**Question 1**

Which is the main risk factor for diastolic dysfunction?

A Hypertrophic cardiomyopathy

B Hypertension

C Diabetic heart disease

D Coronary heart disease

Explanation (B)

Diastolic failure occurs predominantly in patients over 65yrs. Women >men. Hypertension is the most common cause.

Other risk factors include diabetes, obesity and bilateral renal artery stenosis. The reduction in the ability of the left ventricle to relax and fill may stem form myocardial fibrosis (cardiomyopathies and IHD), infiltrative disorders associated with restrictive cardiomyopathies (amyloidosis) and restrictive pericarditis. Diastolic failure may also appear in elderly patients without any known predisposing factors. This may be due to an exaggeration of the normal stiffening of the heart with age.

**Question 2**

Which of the following statements regarding diastolic failure is true?

A It is more common in men

B Pulmonary oedema is an uncommon presentation

C Cardiac output is preserved at rest

D Diabetes mellitus us the most common predisposing condition

Explanation (C)

Diastolic failure occurs mostly in patients>65yrs. It is more common in women. Causes: hypertension (commonest), diabetes mellitus, obesity, bilateral renal artery stenosis. The reduction in the left ventricle to relax and fill may stem from myocardial fibrosis (ischaemic heart disease, cardiomyopathies), infiltrative diseases (amyloidosis), and restrictive pericarditis. Diastolic failure may also occur without any predisposing risk factors as well. With diastolic failure, cardiac output is relatively preserved at rest. However, it is unable to increase its output in response to an increase in metabolic demands-exercise. Because the left ventricle is unable to expand, any increase in filling pressure is immediately referred back into the pulmonary system causing (flash) pulmonary oedema.

**Question 3**

Which of the following is a consequence of a myocardial infarction?

A Irreversible cell injury occurs after 60min

B There is loss of contractility within 60 seconds

C Microvascular injury occurs after 2hrs

D ATP reduction of 50% occurs within 20min

Explanation (B)

Only severe ischaemia, lasting 20-40min or longer, leads to irreversible cell damage (necrosis). The extent of necrosis is largely complete within 3-6hrs in experimental models. Progression of necrosis in humans follows a more protracted course of 6-12hrs. In these patients coronary arterial collateral system, often stimulated by chronic ischaemia, is better developed and thereby more effective.

Approximate time of onset of key events to cardiac myocytes in ischaemia are; Onset of adenosine triphosphate (ATP) depletion occurs in seconds, loss of contractility occurs in <2min, ATP reduced to 50% of normal in 10min and 10% of normal in 40min, irreversible cell injury occurs in 20-40min and microvascular injury occurs >1hr

**Question 4**

What is the most common histological change seen within 24 hours following a myocardial infarction?

A Liquefactive necrosis

B Pallor and oedema

C Disintegration of dead myofibres

D Increased collagen deposition

Explanation (B)

Myocardial infarcts of less than 24hrs show early coagulation necrosis, pallor, oedema and haemorrhage. There is also pyknosis of nuclei. Disintegration of myofibres occurs from day 3 to 7 and collagen deposition at weeks 2 to 8

There is coagulative necrosis not liquifactive necrosis.

A closer look at the TB:

Early morphologic recognition of acute MI is difficult especially when death occurs within a few hours of the onset of symptoms. Mis<12hrs old are usually not apparent on gross examination. If the infarct preceded death by 2-3hrs, it is possible to highlight the infarcted area using a stain. The gross histochemical stain gives a brick red colour to intact no infarcted myocardium. Dead cells of an infarct appear as an unstained pale zone. By 12-24 hrs after the infarction, an MI can usually be identified grossly as a reddish blue area of discolouration caused by stagnated trapped blood.

**Question 5**

Which of the following changes occurs in compensated pressure loaded cardiac hypertrophy?

A Hyperplasia

B Diffuse fibrosis

C Ventricular dilation

D Decreased sarcomeres

Explanation (B)

Compensated cardiac hypertrophy results in diffuse fibrosis, a decrease in the capillary myocyte ratio, an increase in the number and mutations of the sarcomeres, synthesis of abnormal and dysfunctional proteins and extreme hypertrophy. Because adult cardiac myocytes cannot divide, augmentation of myocyte number (hyperplasia) cannot occur. In response to pressure loaded cardiac hypertrophy there is the development of a concentric increase in the ventricular wall. In contrast to volume loaded cardiac hypertrophy where there is a dilation of the ventricle

**Question 6**

Which of the following is a common cause of fungal endocarditis?

A Cryptococcus

B Actinomyces

C Candida

D Aspergillus

Explanation (C)

Candida albicans is the most common cause of fungal endocarditis, causing over half of all cases.

