

# Paper 1 Questions

## MCQs

**[1]** Concerning cardiac failure . . .

- (a) valvular disease is the most common cause in the UK
- (b) raised levels of atrial natriuretic peptide (ANP) are thought to be beneficial in heart failure
- (c) a third heart sound is a common finding on auscultation
- (d) the heart looks small on plain postero-anterior chest radiograph
- (e) all diuretics used in treatment of cardiac failure potentially cause hypokalaemia

**[2]** Concerning hypertension . . .

- (a) approximately 50% of all cases of hypertension are caused by renal dysfunction
- (b) patients with Conn's syndrome usually have a raised serum potassium level
- (c) hypothyroidism does not cause hypertension
- (d) the 'Cushing reflex' is an increase in systolic blood pressure that occurs secondary to Cushing's syndrome
- (e) malignant hypertension can be diagnosed on fundoscopy alone

**[3]** Acute myocardial infarction . . .

- (a) causes chest pain not relieved by glyceryl trinitrate (GTN)
- (b) can be entirely painless in elderly diabetic patients
- (c) should prompt administration of intramuscular morphine to relieve pain
- (d) can be diagnosed on the history alone
- (e) first shows on ECG in the form of 'Q' waves

**[4]** The following are recognized complications of acute MI . . .

- (a) Sheehan's syndrome
- (b) acute aortic regurgitation

- (c) Dressler's syndrome
- (d) electromechanical dissociation
- (e) left ventricular aneurysm

**[5]** The following cardiac events are matched correctly to the corresponding ECG changes . . .

- (a) inferior infarct → ST-segment depression in leads II, III and aVF
- (b) pericarditis → downward-sloping ST-segment depression in all anterior chest leads
- (c) atrial fibrillation → irregularly irregular P-waves
- (d) first degree heart block → broad QRS complexes
- (e) ventricular aneurysm → persistent ST-segment elevation in chest leads

**[6]** In peripheral arterial occlusive disease . . .

- (a) the relationship between radius of artery and flow within it is described by the Fick principle
- (b) claudication means 'cramping' in Latin
- (c) Leriche syndrome is arterial occlusive disease below the popliteal trifurcation
- (d) 'rest pain' commonly occurs in the feet and metatarsal heads
- (e) arterial ulcers are usually painless

**[7]** Concerning aortic stenosis . . .

- (a) all aortic valve murmurs are heard best with the patient on their left side with breath held in end-expiration
- (b) it causes hypertrophy of only the left ventricle
- (c) it causes a 'Waterhammer' character in the radial pulse
- (d) it causes an 'ejection systolic' murmur
- (e) it is a common cause of atrial fibrillation

**[8]** Concerning atrial fibrillation (AF) . . .

- (a) it is commonly caused by dehydration

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- (b) when caused by high levels of circulating thyroid hormone, it is always accompanied by thyroid eye signs
- (c) no P waves are present on the ECG
- (d) the QRS complex is broad
- (e) class Ic antidysrhythmics (e.g. flecainide) are safe to give to most patients with atrial tachyarrhythmias

**[9]** Concerning the respiratory organs of the body . . .

- (a) the right main bronchus lies more vertically than the left
- (b) the lung is an organ with a single blood supply (i.e. the pulmonary artery)
- (c) the alveolar lining of the lungs comprises mainly type I pneumocytes
- (d) only the right lung has an oblique fissure
- (e) in healthy subjects, the strongest stimulator of ventilation is an increase in the arterial partial pressure of CO<sub>2</sub> (PaCO<sub>2</sub>)

**[10]** On examination of the chest of a patient with a unilateral tension pneumothorax . . .

- (a) chest wall movements are decreased on both sides
- (b) the mediastinum is pulled towards the affected side
- (c) the percussion note may be hyperresonant
- (d) breath sounds may be decreased or absent on the affected side
- (e) tactile vocal resonance will be increased on the affected side

**[11]** Concerning chronic obstructive pulmonary disease (COPD) . . .

- (a) the airway obstruction is entirely irreversible
- (b) chronic bronchitis is believed to be due mainly to mucous gland hypertrophy
- (c) patients commonly demonstrate asterixis
- (d) 'cor pulmonale' is defined as lung disease secondary to ventricular hypertrophy
- (e) it mainly leads to type II respiratory failure

**[12]** Concerning respiratory failure . . .

- (a) PaCO<sub>2</sub> is, by definition, elevated in all types of respiratory failure
- (b) the commonest cause of type II respiratory failure is pneumonia
- (c) pulsus paradoxus is a sign commonly associated with respiratory failure
- (d) it always presents with dyspnoea

- (e) it causes 'bat's wing' pulmonary oedema, upper lobe diversion and Kerley-B lines on chest radiograph

**[13]** Concerning asthma . . .

- (a) airway limitation is uniformly reversible in asthma
- (b) typical asthma attacks occur during exercise
- (c) ACE inhibitors should not be given to asthmatic subjects
- (d) symptoms of asthma are usually worse at night
- (e) β<sub>2</sub> adrenoceptor antagonists are commonly used in treatment of asthma

**[14]** Tuberculosis . . .

- (a) is caused by the Gram-negative bacillus *M. tuberculosis*
- (b) is a recognized cause of apical lung fibrosis
- (c) is classically associated with the presence of non-caseating granulomas
- (d) is diagnosed using one test with two different names: Heaf and Mantoux
- (e) is a recognized cause of erythema nodosum

**[15]** Concerning palpation of the abdomen . . .

- (a) in healthy, slim individuals, the liver is normally palpable in the right upper quadrant in deep inspiration
- (b) the normal liver extends superiorly to the 3rd intercostal space
- (c) hepatic enlargement usually proceeds toward the left iliac fossa
- (d) in healthy, slim individuals, the spleen is normally palpable on deep inspiration in the left hypochondrium
- (e) splenic enlargement occurs obliquely, toward the right iliac fossa

**[16]** The following are recognized gastrointestinal causes of finger clubbing . . .

- (a) fibrosis
- (b) coeliac disease
- (c) Plummer–Vinson syndrome
- (d) gastrointestinal lymphoma
- (e) cystic fibrosis

**[17]** The following are recognized medical causes of abdominal pain . . .

- (a) diabetic ketoacidosis
- (b) myocardial infarction
- (c) Addison's disease

## EMQs

## Question 1

Match the following examination findings to the most accurate diagnosis:

<b>(1)</b> JVP	Normal
Apex	'Tapping' in character, not displaced laterally
Auscultation	Grade 3 mid-diastolic murmur at apex, no pre-systolic accentuation
Chest	Clear
BP	155/82 mmHg

<b>(2)</b> JVP	Elevated
Apex	Displaced laterally, and 'thrusting' in character
Auscultation	Pan-systolic murmur at apex
Chest	Bi-basal end expiratory crepitations
BP	143/65 mmHg

<b>(3)</b> JVP	Normal
Apex	'Tapping' in character, not displaced
Auscultation	Grade 3 mid-diastolic murmur at apex, pre-systolic accentuation
Chest	Clear
BP	162/73 mmHg

<b>(4)</b> JVP	8 cm
Apex	Displaced, thrusting in character
Auscultation	Short early diastolic murmur
Chest	Few bi-basal crepitations
BP	180/62 mmHg

<b>(5)</b> JVP	Normal
Apex	'Heaving' in character and displaced laterally
Auscultation	Grade 3 crescendo-decrescendo systolic murmur over whole precordium, not heard in carotid area
Chest	Few bi-basal crepitations
BP	110/93 mmHg

- (a) Mitral stenosis
- (b) Mitral regurgitation
- (c) Aortic regurgitation
- (d) Aortic sclerosis with atrial fibrillation
- (e) Aortic regurgitation with left ventricular failure
- (f) Mitral stenosis with atrial fibrillation
- (g) Mitral regurgitation and stenosis
- (h) Aortic stenosis
- (i) Aortic sclerosis
- (j) Mitral regurgitation with diastolic flow murmur
- (k) None of these

## Question 2

Match the following case histories to the best diagnosis given in the list below.

**(1)** A 47-year-old woman presents with a history of recurrent headaches over 5 years. The pain is described as unilateral, behind the eye and severe. She is well between episodes. There is no vomiting although she feels nauseated and has abdominal discomfort. The onset is typically sudden. She occasionally has 'hazy' disturbance of her vision. There are no abnormal features on systemic review. In the past she has had hypertension, but is currently not taking treatment. She has three children. There are no regular medications. On examination she has a BMI of 29 and a BP recording of 170/95 mmHg. Otherwise a full neurological examination was normal, including fundoscopy.

**(2)** A 15-year-old girl presents with a 5-week history of persistent headaches. These are described as 'all over the head'. There is associated episodic blurring of vision and dizziness. There is no history of head injury. These headaches occur throughout the day. There have been several episodes of vomiting. In the past she has been fit and well. Her mother and sister have both suffered episodes of migraine since childhood. She takes the oral contraceptive pill as her only medication. On examination she is obese and has a BMI of 32. She is normotensive. Cranial nerve examination is normal. Fundoscopy reveals bilateral blurring of the optic disc margins. There are no abnormal findings in the peripheral nervous system.

**(3)** A 52-year-old patient attends complaining of episodic bouts of headache. He has no previous history of headaches. The pain is described as burning and severe. The pain occurs behind the right eye, and is associated with lacrimation, and running of the nostril on the same side. Each episode lasts approximately 1 h and then subsides. In the past he has suffered from diabetes and angina. He smokes 20 cigarettes per day. His current medication includes metformin and GTN spray for occasional use. You happen to see him during an acute episode. He has conjunctival injection on the side of the pain. Cranial nerve examination and fundoscopy findings are normal.

**(4)** A 58-year-old female patient presents with her relatives with a history of increasing headaches. The pain is felt worse at the top of the head. These occur every day and improve towards the end of the day. In addition there have been several reported episodes of irregular 'jerking' of the left arm, each lasting around 5 min. There is no history of head injury. There is a 10-year history of excessive alcohol consumption, 30 units per day. She takes no regular medication. On examination she is afebrile. There is a bilateral coarse tremor. There is a single spider naevus on the upper chest wall, but no jaundice or hepatomegaly. Cranial nerve examination reveals a right-sided homonymous inferior quadrantanopia.

**(5)** A 19-year-old university student presents acutely with a headache, vomiting and photophobia. He had been found confused, wandering around his hall of residence looking for his room. He had been fit and well until a few hours ago. Prior to admission, in the ambulance, he had been observed to have a grand mal fit, which has responded to rectal diazepam. Past medical history includes an admission for observation 6 months previously following a minor head injury after falling off his bike. He had received the meningitis C vaccine before going to university. On examination he was pyrexial at 38.7°C. Confused and GCS 10/15. There was obvious neck stiffness and photophobia. There was no rash. Cranial nerve examination was normal. The pupil size was 3 mm bilaterally and reactive and the optic discs were thought to be normal.

