

Chapter 11 & 12:

The Cardiovascular System: Blood vessels and the Heart

- Robbins Pathology 7th ed. pp511-618
- This topic is worth 7 MCQ questions, but covers *two* chapters.

The syllabus states that these topics are covered;

Blood vessels, Chapter 11:

- Vascular response to injury
- Arteriosclerosis
- Hypertensive vascular disease
- Aneurysms and dissections
- Vasculitides
- Veins and lymphatics

* Normal vessels, congenital abnormalities, Raynaud phenomenon, tumours and interventions are **NOT** covered in the syllabus.

The Heart, Chapter 12:

- Congestive heart failure (*incl. Guyton 10th p235-241*)
- Ischaemic heart disease (*incl. Guyton 10th p229-232*)
- Valvular heart disease
- Cardiomyopathies
- Pericardial disease
- Transplantation

* Congenital heart disease and tumours of the heart are **NOT** covered in the syllabus.

Blood Vessels:

1. Arteries

- (a) that are of the elastic type include the coronary arteries
- (b) that are of the muscular type include the common carotid arteries
- (c) of the muscular type do not contain vasa vasorum
- (d) that are large bore (such as the aorta) have a thick layer of subendothelial connective tissue in the intimal layer
- (e) that are large have vasa vasorum, which are small arterioles arising outside the vessel

1a. From inside to the outside, the layers of the arterial wall are composed of p512

- (a) endothelium, muscularis mucosa, external elastic lamina, adventitia
- (b) tunica interna, tunica media, internal elastic lamina, adventitia
- (c) endothelium, internal elastic lamina, muscle layer, external elastic lamina, adventitia
- (d) endothelium, muscle layer, internal elastic lamina, adventitia, external elastic lamina
- (e) endothelium, internal elastic lamina, muscle layer, adventitia

2. Capillaries

- (a) have an endothelial layer and a minimal media
- (b) are the approximate diameter of a red blood cell (7-8 μ m)
- (c) in most types of inflammation, are the site of vascular leakage, and leukocyte exudation
- (d) individually, have a high cross sectional area
- (e) are the principle points of resistance to blood flow, as they are able to dramatically alter their calibre

3. Endothelial cells

- (a) are phenotypically identical, regardless of site
- (b) uniquely contain Weibel Palade bodies
- (c) have minimal metabolic properties
- (d) in most regions provide a partially permeable membrane for large plasma proteins
- (e) in the inactive state express some adhesion molecules on the vascular surface

4. Endothelial cells are capable of

- (a) inducing thrombosis
- (b) inhibiting thrombosis
- (c) oxidation of low-density lipoprotein (LDL)
- (d) all of the above
- (e) (a) and (b) only

5. Inducers of endothelial activation include

- (a) lipid products
- (b) corticosteroid
- (c) advanced glycosylation end products
- (d) All of the above
- (e) (a) and (c) only

6. Proliferation of smooth muscle cells is promoted by

- (a) heparan sulphates
- (b) nitric oxide (NO)
- (c) Transforming growth factor β (TGF- β)
- (d) corticosteroid
- (e) interferon- γ (IFN- γ)

7. Intimal thickening of vessels

- (a) is a normal part of ageing
- (b) is always abnormal
- (c) is due to a migration of endothelial cells into the media
- (d) is due to proliferation of smooth muscle cells, which are still able to contract and potentially occlude the vessel
- (e) occurs due to smooth muscle cell migration into the adventitia

8. Mönckeberg medial calcific sclerosis

- (a) produces radiographically visible calcifications in muscular arteries
- (b) is rapidly fatal
- (c) involves narrowing of the vessel lumen
- (d) is a disease of childhood
- (e) is a disease that primarily affects arterioles

9. Fatty streaks

- (a) can cause disturbance to blood flow
- (b) are destined to form into atheromatous plaques in later life
- (c) allow platelet aggregation to occur readily
- (d) contain T lymphocytes and extracellular lipid
- (e) appear in all children over the age of 5

10. Concerning the development of atheromatous plaques

- (a) The early stages of plaque development is due to extracellular lipid accumulation
- (b) Atheromatous plaques begin from fatty streak
- (c) Atherosclerosis occurs mainly in small calibre muscular arteries
- (d) Atheromas often undergo calcification, which predisposes the plaques to thromboembolic events
- (e) The coronary vessels are the most heavily involved vessels

11. Atherosclerotic lesions

- (a) in the advanced stages, involve the entire circumference of the vessel
- (b) involving the upper extremities is common
- (c) usually have a soft grumous core of lipid
- (d) usually involve the entire length of smaller vessels
- (e) usually spare the origins (ostia) of major branches

12. In descending order the most heavily involved arterial vessels in forming atherosclerotic plaques are

- (a) coronary> abdominal aorta> popliteal> internal carotid> circle of Willis
- (b) abdominal aorta> coronary> popliteal> internal carotid> circle of Willis
- (c) abdominal aorta> coronary > popliteal> circle of Willis> internal carotid
- (d) abdominal aorta> circle of Willis> coronary> popliteal> internal carotid
- (e) abdominal aorta> coronary> renal> circle of Willis> popliteal

13 Cells which make up atherosclerotic plaques,

- (a) such as smooth muscle cells, can imbibe lipid to become foam cells
- (b) such as endothelial cells, typically make up the superficial fibrous cap
- (c) such as foam cells, are large lipid laden cells derived principally from lymphocytes
- (d) are predominantly smooth muscle cells and fibrous tissue
- (e) such as foam cells are typically derived from tissue macrophages

14. All of the following are major risk factors for the development of atherosclerosis EXCEPT (*from old paper*)

- (a) obesity
- (b) hyperlipidaemia
- (c) smoking
- (d) hypertension
- (e) type II diabetes mellitus

15. Regarding the major risk factors for atherosclerosis development, which of the following statements is TRUE?

- (a) Being male is a major risk factor, and females never statistically catch up to the same rate of myocardial infarction, regardless of age.
- (b) Between the ages of 40 to 60 the risk of myocardial infarction increases five-fold.
- (c) Whilst being a major risk factor, elevated cholesterol levels alone are not sufficient to stimulate lesion development.
- (d) Omega-3 fatty acids are of particular concern in promoting the formation atheroma
- (e) Obesity raises the amount of serum high density lipoprotein (HDL)

16. The pathogenesis of atherosclerosis includes

- (a) accumulation of lipoproteins, mainly high density lipoprotein (HDL) in the vessel wall
- (b) chronic smooth muscle cell injury causing thrombosis
- (c) release of factors from activated platelets, which promote the mitosis and migration of endothelial cells into the intima
- (d) inhibition of lesional lipoprotein oxidation
- (e) elaboration of extracellular matrix by smooth muscle cells

17. The pathogenesis of atheroma

- (a) cannot occur at sites of morphologically intact endothelium
- (b) is due to endothelial dysfunction in response to elevated homocysteine
- (c) is due in part to the migration of monocytes into the media of the vessel
- (d) is directly correlated to the high levels of circulating high density lipoprotein (HDL)
- (e) Is thought to be due in large part to haemodynamic disturbances, and the adverse effects of hypercholesterolaemia

18. The role of smooth muscle cells in the pathogenesis of atheroma include

- (a) migration of smooth muscle cells from the intima to the media
- (b) a decrease in proliferation and an increase in contractile capabilities in intimal smooth muscle cells
- (c) foam cell formation, and death by apoptosis that is induced by immune cells
- (d) modification of lipids by oxidation
- (e) production of growth factors including fibroblast growth factor (FGF) and transforming growth factor α (TGF- α)

19. Cells normally found in an atheromatous plaque include all the following, except

- (a) smooth muscle cells.
- (b) foam cells.
- (c) macrophages.
- (d) lymphocytes.
- (e) platelets

20. Regarding essential hypertension

- (a) Essential hypertension is a distinct disease, made by three readings of systolic blood pressures above 140mmHg
- (b) Essential hypertension is present in 15% of persons in the general population
- (c) Older patients with high systolic blood pressure are at greater risk of cardiovascular event than those with elevated diastolic blood pressure
- (d) 85% of diagnosed hypertension is idiopathic (or essential)
- (e) 10% of patients with hypertension develop malignant hypertension

21. Regarding hypertension

- (a) Peripheral vascular resistance is determined mainly at the smaller artery level
- (b) Resistance vessels are mainly controlled by neural factors
- (c) Angiotensin II acts directly on smooth muscle cells to produce vasoconstriction
- (d) Natriuretic peptides induce vasoconstriction
- (e) Hypertension is considered benign if the diastolic pressure is 140mmHg

