Multiple-choice questions

Cardiology questions for the MRCP (UK) examination

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Answers are given on pages 646-8.

QUESTION 1

- 1 Describe the abnormality on the ECG in figure 1.
- 2 How would you confirm your diagnosis?

Figure 1

QUESTION 2

A 56-year-old man presents with chest pain. What are the two ECG abnormalities shown in figure 2?

Figure 2

QUESTION 3

This 65-year-old man presented with palpitations, nausea and vomiting.

- 1 What are the two ECG abnormalities shown in figure 3?
- 2 What is the most likely underlying cause?
- 3 Suggest a useful therapeutic measure.

Figure 3

QUESTION 4

- 1 Give two abnormalities on the ECG in figure 4.
- 2 What is the underlying diagnosis?

Figure 4

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QUESTION 5

This 50-year-old woman presented with shortness of breath and was found to have a motor neuropathy.

- 1 What does the chest X-ray in figure 5 show?
- 2 Suggest three possible underlying diagnoses.

Figure 5

QUESTION 6

What does the chest X-ray in figure 6A show?

Figure 6A

QUESTION 7

- 1 What investigation is illustrated in figure 7A?
- 2 Name structures A–F.
- 3 What is the diagnosis?

Figure 7A

QUESTION 8

- 1 What investigation has been undertaken in figure 8?
- 2 What does it show?

Figure 8

QUESTION 9

A 40-year-old man is investigated for shortness of breath on exertion. The following catheter data were obtained:

Pressure	(mmHg)
Right atrial end-diastolic	6
Right ventricular end-diastolic	5
Pulmonary artery	54/34
Pulmonary capillary wedge	18
Left ventricular end-diastolic	10

What underlying condition caused the patient to be breathless?

QUESTION 10

A 60-year-old man suddenly deteriorated following a myocardial infarction. A new systolic murmur is detected. The following pressures and oxygen saturations are noted at Swan-Ganz catheterisation:

Mixed venous oxygen saturation (%)	65
Right ventricular oxygen saturation(%)	87
Pulmonary artery pressure (mmHg)	60/25
Pulmonary capillary wedge pressure (mmHg)	20

What is the most likely diagnosis?

QUESTION 11

A 15-year-old schoolgirl was admitted to hospital for further investigation after being non-specifically unwell for 3 weeks. During this period she had lost her appetite and was feeling feverish intermittently. On examination, her temperature was 38°C, blood pressure 90/60 mmHg, pulse 100 beats/min regular, jugular venous pressure not raised and there was a soft systolic murmur at the apex. The lungs were clinically clear and there were no palpable abdominal masses.

On investigation, haemoglobin was 10.0 g/dl, white blood count $11.0 \times 10^9/l$ (85% neutrophils), sodium 138 mmol/l, potassium 3.8 mmol/l, urea 4.7 mmol/l, creatinine 87 mmol/l, glucose 4.3 mmol/l and ESR 90 mm/h. Three sets of blood cultures were negative. On analysis of the urine, there was a trace of protein, with 20 white blood cells per high powered field. During her hospital stay, the fever continued and after 3 days she became aphasic.

- 1 What is the diagnosis?
- 2 Why had she become aphasic?
- 3 Give three further investigations to consider for this patient.

QUESTION 12

- 1 What two abnormalities are shown in figure 12A?
- 2 What may this be associated with?

Figure 12A

Answers

QUESTION 1

1 Atrial flutter with 2:1 atrio-ventricular heart block.

2 Carotid sinus massage.

The rhythm is atrial flutter with 2:1 atrio-ventricular heart block. The usual flutter rate is 300 beats/min. The flutter waves are best seen in lead aVF. Alternate flutter waves are partly obscured by the T-waves. Stimulation of the carotid sinus may well increase the degree of atrio-ventricular block, enabling the flutter waves to be seen more clearly. Carotid sinus massage should never be undertaken in anyone with a history of cerebrovascular disease or with bruits over the carotids. It should be applied on one side at a time and should always be accompanied by continuous monitoring of cardiac activity. As soon as there is any sign of ventricular slowing the carotid sinus massage must be discontinued.