- (a) Trigeminal neuralgia  
 (b) Migraine  
 (c) Acute bacterial meningitis  
 (d) Tension headache

- (e) Giant cell arteritis  
 (f) Space occupying lesion  
 (g) Chronic subdural haematoma  
 (h) Epilepsy  
 (i) Cavernous sinus thrombosis  
 (j) Viral meningitis  
 (k) Benign intracranial hypertension  
 (l) Cluster headache  
 (m) Referred pain  
 (n) Hypertensive encephalopathy  
 (o) Acute encephalitis  
 (p) None of these

### Question 3

Match the following chest examination findings to the most likely diagnosis:

<b>(1)</b> Respiratory rate	36 b.p.m.
Central cyanosis	Present
Trachea	Central
Chest shape	Normal
Expansion	Equal
Percussion note	Resonant bilaterally
Auscultation	Normal breath sounds

<b>(2)</b> Respiratory rate	32 b.p.m.
Central cyanosis	Present
Trachea	Deviated to the right
Chest shape	Normal
Expansion	Reduced on left
Percussion note	Hyperresonant on left
Auscultation	Reduced breath sounds on left

<b>(3)</b> Respiratory rate	16 b.p.m.
Central cyanosis	Absent
Trachea	Deviated to left side
Chest shape	Depressed chest wall at left apex
Expansion	Reduced in left upper zone
Percussion note	Dull over left clavicle
Auscultation	Normal

<b>(4)</b> Respiratory rate	20 b.p.m.
Central cyanosis	Absent
Trachea	Deviated to the right
Chest shape	Normal
Expansion	Reduced at right base
Percussion note	Stony dull at right base
Auscultation	Reduced breath sounds at right base

<b>(5)</b> Respiratory rate	28 b.p.m.
Central cyanosis	Present
Trachea	Central
Chest shape	Normal
Expansion	Normal
Percussion note	Dull at right base
Auscultation	Bronchial area at right base

<b>(6)</b> Respiratory rate	28 b.p.m.
Central cyanosis	Present
Trachea	Central
Chest shape	Hyperinflated bilateral
Expansion	Equal
Percussion note	Resonant equally left and right sides
Auscultation	Diffuse polyphonic expiratory wheezing

- (a) Lung abscess
- (b) Right lower lobe pneumonia
- (c) Right sided pleural effusion
- (d) Old tuberculosis at right apex
- (e) Acute pulmonary oedema
- (f) Asthma
- (g) Tension pneumothorax on the right
- (h) Left ventricular failure
- (i) Left apical lung fibrosis
- (j) Right ventricular failure
- (k) Pulmonary embolism
- (l) Inhaled foreign body in right main bronchus
- (m) None of these

#### Question 4

From the following questions, choose the best diagnosis from the list below.

**(1)** A 7-year-old boy presents with spontaneous onset of swelling and pain, in his right knee. The previous day he

had run a cross-country race at school without problems. There is no family history of haemophilia or other bleeding disorder. On examination he was mildly pyrexial at 37.5°C and he appeared in pain. The right knee was hot, swollen and locally tender. The following investigations were obtained:

Hb	12.3 g/dL
WCC	$5.6 \times 10^9/L$
Platelets	$354 \times 10^9/L$
PT	12 s (NR 10–15 s)
APPT	72 s (NR 25–35 s)
Bleeding time	Normal
Factor VIII:C	5% of normal

**(2)** A 34-year-old man presents with a swollen and painful right knee. There is no preceding history of trauma. His brother had had similar problems in the past. The following investigations were obtained:

Hb	14.7 g/dL
WCC	$10.6 \times 10^9/L$
Platelets	$373 \times 10^9/L$
PT	14 s (NR 10–15 s)
APPT	79 s (NR 25–35 s)
Bleeding time	Normal
Factor VIII:C	Normal

**(3)** A 27-year-old female is investigated for heavy periods. Her mother had similar problems. She had also noticed prolonged bleeding from simple cuts and after blood taking. She was on no medication. The following investigations were obtained:

Hb	10.2 g/dL
WCC	$9.6 \times 10^9/L$
Platelets	$175 \times 10^9/L$
PT	11 s (NR 10–15 s)
APPT	79 s (NR 25–35 s)
Bleeding time	18 min (normal <10 min)
Factor VIII:C	20% of normal

**(4)** A confused 78-year-old patient is admitted and noted to have numerous bruises. Her relatives say she takes a range of medications, but the details are not known and no reliable history can be obtained from the patient. In her bag is found a cardiology outpatients appointment card. On examination she is confused and drowsy with a GCS of 9/15. There are multiple large bruises of the skin. She has an enlarged right pupil that reacts poorly to light. Her pulse is 52/min and irregularly

# Paper 1 Answers

## MCOs

- [1] (a) F (b) F (c) T (d) F (e) F  
 [2] (a) F (b) F (c) F (d) F (e) F  
 [3] (a) F (b) T (c) F (d) F (e) F  
 [4] (a) F (b) F (c) T (d) T (e) T  
 [5] (a) F (b) F (c) F (d) F (e) T  
 [6] (a) F (b) F (c) F (d) T (e) F  
 [7] (a) F (b) T (c) F (d) T (e) F  
 [8] (a) T (b) F (c) T (d) F (e) F  
 [9] (a) T (b) F (c) T (d) F (e) T  
 [10] (a) F (b) F (c) T (d) T (e) F  
 [11] (a) F (b) T (c) T (d) F (e) T  
 [12] (a) F (b) F (c) T (d) F (e) F  
 [13] (a) F (b) F (c) F (d) T (e) F  
 [14] (a) F (b) T (c) F (d) F (e) T  
 [15] (a) T (b) F (c) F (d) F (e) T  
 [16] (a) F (b) T (c) F (d) T (e) T  
 [17] (a) T (b) T (c) T (d) T (e) T  
 [18] (a) F (b) F (c) T (d) F (e) F  
 [19] (a) F (b) F (c) F (d) T (e) F  
 [20] (a) F (b) F (c) F (d) F (e) F  
 [21] (a) F (b) F (c) F (d) T (e) T  
 [22] (a) F (b) T (c) F (d) F (e) F  
 [23] (a) F (b) F (c) T (d) T (e) T  
 [24] (a) F (b) F (c) F (d) F (e) F

[1] (a) F (b) T (c) T (d) F (e) F

(a) Valvular disease only causes 7% of cases of heart failure in the UK. The commonest cause of heart failure in the UK is **atherosclerotic ischaemic heart disease**. In general, the causes of *low-output* cardiac failure can be split up depending on whether they cause predominantly systolic, predominantly diastolic or acute failure of the myocardium . . .

• **Dominant systolic heart failure**

- Ischaemic myocardial disease, coronary artery disease
- Cardiomyopathy (alcoholic, diabetic, drug-induced, idiopathic)
- Myocarditis
- Valvular heart disease
- Congenital heart disease with severe pulmonary hypertension
- Terminal ventricular septal defect or atrial septal defect
- **Dominant diastolic heart failure**
  - Hypertension
  - Severe aortic stenosis
  - Hypertrophic cardiomyopathy
  - Restrictive cardiomyopathy
  - Ischemic myocardial disease, coronary artery disease
- **Acute heart failure**
  - Acute mitral or aortic regurgitation
  - Rupture of valve leaflets or supporting structures
  - Infective endocarditis with acute valve incompetence
  - Myocardial infarction

(b) Atrial cells store and release the 28-amino-acid **atrial natriuretic peptide** (ANP) in response to **stretch of the right atrium** by increased central venous pressure. The relative volume overload of the atria that occurs in heart failure causes levels of ANP to be high, leading to a variety of beneficial effects in heart failure, including . . .

- Increased sodium and water excretion by the kidney (**natriuresis and diuresis**)
- Increased glomerular filtration, hence increased sodium excretion (by selective vasodilatation of renal glomerular afferents, and vasoconstriction of renal glomerular efferents)

- Relaxation of vascular smooth muscle (vasorelaxation of capacitance vessels)
- Increased vascular permeability
- Inhibition of release/action of several undesirable hormones which exacerbate heart failure (e.g. aldosterone, angiotensin II, endothelin, ADH)

When it was first discovered, ANP was cast as the cardiovascular hero, standing opposed to the villainous intents of vasoconstrictors such as angiotensin, ADH and endothelin in heart failure. Unfortunately, its potential therapeutic use foundered with the discovery of its short plasma half-life. Subsequent attempts to devise ANP agonists or agents to block clearance of the endogenous peptide have been thus far unsuccessful.

**(c)** The third heart sound (S3) occurs in **early** diastole due to **rapid ventricular filling** as soon as the mitral and tricuspid valves open. It can be normal in children and young adults, but is abnormal in others and represents **heart failure or volume overload of the heart** (e.g. mitral or aortic regurgitation). It is commonly referred to as a 'distressed' sound.

The fourth heart sound (S4) occurs in **late** diastole in association with ventricular filling due to atrial systole, and is related to **reduced ventricular compliance**. It is a low-frequency oscillation that can be normal at older ages owing to a physiological decline in ventricular compliance, but is nearly always abnormal at younger ages especially if it is of high intensity or is palpable. It is **common in ventricular hypertrophy**, particularly with hypertension and aortic stenosis, and is almost invariably in acute myocardial infarction. S4 may arise from the right ventricle, the left ventricle or both. It is commonly referred to as a 'stressed' sound.

**(d)** The chest X-ray in a patient with heart failure has a classical pattern, comprising . . .

- Cardiomegaly (greater than the width of one hemithorax)
- Upper lobe venous diversion
- Kerley-B lines (fine peripheral septal lines; named after Peter J. Kerley, an English radiologist who also described Kerley-A and Kerley-C lines on chest radiographs)
- 'Bat's wing' hilar oedema
- Bilateral effusions

**(e)** Diuretics commonly used in the treatment of heart failure include . . .

- **Thiazide diuretics** (e.g. bendroflumazide, metolazone): decrease active reabsorption of  $\text{Na}^+$  and  $\text{Cl}^-$  in the dis-

tal convoluted tubule by binding to the chloride site of the electroneutral  $\text{Na}^+/\text{Cl}^-$  co-transport system and inhibiting its action. Potassium loss with these drugs is significant and can be serious. Excretion of uric acid ( $\rightarrow$  gout) and calcium is decreased, whereas that of magnesium is increased.

- **Loop diuretics** (e.g. furosemide, bumetanide): inhibit transport of  $\text{NaCl}$  out of the lumen of the thick segment of the ascending limb of the loop of Henle. These are the most powerful of all currently used diuretics, potentially causing loss of up to 25% of the  $\text{Na}^+$  in the filtrate by direct inhibition of the  $\text{Na}^+/\text{K}^+/2\text{Cl}^-$  carrier in the luminal membrane. Again, these drugs cause significant  $\text{K}^+$  loss. There is an increase in the excretion of calcium and magnesium, and a decrease in the excretion of uric acid ( $\rightarrow$  gout).
- **Potassium-sparing diuretics** (e.g. spironolactone, amiloride): Spironolactone has a limited diuretic action. By acting as an **aldosterone antagonist** it inhibits  $\text{Na}^+$  retention and decreases  $\text{K}^+$  excretion. Similarly, amiloride has limited diuretic efficacy. By blocking  $\text{Na}^+$  reabsorption in the collecting tubules and ducts, it concomitantly decreases  $\text{K}^+$  excretion. Importantly, drugs in this class are the only diuretics that **do not cause hypokalaemia**.

## [2] (a) F (b) F (c) F (d) F (e) F

**(a)** Over 90% of all cases of hypertension arise due to **no known cause**, referred to as '**essential**' hypertension. The remaining cases of hypertension that do have a defined cause are known as '**secondary**' hypertension. These are always important to consider because even though they are rare, they are frequently amenable to treatment.

### **Secondary causes of hypertension**

- **Renal causes:** these account for **>80% of cases of secondary hypertension**. They cause inappropriate retention of salt and water and inappropriate elevation of plasma renin levels. They may be split into:
  - *Renovascular*  
Renal artery stenosis
  - *Renoparenchymal*  
Chronic glomerulonephritis  
Chronic pyelonephritis  
Diabetic nephropathy  
Adult polycystic kidney disease  
Chronic tubulointerstitial nephritis

- **Endocrine causes:** there are eight . . .
  - Conn's syndrome (excess mineralocorticoid) → **(b)**
  - Cushing's syndrome (excess glucocorticoid)
  - Acromegaly (excess growth hormone)
  - Adrenal hyperplasia (congenital)
  - Pheochromocytoma
  - Hyperthyroidism
  - Hypothyroidism → **(c)**
  - Hyperparathyroidism
- **Cardiovascular causes:** the most important is coarctation of the aorta (also polycythaemia rubra vera)
- **Pharmacological causes:** including the oral contraceptive pill, corticosteroids, monoamine oxidase inhibitors (paroxysms when eating tyramine-containing foods), cocaine, amphetamines, vasopressin, etc.
- **Pregnancy**
- **Raised intracranial pressure (ICP):** an acute rise in ICP causes the 'Cushing phenomenon' or 'reflex', which is essentially a rise in systemic blood pressure in response to the increased intracranial pressure. Commonly occurs in head injury or intracranial haemorrhage → **(d)**

To impress examiners with your knowledge of secondary causes of hypertension, also mention **GRA (glucocorticoid remediable aldosteronism)**. In this condition there is a crossover mutation and fusion of the adrenocorticotropic hormone (ACTH)-regulatory element of the 11- $\beta$ -hydroxylase gene and the aldosterone synthase gene, meaning that every time the body attempts to manufacture steroid, it instead produces aldosterone, causing profound hypertension via salt and water retention. Once discovered, the treatment is simple: give exogenous dexamethasone to switch off the need for endogenous steroid production, thereby switching off inappropriate aldosterone production.