22. Licorice ingestion increases blood pressure by

- (a) increasing levels of angiotensinogen
- (b) increasing levels of renin
- (c) increasing mineralocorticoid receptor activity
- (d) acting on the Epithelial sodium channel
- (e) increasing the production of mineralocorticoids

23. The kidneys control blood pressure by

- (a) controlling blood volume, and producing substances that all act as vasopressors.
- (b) producing natriuretic factors
- (c) producing angiotensin I in the juxtaglomerular cells of the kidney
- (d) by producing renin which converts angiotensinogen to angiotensin I
- (e) removing angiotensin converting enzyme (ACE) from the circulation

24. Theories as to the cause of essential hypertension include all EXCEPT

- (a) reduced renal sodium excretion in the presence of normal blood pressure
- (b) variations in the genes encoding components of the renin-angiotensin system
- (c) chronic or repeated vasoconstriction of the resistance vessels
- (d) Mutations in proteins that affect sodium reabsorption
- (e) heavy consumption of salt (NaCl)

25. Hyaline arteriosclerosis

- (a) has a typical onionskin, concentric thickening of the wall of arterioles
- (b) is related to more acute or severe elevations of blood pressure
- (c) shows acute necrosis of the vessel wall
- (d) is a feature of diabetes
- (e) shows none of the above features

26. Hyperplastic arteriosclerosis

- (a) consists of a homogenous hyaline thickening of the walls of arterioles
- (b) is encountered frequently in normotensive elderly patients
- (c) can accompany necrotising arteriolitis
- (d) is common in diabetes
- (e) is due to chronic haemodynamic stress of hypertension

27. An aneurysm

- (a) caused by infection can be due to infective endocarditis
- (b) of the false type is typically seen in tertiary syphilis
- (c) which breaches the vascular wall is called a true aneurysm
- (d) which is fusiform involves a small portion of the vessel wall
- (e) refers exclusively to an abnormal dilatation of a blood vessel

28. A false aneurysm

- (a) is typical of congenital type vascular aneurysms
- (b) in the thoracic aorta can be caused by tertiary syphilis
- (c) does not generally rupture
- (d) is bounded externally by adherent extravascular tissues
- (e) arises when blood enters the wall of the artery as a haematoma dissecting between its layers

29. Abdominal aortic aneurysms

- (a) are most commonly due to trauma
- (b) are more common in women
- (c) generally occur after the age of 40
- (d) are typically described as luetic
- (e) generally expand at a rate of 0.2cm-0.3cm per year

30. The risk of abdominal aortic aneurysm rupture is

- (a) 10% at 4cm
- (b) 25% per year for those larger than 6cm
- (c) 6% per year for those aneurysms of 4.0-5.9cm
- (d) related to the length of the aneurysm rather than the diameter
- (e) none of the above

31. Thoracic aortic aneurysm

- (a) can cause pain due to erosion of bone
- (b) is principally caused by atherosclerosis
- (c) does not generally affect the aortic valve
- (d) is not a complication of giant cell arteritis
- (e) is a feature of Marfan's syndrome

32. Non traumatic aortic dissection
- (a) is associated with marked dilatation of the aorta
 - (b) is a common feature of atherosclerosis
 - (c) Type B involves the ascending aorta
 - (d) can be a complication of pregnancy
 - (e) is a common complication of tertiary syphilis
33. Vasculitis
- (a) caused by Wegener's granulomatosis is due to immune complex deposition
 - (b) of Giant cell type (temporal arteritis) is due to antineutrophil cytoplasmic antibody
 - (c) can be seen in the setting of Hepatitis B infection
 - (d) is drug induced in 25% of cases
 - (e) of Kawasaki disease predominantly affect the small vessels
34. Goodpasture syndrome primarily affects
- (a) capillaries
 - (b) the large arteries.
 - (c) the veins
 - (d) medium sized arteries
 - (e) the aorta
35. Giant cell arteritis
- (a) only affects arteries in the head
 - (b) can often present with only vague constitutional symptoms
 - (c) is of concern because it is painful, rather than because of clinical sequelae
 - (d) is common after the age of 40
 - (e) requires full excision of the offending artery for treatment
36. Takayasu arteritis
- (a) is a disease of capillaries
 - (b) is diagnosed in any patient with Giant cell arteritis diagnosed before age 40
 - (c) is seen predominantly in males younger than 40
 - (d) frequently affects the smaller muscular arteries, and spares the aorta
 - (e) does not affect the coronary circulation
37. Polyarteritis nodosa
- (a) affects small and medium sized muscular arteries
 - (b) is a cause of glomerulonephritis
 - (c) typically affects the pulmonary circulation
 - (d) affects small and medium sized muscular arteries, and the arterioles
 - (e) is a disease of the elderly
38. Concerning Wegener granulomatosis
- (a) it affects males more than females
 - (b) it can present as a persistent pneumonitis
 - (c) left untreated, it causes death in 80% of affected patients within a year
 - (d) is a cause of glomerulonephritis
 - (e) all of the above statements are true

39. Regarding the veins of the lower limb (*old paper*)
- (a) Thrombosis in the superficial vein is a common source of emboli
 - (b) Phlegmasia Alba dolens is associated with iliofemoral vein thrombosis
 - (c) dermatitis is a common consequence of Buerger's disease
 - (d) varicosity has no genetic component
 - (e) 20% of venous thrombi commence in the superficial veins

The Heart:

1. Concerning the heart

- (a) Occlusions of the right coronary artery cannot cause left ventricular damage
- (b) 50% of individuals have a right dominant cardiac circulation
- (c) Myocytes comprise of 25% of the total number of cells in the heart
- (d) The normal heart weighs approximately 500g in adult males
- (e) Cardiomegaly refers to dilatation of the heart

2. The effects of ageing on the heart include

- (a) decreased mass
- (b) tricuspid valve calcific deposits
- (c) increased left ventricular cavity size
- (d) Lambl excrescences
- (e) brown atrophy, which often accompanies dilated cardiomyopathy

3. Cardiac hypertrophy

- (a) due to pulmonary hypertension can produce a heart weight of two times normal
- (b) due to ischaemic heart disease can produce a heart weight of three to four times normal
- (c) due to systemic hypertension can produce a heart weight of up to two times normal
- (d) due to hypertrophic cardiomyopathy can produce a heart of over 1.2kg
- (e) due to aortic regurgitation is usually minimal

4. Cardiac hypertrophy

- (a) pattern reflects the stimulus, and in hypertension is called concentric
- (b) in hypertension leads to increased myocyte cell length
- (c) in volume overload, leads to increased myocyte cell width, but not length, due to extra deposition of sarcomeres
- (d) due to volume overload leads to predominantly increased ventricle wall thickness
- (e) due to hypertension is characterised by dilation with increased ventricular diameter

5. Cardiac hypertrophy

- (a) induced by regular strenuous exercise carries an increased risk of sudden death
- (b) induced by regular strenuous exercise can be explained by comparison to disease states such as high output failure due to Beriberi
- (c) involving the left ventricle is an independent risk factor for sudden death
- (d) increased heart mass is not associated with excess cardiac mortality
- (e) induced by regular strenuous exercise is based on the same processes as early hypertrophic states caused by hypertension

6. Features exclusive to left sided heart failure include

- (a) congestive hepatomegaly
- (b) anasarca
- (c) ascites
- (d) pleural effusions
- (e) haemosiderin containing macrophages in the alveoli

7. Myocardial ischaemia

- (a) is less deleterious than pure myocardial hypoxic states
- (b) due to vasoconstriction can be a consequence of endothelial dysfunction
- (c) due to Prinzmetal angina is the most threatening type for later MI
- (d) due to atherosclerosis is manifested early, as the arteries of the heart are of small diameter
- (e) in 99% of cases is due to atherosclerotic coronary artery obstruction

8. The acute coronary syndromes of ischaemic heart disease include

- (a) unstable angina
- (b) ischaemic cardiomyopathy
- (c) sudden death
- (d) all of the above
- (e) all except (b)

9. Atherosclerotic lesions of the coronary circulation

- (a) often only affect one of the vessels
- (b) causing 75% results in inadequate coronary blood flow at rest
- (c) tend to predominate in the first 3cm of the major artery
- (d) with acute complete occlusion often have a reduced cardiac infarction due to a developed collateral circulation
- (e) causing ischaemic heart disease syndromes, depends primarily on the extent and severity of fixed chronic anatomic atherosclerosis

10. Coronary atherosclerotic plaque (*contains one answer from old paper 2003*)

- (a) with large numbers of smooth muscle cells in the cap is called "vulnerable"
- (b) fissures usually occur at the centre of the plaque
- (c) are most dangerous if they are 50-75% stenotic
- (d) derives most of its tensile strength against rupture directly from smooth muscle cells
- (e) rupture is greatest between the hours of 6pm to 12 midnight