Intravenous adenosine would have a similar effect in increasing the degree of atrio-ventricular block and could be used if carotid sinus massage was unsuccessful.

QUESTION 2

The following ECG abnormalities can be seen: acute inferior myocardial infarction; Mobitz type I second degree heart block (Wenckebach); ST-elevation is present in leads II, III and aVF consistent with an acute inferior myocardial infarction.

Mobitz type I second degree heart block is characterised by progressive prolongation of the P-R interval culminating in a non-conducted P wave. In Mobitz type II second degree heart block the P-R interval remains constant prior to the blocked P wave.

In the setting of an acute myocardial infarction, Mobitz type I second degree heart block more commonly accompanies inferior myocardial infarction and does not usually require pacing. However, Mobitz type II block is more commonly associated with anterior myocardial infarction and may require temporary or permanent pacing.

QUESTION 3

1 Atrial tachycardia with 2:1 atrioventricular heart block and reverse tick ST-depression.

2 Possible digitoxic effect.

3 Correct the serum potassium level.

The ECG abnormalities may be due to primary myocardial ischaemia or digoxin toxicity associated with ischaemia secondary to the tachycardia. In the presence of palpitations, nausea and vomiting, the possibility of digoxin toxicity should certainly be considered. Investigation for and treatment of myocardial ischaemia should also be undertaken.

Lead I shows one P-wave in front of each QRS and one in each ST segment suggesting an atrial tachycardia with 2:1 atrio-ventricular block. There is ST segment depression in leads I, aVL and V2 to V6 consistent with digoxin toxicity or myocardial ischaemia, probably at least part related to the tachycardia. The degree and shape of the ST depression in V3–V5 powerfully suggest an ischaemic element in addition to the probable effects of digoxin.

Effects of high digoxin levels include nausea, vomiting and visual disturbances. An ECG in this situation may show T-wave inversion, particularly associated with downsloping ST segment depression. Other ECG abnormalities associated with digoxin toxicity include increased atrio-ventricular block, ventricular ectopic activity, couplets and supraventricular arrhythmias. Hypokalaemia exacerbates the effects of digoxin toxicity. The ECG features of hypokalaemia include ST segment depression, prolongation of the QT interval, flattening of the T waves and prominent U waves.

The ECG features of hypokalaemia include ST-depression, prolongation of the QT interval, flattening of the T-wave and prominent U-waves. Hyperkalaemia has a tendency to tall pointed T-waves followed by a reduction in P-wave height and R-wave height with widening of the QRS complexes.

QUESTION 4

1 Junctional bradycardia and the presence of J-waves.

2 Hypothermia.

ECG features of hypothermia include sinus bradycardia, junctional escape rhythms, long QT-interval, J-waves and non-specific ST changes.

QUESTION 5

1 The chest X-ray shows pulmonary oedema.

2 A number of systemic conditions are associated with heart failure and a motor neuropathy: amyloidosis, diabetes mellitus, hypothyroidism, beriberi.

QUESTION 6

Calcified left ventricular aneurysm.
Calcification is often more easily visualised in a lateral or more penetrated view (figures 6B and C).

Figure 6B **Figure 6C****QUESTION 7**

- 1 Transthoracic echocardiogram: parasternal long axis view.
- 2

Figure 7B

- 3 Pericardial effusion.

Echocardiography of a heart surrounded by a pericardial effusion produces an echo-free space in front of the right ventricle and behind the posterior wall of the left ventricle.

QUESTION 8

- 1 Coronary angiogram (left coronary artery).
- 2 A severe stenosis is visualised in the distal left main stem.

QUESTION 9

Mitral stenosis.

The pulmonary artery pressures are high (ie, there is pulmonary hypertension). The difference between the pulmonary capillary wedge pressure (8 mmHg) and the left ventricular end-diastolic pressure (8 mmHg) indicates significant obstruction of the mitral valve. This is most probably due to mitral stenosis (other remote possibilities include atrial thrombus or left atrial myxoma). The difference of 1 mmHg between the right atrial end-diastolic pressure and right ventricular end-diastolic pressure is not significant and therefore there is no evidence of tricuspid valve obstruction.