**(e)** Malignant hypertension is said to occur when the BP rises suddenly and precipitously to levels above 140 mmHg diastolic. There is characteristic fibrinoid necrosis of vessel walls and rapid progression to renal failure. There are marked changes in retinal vessels, and grades 3 and 4 on the Keith-Wagener classification of hypertensive retinopathy are diagnostic of malignant hypertension, but *only in the presence of a diastolic BP > 140 mmHg*.

**[3] (a) F (b) T (c) F (d) F (e) F**

**(a)** Although nitrates do not have the marked effect of

lessening chest pain in acute MI that they do in stable angina, they are still able to noticeably lessen the pain of acute MI when given sublingually, buccally or intravenously.

**(b)** So-called 'silent' infarcts occur in the elderly, diabetics and patients with a long history of hypertension. Instead of presenting with pain, these infarcts **present with dyspnoea** from development of acute pulmonary oedema, **syncope or coma** from dysrhythmias, **acute confusional states, diabetic hypoglycaemic crisis, hypotension or shock**.

**(c)** Although administration of morphine (or more correctly diamorphine) is an important early manoeuvre in treating acute MI (since it produces analgesia and lessens subjective distress from dyspnoea), it should be given **intravenously** (or orally if access is difficult) and not intramuscularly. This is because should the patient later be thrombolysed, significant internal or external bleeding can occur if intramuscular injections have been recently given.

**(d)** Myocardial infarction should be *suspected* on the history, but is should only be diagnosed in the presence of either positive ECG findings, or a raised level of cardiac enzymes on testing the blood an appropriate interval after onset of symptoms.

**(e)** The first change of acute MI visible on the ECG is a point of contention, although it most certainly is **not** the appearance of Q-waves.

**ST-segment elevation** is said to occur within minutes of the onset of acute MI and can persist for up to 2 weeks. If elevation persists over 4 weeks, this suggests formation of a ventricular aneurysm.

Occasionally **T-wave inversion** is said to be the first visible ECG change of acute MI. This tends to persist longer than ST-elevation, yet it is still usually only a transient change. T-wave inversion that occurs without subsequent formation of Q-waves is typical of sub-endocardial infarction (non-Q-wave infarction).

The final ECG change associated with acute MI is the appearance of **pathological Q-waves**. These are broad (>1 mm), deep (>2 mm) negative deflections that start the QRS complex. They occur physiologically in aVR, I and III. Pathological Q-waves develop over hours or days, and are the hallmark of transmural infarct. They reflect electrical silence of infarcted cardiac tissue, causing a window through which the normal endo- to epicardial activation of the opposite, non-infarcted ventricular wall is 'seen'. They are **almost always permanent**.

**[4] (a) F (b) F (c) T (d) T (e) T**

The complications of myocardial infarction can be thought of under five main headings . . .

**1. Further chest pain**

- A bruised sensation and musculoskeletal pain are common within the first 48 h, especially if the patient has undergone CPR or repeated attempts at DC cardioversion
- **Pericarditis** may develop in the first 1–3 days post-MI; it is commoner with full thickness infarcts and may cause an audible rub ('like walking in fresh-laid snow'); treat with high dose aspirin
- **Infarct extension** may occur; look for further ST-elevation; treat with repeated thrombolysis or urgent coronary angioplasty
- It may simply be **post-infarction angina**; this usually develops within 10 days of the acute episode and can be treated with standard medical therapy, i.e. nitrates,  $\beta$ -blockers and  $\text{Ca}^{2+}$ -channel antagonists

**2. Fever**

- Fever commonly peaks 3–4 days post-MI and is due to **myocardial necrosis**
- Other causes of fever must be considered, e.g. infection, thrombophlebitis, venous thrombosis, drug reaction, pericarditis, etc.
- **Dressler's syndrome** occurs weeks or even months after MI and consists of the triad of pericarditis, fever and pericardial effusion; it is actually an **autoimmune response** to the damaged myocardium; it may necessitate administration of anti-inflammatories. → (c)

**3. New systolic murmur (four to consider)**

- **Pericardial friction rub** secondary to inflammation of the infarcted myocardium
- **Long-standing murmur** that was missed at presentation
- **Ventricular septal defect**: this classically occurs 5–10 days post-MI and presents as sudden collapse, pulmonary oedema and hypotension; usually diagnosed on echo, where colour flow shows left to right flow across the septum; requires emergent surgical repair
- **Acute mitral regurgitation** occurs 2–10 days post-MI due to infarction and rupture of papillary muscle; can cause torrential regurgitation and sudden death, but if the patient survives, emergent surgical repair is vital

**4. Post-MI arrhythmias (four to consider)**

- **Sinus bradycardia** is common, especially in inferi-

or or posterior MI; treated first with atropine, then with electrical pacing

- **Atrioventricular blockade** is also common; if the infarction is an inferior one, the blockade is often temporary and does not require treatment; however, if the infarction is an anterior one and 2nd or 3rd degree block/bifascicular block arises, then prophylactic pacing is indicated
- **Ventricular ectopics** post-MI have a poor prognosis as they frequently herald the development of ventricular tachycardia or fibrillation; there is no evidence that treatment of these alters prognosis, but it may be prudent to correct  $\text{PaO}_2$ ,  $\text{K}^+$  and  $\text{Mg}^{2+}$ ; if ectopics continue, a magnesium sulphate infusion may be set up
- **Ventricular tachycardia** may arise, and it should be treated in the usual way ( $\text{Mg}^{2+}$ , lidocaine, amiodarone, synchronized DC shock). VT in the first 24 h post-MI has a less sinister prognosis than VT arising later in the post-MI course. Should ventricular fibrillation develop from VT, prompt DC cardioversion should be instigated

**5. Hypotension and shock post-MI**

- Common in large MIs; treatment is with *cautious* plasma volume expansion (i.e. 100–200 mL colloid over 10 min), and then inotropes if the BP should remain low despite adequate filling pressures

Other complications of MI worth considering are **thromboembolism** secondary to prolonged inactivity or cardiac mural thrombus, **left ventricular aneurysm formation** (think persistent ST-elevation), heart failure and, of course, death.

- Sheehan's syndrome is pituitary necrosis due to circulatory collapse following severe post-partum bleeds, and is nothing to do with MI → (a)
- Acute aortic regurgitation is not a complication of acute MI. It happens more often in dissecting aortic aneurysms or with valve destruction associated with the vegetations of bacterial endocarditis → (b)
- Electromechanical dissociation (EMD) occurs when the ECG is relatively normal and yet there is no mechanical activity of the heart (*normal ECG but no output*) → (d)

The causes of EMD can be grouped into the **4 Hs** and the **4 Ts** . . .

<b>H</b> Hypothermia	<b>T</b> Tension pneumothorax
<b>H</b> Hypoxia	<b>T</b> Thromboembolism (pulmonary)

- H Hypovolaemia T Tamponade (cardiac)  
 H Hypo- and T Toxins (i.e. drugs)  
 hyperkalaemia

**[5] (a) F (b) F (c) F (d) F (e) T**

**(a)** Inferior infarcts cause ST-segment **elevation** in leads **II, III and aVF**. Remember: infarction causes elevation, ischaemia causes depression. Inferior infarcts usually reflect occlusion in the RIGHT coronary artery or one of its branches.

**(b)** Pericarditis causes ‘**saddle-shaped**’ ST-segment **elevation** in all leads of the ECG. Downward-sloping ST-segment depression indicates myocardial ischaemia, whereas upward sloping ST-segment depression is non-specific.

**(c)** Atrial fibrillation causes the ECG to show fine oscillations of the baseline (**F-waves**) and **no clear P waves at all**. The QRS segments occur in an irregularly irregular fashion, and at a rapid rate. The causes of AF can be recalled by remembering that **PIRATES** were often dehydrated after long spells at sea . . .

Dehydration

- P Pulmonary disease  
 I Ischaemia, infarction  
 R Rheumatic heart disease  
 A Anaemia, atrial myxoma  
 T Thyrotoxicosis  
 E Ethanol  
 S Sepsis

**(d)** First-degree heart block is manifest on the ECG as **simple prolongation of the PR-interval**. In health, the PR-interval should be between three and five small squares on the ECG, i.e. 0.12–0.2 s. If it is longer than this, then first-degree heart block is present. NB In first-degree heart block, the **PR interval is constant** even though it is prolonged, and **a QRS complex follows every P-wave**. Broad QRS complexes suggest a rhythm of ventricular origin OR a supraventricular rhythm with concomitant bundle branch block.

**(e)** Ventricular aneurysm characteristically produces persistent ST-segment elevation in all leads for greater than 1 month’s duration.

**[6] (a) F (b) F (c) F (d) T (e) F**

**(a)** Flow rate within a vessel is, in fact, governed by **Poiseuille’s law**, which states that the flow within a vessel is proportional to the **fourth power** of the radius. The Fick principle is a means of indirectly calculating cardiac output by dividing total body oxygen

consumption by the difference in oxygen content of arterial and mixed venous blood. It is commonly used in operating theatres to monitor cardiovascular parameters.

**(b)** Claudication means **limping**, being derived from the Latin *claudicatio*, to limp.

**(c)** Leriche syndrome is **aortoiliac occlusive disease**, which causes absent femoral pulses, lower extremity claudication, wasting of buttock muscles and impotence. It can be caused acutely by a ‘saddle embolism’ at the bifurcation of the aorta, but is more commonly seen in the chronic setting of slowly progressive atherosclerosis.

**(d)** Rest pain indicates advanced peripheral vascular disease. It occurs most commonly in the **toes and metatarsal heads** on lying down at night. Temporary relief can be obtained by dangling the legs over the side of the bed, or by standing up and walking, because this makes the feet dependent, increasing gravitational hydrostatic pressure, increasing venous pressure and so temporarily enhancing oxygen delivery. Unlike claudication, which is caused by muscle ischaemia, rest pain is caused by **nerve ischaemia**, and it often corresponds to an **ankle:brachial pressure index (ABPI) of <0.4**. It should be differentiated from benign nocturnal cramps, which usually occur in the calf and are not associated with impaired blood flow.

**(e)** **Arterial ulcers** arise due to arterial insufficiency. They tend to occur **on the toes, heel or dorsum of the foot** in response to minor trauma and are **exquisitely painful**. They have a **punched-out** appearance and a **pale necrotic base**.

**Venous ulcers** usually occur at the **medial or lateral malleolus** in response to venous pooling. They often cause a brownish discoloration of the skin (haemosiderin deposition), have a **granulating base** and whilst **painful**, do not cause anywhere near as much pain as arterial ulcers. They are associated with significant oedema. Treatment is usually conservative.

**Neuropathic ulcers** are **painless**, and usually located on the **plantar or lateral aspects of the foot**. They are the direct result of diabetic neuropathy and are often associated with destruction of the ankle joint (Charcot’s foot). Because of the nature of diabetes, these ulcers often co-exist with arterial ulceration.

**[7] (a) F (b) T (c) F (d) T (e) F**

**(a)** Aortic stenosis is heard best in the **aortic area** (2nd right intercostal space) with the patient **leaning forward** and the **breath held in end-expiration** (‘breathe in,

breathe out, hold it'). Aortic regurgitation is best heard at the left sternal border with the patient sitting forward in end expiration.

**(b)** Aortic stenosis causes isolated hypertrophy of the left ventricle. The left atrium does not become enlarged or hypertrophied because the mitral valve is intact (unless there is mixed valvular disease).

**(c)** A water-hammer pulse describes a pulse with a **forcible impulse but immediate collapse**, and is characteristic of aortic regurgitation. It is also known as a **collapsing** pulse. The pulse character associated with aortic stenosis is **slow-rising** because of the resistance encountered by the left ventricle when pushing blood past the stenosed valve.