11. Regarding coronary atherosclerotic plaque

- (a) Rupture is a potentially deadly, but fortunately rare occurrence
- (b) Disruption of plaque, with intraluminal thrombosis is common, often clinically silent occurrence
- (c) Plaque is stabilised by statin drugs, which act by directly inhibiting foam cell formation
- (d) Plaque rupture is the most likely cause of subendocardial infarction
- (e) None of the above statements are true

12. Regarding Angina Pectoris

- (a) Prinzmetal angina can be related to hypertension
- (b) Typical angina pectoris refers to the classic pattern of retrosternal chest pain relieved by rest or glyceryl trinitrate
- (c) Prinzmetal angina is a relatively common variant, which is typically difficult to control with the usual angina medications
- (d) Stable angina typically has coronary artery stenosis of less than 75%
- (e) Prinzmetal angina does not cause ECG changes, and is diagnosed with angiography

13. Regarding myocardial infarction

- (a) Myocardial infarction refers to the prolonged ischaemia of cardiac myocytes
- (b) Subendocardial infarction constitutes an area of necrosis limited to the outer third of the ventricular wall
- (c) Subendocardial infarcts can be limited to the distribution of a single major coronary artery
- (d) The subendocardium is well perfused, and true infarcts of this region are therefore uncommon
- (e) Myocardial infarction are generally rare with atherosclerotic lesions affecting 50% or less of the coronary arteries

14. Myocardial infarction

- (a) 10% of myocardial infarctions occur in people under the age of 40 years
- (b) 45% of infarcts occur in people under the age of 75 years
- (c) Infarcts can be prevented by hormone replacement therapy in postmenopausal women
- (d) Black populations have higher rates of infarcts
- (e) In 20% of cases, transmural acute infarct is not associated with atherosclerotic plaque rupture, with thrombosis or embolism

15. In myocardial infarction

- (a) thrombus generally takes hours to evolve and occlude the lumen of the affected vessel
- (b) vasospasm increases the area of infarct, and is a spontaneous unmediated event
- (c) the initial event is spontaneous platelet aggregation over stable plaque
- (d) the observation of occlusion of an artery is seen in 90% of patients undergoing angiography at 12 hours post infarct
- (e) none of the above statements are true

16. In myocardial infarction

- (a) onset of ATP depletion occurs after 5 minutes of artery occlusion
- (b) loss of contractility is seen after 2 minutes of artery occlusion
- (c) ATP level is reduced to 50% of normal after 40 minutes of artery occlusion
- (d) irreversible cell injury is seen after one hour of artery occlusion
- (e) microvascular injury is seen within one hour of artery occlusion

17. The consequences of myocardial ischaemia include
- (a) cessation of aerobic glycolysis in myocytes within seconds
 - (b) development of acute heart failure long before myocyte cell death
 - (c) mitochondrial swelling within minutes of onset
 - (d) myocardial necrosis after approximately 30 minutes of severe ischaemia
 - (e) all of the above
18. Regarding myocardial infarction
- (a) about 50% of transmural infarcts involve at least a portion of the left ventricle
 - (b) isolated infarction of the right ventricle is common
 - (c) Transmural infarcts encompass the entire perfusion zone of the occluded coronary artery
 - (d) The left anterior descending artery is occluded in 40-50% of infarcts
 - (e) New infarcts (less than 12 hours) are apparent on gross examination at autopsy
19. Regarding changes to the myocardium after myocardial infarction (*old paper*)
- (a) there is pallor seen at 24 hours
 - (b) wavy fibres are found centrally
 - (c) decreased contractility after 5 minutes
 - (d) liquefactive necrosis is typical
 - (e) the sarcoplasm is resorbed by leukocytes
20. With regard to myocardial infarction (*old paper*)
- (a) Gross necrotic changes are seen at 3-5 hours post MI
 - (b) irreversible cell injury occurs in less than 10 minutes
 - (c) fibrotic scarring is complete in less than two weeks
 - (d) death occurs in 20% of patients in the first 2 hours
 - (e) is most commonly caused by occlusion of the L circumflex coronary artery
21. Myocardial reperfusion injury
- (a) Generally counteracts the beneficial effects of thrombolysis
 - (b) can cause endothelial swelling that may prevent local reperfusion by occluding capillaries
 - (c) causes prominent necrosis
 - (d) prevents haemorrhage into areas of ischaemia
 - (e) is produced in large part, by oxygen free radicals generated by ischaemic myocytes
22. In regard to myocardial infarction
- (a) 10-15% of patients are asymptomatic whilst having an acute MI
 - (b) creatine kinase MB is exclusively produced by cardiac cells
 - (c) creatine kinase returns to normal levels within 24 hours post MI
 - (d) Troponin I can remain elevated for up to 3 weeks post MI
 - (e) C-reactive protein levels are unchanged in acute myocardial infarct

23. Cardiogenic shock
- (a) occurs in 10 to 15% of patients following an MI
 - (b) is likely after a large infarct that involves 30% of the left ventricle
 - (c) has a 50% mortality rate
 - (d) is generally due to arrhythmia
 - (e) is due to myocardial rupture
24. Myocardial rupture following AMI
- (a) most commonly causes septal rupture
 - (b) most commonly causes papillary muscle rupture
 - (c) is most frequent 3-7 days post event
 - (d) occurs in 10% of cases within 24 hours of MI
 - (e) is more common in males
25. Sudden cardiac death has associated following pathologies except
- (a) aortic valve stenosis
 - (b) mitral valve prolapse
 - (c) tricuspid valve regurgitation
 - (d) myocarditis
 - (e) dilated cardiomyopathy
26. Cor pulmonale
- (a) can be caused by left ventricular failure
 - (b) that is acute causes hypertrophy of the right ventricle
 - (c) is not a feature of pulmonary embolism
 - (d) can be caused by severe obesity
 - (e) can be caused by metabolic alkalosis
27. Regarding valvular pathology
- (a) Functional regurgitation refers to the level of regurgitation of a valve that causes functional symptoms
 - (b) Combined valvular disease implies that the valve is dysfunctional because of a combination of valvular pathology and ventricular hypertrophy
 - (c) Destruction of a valve caused by infection causes slowly evolving stenosis
 - (d) Valvular stenosis is almost always due to a primary cuspal abnormality
 - (e) The causes of mitral stenosis are many and varied
28. Regarding valvular pathology
- (a) Bicuspid aortic valve is seen in 3% of the population
 - (b) Extensive calcium deposits around the mitral valve can cause arrhythmia and sudden death
 - (c) 20% of calcific aortic stenosis is due to rheumatic disease
 - (d) Aortic valve sclerosis refers to serious calcification
 - (e) Calcific aortic stenosis is an example of metastatic calcification

29. Rheumatic fever (*Aschoff bodies in old paper*)

- (a) is seen two weeks after infection with group B streptococcus
- (b) causes acute rupture of the aortic valve in the acute phase of the illness
- (c) produces Aschoff bodies
- (d) can produce a myocarditis, but not an endocarditis
- (e) can produce an arthritis that is far more common in children than adults with the disease

30. Infective endocarditis

- (a) can be cured with antibiotic therapy alone, even if the infection is caused by highly virulent organisms
- (b) leads to death within days or weeks in 50% of cases, despite surgery and antibiotics
- (c) is seen most commonly in patients with previous rheumatic heart disease
- (d) in native valves is most commonly caused by staphylococcus epidermidis
- (e) in prosthetic valves is most commonly caused by staphylococcus aureus

30a. The most common cause of infective endocarditis is (*2004 old paper*)

- (a) *Streptococcus viridans*
- (b) *Staphylococcus aureus*
- (c) *Candida albicans*
- (d) *Haemophilus* species
- (e) *Staphylococcus epidermidis*

31. Libman-Sacks endocarditis

- (a) is a complication of rheumatic heart disease
- (b) produces aortic valvular lesions
- (c) is caused by haemophilus infection
- (d) produces lesions on both sides of the valve leaflets
- (e) produces large lesions, and is a common consequence of systemic lupus

32. Diagnosis of endocarditis

- (a) is based on the Duke criteria
- (b) is based mainly on blood culture
- (c) is purely based on examination
- (d) is diagnosed in the presence of Janeway lesions, Roth spots or Osler's nodes
- (e) is made on the finding of a new murmur and a fever

33. Regarding prosthetic valves all these statements are true except

- (a) Patients are prone to staph epidermidis endocarditis
- (b) Patients can develop a haemolytic anaemia
- (c) Structural deterioration occurs commonly in mechanical valves
- (d) Thromboembolic events are common and require long-term anticoagulation
- (e) 60% of patients with prosthetic valves will develop a valve related problem within 10 years of surgery