Fungal endocarditis

Candida albicans 24%

Non-albicans sepices of Candida 28%

Aspergillus species 24%

Histoplasma capsulatum 6%

Other 17%

The vegetations caused by fungal organisms tend to be larger than bacterial infections

**Question 7**

High output failure occurs in all of the following conditions with the exception of?

A Paget's disease

B Hyperthyroidism

C Systemic arteriovenous fistulas

D Sickle cell disease

Explanation (D)

Causes of high output failure are: anaemia including iron deficiency, vitamin B12, folate defeciency, renal failure (lack of erythropoietin).Others include: pregnancy, beriberi (vitamin B1/thiamine deficiency), thyrotoxicosis, Paget’s disease, arteriovenous fistulae and arteriovenous malformations, morbid obesity, cor pulmonle, carcinoid syndrome, multiple myeloma, beta-thalassemia intermedia and cirrhosis.

**Question 8**

Which of the following statements regarding infective endocarditis is correct?

A Cryptococcus is the most common fungal cause

B Is most commonly caused by staphylococcus epidermidis on prosthetic vaves

C Is most commonly caused by streptococcus viridans on healthy valves

D Is most commonly caused by staphylococcus aureus on damaged valves

Explanation (B)

According to prescribed texts- the causative organisms differ in the major high risk groups. Endocarditis of native but previously damaged or abnormal valves is caused most commonly by Strep Viridans (50-60% of cases). in contrast, more virulent Staf Aureus organisms commonly found on the skin can infect healthy and deformed valves and are responsible for 10-20% of cases overall. Staf Aureus is the most common offender in IVDU. Prosthetic valves are most commonly caused by S. epidermidis. The remaining organism include enterococci and the HACEK organisms (Haemophilus, Actinobacillus, Cardiobacterium, Eikenellla and Kingella).

Emergency text books of Australia have noted that Staf Aureus entering through a breach in the skin, has surpassed Strep Viridans as the commonest pathogen in both NVE (native valve endocarditis) and prosthetic valve endocarditis. This change reflects better dental care and an increase in nosocomial infections. Proven Staf Aureus bacteraemia the incidence of infective endocarditis is 13-25%

Candida is the most common fungal cause of infective endocarditis. The aortic and mitral valves are the main sites of infection

NOTE: For the primary exam, this may be a contentious question. Apparently this question is featured on the emergency medicine website and the answer is STREP VIRIDANS

**Question 9**

Which of the following is the key microscopic feature of Rheumatic fever?

A Nutmeg cells

B Aschoff bodies

C Curshmann's spirals

D Reed-Sternberg cells

Explanation (B)

Aschoff bodies are foci of swollen eosinophilic collagen surrounded by lymphocytic T cells, and they are found in Rheumatic fever. Occasionally plasma cells and plump macrophages called Anitschkow cells are also seen. Curschmann's spirals are spiral-shaped mucus plugs of sputum that may occur in several different lung diseases. Reed-Sternberg cells are giant cells found in individuals with Hodgkin's lymphoma, and are usually of B lymphocyte origin. Nutmeg cells do not exist, but nutmeg liver is due to a perfusion abnormality of the liver usually as result of hepatic venous congestion.

**Question 10**

In compensated cardiac hypertrophy, which of the following changes occur?

A Increased capillary density

B Diffuse fibrosis

C Hyperplasia

D Decreased sarcomeres

Explanation (B)

Compensated cardiac hypertrophy results in diffuse fibrosis, a decrease in the capillary myocyte ratio, an increase in the number and mutations of the sarcomeres, synthesis of abnormal and dysfunctional proteins and extreme hypertrophy. Because adult cardiac myocytes cannot divide, augmentation of myocyte number (hyperplasia) cannot occur.

Question 11

Which of the following is correct with regard to endocarditis in intravenous drug users (IVDUs)?

A Has a better prognosis than other types of endocarditis

B Involves the mitral valve

C Is caused by candida albicans

D Is caused by staphylococcus aureus

Explanation (D)

Staphylococcus aureus causes infective endocarditis on healthy valves in up to 10-20% of cases. Although the aortic and mitral valves are more common sites of infective endocarditis, the right sided heart valves are more common in IVDUs

Extra:

According to the prescribed texts- the causative organisms differ in the major high risk groups. Endocarditis of native but previously damaged or abnormal valves is caused most commonly by Strep Viridans (50-60% of cases). in contrast, more virulent Staf Aureus organisms commonly found on the skin can infect healthy and deformed valves and are responsible for 10-20% of cases overall. Staf Aureus is the most common offender in IVDU. Prosthetic valves are most commonly caused by S. epidermidis. The remaining organism include enterococci and the HACEK organisms (Haemophilus, Actinobacillus, Cardiobacterium, Eikenellla and Kingella).