The character of pulsation may also be described in the following ways . . .

- *Pulsus paradoxus*: an **exaggeration of the normal tendency of the systolic pressure to fall on inspiration** (should be <10 mmHg in health, but is greater than this in pulsus paradoxus); characteristically seen in **severe airway obstruction** (e.g. acute severe asthma), cardiac tamponade and constrictive pericarditis.
- *Pulsus alternans*: alternating **weak then strong** (but regular) beats; characteristic of **severe myocardial failure**, indicating very poor prognosis.
- *Pulsus bigeminus*: **ectopic beats follow each sinus beat**, causing beats to occur in pairs; rhythm is not regular.
- *Pulsus bisferiens*: an arterial **pulse with palpable, separated peaks**; characteristic of **hypertrophic, obstructive cardiomyopathy (HOCM)** and when aortic stenosis co-exists with aortic regurgitation.

**(d)** The murmur caused by aortic stenosis is a **diamond-shaped ejection systolic murmur**, best heard in the aortic area. It is said to be **rough** in quality, and is longer the more severe the stenosis. It **radiates to the carotids**. A systolic **ejection click** may be heard when the valve opens if it is calcified and has become immobile (severe disease). There may be a **prominent fourth heart sound (S4)** if the left ventricle is hypertrophied and has become stiff.

**(e)** Aortic stenosis alone is not associated with the development of AF as long as the mitral valve remains intact.

**[8] (a)T (b)F (c)T (d)F (e)F**

**(a)** Dehydration and hypovolaemia, whether acute or chronic, are common causes of atrial fibrillation, especially in hospital patients.

**(b)** Thyroid eye signs (i.e. retro-orbital swelling, proptosis, exophthalmos, limitation of eye movements, visual impairment due to optic nerve pressure) are **only seen in Graves' disease**. All causes of hyperthyroidism, however, may cause atrial fibrillation, because it is the high level of circulating thyroid hormone and *not* the high levels of IgG (thyroid stimulating antibody) seen in Graves' that is responsible for precipitating atrial fibrillation.

**(c)** Because there is no ordered contraction of the atria in AF (i.e. electrical activity does not start at and is not propagated from the sino-atrial node), there are no P-waves visible on the ECG.

**(d)** Anti-dysrhythmic agents are traditionally classified into four groups according to the **Vaughan-Williams classification** . . .

- Class 1: Membrane depressants
  - Class 1a: disopyramide, quinidine
  - Class 1b: lidocaine
  - Class 1c: flecainide, propafenone
- Class 2: Antisymphathetics
  - e.g.  $\beta$ -blockers, the cardioselective (i.e.  $\beta$ -1 specific) ones being metoprolol and atenolol
- Class 3: Prolongers of the action potential
  - e.g. amiodarone, sotalol
- Class 4: Slowers of conduction in nodal tissue
  - e.g. the non-dihydropyridine calcium channel antagonists—verapamil, diltiazem

Class 1c agents (like flecainide) and all other class 1 anti-dysrhythmics should only really be used in the treatment of **intractable or life-threatening arrhythmias**; they should *never* be used where the patient has significant left ventricular dysfunction (e.g. heart failure), prior history of MI or acute coronary ischaemia.

**(e)** Treatment of AF can be approached in two ways:

1. *Control the ventricular rate and ignore what the atria are doing*: this is usually achieved using an **AV-nodal blocking drug** (e.g. digoxin,  $\beta$ -blockers or verapamil).
2. *Cardiovert in an attempt to stop the atria fibrillating*: this can be performed **electrically** (80% of patients convert to sinus rhythm with synchronized DC cardioversion) or **medically** (with an IV infusion of class 1a, 1c or 3 drug, usually amiodarone). Recurrent paroxysms can be prevented with prophylactic oral treatment with a class 1a, 1c or 3 drug, again usually amiodarone.

Amiodarone is one of a select number of drugs whose

side effect profile should be known for undergraduate final examinations . . .

- Sensitivity to sunlight (i.e. phototoxicity)
- Corneal microdeposits
- Slate-grey skin discolouration
- Peripheral neuropathy
- Hypo- or hyperthyroidism
- Pulmonary fibrosis (irreversible)

**[9] (a)T (b)F (c)T (d)F (e)T**

**(a)** The right main bronchus is **wider, shorter and more vertical** than the left; this is why a swallowed foreign object or aspirated material is more likely to impact in the lower lobe of the right lung rather than the left. Material aspirated when a patient is lying flat is most likely to impact in the middle lobe of the right lung.

**(b)** The lung has a **dual blood supply**. The bronchi, connective tissue of the lung and the visceral pleura receive their blood supply from the **bronchial arteries**, which are branches of the descending aorta. The alveoli on the other hand receive deoxygenated blood from the terminal branches of the **pulmonary arteries**.

**(c)** The epithelial lining of the alveoli is mainly made up of type 1 pneumocytes. These form a **thin barrier for gas exchange**. They are, however, derived from **surfactant-producing** type 2 pneumocytes, which are slightly more in number but cover less surface area.

**(d)** Both lungs have oblique fissures, but **only the right has a horizontal fissure**, hence there are three lobes in the right lung (upper, middle and lower) but only two in the left (upper, lower and the lingula—a redundant segment of pulmonary tissue anterior to the surface of the heart).

**(e)** In health **the strongest stimulation to ventilation is an increase in the arterial partial pressure of CO<sub>2</sub>** (PaCO<sub>2</sub>), which causes an increase in the concentration

of hydrogen ions in cerebrospinal fluid (CSF), stimulating the brainstem respiratory centre. Sensitivity to increasing PaCO<sub>2</sub> can be lost in chronic obstructive pulmonary disease (COPD) due to chronic over-exposure of the brainstem to high levels of CO<sub>2</sub>, and in these patients the chief stimulus to breathe becomes the partial pressure of oxygen (PaO<sub>2</sub>), as detected by the carotid and aortic arch bodies. Patients with chronic type II respiratory failure should therefore not be administered high concentrations of oxygen because it abolishes their respiratory stimulus, and they would quickly have a respiratory arrest. Pyrexia, large doses of aspirin and the drug doxapram may directly stimulate the brainstem respiratory centre, whereas severe hypoxaemia and sedatives, especially opioids, may depress it.

**[10] (a)F (b)F (c)T (d)T (e)F**

On examination of the chest in a patient with a unilateral tension pneumothorax . . .

- Chest wall movements will be decreased on the affected side only → **(a)**
- The mediastinum will be displaced away from the affected side → **(b)**
- The percussion note will be hyperresonant on the affected side → **(c)**
- Breath sounds will be decreased or absent on the affected side → **(d)**
- Tactile vocal resonance will be decreased on the affected side → **(e)**
- There are no added sounds

It is very useful for exams to be exactly sure of the chest signs of the most common respiratory complaints. Recent exams have contained MCQs and EMQs that tested precisely the candidate's knowledge of such signs. A useful table to memorize is shown below, but there is no substitute for having seen/discovered such signs for yourself: find patients with signs and examine them!

<i>Pathological process</i>	<i>Chest wall movement</i>	<i>Mediastinal displacement</i>	<i>Percussion note</i>	<i>Breath sounds</i>	<i>Vocal resonance</i>	<i>Added sounds</i>
Consolidation	Decreased ipsilaterally	None	Dull	Bronchial	Increased	Fine creps
Collapse	Decreased ipsilaterally	Towards	Dull	Decreased or absent	Decreased or absent	None

*Continued on p. 56*

<i>Pathological process</i>	<i>Chest wall movement</i>	<i>Mediastinal displacement</i>	<i>Percussion note</i>	<i>Breath sounds</i>	<i>Vocal resonance</i>	<i>Added sounds</i>
Fibrosis (general)	Decreased bilaterally	None	Normal	Vesicular	Increased	Fine creps
Pleural effusion (>500 mL)	Decreased ipsilaterally	Away	Stony dull	Vesicular (decreased or absent)	Decreased or absent	None
Large pneumothorax	Decreased ipsilaterally	Away	Hyper-resonant	Decreased or absent	Decreased or absent	None
Asthma	Decreased bilaterally	None	Normal	Vesicular, with prolonged expiration	Normal	Expiratory polyphonic wheeze
COPD	Decreased bilaterally	None	Normal	Vesicular, with prolonged expiration	Normal	Coarse creps plus expiratory polyphonic wheeze

**11] (a) F (b) T (c) T (d) F (e) T**

**(a)** COPD describes airways obstruction that occurs **mainly in smokers or ex-smokers**. The clinical distinction between COPD and asthma is blurred, because virtually all patients with COPD have a reversible element to their disease, and is the reason these patients are treated with **bronchodilators** [( $\beta$ -adrenoceptor agonists such as salbutamol (Ventolin<sup>®</sup>) and antimuscarinics such as ipratropium bromide (Atrovent<sup>®</sup>)] and **corticosteroids** (initially prednisolone orally, and if this is successful, wean to inhaled beclomethasone).

**(b)** Chronic bronchitis is defined on the basis of the **history** as ‘**cough productive of sputum on most days for at least 3 months of the year, for more than 1 year**’. This reflects the nature of the underlying condition: hypertrophy of the mucous-secreting glands of the bronchial tree (mainly large bronchi). Emphysema, however, is defined **pathologically** as ‘**dilatation and destruction of the lung tissue distal to the terminal bronchioles**’. Thus, it affects only the small airways, leading to loss of elastic re-

coil of the lungs, decreased gas transfer, expiratory air-flow limitation and air-trapping. Whilst bronchitis and emphysema are described as two separate clinical entities, they usually co-exist in all patients with COPD.

**(c)** Patients with COPD commonly demonstrate asterixis, which is a coarse, flapping tremor due to chronic retention of CO<sub>2</sub>. It is also seen in patients with hepatic encephalopathy (‘liver flap’), although it can occur in a wide variety of metabolic and toxic encephalopathies. CO<sub>2</sub> retention also causes the hands to be warm (vasodilatation) and the pulse to be bounding (hyperdynamic circulation).

**(d)** *Cor pulmonale* is the term used to describe **the process of adaptation and failure the right side of the heart undergoes as a result of lung disease**. Normally the pulmonary vasculature responds to local hypoxia by arteriolar constriction. So, for example, in lobar pneumonia, blood flow to the affected lobe is decreased, minimizing the amount of poorly oxygenated blood reaching the systemic circulation. When the pneumonia

resolves, the vasoconstriction ceases and the flow returns to normal. However, if hypoxia is widespread and irreversible, as is the case in COPD, the resultant arteriolar constriction is equally widespread and irreversible, thus causing pulmonary hypertension. Over time the right atrium and ventricle hypertrophy, eventually the limit of the heart's ability to adapt is reached, and the right heart fails, leading to the typical signs and symptoms of right-sided cardiac failure.

**(e)** Respiratory failure occurs when pulmonary gas exchange is sufficiently impaired to cause **hypoxaemia** ( $\text{PaO}_2 < 8 \text{ kPa}$ ) with or without **hypercapnia** ( $\text{PaCO}_2 > 7 \text{ kPa}$ ). There are two types of respiratory failure.