34. Regarding cardiomyopathy

- (a) Dilated cardiomyopathy is the most common type
- (b) is mostly commonly caused by ischaemia
- (c) Restrictive cardiomyopathy is most often seen in long-term alcohol abusers
- (d) Hypertrophic cardiomyopathy causes heart failure due to impairment of contractility
- (e) Dilated cardiomyopathy has no associated familial occurrence

35. Concerning myocarditis

- (a) bacteria are the most common causative agent
- (b) Chagas disease is endemic in parts of Europe
- (c) Lyme carditis can cause conduction defects such as atrioventricular block
- (d) HIV is a very common cause of myocarditis
- (e) none of the above statements are true

36. Concerning pericardial disease

- (a) The pericardium normally contains 30-50 mL of straw coloured fluid
- (b) Primary pericardial disease is quite common
- (c) If it accumulates slowly, the pericardium can expand to hold up to 700mL of fluid, before symptoms occur
- (d) Pericarditis is rare after transmural MI, as the epicardium is destroyed and no longer produces fluid
- (e) Unusually, pericarditis is never produced in the setting of infective endocarditis

37. Causes of pericarditis include all except

- (a) Neoplasia
- (b) Rheumatoid disease
- (c) Scleroderma
- (d) SLE
- (e) Chronic hypercalcaemia

38. Causes of dilated cardiomyopathy include all of the following except

- (a) Thiamine deficiency
- (b) Hyperkalaemia
- (c) Lithium
- (d) Coxsackie B virus
- (e) Wilson's disease (excessive copper accumulation)

39. Regarding cardiac transplantation

- (a) Myocardial infarctions in transplant patients present similarly to non-transplant patients
- (b) Lymphoma is a common complication
- (c) has a 5 year survival of 20%
- (d) The major current limitation with transplantation is immunosuppressant therapy induced cardiomyopathy
- (e) none of the above is true

40. Regarding acute endocarditis (*old paper*)

- (a) has a less than 20% mortality
- (b) is caused by virulent organisms
- (c) 30% is caused by bacteria
- (d) There is a clear delineation between the sub-acute and acute types
- (e) *Kingella* is the common pathogen associated with IV drug users with endocarditis

41. Regarding pericarditis (old paper)

- (a) constrictive pericarditis rarely follows suppurative pericarditis
- (b) primary pericarditis is usually bacterial in origin
- (c) serous pericarditis may be due to uraemia
- (d) fibrinous pericarditis is tuberculous until proven otherwise
- (e) haemorrhagic pericarditis is most commonly caused by bacterial infection

Answers:

1. Arteries (**not covered in the syllabus, but worth knowing*) (p512)
 - (a) that are of the **muscular type include the coronary arteries, renal arteries and other branches of the aorta**
 - (b) that are of the **(large) elastic type include the common carotid arteries, aorta, brachiocephalic, iliac, pulmonary.**
 - (c) of the muscular type **contain vasa vasorum**
 - (d) that are large bore (such as the aorta) have a **thin layer** of subendothelial connective tissue in the intimal layer
 - (e) **that are large have vasa vasorum, which are small arterioles arising outside the vessel**

- 1a. From inside to the outside, the layers of the arterial wall are composed of p512
 - (a) endothelium, muscularis mucosa, external elastic lamina, adventitia
 - (b) tunica interna, tunica media, internal elastic lamina, adventitia
 - (c) **endothelium, internal elastic lamina, muscle layer, external elastic lamina, adventitia**
 - (d) endothelium, muscle layer, internal elastic lamina, adventitia, external elastic lamina
 - (e) endothelium, internal elastic lamina, muscle layer, adventitia

2. Capillaries **not covered in the syllabus, but worth knowing* (p513)
 - (a) have **an** endothelial layer and **NO** media
 - (b) **are the approximate diameter of a red blood cell (7-8µm)**
 - (c) in most types of inflammation, **post capillary venules are the site of vascular leakage, and leukocyte exudation**
 - (d) **collectively**, have a high cross sectional area
 - (e) **arterioles** are the principle points of resistance to blood flow, as they are able to dramatically alter their calibre

3. Endothelial cells p513
 - (a) have **remarkable phenotype variability**, depending on the anatomical site
 - (b) **uniquely contain Weibel Palade bodies, which contain vWF**
 - (c) have **many metabolic** properties
 - (d) in most regions provide **an impermeable membrane** for large plasma proteins
 - (e) in the inactive state express **no adhesion molecules** on the vascular surface

4. Endothelial cells are capable of, table 11-1 Endothelial cell properties & functions p514
 - (a) inducing thrombosis **via vWF, tissue factor, and plasminogen activator inhibitor**
 - (b) inhibiting thrombosis via **PGI₂, thrombomodulin, heparin like molecules, plasminogen activator**
 - (c) oxidation of low density lipoprotein (LDL) ✓
 - ∴ **(d) all of the above: they are also capable of**; regulation of vasodilation (NO, PGI₂), vasoconstriction (endothelin, ACE); regulation of immunity (IL-1, IL-6, chemokines, adhesion molecules, MHC molecules; extracellular matrix production (collagen, proteoglycans); Regulation of cell growth (↑PDGF, CSF, FGF; ↓heparin, TGF-β)
 - (e) (a) and (b) only. ✗

5. Inducers of endothelial activation include p514

(a) **lipid products (atherosclerosis), cytokines, bacterial products (septic shock), haemodynamic stresses, complement components and viruses**

(b) corticosteroid

(c) **advanced glycosylation end-products (diabetes)**

(d) All of the above **x**

∴ (e) = (a) and (c) only

6. Proliferation of smooth muscle cells is promoted by

a-d inhibit smooth cell proliferation.

(a) heparan sulphates

(b) nitric oxide (NO)

(c) TGF- β

(d) corticosteroid

(e) **interferon- γ (IFN- γ), endothelin-1, fibroblast growth factor, PDGF, thrombin, IL-1. Other regulators include angiotensin II, catecholamines, oestrogen receptor and osteopontin (from extracellular matrix)**

7. Intimal thickening of vessels p515

(a) **is a normal part of ageing, seen in coronary vessels, without evidence of harm**

(b) is ***mostly abnormal***, but, if even, can be part of ageing as in (a)

(c) is due to a migration of SMC's into the ***intima***

(d) is due to proliferation of smooth muscle cells, ***which lose their ability to contract but potentiate their ability to produce protein***, and divide...***hyperplasia***

(e) occurs due to smooth muscle cell migration into ***the intima***

8. Mönckeberg medial calcific sclerosis p515 -6

(a) **produces radiologically visible calcifications in muscular arteries that are sometimes palpable**

(b) ***does not cause fatality by itself***

(c) ***produces calcific deposits that do not encroach on the vessel lumen***

(d) is a ***disease of people over the age of 50***

(e) is a disease that primarily affects muscular arteries

9. Fatty streaks p516 fig 11-4

(a) ***do not cause*** disturbance to blood flow as they are not particularly raised

(b) do ***not necessarily become fibrous plaques or more advanced lesions, but there is a link***

(c) ***are not thrombogenic***

(d) **contain T lymphocytes and extracellular lipid in lesser amounts than in plaques**

(e) appear in all children ***over the age of 10, regardless of sex, race, environment or geography***

10. Concerning the development of atheromatous plaques pp516-8
- (a) The early stages of development is due to ***intracellular lipid accumulation***
 - (b) Atheromatous plaques begin from ***isolated macrophage foam cells***
 - (c) Atherosclerosis occurs mainly in elastic and ***large and medium sized calibre muscular arteries***
 - (d) Atheromas often undergo calcification, which predisposes plaques to thromboembolic events**
 - (e) The ***abdominal aorta*** is the most heavily involved vessels

11. Atherosclerotic lesions p517
- (a) in the advanced stages, ***involve only the partial circumference of the vessel (eccentric lesion)***
 - (b) involving the ***upper extremities is rare, mesenteric, and renal except at the ostia***
 - (c) usually have a soft grumous core of lipid**
 - (d) are ***patchy and variable along the vessel length***
 - (e) usually ***heavily involve the origins (ostia) of major branches***

12. In descending order the most heavily involved arterial vessels in forming atherosclerotic plaques are p517
- (a) coronary> abdominal aorta> popliteal> internal carotid> circle of Willis ✗
 - (b) abdominal aorta> coronary> popliteal> internal carotid> circle of Willis**
 - (c) abdominal aorta> coronary> popliteal> circle of Willis> internal carotid ✗
 - (d) abdominal aorta> circle of Willis> coronary> popliteal> internal carotid ✗
 - (e) abdominal aorta> coronary> **renal*> circle of Willis> popliteal ✗
- *Vessels of the upper extremities are spared, as are the mesenteric and *renal*, except at the ostia. Severity in one vessel does not mean severity in another.

