site of involvement: Stanford Type A prox, Type B distal OR Debakey I – asc and desc; II asc only, III desc only	Either classification to pass.
	c) What are the potential consequences of aortic dissection? Give examples	- Rupture back into intima or through adventitia - Rupture out or into pericardial, pleural or peritoneal cavities - Cardiac tamponade, aortic insufficiency, MI, distal ischaemia, spinal cord ischaemia - Death	3 examples to pass.

Aortic Dissection 2015-1-C

Stem: Moving to Pathology. You suspect aortic dissection			
Question 3 Aortic Dissection **Subject:** Path LOA: 1	What sequence of changes occur in the vessel wall in aortic dissection?	**Intimal tear** into media of aorta, **strips along laminar planes**, formation of blood filled channel which may then rupture outwards.	Bold (conceptually)
	What are the risk factors?	Men aged 40-60 with **hypertension** Connective tissue disorders eg Marfans Complication of arterial cannulation Trauma	**Hypertension + one other**
	What are the types of aortic dissection? Prompt = classification?	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)	**Concept (prox & distal)**

Aortic Dissection 2012-2

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.

Aortic Dissection 2010-1

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

Aortic Dissection 2007-1

5. Aortic dissection	1. List the major risk factors for aortic dissection.	**1. Hypertension** **2. Connective tissue diseases eg. Marfan Syndrome,** 3. Iatrogenic: coronary artery catheterisation, Coronary artery by pass. 4. Pregnancy	First 2 and 1 other
	2.Describe the morphological features of aortic dissection	1. Most frequent pre-existing = medial degeneration of elastic tissue 2. Intimal tear aorta extends into the media. 3. Haematoma spread between the middle and outer thirds along the laminar planes of the media and formed a blood filled channel. 4. Disrupts outward causing massive haemorrhage or re-rupture into the lumen of the aorta producing a false lumen.	At least 2
	3.What are the consequences of aortic dissection?	1. Dissects proximally towards the aortic valve and vessels of the neck and causes disruption of the aortic valve, cardiac tamponade, myocardial infarction, cerebral vascular accident. 2. Dissects distally into the renal, mesenteric, iliac & femoral arteries causing ischaemia. 3. Compression of the spinal vessels causing transverse myelitis.	At least 1 prox, 1 distal + 2 others

Aortic Dissection 2004-2

TOPIC: Aortic Dissection **NUMBER: 4**

OPENING QUESTION	What is aortic dissection?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	Aortic dissection is the dissection of blood along the laminar planes of the aortic media, with the formation of a blood filled channel within the aortic wall, which often ruptures, causing massive haemorrhage. There is usually an intimal tear that extends into but not through the media of the ascending aorta, usually within 10 cm of the aortic valve. The dissection can extend proximally into the heart, as well as distally along the aorta into the iliac and femoral arteries. Sometime, the blood re-ruptures into the lumen of the aorta, producing a second intimal tear. There is usually no marked dilatation of the aorta. The dissection does not affect the aorta that is affected by substantial atherosclerosis.	Adequate definition to pass	
SECOND QUESTION (if needed)	What are the predisposing factors for aortic dissection?	Need 1 to pass	
POINTS REQUIRED	Hypertension Connective tissue diseases (eg: Marfan) Iatrogenic		
THIRD QUESTION	What are the complications of aortic dissection?	Need 3 specifics to pass	
	Death - rupture 3 body cavities Extension/Obstruction - Carotid, renal, mesenteric arteries Retrograde to aortic valvular apparatus (root) - Cardiac tamponade - Aortic insufficiency - Coronary ostia (AMI) - Spinal arteries		

Stem: Moving onto Pathology. His past history includes severe aortic stenosis.			
Question 4 Calcific aortic stenosis LOA: 2	*a. What are the pathological consequences of aortic stenosis?* *Prompts – 'What type of ventricular hypertrophy?'* *'Which ventricle is hypertrophied?'*	**Concentric left ventricular hypertrophy.** Left ventricular outflow obstruction. Myocardial ischaemia (without coronary artery disease needing to be present) Syncope; Aortic dissection; Heart failure (diastolic or systolic); Endocarditis (uncommon)	Bold and 3 others
	b. What are the likely causes of aortic stenosis in this man?	Calcific/degenerative Bicuspid valve Rheumatic heart disease	2 to pass (do not accept congenital)
	BONUS: What clinical signs may differentiate calcific aortic stenosis from rheumatic aortic stenosis?	Rheumatic disease involves more than one valve (ie aortic and mitral) Absence of features of MS/MR Absence of features of aortic regurgitation	