- **Type I respiratory failure** — 'acute hypoxaemic'
  - mainly due to ventilation–perfusion mismatch
  - $\text{PaO}_2$  low ( $< 8 \text{ kPa}$ )
  - $\text{PaCO}_2$  normal or low ( $< 5 \text{ kPa}$ )
  - Common causes include pulmonary oedema, pneumonia, ARDS, pulmonary embolism (PE)
- **Type II respiratory failure** — 'ventilatory failure'
  - $\text{PaO}_2$  low ( $< 8 \text{ kPa}$ )
  - $\text{PaCO}_2$  high ( $> 7 \text{ kPa}$ )
  - Occurs when alveolar ventilation is insufficient to excrete the volume of  $\text{CO}_2$  being produced by tissue metabolism
  - Due to reduced ventilatory effort, inability to overcome increased resistance, failure to compensate for increased dead space or  $\text{CO}_2$  production, or a combination of these
  - Most common cause is COPD
  - Other causes include chest wall deformity, respiratory muscle weakness [e.g. Guillain–Barré syndrome, motor neurone disease (MND)] and depression of the respiratory centre (e.g. opioid excess)

**[12] (a) F (b) F (c) T (d) F (e) F**

- (a)**  $\text{PaCO}_2$  is not elevated in type I respiratory failure.
- (b)** The commonest cause of type II respiratory failure is COPD. Pneumonia tends to cause type I respiratory failure.
- (c)** Pulsus paradoxus is explained in the answers to MCQ [7], above.
- (d)** Respiratory failure **does not always present with dyspnoea**, e.g. opioid overdose, Guillain–Barré syndrome. Chronic retainers of  $\text{CO}_2$  may also present drowsy and not obviously dyspnoeic.
- (e)** The CXR described in the question is one of classical heart failure. The CXR in respiratory failure is one of so-

called **non-cardiogenic pulmonary oedema**, where the typical features are no upper lobe venous distension, no septal lines or pleural effusion, a normal heart size and the most reliable sign, which is **peripheral alveolar shadowing and/or an air bronchogram**. The CXR in someone with suspected respiratory failure should also be checked carefully for pneumothorax, consolidation, bronchogenic cancer and oligaemia or wedge-shaped infarcts (i.e. signs of PE). Localized oligaemia on CXR is also known as 'Westermarck's sign', and is usually indicative of PE.

The main causes of respiratory failure can be remembered if you remember that **A DIMPLE can cause respiratory failure . . .**

- A Asthma, ARDS, aspirin
- D Drugs (opiates)
- I Infection (pneumonia)
- M Metabolic acidosis (e.g. diabetic ketoacidosis)
- P PE, pneumothorax
- L Left ventricular failure
- E Effusions

**[13] (a) F (b) F (c) F (d) T (e) F**

**(a)** Asthma is characterized by chronic inflammation of the airways which normally presents with a classical triad of symptoms . . .

1. **Wheeze**
2. **Nocturnal cough**
3. **Dyspnoea**

Within the airways themselves, it has three main characteristics: **bronchial inflammation** (mainly involving eosinophils and mast cells with associated plasma exudation, oedema, smooth muscle hypertrophy and mucus plugging), **airways hyper-responsiveness** and **reversible airflow limitation**. It is important to realize, however, that **with chronic asthma, the airflow limitation does become irreversible to a certain degree**. It is also the case that aspects of the acute severe asthma that continues to kill young people are also irreversible (otherwise no-one would die from asthma as long as they were treated with the correct medications).

- (b)** Typical asthma attacks occur **after the conclusion of, and not during, exercise**. Cold, dry air cools and dries the epithelial lining of the bronchi, precipitating attacks.
- (c)** ACE-inhibitors are safe to give to asthmatic patients. ACE-inhibitors *should never*, however, be given to patients with **renal artery stenosis** as they block production of angiotensin II and aldosterone, leading to a decrease in the already threatened renal perfusion, and

[24] (a) F (b) F (c) F (d) F (e) F

(a) The commonest cause of small bowel obstruction worldwide is **herniae**, whilst the most common cause in industrialized countries is **postoperative adhesions**.

(b) This is the description of an **intussusception**. A volvulus occurs when a length of large bowel becomes twisted on itself, using the mesentery as the axis about which it twists.

(c) In gallstone ileus, a large (>2.5 cm) stone passes from gallbladder or common bile duct into the GIT **through a fistula**. The stones usually lodge in the terminal ileum.

(d) Bowel sounds are characteristically infrequent, high-pitched and 'tinkling' in small bowel obstruction. They would be absent in paralytic ileus.

(e) In paralytic ileus, there is characteristically **no physical obstruction** to passage of the luminal contents of the bowel, i.e. it is **functional**. Causes of paralytic ileus include . . .

- Sepsis (either within or adjacent to the peritoneal cavity, e.g. pneumonia)
- Drugs (e.g. opiates)
- Retroperitoneal haematoma
- Electrolyte abnormalities (e.g. hypokalaemia)
- Physical inactivity
- Laparotomy

Paralytic ileus classically demonstrates **absent bowel sounds**, and may be definitively diagnosed from physical obstruction by contrast studies and a history of less abdominal pain.

## EMQs

### Answer 1

#### (1) Mitral stenosis with atrial fibrillation

On examination the patient may have a **malar flush** and be in **atrial fibrillation**. There may be evidence of a cerebrovascular accident (hemiplegia, facial weakness). An **opening snap** is followed by a '**rumbling**' mid-diastolic murmur, localizing (often very localized) over the '**tapping**' apex beat (palpable first heart sound). Listen with the patient in the left lateral position with the bell of the stethoscope in several positions over the apex. If difficult to hear, the murmur may be accentuated by asking the patient (if this is possible) to sit up and lie down five times so raising the cardiac output. There is **pre-systolic accentuation only if the heart is in sinus rhythm** and is due to atrial systole.

In addition if **pulmonary hypertension** has developed there may be a . . .

- **Left para-sternal heave** (right ventricular hypertrophy)
- **Loud second heart sound** over the pulmonary area
- Possible murmur of **pulmonary regurgitation** (Graham Steell)
- Murmur of **tricuspid regurgitation** [systolic murmur at lower left sternal edge plus large 'V' waves in the JVP (jugular venous pulse)]
- **Right ventricular failure** (elevated JVP, hepatomegaly, ascites and peripheral oedema)

These findings will also be seen in pulmonary hypertension due to other causes (e.g. congestive cardiac failure, COPD, chronic lung pathology, primary pulmonary hypertension).

#### **Causes of mitral stenosis**

- Rheumatic fever (a common cause)
- Congenital
- Calcification

#### **Complications (often the patient is asymptomatic)**

- Recurrent chest infections (wet lungs)
- Atrial fibrillation and embolization (stroke)
- Pulmonary hypertension and pulmonary oedema
- Right ventricular failure
- Tricuspid regurgitation
- Bacterial endocarditis

#### **The severity of the mitral stenosis is indicated by:**

- Signs of pulmonary hypertension (see above)
- Close proximity of the opening snap after the second heart sound
- The length of the mid-diastolic murmur (directly proportional, however, very tight lesions lead to quiet murmurs)

**ECG findings**

- Atrial fibrillation
- P mitrale (bifid 'M' shaped P waves best seen in lead II)

**CXR appearances**

- Prominent left atrium may be the only abnormality
- Calcification of the valve ring
- Possible features of congestive cardiac failure
  - Kerley-B lines
  - Upper lobe venous diversion
  - Bilateral effusions
  - Cardiomegaly
  - 'Bat's wing' oedema spreading from the hilar areas

**Management**

- Diuretics for symptoms of breathlessness
- Anticoagulation and digoxin if atrial fibrillation develops
- Prophylactic antibiotics for 'dirty' procedures to prevent valve infection (dental procedures or pelvic surgery). Check the British National Formulary for the current recommendations
- Surgical relief of stenosis if secondary pulmonary hypertension develops
- Open surgery can be avoided in those unfit for a general anaesthetic by percutaneous *trans*-septal balloon valvotomy. Here a balloon catheter is passed via a peripheral vein into the right atrium. The atrial septum is then perforated and the balloon is passed across the mitral valve and inflated. Mitral regurgitation is an inevitable consequence. Patients most suitable for this approach include those with:
  - Minimal mitral regurgitation
  - Mobile valves (no calcification)
  - Minimal subvalvular disease

Patients more suitable for formal valve replacement include those with:

- Significant mitral regurgitation
- Extensive valve calcification
- Thrombus in the left atrium

**(2) Mitral regurgitation**

The patient may be in **atrial fibrillation** (left atrial dilatation). The **JVP** may be elevated if associated with right ventricular failure (secondary to pulmonary hypertension). The **apex is displaced and 'thrusting'** (a dynamic low-pressure movement indicating volume overload). The **first heart sound is soft**. There is a **pan-systolic murmur heard at the apex radiating to the left axilla**.

There may be a **third heart sound** (volume overload) and a diastolic flow murmur due to the re-entry of the regurgitated blood back into the left ventricle.

Clinically the main **differential** will be aortic stenosis causing an ejection systolic murmur, a ventricular septal defect or tricuspid regurgitation which both cause pan-systolic murmurs.

Tricuspid regurgitation, however, will result in prominent V waves in the JVP, a murmur that *increases* during inspiration and possible hepatic pulsations.

**Causes of mitral regurgitation**

- Myocardial infarction (acute presentation following an acute MI due to rupture of the chordae tendineae)
- Rheumatic fever (acute or old)
- Left ventricular dilatation
  - Severe hypertension
  - Left ventricular failure
  - Cardiomyopathy
- Bacterial endocarditis
- Connective tissue diseases
  - SLE
- Elastic tissue disorders
  - Marfan's syndrome
  - Ehlers–Danlos syndrome
- Prolapsing mitral valve; 'floppy mitral valve'. This occurs in young women. Embolic events and atypical chest pain (localized over the apex) may occur. A mid-systolic click is heard.

The **severity of mitral regurgitation** can be assessed by the presence of a . . .

- Third heart sound
- Mid-diastolic flow murmur
- Left ventricular enlargement
- Thrill

**(3) Mitral stenosis**

Here the patient is not in atrial fibrillation so there is pre-systolic accentuation of the murmur.

**(4) Aortic regurgitation with left ventricular failure**

The patient has a **wide pulse pressure** on measuring the blood pressure. The **pulse is collapsing** when the left brachial artery palpated with arm elevated. The **apex beat is displaced** (state the location) and is **thrusting** in character, indicating volume overload.

With the **patient sat forward** and the breath held in end expiration, a short early **decrescendo murmur radi-**

**ating down the left sternal edge** is heard (listen in several positions down the left sternal edge with the **diaphragm**). **The longer and louder the murmur, the more severe the regurgitation.**

In this case, left ventricular failure is suggested by the elevated JVP, displaced apex and bi-basal crepitations. There may be an additional ejection systolic murmur in the aortic area due to increased flow across the aortic valve. The aortic regurgitant jet can cause interference with the anterior mitral valve cusp resulting in a mid-diastolic murmur to be heard at the apex (Austin Flint murmur).

Inspect the patient for Marfan's body habitus, ankylosing spondylitis (kyphosis of the spine) features of rheumatoid arthritis and the small, irregular Argyll Robertson pupils (which react to accommodation but not to light) of syphilis.

#### **Causes of aortic valve regurgitation**

- Aortic root dilatation
  - Severe hypertension
  - Connective tissue disorders:
    - Marfan's syndrome
    - Rheumatoid arthritis
    - Ankylosing spondylitis
  - Aortic aneurysm
  - Dissection of the aorta
  - Syphilis
- Valve disruption
  - Congenital (e.g. bicuspid valves)
  - Rupture of the sinus of Valsalva
  - Endocarditis
  - Rheumatic fever
  - Ventricular septal defect (loss of support)

#### **Associated eponymous signs**

- **Corrigan's sign:** vigorous arterial pulsations in neck
- **De Musset's sign:** head nodding with arterial pulsations
- **Quinke's sign:** visible nail bed capillary pulsations. Partially compress the nail bed and look for pulsations in the subungual capillaries
- **Durozier's sign:** partially compress the femoral artery in the inguinal area with a digit and listen proximally for a regurgitant murmur as blood turbulently flows back under the 'stenosis' during diastole

Patients with aortic regurgitation may have few symptoms until left ventricular failure occurs. It is important

to replace the valve before irretrievable ventricular dysfunction occurs as indicated by an enlarging heart on echocardiography. Once the end systolic diameter of the left ventricle has reached 55 mm, then surgery is recommended.

#### **(5) Aortic stenosis**

This patient has a **slow rising pulse** and a blood pressure that has a **narrow pulse pressure**. The apex is displaced and **heaving** indicating pressure overload. There may be a **thrill** felt with the ulnar border of the palm over the aortic area (indicating at least a grade 4 murmur and often reflects a pressure gradient across the valve of at least 40 mmHg). An **ejection click** may be heard over the left sternal border indicating the stenosis is occurring from the valves (and not subvalvular or supra-valvular in origin) and is due to opening of the valve. A click is especially heard in bicuspid aortic stenosis. Note that clicks may also be heard in pulmonary stenosis or with a prolapsing mitral valve.