- 13 Cells which make up typical atherosclerotic plaques, p517-18
- (a) such as smooth muscle cells, can imbibe lipid to become foam cells**
 - (b) such as ***smooth muscle cells***, typically make up the superficial fibrous cap
 - (c) such as foam cells, ***are large lipid laden cells derived principally from blood monocytes***
 - (d) ***fibrous plaques*** are not typical atheromas. They are predominantly smooth muscle cells and fibrous tissue
 - (e) such as foam cells are ***typically derived from blood monocytes***

14. All of the following are major risk factors for the development of atherosclerosis EXCEPT p520 (*from old paper*)

- (a) obesity**
- (b) hyperlipidaemia
- (c) smoking
- (d) hypertension
- (e) type II diabetes mellitus

Major Non modifiable risks: ↑Age, male, family Hx, genetic abnormality

Major Modifiable risks: ↑Lipids, HTN, smoking, diabetes,

Lesser/ uncertain/ unqualified risks: Stress:-type A personality, **obesity**, homocystine levels, physical inactivity, saturated fat intake, Chlamydia pneumoniae infection

15. Regarding the major risk factors for atherosclerosis development, which of the following statements is TRUE? p521
- (a) Being male is a major risk factor, but *females catch up to the same rate of myocardial infarction in the 7th-8th decade because of the withdrawal of oestrogen*
 - (b) Between the ages of 40 to 60 the risk of myocardial infarction increases five-fold.**
 - (c) Whilst being a major risk factor, elevated cholesterol levels alone *are sufficient* to stimulate lesion development.
 - (d) Omega-3 fatty acids *are likely to be beneficial in preventing* the formation atheroma
 - (e) ***Obesity and smoking lowers*** the amount of *serum high density lipoprotein (HDL)*: HDL mobilises cholesterol from developing atheromas, and transports it to the liver for transport to the bile ∴ “good cholesterol”
16. The pathogenesis of atherosclerosis includes p521-522
- (a) accumulation of lipoproteins, mainly *low density lipoprotein (LDL) in the vessel wall*
 - (b) chronic *endothelial cell* injury, usually subtle, with resultant endothelial dysfunction, increased permeability, leukocyte adhesion and *potential for thrombosis*
 - (c) release of factors from activated platelets (and adhesion of the same), mφ, vascular cells, which promote the mitosis and migration of *smooth muscle cells from the media* into the intima: then production of extracellular matrix (collagen, proteoglycans.
 - (d) *accumulation of cholesterol and LDL, and modification of lesional lipoproteins by oxidation, then enhanced accumulation of intra/extracellular lipids in and around mφ and SMCs*
 - (e) elaboration of extracellular matrix by smooth muscle cells, along with proliferation of the SMCs in the intima.**
17. The pathogenesis of atheroma
- (a) *can occur* at sites of morphologically intact endothelium, as endothelial dysfunction has been implicated
 - (b) is *UNKNOWN*, but several hypotheses include elevated homocysteine, smoking by-products and viruses or other infectious agents as a cause for endothelial dysfunction
 - (c) is due in part to the migration of monocytes into the *intima of the vessel: atherosclerosis is primarily a disease of the intima. The media is weakened by intimal pressures late in the disease.*
 - (d) is directly correlated to the high levels of circulating *low density lipoprotein (LDL)*
 - (e) is thought to be due in large part to haemodynamic disturbances, and the adverse effects of hypercholesterolaemia**

18. The role of smooth muscle cells in the pathogenesis of atheroma include p523
- (a) migration of smooth muscle cells from the *media to the intima*
 - (b) an *INcrease in proliferation* and a *DEcrease in contractile capabilities* in intimal smooth muscle cells
 - (c) foam cell formation, and death by apoptosis that is induced by immune cells**
 - (d) *SMCs take up modified lipids*, but do not oxidise these molecules
 - (e) *Respond to* growth factors including follicular growth factor (FGF) and transforming growth factor α (TGF- α), but do not generally produce these factors
19. Cells normally found in an atheromatous plaque include all the following, except
- (a) smooth muscle cells
 - (b) foam cells.
 - (c) macrophages.
 - (d) lymphocytes.
 - (e) platelets. Platelets attach to the outside of plaque and form thrombus**
20. Regarding essential hypertension p526
- (a) Essential hypertension is considered to be a *continuously distributed variable*, rather than a distinct disease, however it is considered to be sustained systolic BP 140, or diastolic of 90
 - (b) Essential hypertension is *present in 25% of persons* in the general population
 - (c) Older patients with high systolic blood pressure are at greater risk of cardiovascular event than those with elevated diastolic blood pressure**
 - (d) *95%* of diagnosed hypertension is idiopathic (or essential)
 - (e) *5% of patients* with hypertension develop malignant hypertension
21. Regarding hypertension p526
- (a) Peripheral vascular resistance is determined mainly at the *arteriolar level*
 - (b) Resistance vessels are controlled by neural α -adrenergic (constriction) β -adrenergic (dilation), humoral (AII, catecholamines, TXA₂, LTBs, Endothelin:- constrict); PGs, kinins, NO:- dilate) and local factors (pH, hypoxia, autoregulation)
 - (c) Angiotensin II acts directly on smooth muscle cells to produce vasoconstriction**
 - (d) Natriuretic peptides induce *vasodilation*
 - (e) Hypertension is considered malignant if the diastolic pressure is 140mmHg
22. Liquorice ingestion increases blood pressure by p527 fig 11-14
- (a) increasing levels of angiotensinogen (*oral contraceptive*)
 - (b) increasing levels of renin (*renal artery stenosis*)
 - (c) increasing mineralocorticoid receptor activity, ?Cushing syndrome**
 - (d) acting on the epithelial sodium channel (*genetic disorder only*)
 - (e) increasing the production of mineralocorticoids (*aldosteronoma*)

23. The kidneys control blood pressure by p527
- (a) controlling blood volume, and producing substances that act as vasopressors and *vasodilators (PGs and NO)*.
 - (b) production of natriuretic factors is performed by the *atrial and ventricular myocardium*
 - (c) producing *renin* in the juxtaglomerular cells of the kidney
 - (d) by producing renin which converts angiotensinogen to angiotensin I**
 - (e) removing angiotensin converting enzyme (ACE) from the circulation **X**
24. Theories as to the cause of essential hypertension EXCEPT
- (a) reduced renal sodium excretion in the presence of normal blood pressure: perhaps the sentinel event, which resets the “pressure natriuresis” point
 - (b) variations in the genes encoding components of the renin-angiotensin system; *partially explains race variability*
 - (c) chronic or repeated vasoconstriction of the resistance vessels
 - (d) Mutations in proteins that affect sodium reabsorption: rare cause of moderately severe hypertension; Liddle’s Syndrome ∴ not “essential”**
 - (e) heavy consumption of salt *could modify the expression of genetic determinants of increased BP.*
25. Hyaline arteriosclerosis p529-530
- (a) has a typical onionskin, concentric thickening of the wall of arterioles (*Hyperplastic*)
 - (b) is related to more acute or severe elevations of blood pressure (*Hyperplastic*)
 - (c) shows acute necrosis of the vessel wall (*Hyperplastic*)
 - (d) is a feature of diabetes**
 - (e) shows none of the above features **X**
26. Hyperplastic arteriosclerosis
- (a) consists of a homogenous hyaline thickening of the walls of arterioles (*hyaline*)
 - (b) is encountered frequently in normotensive elderly patients (*hyaline*)
 - (c) can accompany necrotising arteriolitis, associated with malignant hypertension**
 - (d) is common in diabetes (*hyaline*)
 - (e) is due to chronic haemodynamic stress of hypertension (*hyaline*)
27. An aneurysm p530
- (a) caused by infection can be due to infective endocarditis**
 - (b) of the *true type, bounded by arterial wall components*, is typically seen in tertiary syphilis, atherosclerosis, congenital vascular aneurysms, post MI.
 - (c) which breaches the vascular wall is called a *false* aneurysm: post MI, post vascular graft.
 - (d) which is *fusiform involves long portions* of the vessel wall; saccular aneurysm involves a small portion eg. Berry aneurysm
 - (e) refers to an abnormal dilatation of a blood vessel *or a wall of the heart*

28. A false aneurysm

- (a) A **true aneurysm** is typical of congenital type vascular aneurysms
- (b) A **true aneurysm** in the thoracic aorta can be caused by tertiary syphilis
- (c) **CAN** generally rupture
- (d) is bounded externally by adherent extravascular tissues**
- (e) A **dissection** arises when blood enters the wall of the artery as a haematoma dissecting between its layers