Stem: The patient has aortic stenosis.			
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

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

TOPIC: Calcific aortic stenosis **NUMBER:** 4

OPENING QUESTION	What are the causes of calcific aortic stenosis?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	- Senile calcific aortic stenosis - Calcification of congenitally deformed valve	Need 1 to pass	
SECOND QUESTION (if needed)	What are the complications of aortic stenosis?	Need 2 to pass	
POINTS REQUIRED	Increasing obstruction to left ventricular outflow Cardiac output maintained (left ventricular hypertrophy) Angina (? ↓ microcirculatory myocardium) Syncope (? Poorly understood) Cardiac decompensation – congestive failure		

Stem: She has a history of coronary artery disease. Moving onto Pathology.			
Question 2 Atherosclerosis **Subject:** Path LOA: 1	1. What are the systemic and local factors that lead to atherosclerosis?	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.	1. Bold to pass
	2. Which arteries are most often affected by atherosclerosis?	2. **Lower abdominal aorta, the coronary arteries, the popliteal arteries, the internal carotid arteries, and the vessels of the circle of Willis.**	2. 3 of 5 bold to pass
	3. How does an atherosclerotic plaque suddenly cause symptoms?	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	3. 2 of 4 bold to pass

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

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

Atherosclerosis 2003-1

TOPIC: Atherosclerosis **NUMBER:** ____

OPENING QUESTION	Draw a typical atheromatous plaque	**COMMENTS**
POINTS REQUIRED	1 Endothelium	4 of 6
	2 Fibrous Cap	
	3 Necrotic Centre	
	4 Foam Cells	
	5 Cholesterol crystals	
	6 Calcium	
	7	
PROMPTS		
SECOND QUESTION (if needed)	What are the pathological consequences	
POINTS REQUIRED	1 Weakening wall -Rupture of Vessel, aneurysm	3 of 5
	2 Gradual Occlusion – Ischaemia	
	3 Disruption – Acute Occlusion	
	4 Embolisation – Distal occlusion	
	5 Calcification	
	6	
PROMPTS	Prompt for process and consequence	
THIRD QUESTION (if needed)	Which arteries are commonly affected	
POINTS REQUIRED	1 Large elastic - aorta	
	2 Medium sized muscular	
	3 Lower Limbs more than Upper	
	4 Coronary	
	5 Circle of Willis	
	6 Carotids	
PROMPTS		

Stem: A 65-year-old alcoholic woman presents following a fall. She is short of breath. We will start with Pathology.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Cardiomyopathy **Subject:** Path LOA: 1	a) What is the definition of cardiomyopathy?	• Heterogenous group of diseases of the myocardium associated with **mechanical and/or electrical dysfunction** that usually (but not invariably) exhibit inappropriate **ventricular hypertrophy or dilation** • Primary cardiomyopathies can be congenital or acquired • Secondary cardiomyopathies have myocardial involvement as a component of a systemic or multisystem disorder	Bold
	b) What are the types of cardiomyopathy? Give a cause of each. **PROMPT**: Structural changes?	**Hypertrophic**. 75% genetic cause. Autosomal dominant HCM. **Dilated –** alcohol, myocarditis, idiopathic, peripartum, genetic **Restrictive**. Infiltrative: Amyloidosis. Sarcoidosis. Non-infiltrative: Idiopathic, scleroderma	Bold, with one example for each
	c) What type of cardiomyopathy is alcoholic cardiomyopathy?	Dilated	