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Candida is the most common fungal cause of infective endocarditis. The aortic and mitral valves are the main sites of infection

NOTE: For the priamry exam, this may be a contentious question

Question 12

With regard to acute myocardial infarction, which of the following statements is correct?

A It is most commonly caused by occlusion of the left circumflex coronary artery (LCA)

B Gross necrotic changes are present within 4-12h

C Irreversible cell injury occurs in less than 10 minutes

D Fibrotic scarring is completed in less than 2 weeks

Explanation (B)

Irreversible cell injury occurs in 20-40min, fibrotic scaring in 2 months. The most common occlusion occurs in the left anterior descending (LAD) 40-50%, right coronary artery (RCA) 30-40% and the left circumflex (LCA) 15-20%. The overall death rate from an acute myocardial infarction (AMI) in hospital is 10-13% and is reduced if thrombolysis or angioplasty is performed. Out of hospital the death rate is up to 50% in the first hour.

(Not a great question-but seems to come up)

Note: according to the current text. Gross features of a myocardial infarction are present at 4-12hrs. The features are occasional dark mottling

**Question 13**

Which of the following statements is correct in relation to pericarditis?

A Haemorrhagic pericarditis is most commonly due to Klebsiella infection

B Constrictive pericarditis only rarely follows suppurative pericarditis

C Primary pericarditis is usually bacterial in origin

D Serous pericarditis may be due to uraemia

Explanation (D)

Constrictive pericarditis frequently follows suppurative pericarditis. Viral organisms often cause pericarditis. Primary pericarditis is usually and almost always of a viral origin. Haemorrhagic pericarditis is commonly caused by malignant neoplasm. Fibrinous and serofibrinouse pericarditis are the most common types of pericarditis and is caused by uraemia, radiation, rheumatoid arthritis, systemic lupus erythematosus (SLE), trauma and following a myocardial infarction (Dressler syndrome)

Note: the text also talks about acute pericarditis-serous pericarditis, produced by non-infective inflammatory diseases like RA, SLE, scleroderma, tumours and uraemia

**Question 14**

Following myocardial infarction, which of the following is correct?

A Microvascular injury occurs within 30 minutes

B Adenosine triphosphate (ATP) is depleted to 50% at 10 minutes

C Irreversible cell injury occurs within 5 minutes

D Adenosine triphosphate (ATP) depletion begins at 2 minutes

Explanation (B)

Approximate time of onset of key events in myocytes following cardiac ischaemia: Onset of adenosine triphosphate (ATP) depletion occurs in seconds, loss of contractility occurs in <2min, ATP reduced to 50% of normal in 10min and 10% of normal in 40min, irreversible cell injury occurs in 20-40min and microvascular injury occurs >1hr

**Question 15**

Which of the following is the cause of peripheral fluid retention in congestive cardiac failure (CCF)?

A Increased aldosterone

B Increased renin

C Increased glomerular filtration rate (GFR)

D Increased angiotensin II

Explanation (A)

In CCF there is a decreased renal blood flow which activates the renin-angiotensin system. However, an increase in renin and angiotensin II does not of itself causes sodium and water retension. Rather the renin-angiotensin system leads to an increased release of aldosterone which causes sodium and water retension, expanding the extracellular fluid (ECF), increasing hydrostatic pressure leading to the formation of peripheral oedema.

**Question 16**

Rheumatic fever is associated with the following major criteria EXCEPT?

A Erythem marginatum

B Migratory polyarthritis

C Fever

D Pancarditis,

Explanation (C)

Rheumatic fever is characterised by a group of major criteria-JONES criteria

Migratory polyarthritis of the large joints, pancarditis, endocarditic subcutaneous nodules (painless firm subcutaneous nodules of collagen over bones/tendons), erythema marginatum of the skin and Sydenham chorea (involuntary rapid purposeless movements. Acute rheumatic fever appears 10d to 6w after an episode of pharyngitis caused by a group A streptococcal infection in about 3% of infected patients.