29. Abdominal aortic aneurysms

- (a) are most commonly due to **atherosclerosis**
- (b) are more common in **men**
- (c) generally occur after the **age of 50**
- (d) **syphilitic aneurysms** are typically described as luetic: end-arteritis affects the vasa vasorum of the thoracic aorta and causes dilatation
- (e) generally expand at a rate of 0.2cm-0.3cm per year**

30. The risk of abdominal aortic aneurysm rupture is 532

- (a) **virtually 0%** at **<4cm**
- (b) 25% per year for those larger than 6cm**
- (c) **4.0-4.9cm 1%; 11% per year** for those aneurysms of 5.0-5.9cm
- (d) **directly related** to the diameter of the aneurysm
- (e) none of the above **X**. The most important clinical factor affecting growth of the aneurysm is BP. Wall tension is \propto to Diameter and internal pressure of the lumen. Mortality for unruptured AAA repairs is 5%; ruptured 50+%

31. Thoracic aortic aneurysm p532

- (a) can cause pain due to erosion of bone; eg ribs and thoracic vertebrae**
- (b) is principally caused by **trauma**
- (c) **can affect the aortic valve**, leading to aortic incompetence
- (d) **is a complication of giant cell arteritis**
- (e) is not a feature of Marfan's syndrome**

Other clinical features of thoracic aortic aneurysm include respiratory difficulties due to encroachment on the lungs and airways, swallowing difficulties due to oesophageal compression, persistent cough- laryngeal nerve compression.

32. Non traumatic aortic dissection p532-4

- (a) **does not necessarily require** marked dilatation of the aorta
- (b) is **unusual in** atherosclerosis
- (c) Type A (proximal type) involves the ascending aorta whereas Type B (distal type) **does not** (fig 11-22)
- (d) can be a complication of pregnancy**
- (e) is **an unusual complication** of tertiary syphilis

Occurs in connective tissue disorders such as Marfan's disease. Dissection is most common in men with antecedent hypertension between the ages of 40-60

33. Vasculitis p535-536

- (a) caused by Wegener's granulomatosis is *ANCA mediated*
- (b) of Giant cell type (temporal arteritis) is *unclear (?immune reaction to elastin)*
- (c) can be seen in the setting of Hepatitis B infection, which can cause polyarteritis nodosa**
- (d) is drug induced in *10% of cases*
- (e) of Kawasaki disease predominantly affect the *medium sized* vessels

34. Goodpasture syndrome typically affects p536 (fig 11-23)

- (a) capillaries:** other small vessel vasculitides include Wegener, microscopic polyangiitis
- (b) the large arteries (*giant cell, Takayasu*)
- (c) the veins **X**
- (d) medium sized arteries (*polyarteritis nodosa, Kawasaki disease: antibodies raised against endothelial cells*)
- (e) the aorta (*giant cell, Takayasu*)

35. Giant cell arteritis pp537-8

- (a) not only affects arteries in the head, *but can affect the aorta, and be a cause of thoracic aortic aneurysm (giant cell aortitis)*
- (b) can often present with only vague constitutional symptoms, fever, weight loss, fatigue, without localising signs**
- (c) is of concern because of clinical sequelae such as *blindness, aortic aneurysm,*
- (d) *is very uncommon before the age of 50*
- (e) requires *anti-inflammatory medication* for treatment, which is very effective.

36. Takayasu arteritis 538-9

- (a) is a disease *of large vessels*
- (b) is the diagnosis in any patient with giant cell aortitis before age 40**
- (c) is seen predominantly *in females younger than 40*
- (d) *classically affects the aortic arch*, and in 1/3 of patients it also affects the remainder and the branches
- (e) *can affect* the coronary circulation, and cause MI

37. Polyarteritis nodosa p539

- (a) affects small and medium sized muscular arteries**
- (b) *does not* cause glomerulonephritis, as small vessel involvement is absent, but frequently affects the renal arteries, which is a common cause of death.
Microscopic polyarteritis causes glomerulonephritis in 90% of cases
- (c) typically *sparing* the pulmonary circulation, unlike microscopic polyarteritis
- (d) affects small and medium sized muscular arteries, *but not capillaries, venules or the arterioles*
- (e) is a disease of the young predominantly, but all ages are affected.
30% patients have Hep B antigen in their serum. Universally fatal if left untreated, steroid and immunosuppressants results in remission in 90%.

38. Concerning Wegener granulomatosis p541

- (a) it affects males more than females, peak incidence 5th decade
- (b) it can present as a persistent pneumonitis with cavitory and nodular infiltrates (95%), sinusitis (90%), ulcerations of the nasopharynx (70%) and renal disease (80%)
- (c) left untreated, it causes death in 80% of affected patients within a year
- (d) is a cause of glomerulonephritis

(e) all of the above statements are true

It affects the small to medium sized arteries, arterioles, capillaries and venules.

Similar presentation to PAN, but includes the lungs

39. Regarding the veins of the lower limb p544 (*old paper*)

- (a) Thrombosis in the superficial vein is an ***uncommon source of emboli: The deep veins account for 90% of phlebothrombosis, or thrombophlebitis***
- (b) Phlegmasia alba dolens (painful white leg) is associated with iliofemoral vein thrombosis, seen in pregnancy**
- (c) ***ulceration of the toes and feet and frank gangrene is a common consequence*** of Buerger's disease. Heavy smoking is the main cause. Causes acute and chronic vasculitis of the upper and lower extremities. Begins before age 35 in most cases.
- (d) varicosity ***has a very high genetic component***
- (e) The great preponderance of venous thrombi commence in ***either*** the superficial or the deep veins of the leg (p134)

The Heart:

1. Concerning the heart p554-556

- (a) ***75% of persons have a right dominant circulation, so*** occlusions of the right coronary artery or the left can cause left ventricular damage
- (b) ***75% of individuals*** have a right dominant cardiac circulation
- (c) Myocytes comprise of 25% of the total number of cells in the heart**
- (d) The normal heart weighs approximately ***300-350g*** in adult males
- (e) Cardiomegaly refers to dilatation ***or hypertrophy*** of the heart

2. The effects of ageing on the heart include p558-9

- (a) ***increased mass***
- (b) ***mitral and aortic*** valve calcific deposits
- (c) ***decreased left ventricular cavity size***, increased L atrial size
- (d) Lambl excrescences, on the closure lines of the aortic and mitral valves, probably arising from the organization of small thrombi on the valve contact margins**
- (e) brown atrophy, which often accompanies ***cachectic weight loss, as seen in terminal cancer***

3. Cardiac hypertrophy p560

- (a) **due to pulmonary hypertension can produce a heart weight of two times normal (350-600g)**
- (b) due to ischaemic heart disease can produce a heart weight of *two to three* times normal **(400-800g)**
- (c) due to systemic hypertension can produce a heart weight of *three or more* times normal **(600g-1kg)**
- (d) due to hypertrophic cardiomyopathy can produce a heart weight of *three or more* times normal **(600g-1kg). Hearts over 1kg are rare**
- (e) due to aortic regurgitation can produce a heart weight of *three or more* times normal **(600g-1kg)**

4. Cardiac hypertrophy p560-561

- (a) **pattern reflects the stimulus, and in hypertension is called concentric**
- (b) in hypertension leads to increased myocyte cell width **(but length is not increased), due to extra deposition of sarcomeres**
- (c) in volume overload, leads to increased myocyte cell *width and length*
- (d) due to volume overload leads to **increased dilation and increased ventricle wall diameter**, but proportionately to diameter, this thickness may be less than normal
- (e) due to hypertension is characterised by **reduced cavity size of the ventricle**

5. Cardiac hypertrophy p561

- (a) induced by regular strenuous exercise carries **no, or minimal risk of sudden death**
- (b) induced by regular strenuous exercise **is an extension of physiological growth** and has no deleterious effect. The explanation for this is lacking.
- (c) **involving the left ventricle is an independent risk factor for sudden death**
- (d) increased heart mass **is associated** with excess cardiac mortality
- (e) induced by regular strenuous exercise is an *extension of physiologic growth*

6. Features exclusive to left sided heart failure include p562-563

- (a) congestive hepatomegaly **(R)**
- (b) anasarca **(R)**
- (c) ascites **(R)**
- (d) pleural effusions **(R)**
- (e) **haemosiderin containing macrophages in the alveoli, secondary to Fe containing macrophages that have taken up Fe spilled into the interstitium of the lung by proteins and erythrocytes. (L) In pure RHF, pulmonary congestion is rare.**

7. Myocardial ischaemia p571, 574 para 3

- (a) is **more deleterious than pure myocardial hypoxic states**, as the metabolites are not transported to & from the myocardium
- (b) **due to vasoconstriction can be a consequence of endothelial dysfunction**
- (c) due to **unstable** angina is the most threatening type for later MI
- (d) due to atherosclerosis is manifested late, as the **disease is silent for decades**
- (e) in **90% of cases** is due to atherosclerotic coronary artery obstruction