Cardiomyopathy 2016-1-A

Stem: Moving onto Pathology. He is found to be in heart failure, and on bedside echocardiogram is found to have a grossly dilated heart with poor contractility in all chambers.			
Question 2 Cardiomyopathy – focussing on alcoholic **Subject:** Path LOA: 2	1) What pathological process is likely to be causing his heart failure?	Alcohol related **Dilated Cardiomyopathy**	Bold to pass
	2) Name some causes of dilated cardiomyopathy	Causes include **myocarditis** (viral causes?), **toxins** (alcohol, chemo, cobalt), **congenital**, **pregnancy**.	Any 2 bolded to pass
	3) What are potential pathologic consequences of dilated cardiomyopathy.	**Valve dysfunction** (incompetent mitral/tricuspid), **mural thrombi** and **embolization**, lethal **arrhythmia, atrial fibrillation, death** from progressive failure	Any 2 bolded to pass

Cardiomyopathy 2015-1-A

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

Cardiomyopathy 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

Cardiomyopathy 2006-1

TOPIC: Friday AM – Q 3 – Hypertrophic Cardiomyopathy **NUMBER:** _____

OPENING QUESTION	What are the different types of cardiomyopathy?	**COMMENTS**
POINTS REQUIRED	1. Dilated	2 to pass
	2. Hypertrophic	
	3. Restrictive	
PROMPTS		
SECOND QUESTION (if needed)	What are the pathological features of hypertrophic cardiomyopathy?	2 to pass
POINTS REQUIRED	1. Macroscopic: hypertrophy without dilatation, asymmetric (sub-aortic), LV outflow	
	2. Microscopic: myocyte hypertrophy, disarray of myocytes, interstitial fibrosis	
	3.	
	4.	
PROMPTS		
THIRD QUESTION (if needed)	What is the complications of HOCM?	
POINTS REQUIRED	1. AF	
	2. CCF	
	3. Sudden death	
PROMPTS		

Cor Pulmonale 2012-1

Question 3 Cor pulmonale	**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
	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.

Endocarditis 2016-2-B

Stem: Moving onto Pathology. He has multiple track marks and has a loud cardiac murmur			
Question 2 Endocarditis **Subject:** Pathology LOA: 2	1. What factors predispose to infective endocarditis?	**Cardiac Factors** – Degenerative mitral valve prolapse (myxomatous), calcific aortic stenosis, bicuspid aortic valve, prosthetic valves, congenital valve defects, rheumatic heart disease **Host Factors** – Bacteraemia (dental or surgical procedure, loss of skin integrity), Intravenous drug use, immunodeficiency, drug induced immunosuppression, malignancy, neutropaenia, diabetes, alcohol	**4 to pass** (2 from each group) Prompt "Any other factors for infective endocarditis in general?"
	2. Which organisms commonly cause infective endocarditis?	**Streptococcus viridans, Staph aureus**, staph epidermidis, enterococci, gram negative bacilli, HACEK (Haemophilus, Actinobacillus/Aggregatibacter, Cardiobacterium, Eikenella, Kingella); Fungal	**Bold** + 1 other to pass
	3. What are the complications of infective endocarditis?	**Local** – erosion/destruction of tissue (valve or myocardium, abscess formation (ring abscess) **Systemic** – Septic infarcts – brain, lung, kidneys, mycotic aneurysms. Embolic phenomena – Subcutaneous tissues (splinter haemorrhages, janeway lesions, oslers nodes) Retina (roth spots) Glomerulonephritis (immune mediated)	**1 local and 1 systemic** to pass Prompt "Any local/systemic complications"

Heart Failure 2015-2-A

Stem: An 80 year old woman is noted to be in heart failure. Starting with Pathology			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Heart Failure **Subject:** Path LOA: 1	What is heart failure?	When **cardiac function is impaired** and/or the **heart is unable to maintain a cardiac output** sufficient for the body's metabolic needs.	Bold to pass
	Please classify the types of heart failure? Prompt: Examples?	**Pump failure:** **Systolic dysfunction (Contractile dysfunction)** eg myocardial contractile dysfunction secondary to ischaemia, AMI, pressure or volume overload, dilated cardiomyopathy. **Diastolic dysfunction (Inadequate filling)** eg LV hypertrophy, myocardial fibrosis, amyloidosis, pericarditis. **Others:** arrhythmias, regurgitant flow eg MR, outflow obstruction eg AS, HOCM **Left heart failure** (IHD, HT, Valvular diseases eg AS, rheumatic heart disease, myocardial disease) **Right heart Failure** (eg secondary to left heart failure, PE, Pulmonary HT etc)	One of the classifications with examples
	What are the clinical features of heart failure? Prompt: What symptoms or signs from other organ systems might occur with heart failure?	Lung –breathlessness, orthopnoea, PND, APO, pleural effusions Cardiac – 3rd HS, gallop, displaced apex beat, AF, murmur, JVP elevation Renal – RAA activation – with fluid retention, pedal oedema, AKI Brain – confusion secondary to hypoxia Hepatic –engorgement, ascites, cirrhosis(late)	3/5 organ system symptoms to pass