Extra

Fever: is a minor criteria of rheumatic fever. Other minor criteria include: Arthralgia, Raised CRP/ESR, leukocytosis and a prolonged PR interval

**Question 17**

Baroreceptors

A Are located in the carotid bodies

B Have afferent fibres which use glutamate as an excitatory transmitter

C Are reset in chronic hypertension to bring blood pressure back to normal

D Are located in the tunica media of vessels

Explanation (B)

Baroreceptors are stretch receptors in the walls of the heart and blood vessels (tunica adventitia). The carotid sinus and aortic arch receptors monitor the arterial circulation. Receptors are also found in the walls of the right and left atria, entrance to the superior and inferior venae cavae, pulmonary veins and pulmonary circulation. The baroreceptors are stimulated by distension of the structures in which they are found, and so they discharge at an increased rate when the pressure in these structures rises. Their afferent fibres pass via CN IX and X to the medulla. Most of them end in the nucleus tractus solitarus (NTS), and the excitatory transmitter is glutamate. Excitatory (glutamate) transmissions extend from the NTS to the caudal venterolateral medualla where they stimulate GABA secreting inhibitory neurons that project to the rostal ventrolateral medulla and the vagal motor neurons in the nucleus ambiguus and the dorsal motor nucleus. Increased baroreceptor discharge INHIBITS the tonic discharge of the vasoconstrictor nerves and excites vagal innervation of the heart, producing vasodilatation, venodilation, a drop in BP, bradycardia and a decrease in cardiac output.

In chronic hypertension the baroreceptor reflex mechanism is reset to maintain an elevated rather than a normal blood pressure.

**Question 18**

Which is NOT a feature of tetralogy of Fallot?

A Subpulmonary stenosis

B Obstruction of the right ventricular outflow tract

C ASD

D Right ventricular hypertrophy

Explanation (C)

The four features of tetralogy of Fallot are

VSD, obstruction of the right ventricular outflow tract (subpulmonary stenosis), an aorta that overrides the VSD and right ventricular hypertrophy. All of the features arise from anterosuperior displacement of the infundibular septum in the embryological stage of development.

Note: Combination of ASD + Tetralogy of fallot is often called Pentad of Fallot

**Question 19**

Which of the following statements regarding dilated cardiomyopathy is true?

A Pregnancy is not a risk factor

B End stage disease, patients have an ejection fraction <25%

C Most commonly affects adults between ages of 30-50

D 5yr mortality rate is 50%

Explanation (B)

Dilated cardiomyopathy (DCM) is characterised by progressive cardiac dilation and contractile dysfunction, usually with concomitant hypertrophy. Many individuals with DCM have a familial (genetic) form. Note that DCM can arise from myocardial insults or interaction of genetic factors and the environment that yield a similar clinicopathologic pattern. These include: toxicities-alcohol and chemotherapeutics, peripartum cardiomyopathy (occurring late in pregnancy or up to 5 months after delivery). End stage DCM often has an ejection fraction (EF) of <25%. 50% of patients die within 2yrs and the 5yr survival rate is 25%. DCM most commonly affects patients aged between 20-50yrs

**Question 20**

In the aging heart which of the following pathological features is correct?

A Decreased cross section luminal areas of the epicardial coronary arteries

B Decreased myocardial mass

C Red atrophy of the myocardium

D Sigmoid shaped ventricular septum

Explanation (D)

Changes in the aging heart

Chambers:

Increased left atrial cavity size, decreased left ventricular cavity size, sigmoid shaped ventricular septum

Valves:

Aortic valve calcific deposits, mitral valve annular calcific deposits, fibrous thickening of leaflets.

Epicardial coronary arteries:

Tortuosity, increased cross sectional luminal area, calcific deposits and atherosclerotic plaque.

Myocardium:

Increased mass, increased sudepicardial fat, brown atrophy and amyloid deposits.

NOTE: in the current TB the table says: DECREASED MASS

Aorta:

Dilated ascending aorta with rightward shift, elongated tortuous thoracic aorta, atherosclerotic plaques

Note: there are other changes not mentioned in this list

**Question 21**

A man presents to the emergency department with severe tearing like pain up and down his back. A CT scan shows an intimal tear and blood between the vessel layers in his ascending aorta only. What is the diagnosis?

A Debakey type III

B Debakey type IV

C Debakey type I

D Debakey type II

Explanation (D)

Aortic dissection are divided into two types

More common and more dangerous proximal lesions- called type A dissections, involving either both the ascending and descending aorta (DeBakey type I) or just the ascending aorta (DeBakey type II)

Distal lesions not involving the ascending part and usually beginning distal to the subclavian artery (called type B dissections or DeBakey type III)

The most common cause of death is rupture of the dissection outward into the pericardium, pleural or peritoneal cavities.

The other classification system is Stanford and groups them based on whether they are managed with surgery or conservatively (with BP control):

DeBakey type 1 and 2 are grouped together as type A dissections --> surgery indicated

DeBakey type 3 are also classified as type B dissections --> conservative management