8. The acute coronary syndromes of ischaemic heart disease include p572

- (a) **unstable angina**
- (b) ischaemic cardiomyopathy ✗
- (c) **acute MI and sudden death**
- (d) all of the above ✗
- (e) **all except (b), which is a chronic, not an acute state** ✓

9. Atherosclerotic lesions of the coronary circulation p572-3

- (a) often ***affect all three of the vessels***
- (b) causing **90%** stenosis often results in inadequate coronary blood flow at rest
- (c) **tend to predominate in the first 3cm of the major artery**
- (d) ***with chronically developed complete occlusion*** often have a reduced cardiac infarction area due to developed collateral circulation
- (e) causing ischaemic heart disease syndromes, depends on the dynamic interaction of the extent and severity of fixed chronic anatomic atherosclerosis, plaque rupture, platelet aggregation, intramural thrombosis and vasospasm

10. Coronary atherosclerotic plaque p573

- (a) with ***small numbers of smooth muscle cells, or with a thin cap*** is called “vulnerable” and are likely to rupture
- (b) ***fissures usually occur frequently occur at the junction of the fibrous cap*** and the adjacent plaque free arterial segment, where the cap is thinnest, and the mechanical stresses are highest.
- (c) **rupture of mild to moderate luminal stenotic (<50%) lesions is common**, making up 2/3^{rds} of ruptured plaque. At <70% stenosis, it comprises 85% of plaque rupture numbers. Once formed plaque is ***a dynamic lesion and remodels***, and the cap remodels, making it potentially labile.
- (d) derives most of its tensile strength against rupture from smooth muscle cell ***production of collagen***, rather than the cells themselves. Remember that they migrate and lose their contractile capabilities when they migrate into the intima
- (e) ***rupture is greatest between the hours of 6am to 12 noon ?due to waking and adrenergic influences on the blood vessels***

11. Regarding coronary atherosclerotic plaque p573-4

- (a) Rupture is a potentially deadly, but fortunately rare occurrence (*see b*) ✗
- (b) **Disruption of plaque, with intraluminal thrombosis is common, often clinically silent occurrences**
- (c) Plaque is stabilised by statin drugs, ***which act by reducing lipid levels in plaque***, thereby inhibiting foam cell formation, and stabilising the lesions
- (d) ***Plaque rupture is often not seen in subendocardial infarction***
- (e) None of the above statements are true ✗

12. Regarding Angina Pectoris p575

- (a) Prinzmetal angina **is not related** to hypertension
- (b) Typical angina pectoris refers to the reduction of the coronary perfusion to a critical **level by chronic stenosing coronary atherosclerosis**. It **presents** as the classic pattern of retrosternal chest pain relieved by rest or glyceryl trinitrate
- (c) Prinzmetal angina **is an uncommon variant**, which is controllable with the usual angina medications such as GTN and calcium channel blockers
- (d) Stable angina typically has coronary artery stenosis of less than 75%**
- (e) Prinzmetal angina **causes ECG changes, and is unrelated to HTN, HR or exercise**

13. Regarding myocardial infarction p575

- (a) Myocardial infarction refers to the **prolonged ischaemia, and death of cardiac myocytes**
- (b) Subendocardial infarction constitutes an area of necrosis limited to the **inner third, and no more than a half** of the ventricular wall
- (c) Subendocardial infarcts can be limited to the distribution of a single major coronary artery, if the clot is lysed before affecting the whole are of myocardium**
- (d) The **subendocardium is least perfused (watershed region)**, and is vulnerable to infarcts
- (e) **Two thirds of myocardial infarctions are caused by atherosclerotic lesions affecting 50% or less** of the coronary arteries (see 10c)

14. Myocardial infarction p576

- (a) 10% of myocardial infarctions occur in people under the age of 40 years**
- (b) 45% of infarcts occur in people **under the age of 65 years**
- (c) Infarcts **cannot be prevented** by hormone replacement therapy in postmenopausal women
- (d) Black populations have **equal rates of infarcts** with whites
- (e) In **10% of cases**, transmural acute infarct is not associated with atherosclerotic plaque rupture, with thrombosis or embolism

15. In myocardial infarction p576

- (a) thrombus generally takes **minutes to evolve** and occlude the lumen of the affected vessel
- (b) vasospasm **potentially** increases the area of infarct, **and is mediated by TXA₂ and endothelin, and platelet aggregation**
- (c) the initial event is change in the morphology of an atheroma: **eg intraplaque haemorrhage, erosion or ulceration**.
- (d) the observation of occlusion of an artery is seen in **60% of patients undergoing angiography at 12 hours post infarct**
- (e) none of the above statements are true ✓**

16. In myocardial infarction (table 12-4 p577)
- (a) the onset of ATP depletion occurs within seconds of artery occlusion
 - (b) loss of contractility is seen after 2 minutes of artery occlusion**
 - (c) ATP is reduced *to 50% of normal after 10 minutes and 10% of normal after 40 minutes* of artery occlusion
 - (d) irreversible cell injury is seen in *20-40 minutes* of artery occlusion
 - (e) microvascular injury *occurs after an hour of artery occlusion*
17. The consequences of myocardial ischaemia include p577
- (a) cessation of aerobic glycolysis in myocytes within seconds ✓
 - (b) development of acute heart failure long before myocyte cell death, *due to loss of contractility very early on in ischaemia* ✓
 - (c) mitochondrial swelling within minutes of onset ✓
 - (d) myocardial necrosis after approximately 30 minutes of severe ischaemia ✓
 - (e) all of the above** ✓
18. Regarding myocardial infarction p577-8
- (a) ***Almost all transmural infarcts*** involve at least a portion of the left ventricle
 - (b) isolated infarction of the right *ventricle is uncommon...1-3%*
 - (c) Transmural infarcts encompass *almost the entire perfusion zone* of the occluded coronary artery, as there is a narrow rim (0.1mm) that is preserved by diffusion of O₂ from the lumen
 - (d) The left anterior descending artery is occluded in 40-50% of infarcts** causing anterior infarct, the R coronary artery is involved 30-40% of times causing inferior/posterior. L circumflex (15-20%) involves the LV lateral wall
 - (e) New infarcts (less than 12 hours) ***are not apparent*** on gross examination at autopsy
19. Regarding changes to the myocardium after myocardial infarction p579 table 12-5
- (a) there is ***dark mottling*** seen at 0.5-4 hours
 - (b) variable wavy fibres are seen ***at the border after 0.5-4 hours. These are probably produced by forceful systolic tugs by the viable fibres adjacent.***
 - (c) decreased contractility ***after 2 minutes***
 - (d) ***coagulative*** necrosis is typical
 - (e) the sarcoplasm is resorbed by leukocytes (in the current text it says macrophages remove necrotic myocytes).**
20. With regard to myocardial infarction: p p577-8 p579 table 12-5 (*old paper*)
- (a) Gross necrotic changes are seen at ***4-12 hours post MI (occasionally dark mottling)***
 - (b) ***irreversible cell injury occurs in 20-40 minutes***
 - (c) fibrotic scarring is ***complete in eight weeks+*** (dense collagenous scar)
 - (d) death occurs in 20% of patients in the first 2 hours**
 - (e) is most commonly caused by occlusion of the ***L anterior descending coronary artery (40-50%)***

21. Myocardial reperfusion injury p583

- (a) Has *unknown clinical significance*
- (b) can cause endothelial swelling that may prevent local reperfusion by occluding capillaries**
- (c) causes *prominent apoptosis*
- (d) *produces haemorrhage* into areas of ischaemia
- (e) is produced in large part, by oxygen free radicals *generated by infiltrating leukocytes*

22. In regard to myocardial infarction p583-584

- (a) 10-15% of patients are asymptomatic whilst having an acute MI**
- (b) creatine kinase MB is produced by cardiac cells *and a small amount is produced by skeletal muscle*
- (c) creatine kinase returns to *normal levels within 72 hours post MI*
- (d) Troponin I can remain elevated for up to *10 days* post MI
- (e) C-reactive protein levels *are elevated* in acute myocardial infarct

23. Cardiogenic shock p584

- (a) occurs in 10 to 15% of patients following an MI**
- (b) is likely after a large infarct that involves *40% of the left ventricle*
- (c) has a *70% mortality rate*
- (d) is generally due to severe pump failure *following massive loss of contractile function*
- (e) is due to myocardial rupture **✗**