Heart Failure 2012-2

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

Hypertension 2016-2-B

Stem: A 40-year-old woman presents with a blood pressure of 220/160. Starting with Pathology			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Hypertension **Subject:** Path LOA: 2	How is hypertension classified? What are the causes of secondary hypertension?	**Primary (essential)** **Secondary** **Renal** (Acute glomerulonephritis, Chronic renal disease, Polycystic disease, Renal artery stenosis, Renal vasculitis, Renin-producing tumors) **Endocrine** (Adrenocortical hyperfunction [Cushing syndrome, primary aldosteronism, congenital adrenal hyperplasia, licorice ingestion], Exogenous hormones [glucocorticoids, estrogen {including pregnancy-induced and oral contraceptives}, sympathomimetics and tyramine-containing foods, monoamine oxidase inhibitors], Pheochromocytoma, Acromegaly, Hypothyroidism, Hyperthyroidism, Pregnancy-induced **Cardiovascular** (Coarctation of aorta, Polyarteritis nodosa,, Increased intravascular volume, Increased cardiac output, Rigidity of the aorta. **Neurologic** (Increased intracranial pressure, Sleep apnea) **Psychogenic** (Acute stress, including surgery, Pain)	**Bold** **6 examples from at least 3 different systems.** Prompt "Diseases in what (other) systems cause secondary hypertension?"

Hypertension 2011-1

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.**

Hypertensive Heart Disease 2003-1

TOPIC: Hypertensive Heart Disease **NUMBER:** _____

OPENING QUESTION	What are the criteria for systemic hypertensive heart disease	**COMMENTS**
POINTS REQUIRED	1 Left Ventricular Hypertrophy	
	2 Absence of another cause	
	3 Systemic Hypertension	
	4	
	5	
	6	
	7	
PROMPTS		
SECOND QUESTION (if needed)	What are the gross morphology findings in hypertensive heart disease	
POINTS REQUIRED	1 Thick L ventricular wall	3 of 4
	2 No dilation	
	3 L atrial enlargement	
	4 Increased weight of heart	
	5	
	6	
PROMPTS		
THIRD QUESTION (if needed)	What are the pathological consequences	
POINTS REQUIRED	1 Stiffness	
	2 Impaired diastolic filling	
	3 Atrial dilation/fibrillation	
	4 Heart failure	
	5 Sudden Cardiac Death	
	6	
PROMPTS		

Infective Endocarditis 2012-1

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**

Myocardial Infarction 2016-2-B

Stem: Moving onto Pathology.			
Question 2 Acute coronary syndromes **Subject: Pathology** LOA: 1	1. Describe the pathogenesis of Myocardial infarction due to atherosclerosis	**1. Acute plaque change** a. Rupture / fissuring b. Erosion / ulceration c. Haemorrhage into atheroma **2. Thrombosis** a. Platelet adhesion, aggregation & micro-thrombi formation b. Platelet release of mediators causing vasospasm c. Activation of coagulation pathway leading to thrombus **3. Vasoconstriction** stimulated by: a. Circulating adrenergic agonists b. Locally released platelet contents c. Endothelial cell dysfunction causing decrease NO d. Perivascular inflammatory cell mediators **4. Vessel occlusion leading to:** a. decreased myocardial blood flow b. myocyte necrosis	**3 of 4 Bold** and demonstrate understanding of processes "Can you describe the process" – for each bold
	2. What are the complications of acute myocardial infarction?	• Contractile dysfunction o LVF / RVF / cardiogenic shock • Arrhythmias • Myocardial rupture o Free wall / Vent septum / Pap muscle • Ventricular aneurysm • Pericarditis/effusion/tamponade • Mural thrombus • Papillary muscle dysfunction	3 to pass