An **ejection systolic (rough, sawing, diamond shaped) murmur** is heard all over the precordium, but especially in the aortic area and possibly radiating to the carotids. The **second heart sound is quiet**. The differential diagnosis includes mitral regurgitation, hypertrophic obstructive cardiomyopathy and aortic sclerosis.

Aortic sclerosis is simply a noisy valve due to calcification frequently seen in the elderly, and has no abnormal increase of the pressure gradient across the valve. Thus in aortic sclerosis the patient has a normal pulse character, a normal pulse pressure (blood pressure) and a normal apex beat position and character.

#### **Clinical features of aortic stenosis**

- Palpitations due to arrhythmias (ventricular)
- Syncope due to arrhythmias
- Chest pain (angina)
- Dyspnoea (left ventricular failure)
- Sudden death (10–20%)

In aortic stenosis the ventricular function is relatively well preserved, unlike in aortic regurgitation. However with significant stenosis exercise fails to improve cardiac output leading to ischaemia and cardiac arrhythmias. For this reason exercise tolerance tests are avoided in these patients. In patients with aortic stenosis **symptoms are a good indication of the disease severity.**

Aortic stenosis is especially prone to bacterial endocarditis as it is a high-pressure valve lesion.

**Causes of aortic stenosis**

- Valvular
  - Rheumatic fever
  - Calcification
  - Congenital (e.g. bicuspid valve)
- Supravalvular
  - William's syndrome (supravalvular aortic diaphragm, mental retardation and hypercalcaemia)
- Subvalvular
  - Diaphragm below the aortic valve
- Septal
  - Hypertrophic obstructive cardiomyopathy

**The severity of aortic stenosis is indicated by:**

- A narrow pulse pressure on blood pressure recording
- Reverse splitting of the second heart sound (i.e. pulmonary before aortic)
- A soft second heart sound
- A fourth heart sound (indicating ventricular stiffness due to left ventricular Hypertrophy)
- A thrill
- A heaving apex beat

The ECG may show signs of **left ventricular hypertrophy** (R wave in V6 plus S in V2 summate to more than 35 mm) plus signs of **ventricular strain** (ST depression and T wave inversion) in the leads, which look at the left ventricle (leads I, AVL, II, V5 and V6).

The **chest radiograph** may show **post-stenotic dilatation**. There may be **calcification** of the aortic valve. Alternatively there may be feature of **congestive cardiac failure**.

In the **management**, valve replacement is indicated for all patients with symptoms and where the pressure gradient across the valve exceeds 50 mmHg. Balloon valvotomy is reserve for those unfit for surgery or as a temporary measure. Antibiotic prophylaxis is required for surgery.

**Answer 2**

Know the features for patients with a serious cause for headaches:

**History alarm features**

- **Morning headache:** improving after getting up
- **Morning vomiting**
- **Pain exacerbated by coughing, straining or bending:** note that this occurs in many headaches to a degree, so

avoid using a leading question when asking about exacerbating factors

- **Progressively getting worse**
- **No 'days off':** relentless daily symptoms suggest a possible serious cause
- **New onset** in middle life (i.e. uncharacteristic, no previous history of headaches)
- **Risk factors for intracranial bleeding:** e.g. anticoagulation, alcoholism, old age
- **Trauma:** recent falls/head injury (frequently the trauma is forgotten)

**Examination alarm features**

- Any reduction in the **Glasgow Coma Scale**
- **Confusion** (reduction in abbreviated mental test scoring)
- **Focal neurology:** motor/sensory/coordination/visual/meningeal irritation
- **Seizures**
- **Purpuric rash**
- **Signs of meningeal irritation**
- **Fever**
- **Papilloedema**
- **Severe hypertension**

The **diagnosis of headache is made largely on the history**. A good history will pick out those where there should be heightened concern.

**(1) Migraine**

The features of migraine may be 'classical' in (only) 20%, presenting with a prodrome of visual auras (e.g. flashing lights or zig-zag lines due to cerebral vasospasm) often taking a stereotypical pattern for a given patient. This is followed by a severe unilateral headache with photophobia and phonophobia (intolerance of noise). Nausea and abdominal pain may occur along with focal neurology, e.g. dysphasia, hemiplegia, sensory symptoms.

However, some of these features may be lacking in 'common migraine' and differentiation from tension headaches (the main differential) can be difficult. The approach to first-line treatment is with paracetamol or ibuprofen (or both) used together with an anti-emetic, as gastric stasis during migraine can impair absorption. Codeine is avoided due to its predisposition to cause 'analgesic' headache, in addition to the risk of dependency and side effects such as constipation and drowsiness. Asking about triggers (food, stress, menstruation) and avoidance of these is helpful. Note that there is often a family history in migraine sufferers.

**(2) Benign intracranial hypertension**

A classical history. The problem is a recirculation disorder of CSF. There is no focal neurology and the CT scan is normal without ventricular dilatation. Prominent papilloedema is found on examination. Enlargement of the blind spot on testing the visual fields occurs; if untreated, blindness can occur. A CT scan is required, however, to exclude mass lesions and other causes of raised intracranial pressure. Treatment involves stopping any causative drugs (e.g. steroids or oral contraceptives), weight loss and thiazide diuretics. Repeated lumbar puncture and shunt operations are second line therapy.

**(3) Cluster headache**

Typically a pattern of recurrent headaches for a few days or weeks followed by temporary remission occurs. 20% of cases are chronic. The male:female ratio is 5:1. The exact cause is unknown; however, alcohol may precipitate an attack. Sufferers are more commonly smokers. The episodes may be brief (up to an hour) but are recurrent in bouts, often with periods of remission lasting several months. Severe pain occurs behind the eye. A partial Horner's syndrome may occur along with vasomotor dysfunction causing lacrimation and nasal discharge. The treatment is similar to migraine and 5HT (serotonin) antagonists (e.g. sumatriptan) are often helpful. In addition 100% oxygen (which may abort attacks), verapamil, lithium or methysergide are alternatives. Treatment can be very difficult (like some migraine).

**(4) Space occupying lesion**

This is a worrying history. The progression of focal neurology and seizures suggest an expanding mass lesion. The exact neurological presentation depends on the location; however, common patterns of presentation include;

- Features of rising intracranial pressure (headache, vomiting)
- Neurological deficit (see patterns below)
- Epilepsy

Oedema around the lesion may exaggerate the picture.

An urgent CT scan is indicated. Remember the approach to neurology: 'Where is the lesion? Then, what is the lesion?'

**Localizing mass lesions**

- Frontal lobe
  - Personality change
  - Disinhibition

- Memory/intellect deterioration
- Primitive reflexes return (Plantar response, grasp and suck reflex)
- Expressive dysphasia (Broca's area)
- Anosmia
- Parietal lobe
  - Apraxias (learned movements).
  - Asteriognosis (shape recognition)
  - Homonymous inferior quadrantanopias
  - Extinction (sensory inattention for stimuli on the affected side when both sides stimulated)
- Temporal lobe
  - Receptive dysphasia (Wernicke's area)
  - Memory loss
  - Cortical deafness
  - Homonymous superior quadrantanopias
- Occipital lobe
  - Cortical blindness
  - Homonymous hemianopias

**Causes of cerebral mass lesions**

- Tumour
  - Secondary > Primary lesions
  - Malignant > Benign
- Abscess
  - Pneumonia
  - Skin sepsis
  - ENT sepsis
- Haemorrhage
  - Extradural haemorrhage
  - Subdural haemorrhage
  - Subarachnoid haemorrhage
- Granulomatous
  - Cerebral sarcoidosis
- Vascular
  - AV malformations
  - Hydrocephalus

**(5) Acute bacterial meningitis**

From the given history it may seem difficult to separate viral from bacterial meningitis. However, the lack of any viral prodromal illness and the importance of not missing acute bacterial meningitis, make this the best answer. The main other differential diagnoses include encephalitis, subarachnoid haemorrhage, severe migraine, cerebral abscess, subdural and extradural haemorrhage.

**Immediate management** includes **antibiotics**, which should not be delayed awaiting investigations or even admission to hospital as this is associated with an adverse

outcome. Penicillin V 1.2 g should be given immediately. A CT scan should *ideally* be performed before a lumbar puncture ideally to exclude raised intracranial pressure and other differential diagnoses. If meningococcal disease is suspected a lumbar puncture is avoided due to the high risk of coning because of the prevalence of associated cerebral oedema. Blood cultures (several sets) should be taken. The organism can also be looked for on PCR of blood or CSF. General resuscitation measures (IV fluids and oxygen) are required for septic shock, as well as close monitoring and support usually in intensive care. Despite optimum management overall mortality is still around 10%. Early recognition of symptoms is therefore vital.

#### **Presentation of acute bacterial meningitis**

- **Headache** (bursting, gradual or abrupt onset)
- **Pyrexia**
- Signs of **meningeal irritation**, e.g. neck stiffness, Kernig's sign
- **Photophobia** (a non-specific feature for meningitis)
- **Rash**: A non-blanching purpuric rash due to disseminated intravascular coagulation (DIC) occurs in meningococcal meningitis only. The lesions may occur anywhere, and the patient should be completely inspected as even a single lesion may be significant
- **Global cerebral dysfunction** (cerebral oedema, toxæmia): confusion/drowsiness/convulsions/vacant staring

- **Focal neurological signs** are possible. Infarction or false localizing signs due to raised ICP (C III, CVI palsy or hemi-paresis)
- **Shock** (septicaemic)
- **Bleeding** (due to DIC) e.g. bleeding from venepuncture sites

Note that many of these features may be absent in certain patients with meningitis and that the disease may present in a non-acute and non-specific manner.

#### **Which blood tests?**

FBC/U and Es/CRP/PT/APTT/FDPs (fibrin degradation products), blood cultures.

#### **Which antibiotics?**

**Penicillin V** (2.4 g i.v. or 1 g i.m. stat if no IV access) and **Cefotaxime** (2 g/8 h i.v.). Usually hospitals will have their own policy.

#### **Why CT scan?**

- Helps exclude other causes, e.g. IC bleed/subarachnoid haemorrhage/cerebral abscess
- Ideally prior to lumbar puncture to exclude raised ICP

#### **Lumbar puncture patterns**

Normal opening pressure: <16 cm manometric height of water.

In general the following apply (for exam purposes):

	<i>Bacterial</i>	<i>Viral</i>	<i>TB</i>	<i>Normal</i>
Appearance	Cloudy	Clear	Clear	Clear
Cells	Neutrophils	Lymphocytes	Lymphocytes	<5/mm <sup>3</sup>
Glucose	<1/3 levels in blood	Normal	<1/2 blood levels	1/2–2/3 blood levels
Protein	>1 g/L	<1 g/L	>1 g/L	0.2–0.4 g/L

### **Answer 3**

#### **(1) Pulmonary embolism**

The diagnosis of pulmonary embolism relies on a combination of the following . . .

#### **An assessment of the clinical symptoms**

- Shortness of breath of abrupt onset (occasionally gradual onset occurs with multiple small emboli)
- Cough

- Haemoptysis
- Chest pain (pleuritic type; a late feature)
- Sudden collapse (cardiac arrest)

#### **An assessment of the risk factors for thromboembolism**

- Plaster casts/immobility\*

\*Major risk factors

- **Postoperative/trauma\*** (hypercoagulable state, dehydration)
- **Previous deep vein thrombosis (DVT)/PE\***
- **Pregnancy\***
- **Poorly (major illness)\***
- Oral contraception/oestrogens
- Obesity
- Smoking
- Carcinoma
- Travel
- Old age (PE is rare under the age of 40 years without risk factors)
- Hereditary thrombophilic states:
  - Protein S deficiency
  - Protein C deficiency
  - Antithrombin III deficiency
  - Factor V Leiden (fails to bind Protein C)
- Secondary to other medical conditions:
  - Nephrotic syndrome (renal loss of anti-thrombin III)
  - Diabetic ketoacidosis (dehydration)
  - Antiphospholipid syndrome (poorly understood mechanism; antibodies to phospholipids involved in coagulation occur, the APTT is prolonged *in vitro* but *in vivo* thrombosis occurs)
  - Homocystinuria
  - Paroxysmal nocturnal haemoglobinuria
  - Behçet's disease (arterial and venous thromboses)

#### **An assessment of the clinical findings**

- Tachycardia (sensitive sign)
- Tachypnoea (sensitive sign)
- Cyanosis
- Chest findings (often normal):
  - Pleural rub
  - Reduced air entry
  - Hypotension
- Clinical evidence of DVT (frequently lacking)

#### **An assessment of basic investigations**

- White cell count; to help exclude infection from the differential diagnosis
- Arterial blood gases; type I respiratory failure pattern (hypoxia with normal or reduced CO<sub>2</sub>)
- ECG
  - Tachycardia is the most frequent finding
  - Right axis deviation
  - The classic S1Q3T3 is far less common
- D-dimer
  - Sensitive but not specific
  - Good for excluding PE if normal
  - Should not be performed if there is strong clinical suspicion
- Chest radiograph
  - Often normal
  - Wedge-shaped infarction (opacity) which may cavitate after several days

#### **Assessing the probability of PE**

This is assessed by scoring the above as follows . . .