24. Myocardial rupture following AMI p584

- (a) most commonly causes *lateral wall rupture at the mid-ventricular level*
- (b) *least commonly* causes papillary muscle rupture
- (c) is most frequent 3-7 days post event**
- (d) occurs in *25% of cases within 24 hours* of MI
- (e) is more common in *females*, over 60, no previous infarct as there is no fibrosis that prevents rupture, and no hypertrophy

25. Sudden cardiac death has associated following pathologies **except** p586

- (a) Aortic valve stenosis
- (b) Mitral valve prolapse
- (c) tricuspid valve regurgitation ✗**
- (d) myocarditis
- (e) dilated or hypertrophic cardiomyopathy, isolated hypertrophy, abnormal conduction system, marked stenosis of 1 or more of the vessels is present (>75%)

26. Cor pulmonale p588

- (a) cannot be caused by left ventricular failure by definition. ***R sided failure secondary to lung pathology***
- (b) that is acute causes ***dilation*** of the right ventricle
- (c) is a feature of ***chronic pulmonary embolism***
- (d) can be caused by severe obesity (Pickwickian syndrome)**
- (e) can be caused by ***metabolic acidosis***, which causes ***pulmonary vasoconstriction***
Can be caused by parenchymal disorders (COAD, CF, bronchiectasis), vessel disorders (recurrent PE, tumour micro embolism, pulmonary HTN, Wegener's, R_x, radiation) chest movement disorders (kyphoscoliosis, obesity, NM disease) Metabolic disorders (acidosis, hypoxaemia, chronic altitude sickness, major airway obstruction)

27. Regarding valvular pathology p589

- (a) Functional regurgitation refers to ***regurgitation caused by ventricular dilation or dilation of the artery***
- (b) Combined valvular disease implies that ***more than one valve is affected***
- (c) Destruction of a valve caused by infection causes ***acute destruction of the valve***
- (d) Valvular stenosis is almost always due to a primary cuspal abnormality**
- (e) The causes of mitral stenosis is ***post inflammatory scarring (eg rheumatic fever)***

28. Regarding valvular pathology p590

- (a) Bicuspid aortic valve is seen in ***1% of the population***
- (b) Extensive calcium deposits around the mitral valve can cause arrhythmia and sudden death, by potentially interfering with the conduction system**
- (c) ***<10% of calcific aortic stenosis*** is due to rheumatic disease
- (d) Aortic valve sclerosis refers to a haemodynamically inconsequential calcification
- (e) Calcific aortic stenosis is an example of dystrophic calcification

29. Rheumatic fever (*Aschoff bodies in old paper*) p594

- (a) is seen two weeks after infection with ***group A streptococcus***
- (b) ***Acute*** infections cause acute rupture of the aortic valve in the acute phase of the illness. ***Post rheumatic change occurs over years***
- (c) produces Aschoff bodies (focal inflammatory lesions, which can be found in all layers of the heart in acute RF; hence the lesion is called a pancarditis)**
- (d) can produce a ***carditis: epicarditis, myocarditis, and endocarditis***
- (e) can produce a migratory polyarthritis of the large joints that is ***far more common in adults than children*** with the disease

30. Infective endocarditis p596

- (a) ***difficult to cure with antibiotic therapy alone***, and often requires surgery if the infection is caused by highly virulent organisms
- (b) leads to death within days or weeks in 50% of cases, despite surgery and antibiotics**
- (c) is seen most commonly with background of ***calcific stenosis, bicuspid valve and artificial valves***.
- (d) in ***prosthetic valves*** is most commonly caused by ***staphylococcus epidermidis***
- (e) in ***native valves*** is most commonly caused by ***strep viridans*** (50-60%), ***staphylococcus aureus*** esp. in IVDU's (10-20%), then commensals in the oral cavity (HACEK). 10% no organism is found.

30a. The most common cause of infective endocarditis is differ somewhat in in the major risk groups (2004 old paper) p596

- (a) ***Streptococcus viridans is commonest in endocarditis of native valves, damaged or otherwise (50-60%)***
- (b) *Staphylococcus aureus, but in IVDU's very high (10-20%)*
- (c) *Candida albicans (rare)*
- (d) *Haemophilus species (HACEK group is not the most common)*
- (e) *Staphylococcus epidermidis (commonest in prosthetic heart valves)*

31. Libman-Sacks endocarditis p598

- (a) is a complication of rheumatic heart disease ✗
- (b) produces valvular lesions on the ***atrioventricular valves (mitral, tricuspid)***
- (c) is ***sterile, with granular fibrinous material***
- (d) **produces lesions on both sides of the valve leaflets**
- (e) produces ***small*** lesions, and is a ***rare*** consequence of systemic lupus

32. Diagnosis of endocarditis

(a) is based on the Duke criteria:

Major: +ve blood culture indicative of causative organism, or persistent unusual one, Echo findings, new regurgitation. Minor: Predisposing lesion, IVU, vascular lesions eg emboli, splinter haemorrhages. Single culture of unusual organism, Echo finding consistent, but not diagnostic eg pericarditis. For diagnosis to be made 2 major; 1 major + 3 minor or 5 minor criteria

- (b) is based mainly on blood culture (***can be major or minor***)
- (c) is purely based on examination ✗
- (d) is diagnosed in the presence of Janeway lesions, Roth spots or Osler's nodes (***minor criteria***)
- (e) is made on the finding of a new murmur and a fever

33. Regarding prosthetic valves all these statements are ***true*** except p600

- (a) Patients are prone to staph epidermidis endocarditis
- (b) Patients can develop a haemolytic anaemia
- (c) Structural deterioration occurs commonly in ***bioprosthetic valves, but modern valves failure is very uncommon***
- (d) Thromboembolic events are common and require long-term anticoagulation
- (e) 60% of patients with prosthetic valves will develop a valve related problem within 10 years of surgery

34. Regarding cardiomyopathy p602

- (a) **Dilated cardiomyopathy is the most common type (90%)> hypertrophic> restrictive**
- (b) is described as a myopathy ***that excludes ischaemia***
- (c) ***Dilated cardiomyopathy*** is most often seen in long-term alcohol abusers
- (d) Hypertrophic cardiomyopathy causes heart failure due to impairment of compliance (***diastolic dysfunction***)
- (e) Dilated cardiomyopathy has ***25-35% associated familial occurrence***

35. Concerning myocarditis
- (a) **Viruses (Coxsackievirus A & B) are the most common causative agent**
 - (b) Chagas disease is endemic in parts of *South America*
 - (c) **Lyme carditis (5% of patients with the disease) can cause conduction defects such as atrioventricular block which require pacing in 30% of cases**
 - (d) **HIV is an uncommon cause of myocarditis**
 - (e) none of the above statements are true ✘
36. Concerning pericardial disease p611
- (a) **The pericardium normally contains 30-50 mL of straw coloured fluid**
 - (b) **Primary pericardial disease is rare**
 - (c) The pericardium can expand to hold up to *500mL of fluid, if it accumulates slowly, before symptoms occur*
 - (d) Pericarditis is seen after transmural MI, *known as Dressler's Syndrome*
 - (e) **Pericarditis is sometimes produced** in the setting of infective endocarditis
37. Causes of pericarditis include all except p611
- (a) Neoplasia
 - (b) Rheumatoid disease
 - (c) Scleroderma
 - (d) SLE
 - (e) **Chronic hypercalcaemia ✘**
38. Causes of dilated cardiomyopathy include all of the following except
- (a) Thiamine deficiency
 - (b) Hyperkalaemia
 - (c) Lithium
 - (d) Coxackie B virus
 - (e) **Wilson's disease** (excessive copper accumulation) especially in the eye, the brain and the liver causing cirrhosis, Parkinson like features, and Kayser-Fleischer rings in the eyes. ✘
39. Regarding cardiac transplantation p615
- (a) Myocardial infarctions in transplant patients present differently to non-transplant patients, as the hearts are denervated.
 - (b) **Lymphoma is a common complication of long-term immunosuppressant agents**
 - (c) **Has a 5 year survival of 60%**
 - (d) The major current limitation with transplantation is *graft arteriopathy (stenoses off the coronaries)*
 - (e) None of the above is true ✘
40. Regarding acute endocarditis (*old paper*)
- (a) Has a **50%+ mortality**
 - (b) **Is caused by virulent organisms**
 - (c) **Most are caused by bacteria**
 - (d) There is *no clear delineation between the sub-acute and acute types*
 - (e) **S. Aureus** is the common pathogen associated with IV drug users with endocarditis

41. Regarding pericarditis p612 (*old paper*)

(a) Constrictive pericarditis *may follow* suppurative pericarditis

(b) Primary pericarditis is *usually viral* in origin

(c) Serous pericarditis may be due to uraemia

(d) *Caseous pericarditis* is tuberculous until proven otherwise

(e) Haemorrhagic pericarditis is most commonly caused by *malignancy*