Myocardial Infarction 2013-2-C

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

Myocardial Infarction 2013-1

Question 3 IHD LOA: 1	1. In myocardial infarction, what sequence of events leads to acute coronary artery occlusion? Prompt- pathological events 2. Describe the time course of myocardial injury after acute coronary artery occlusion. Prompt- What happens to the myocardial tissue over time?	**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 **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** **Bold to pass with minutes to hours concept**

Myocardial Infarction 2007-2

TOPIC: Myocardial Infarction **NUMBER: Q3**

OPENING QUESTION	**What is the sequence of events in acute coronary artery occlusion?**	**COMMENTS**
POINTS REQUIRED	1. Atheromatous plaque rupture / erosion 2. Platelet adhesion, activation and aggregation, with release of aggregators (Thromboxane, serotonin and platelet factors) 3. Vasospasm 4. Intrinsic coagulation cascade activation Thrombus evolves to occlude artery Prompt: What happens to an atheromatous plaque to initiate acute coronary occlusion?	General outline of plaque rupture, platelet aggregation and thrombus formation = Red thrombus
SECOND QUESTION	**Describe the time course of myocardial injury after coronary occlusion.**	Prompt: When does irreversible cell injury occur?
POINTS REQUIRED	1. ATP depletion – seconds 2. Loss of contractility – 2 mins 3. ATP reduction – 10-40 mins 4. Irreversible cell injury – 20-40 mins 5. Myonecrosis begins after ~ 30 mins 6. Microvascular injury at 1 hour 7. Extensive necrosis needs 2-4 hours of ischaemia (blood flow < 10%)	Minutes to hours concept
	Anaerobic metabolism begins immediately; cell death occurs begins after half an hour or so; extensive necrosis occurs after 2 hours.	Desired knowledge

Myocardial Infarction 2003-2

3.3 Consequences of AMI	Describe the pathological changes in myocardium following occlusion of a coronary artery.	**Loss of contractility (<2mins)**; loss of ATP (50%at10min,10%at40min); **irreversible cell injury (20-40min)**; **microvascular injury (>1hour); coagulative necrosis.** Minutes: myofibrillar relaxation, glycogen depletion, mitochondrial and cell swelling. 40minutes: sarcolemmal disruption, mitochondrial amorphous densities. Necrosis first in subendocardium, endocardium is spared. 4-12hour coag necrosis, edema, hemorrhage.	
	What are the potential consequences of reperfusion?	Early: no damage. Later: reperfusion hemorrhage; acceleration of disintegration of **damaged** myocytes; exaggerated contraction of myofibrils; **some new injury** from oxygen free radicals. 'Prolonged post-ischaemic ventricular dysfunction'.	

Pericarditis 2016-2-B

Stem: A 40 year old lady presents with suspected pericarditis. We will start with Pathology

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Acute Pericarditis **Subject:** Pathology LOA: 2	Describe the characteristic clinical features of pericarditis	**Chest pain** (dull or sharp, pleuritic, positional), fever, congestive failure, p*ericardial* ***friction rub*** Constrictive pericarditis: distant or muffled heart sounds, elevated JVP, peripheral edema.	2 Features to pass Prompt "History and examination features?"
	What are the causes of pericarditis?	**Infectious: viral**, pyogenic bacteria, TB, fungal **Immune mediated**: Rheumatic fever, SLE, Scleroderma, post cardiotomy. Post MI (Dressler's), Drug hypersensitivity reaction. AMI, uraemia, post cardiac surgery, neoplastic, trauma, radiation	Need viral, immune example and one other
	What types of pericardial fluid exudate occur?	1. Serous: usually non-infectious inflammation (RF, SLE), but also viral, 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 (TB)	2/5 types to pass

Pericarditis 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

Pericarditis 2003-2

1.3 Genesis of acute pericarditis	What are the causes of pericarditis?	**Infectious, Inflammatory, Malignant., example of each.** Non-infectious inflammations (Rhfever, SLE, uremia), viral; fibrinous reaction occurs with these, plus AMI, post AMI; pus from adjacent invasion, haem spread, lymphatic spread, surgical introduction; blood from TB, malignant spread, bacteria, post-surgery; caseous is TB.	
	What pathological types of pericarditis can occur?	Serous; fibrinous/serofibrinous; purulent (suppurative); haemorrhagic; caseous	3/5