- No other likely diagnosis on the basis of the above clinical findings and the basic investigations? → 1 point
- Presence of a major risk factor (as above in bold)? → 1 point

#### **Definitive tests**

- V/Q scan
- Helical CT scanning
- Pulmonary angiography (the most accurate test)

Following the diagnosis of PE anticoagulation is commenced with warfarin by giving a loading dose of 5–10 mg for 2 days, followed by a dose adjusted to give an INR between 2× and 3× normal. Warfarin is continued

---

\*Major risk factors

<i>Action</i>	<i>2 points (high risk)</i>	<i>1 Point (medium risk)</i>	<i>0 points (low risk)</i>
Heparinize?	Yes	Yes	Wait
Arrange definitive tests?	Urgent	Soon	Possibly
Alternative diagnosis?	Possible	Likely	Very likely

for 3–6 months. If the PE is a recurrent episode, consideration is given to lifelong warfarin treatment. Following massive PE with collapse, intravenous thrombolysis is given using rtPA or streptokinase.

### (2) None of these

Tension pneumothorax on the left pushing all the mediastinal structures over to the right. Patients with tension pneumothorax have respiratory compromise, frequently extreme and rapidly progressive as the ‘valve’ type leak causes further compromise with each breath. The impedance of venous return to the right side of the heart leads to reduced cardiac output (tachycardia and hypotension) and an elevated JVP. The management involves urgent release using a green needle followed by insertion of an intercostal drain. (See also OSCE 12, p. 244)

### (3) Left apical lung fibrosis

#### Causes

- Old TB
- Silicosis
- Sarcoidosis
- Aspergillosis
- Extrinsic allergic alveolitis
- Ankylosing spondylitis

#### Differential on CXR of apical opacity

- Pancoast tumour
- TB
- Pneumonia
  - *Klebsiella*
  - Pneumocysti
- Fibrosis (as above)

### (4) Right-sided pleural effusion

Stony dull percussion. Normal respiratory rate and lack of cyanosis makes pneumonia less likely. Know causes of effusions (exudates and transudates), CXR appearances of and basic management. (See OSCE 26, p. 271.)

### (5) Right lower lobe pneumonia

The patient is breathless, cyanosed with evidence of right lower lobe consolidation. Other clinical features might include pyrexia, tachycardia, herpes labialis. The patient might complain of chest pain that is

pleuritic on closer questioning. Diaphragmatic pleural pain may radiate to the epigastrium and cause confusion with an acute abdomen. Pneumonia is an inflammation of the lung. The causes may be divided into . . .

- Infective
  - Bacterial, viral, fungal
- Allergic
  - e.g. allergic bronchopulmonary aspergillosis
- Physical agents
  - Chemical; aspiration of vomit or caustic material
  - Radiotherapy

Remember the right lower lobe as the most likely location of a pneumonia following aspiration.

### (6) Asthma

Beware similar but late acute asthma with respiratory failure and silent chest. Know the blood gases patterns in acute asthma;

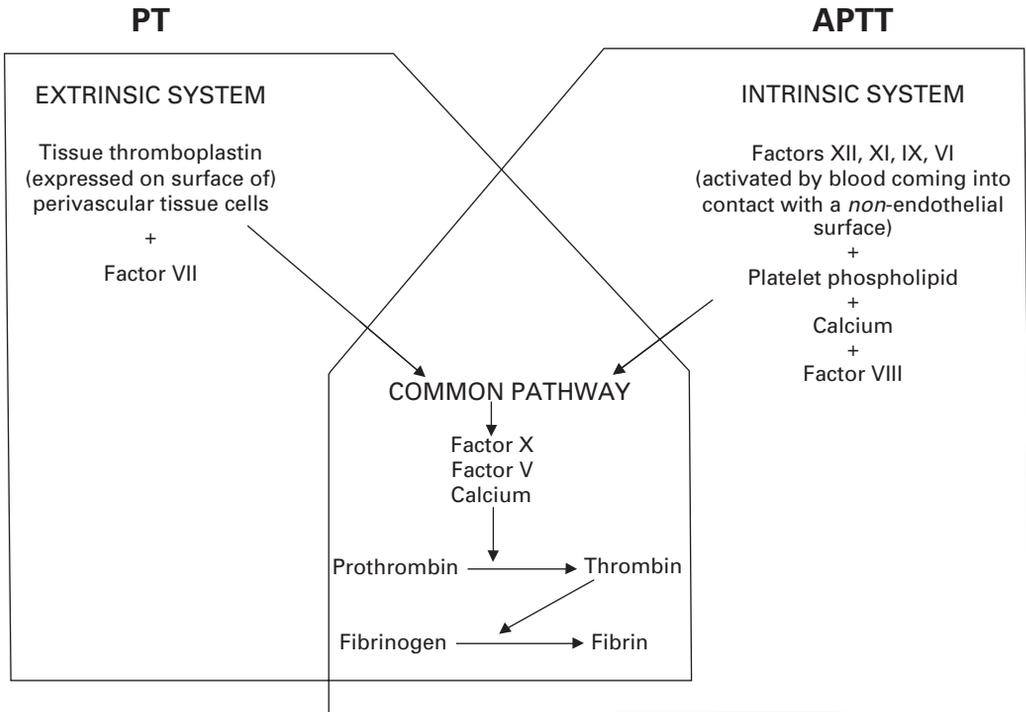
- **Type I respiratory failure early** with low PaO<sub>2</sub> and normal (or even elevated) PaCO<sub>2</sub> progressing to fatigue and eventual;
- **Type II respiratory failure** and rising PaCO<sub>2</sub> and low PaO<sub>2</sub>. In asthma, acidosis is an indication that urgent respiratory support (ventilation on ITU) is needed immediately, so call for anaesthetic help. (See EMQ Answers Q24 (4), p.158 and OSCE 1, p. 219.)

## Answer 4

#### Notes

- **Extrinsic pathway:** utilizes factor VII. Tissue factor initiates coagulation
- **Intrinsic pathway:** utilizes factors VIII, IX and XI. Tissue factor/factor VII complex activates
- **Common pathway:** Factor X dependent
- The **PT** (prothrombin time) measures factors II, V, VII, and X, i.e. the **extrinsic and common pathways**
- **APTT** (activated partial thromboplastin time) measures VIII, X and XI, i.e. the **intrinsic and common pathways**

See diagram overleaf.



- **Vitamin K** is a co-factor required for the gamma carboxylation of factors II, VII, IX and X, and proteins S and C. This is required so that these factors can bind calcium in the normal process of coagulation. The **intrinsic and extrinsic system** will thus be affected by deficiency of vitamin K. Warfarin inhibits the reduction of Vitamin K epoxide to Vitamin K. As Vitamin K is a fat soluble vitamin (along with vitamins A, D and E) deficiency may arise with cholestatic jaundice. Vitamin K deficiency can also affect the newborn (haemorrhagic disease of the newborn) due to poor placental transfer of the vitamin. For this reason vitamin K is given immediately after birth
- **Coagulation disorders produce:**
  - Bleeding into muscles and joints
  - Large bleeds into the skin
  - Small cuts stop spontaneously but larger deeper cuts do not
  - Healing is delayed
- **Platelet disorders produce:**
  - Multiple small areas of bleeding into the skin (purpura/petechiae)
  - Prolonged bleeding of smaller cuts (which depend on platelet adhesion more than coagulation)

- Bleeding into mucus membranes
- Heavy periods
- Prolongation of the bleeding time

Note that the **platelet count must drop below around  $50 \times 10^9$  before any bleeding disorder is likely to occur.**

Therefore:

### (1) Haemophilia A

Prolongation of the APTT with normal PT and bleeding time. Although the inheritance is X linked recessive, one third of haemophilia A cases are spontaneous mutations without family history. There is a range of severity. Factor VIII:C levels less than 5% are associated with spontaneous haemarthrosis and bleeding into muscles. Restoring factor VIII:C levels to 30% of normal is required in the management.

### (2) Haemophilia B, factor IX deficiency (Christmas disease)

As for Haemophilia A, there is prolongation of the APTT, with normal PT and bleeding time. Factor VIII:C levels are normal, however. There is identical inheritance and presentation to Haemophilia A, but the condition is 20% as common as Haemophilia A.

**(3) VonWillebrand's disease**

Deficiency in factor VIII:WF. Three types occur (Types I and II are dominant; Type III is recessive and produces a severe form of the disease). Chromosome 12. The commonest coagulation disorder. VIII:WF binds platelets, so deficiency behaves like platelet problem. Confusingly factor VIII:C (haemophilia A) may (or may not) also be low, with a resultant prolongation of the APTT. However, haemarthrosis is rare. Ristocetin (an antibiotic) normally clumps platelets, but fails to do so in Von Willebrand's disease and so forms the basis of a diagnostic test.

**(4) Warfarin medication**

Although Vitamin K deficiency will produce the same pattern. The raised PT and APTT in this case are suggestive of warfarin therapy given the clinical scenario.

The normal bleeding time counts against liver disease, which causes a coagulopathy via . . .

- Impaired protein synthesis; fibrinogen/coagulation factors
- Malabsorption of fat soluble vitamin K due to cholestasis
- Hypersplenism; consumes platelets
- DIC
- Functional platelet abnormalities. Also seen in uraemia/myeloma/scurvy/antiplatelet drugs/essential thrombocythemia/leukaemia/congenital conditions

**(5) Easy bruising syndrome**

Common and benign. Reassurance required. Similar to senile purpura in that vessel fragility is to blame. All measurements of coagulation are normal.

**Answer 5****(1) Tietze's syndrome**

Costochondritis of the sterno-costal junctions. The aetiology is uncertain. Symptomatic treatment and reassurance is all that is required.

**(2) Lobar pneumonia**

The main differential being pulmonary embolism, given the patient's clear major risk factors for thrombosis. There has been no haemoptysis (although this does not exclude a PE) and septic features dominate the clinical picture. In addition the prodromal symptoms and the 'gradual' onset of breathlessness make pneumonia the more likely diagnosis. She is clearly very unwell given the hypotension (septic shock) and in practice would al-

most certainly be investigated for both these possibilities. Note that pregnancy does not contraindicate emergency radiography. Other investigations include urgent blood cultures, FBC, U and Es, and arterial blood gases. Intravenous fluids and broad spectrum antibiotics (e.g. cefuroxime 1.5 g t.d.s., plus erythromycin 50 mg q.d.s.) are required. Inotropic support may be required on ICU.

**(3) Epidemic myalgia (Bornholm's disease)**

Coxsackie B virus. A typical history. A seasonal illness often occurring in late summer. The pain may be unilateral and severe ('the devil's grip'). There are no significant clinical findings on examination. Symptomatic treatment is all that is required and resolution occurs spontaneously.

**(4) Aortic dissection of the thoracic aorta**

The patient has a history of hypertension, which is a major risk factor. Often a history of a valsalva type manoeuvre (e.g. lifting, bending) before the event is obtained. The pain is usually very severe and described as 'ripping' or 'tearing' classically. Two thirds are of the ascending aorta, one third are in the descending aorta. Clinical examination is frequently normal, the diagnosis being made from the history and absence of the acute ECG changes of myocardial infarction (the major differential diagnosis).