Normal values:

	Intracardiac pressure (mmHg)	Oxygen saturation (%)
Right atrial pressure	3	65–75
Right ventricular pressure	20/4	65–75
Pulmonary artery pressure	20/12	65–75
Pulmonary capillary wedge pressure	8	
Left atrial pressure	8	96–98
Left ventricular pressure	130/8	96–98
Aortic pressure	130/75	96–98

QUESTION 10

Ruptured intraventricular septum, causing a ventricular septal defect.

A new systolic murmur following a myocardial infarction may be due to a ventricular septal defect or ruptured papillary muscle leading to mitral regurgitation. Distinction between these two possibilities may be very difficult on purely clinical grounds.

Swan-Ganz catheterisation can be used to identify a ruptured intraventricular septum following a myocardial infarction. There is a raised pulmonary artery pressure, raised pulmonary capillary wedge pressure and an increase in oxygen saturation from right atrium to right ventricle.

QUESTION 11

- 1 Infective endocarditis ('culture negative').
- 2 Septic emboli to the brain.
- 3 Blood cultures; echocardiography (transthoracic/transoesophageal); brain computed tomography (CT) scan.

Transthoracic echocardiography may identify the presence of vegetations. In a patient with a good history for endocarditis but no vegetations seen on transthoracic echocardiography, more detailed valve assessment is obtained using transoesophageal echocardiography.

Infective endocarditis often presents gradually with fevers, sweats, flu-like symptoms, joint aches, muscle aches and other flu-like symptoms. The patient may become more short of breath with further chills and fevers. On examination, the patient may be pale and unwell with evidence of weight loss. Petechiae may be seen on the legs, chest, mucous membranes, conjunctiva or on the fundi as Roth's spots. Splinter haemorrhages may be seen more frequently than usual. Small tender areas in the finger and toe pads may be seen (Osler's nodes). Various heart murmurs may be noted, and destruction in a valve may lead to a change in character of the murmur. Acute regurgitation through a valve can lead to severe pulmonary oedema. Vegetations from valve cusps can embolise into coronary arteries, causing diffuse myocardial damage. Systemic emboli may occur to any part of the body. Embolisation to the brain cause acute neurological disturbances and renal emboli sudden loin pain. Right-sided lesions or ventricular septal defects often produce pulmonary emboli. Infected emboli may lead to multiple lung abscesses. In certain circumstances, right-sided emboli may pass via the foramen ovale into the systemic circulation due to the high right-sided pressures ('paradoxical emboli') and cause systemic embolic features.

Initially negative cultures do not necessarily exclude infective endocarditis. Blood serology should also be checked to exclude some of the rarer causes of endocarditis. In some cases multiple repeated cultures over a number of days may prove negative. The diagnosis is, of course much easier to sustain when the blood cultures are positive. When there is a strong presumptive clinical case of infective endocarditis (known cardiac lesion, the presence of a murmur, previous good health, pyrexia, anaemia, leucocytosis, raised ESR and C-reactive protein level, the absence of any other evidence of inflammation or specific localised infection) the condition may be treated as 'culture negative endocarditis'. During the initial diagnostic period and subsequent treatment period, frequent (two or three times weekly) checks on the haemoglobin, white cell count, ESR, C-reactive protein and creatinine levels are helpful.

QUESTION 12

1 Xanthelasmata; corneal arcus.

2 Hypercholesterolaemia.

Xanthelasmata are subcutaneous cholesterol deposits at the inner margins of the eyelids. A corneal arcus is a white circle at the junction of the cornea and sclera. The presence of xanthelasmata and a corneal arcus are often associated

with hypercholesterolaemia. Cholesterol may also be deposited in subcutaneous nodules over tendons as 'tendon xanthomata'. Common sites include areas over the patella tendon, Achilles tendon and hand tendons.

Figure 12B

Figure 12C

Figure 12D