**Risk factors for aortic dissection**

- Hypertension
- Coarctation of the aorta (Turner's Syndrome; XO)
- Marfan's syndrome
- Giant cell arteritis
- Major thoracic trauma

**Complications for dissection of the aorta**

- Inferior MI (occlusion of the right coronary artery origin)
- Aortic regurgitation (causing cardiac failure)
- Cardiac tamponade (retrograde dissection)
- Dissection of the major arteries coming off the aortic arch leading to strokes and limb ischaemia.
- Rupture of the false lumen back into the aorta or externally (usually resulting in death). In this case pulmonary haemorrhage had occurred

**Investigations**

- ECG: non-specific changes e.g. tachycardia, left ventricular strain, possible inferior MI

### OSCE station 3

Q. What are the causes of a mass in the right iliac fossa?

**A.**

- Carcinoma of the caecum
- Crohn's disease (usually an underweight, frequently young patient with scars from previous resections)
- Appendix mass or abscess
- Lymphoma
- Ileocaecal TB
- Ovarian tumour (NB ovarian tumours often rise out of the pelvis centrally)
- Transplanted kidney
- (Amoebiasis)
- (Schistosomiasis)
- (Carcinoid tumour)

Q. What would be your next investigation for this right iliac fossa mass?

**A.**

- Plain abdominal radiograph, looking for . . .
  - Obstruction (air/ fluid levels)
  - Calcification (tuberculosis)
  - Bowel wall oedema thickening (CD)
- Chest radiograph
  - TB
  - Metastatic disease
- Abdominal ultrasound scan
- Abdominal CT scan
- Small bowel meal and follow through for Crohn's disease
- Diagnostic laparoscopy

Q. What are the causes of a mass in the left iliac fossa?

**A.**

- Colonic carcinoma
- Diverticular mass
- Transplanted kidney
- Ovarian tumour
- Faeces

Q. What are the causes of an epigastric mass?

**A.**

- Hepatomegaly (see OSCE 4, p. 230)
- Gastric malignancy
- Pancreatic
  - Pseudocyst
  - Malignancy
- Lymphoma

Q. What are the causes of an enlarged kidney?

**A.**

- Renal carcinoma
- Hydronephrosis
- Hypertrophy of a solitary kidney
- Adult polycystic kidney disease
  - May have had a previous nephrectomy (for a painful non-functioning kidney) so that only one kidney is palpable
  - Cysts develop from all segments of the nephron during adolescence. Patients present with renal pain, treatment resistant hypertension, haematuria and stone symptoms
- Renal failure occurs later in life (10% of chronic renal failure)
- Various associations exist [see EMQ Answers 19 (2), p. 142]

Q. What are the symptoms and signs of uraemia?

**A.**

**Clinical features of uraemia . . .**

- Symptoms
  - Nausea, vomiting
  - Diarrhoea
  - Fatigue
  - Breathlessness:
    - Anaemia
    - Pleural effusion
    - Pericardial effusion
    - Impaired ventricular function
  - Weakness
  - Itching
- Signs
  - Pallor (anaemia)
  - Scratch marks (pruritus)
  - Bruising
  - Pericarditis (pericardial rub or effusion)
  - Hypertension
  - Oedema
  - Confusion
  - Muscle weakness
  - Fistula (for dialysis)

## OSCE station 4

Q. What are the causes of hepatomegaly (without splenomegaly)?

A.

- **Malignancy**
  - Secondary tumours mainly
  - Primary hepatocellular carcinoma
  - Lymphoma
  - Leukaemias:
    - CML
    - CLL
    - Myelofibrosis
- **Cirrhosis**
  - NB Late alcoholic cirrhosis causes a small fibrotic liver
- **Blood**
  - **Cardiac failure**
  - Tricuspid regurgitation (pulsatile liver)
  - Budd Chiari syndrome (hepatic vein thrombosis)
  - NB Hepatic engorgement can give abnormal LFTs (obstructive picture)
- **Bile**
  - Primary biliary cirrhosis
  - Common bile duct obstruction:
    - Stones
    - Strictures
    - Tumours
    - Lymph nodes
- **Infections**
  - **Viral hepatitis A, B (D), C**
  - Leptospirosis
  - Infectious mononucleosis
  - Others:
    - TB, syphilis, brucellosis
    - Tropical, e.g. malaria, kala-azar
- **Infiltrations**
  - Amyloid
  - Sarcoid
  - Wilson's disease
  - Haemochromatosis
  - Glycogen storage diseases

Q. What are the causes of splenomegaly?

A.

- **Blood**
  - Portal hypertension (late cirrhosis with a small liver)
- **Busy spleen**
  - Infections:

- Hepatitis A, B (D), C
- Leptospirosis (Weil's disease)
- Infectious mononucleosis
- Others:
  - TB, syphilis, brucellosis
  - Tropical infections, e.g. malaria, kala-azar
- Haematological malignancy:
  - CML\* and CLL\*
  - Polycythemia vera
  - Myelofibrosis\*
  - Lymphoma
- Haemolytic anaemias (splenic filtration)
- **Others**
  - Connective tissue disease:
    - SLE
    - RA
  - Sarcoidosis
  - Amyloidosis

Q. What are the causes of hepatosplenomegaly?

A.

As above; however, haematological malignancy and infections feature more prominently.

Q. How can you differentiate an enlarged spleen from an enlarged liver?

A.

There is a mass in the left/ right hypochondrium extending down towards the pelvis. It moves with respiration. It is resonant to percussion (over lying air filled bowel). The upper border can be palpated (unlike a spleen or liver which have their upper pole under the ribs). You can ballot the mass bimanually assessing the size, shape and consistency. Look for abdominal scars (?nephrectomy on the opposite side). Look also for an AV dialysis fistula over the distal radius.

	Kidney	Spleen
Enlarges?	Inferiorly	To RIF
Ballotable?	Yes	No
Resonant?	Yes	No
Get above?	Yes	No

\*Cause massive splenomegaly (as likely to be found in an examination)

## OSCE station 5

A 48-year-old female sits in front of you in your OSCE. You are asked to take a thyroid-based history from her.

*Q. What questions would you ask her?*

**A.**

**Has your weight changed recently?**

‘Oh yes, I’ve lost over a stone in the last 3 months alone.’

**Have you been trying to lose weight?**

‘No.’

**Has your appetite changed?**

‘It has. I eat like a horse and yet I continue to lose all this weight. In fact, I’m getting a bit concerned, as this is what happened to my mother shortly before she was diagnosed with cancer.’

**Have you noticed any change in your behaviour?**

‘Not really, but my husband says that I’ve changed of late; that I’m crabby and irritable all the time. I just thought it was “the change”, you know. He also says that I’m unable to sit still and I suppose he’s right about that really. I just like to keep active.’

**Have you noticed any weakness of your muscles?**

‘Not as such, though in saying that, I have noticed that of late I’ve found it a bit of a trial climbing up the stairs, especially if I’m carrying the washing.’

**Have you noticed any trembling of your fingers or hands?**

‘It’s strange you should say that, because just the other day I was writing a letter and I noticed that I couldn’t stop my hand trembling. Now I’m beginning to notice it more and more, even when I’m holding a cup of tea.’

**Have you had any palpitations?**

‘All the time. But that’s nothing new, I’ve had them for years. I’ve got an irregular heart beat, you know.’

**Are you as a rule a ‘hot’ or a ‘cold’ person?**

‘I’m always red hot. And sweat? I could sweat for England, even when everyone else in the room is perfectly happy with the temperature.’

**Have you noticed any change in your bowel habit over the last year or so?**

‘Well I am a bit “looser” than I used to be in that department.’

**Have you noticed any change in your appearance?**

‘Well my son says that my eyes are “goggly”, but I don’t really see any change.’

**Have you noticed any changes in your vision?**

‘I do occasionally suffer a bit of double vision, mainly when I’m tired.’

**Have you noticed any changes in your periods?**

‘Yes, they’re becoming much more infrequent. Very hit-and-miss. But I suppose I’m at that time of life, aren’t I?’

You are asked to perform a thorough examination of the person’s thyroid status.

From the end of the bed you note a **thin**-looking woman who is very jittery and **fidgits** incessantly with her hands. You ask her to hold her hands out straight in front of her and close her eyes. You place a sheet of paper on top of her hands to accentuate the **very fine tremor** that is present. Her hands are **warm** and her palms are **flushed**. There is neither clubbing, swollen fingers nor periosteal bone formation. You palpate her pulse, and note a rate of 98/min, **irregularly irregular**. Blood pressure is **161/91**. JVP is not raised. In her face you note exophthalmos, and to confirm this you stand behind the patient, looking down from on top of their forehead to check that there is genuine protrusion if the eyeballs. You check for lid lag and there is none present. There is no clear loss of acuity, ophthalmoplegia or conjunctival oedema, and no goitre in the neck. The apex beat is normally located, but it is **hyperdynamic**. Heart sounds are normal and the chest is clear. There is no peripheral oedema, although there is evidence of a rash on both shins. Comprehensive neurological examination reveals questionable wasting of the limb girdle musculature, and **decreased power** in both pectoral and pelvic muscles (MRC grade 4).

*Q. What is the cause of this woman’s clinical situation?*

**A.**

This woman is hyperthyroid and is displaying nearly all of the characteristic signs and symptoms. Like all types of thyroid disease, hyperthyroidism is more common in females, patients with autoimmune disease and those with a family history of thyroid disease.

*Q. What are the common causes of hyperthyroidism?*

**A.**

The commonest cause of hyperthyroidism, and the only one associated with thyroid eye disease, is **Graves’ disease**. It is due to autoantibodies directed at the **TSH re-**

**ceptor.** It typically causes a **fluctuating** clinical picture, with alternating relapse and remission.

Other causes of hyperthyroidism include . . .

- **Toxic multinodular goitre:** difficult to treat, traditional antithyroid drugs rarely successful
- **Single toxic adenoma (Plummer's disease):** similarly difficult to treat
- **Iatrogenic hyperthyroidism** (excessive levothyroxine replacement)
- **Drug-induced hyperthyroidism** (e.g. due to lithium, amiodarone)
- **Transient thyroiditis** (e.g. de Quervain's viral-mediated transient thyroiditis, post-partum thyroiditis)
- **Thyrotoxicosis factitia** (patient takes thyroxine, but attempts to conceal this)

*Q. What investigations would you perform to confirm your diagnosis?*

**A.**

In all cases of primary hyperthyroidism, **TSH will be suppressed**, and this is the initial investigation of choice. When this is combined with a **raised serum T<sub>3</sub> or T<sub>4</sub>**, then the diagnosis of hyperthyroidism is made. **TSH-receptor autoantibodies** will be present in cases of Grave's disease, but these are not commonly measured. Surprisingly, the microsomal and thyroglobulin autoantibodies associated with hypothyroidism are also often found in Grave's disease.

In very rare situations, a pituitary tumour will be the cause of hyperthyroidism, and TSH will be raised. The differing laboratory findings in thyroid diseases are summarized in the table below . . .

Thyroid state	Thyroxine (T <sub>4</sub> )	Triiodothyronine (T <sub>3</sub> )	TSH (pituitary)	TRH (hypothalamus)
<b>Hyperthyroidism</b>				
Graves' disease	Raised	Raised	Decreased	Decreased
Toxic multinodular goitre	Raised	Raised	Decreased	Decreased
Pituitary tumour	Raised	Raised	Raised	Decreased
<b>Hypothyroidism</b>				
Primary	Decreased	Decreased	Increased	Increased
Secondary	Decreased	Decreased	Decreased	Increased
Tertiary	Decreased	Decreased	Decreased	Decreased

*Q. What are the treatment options available?*

**A.**

Treatment of hyperthyroidism is complex, and depends on the underlying cause and the clinician's choice. In general, however, it can be split into three options . . .

1. **Radioiodine:** a safe treatment option that usually leads to euthyroidism in **3–6 months**. I<sup>131</sup> is given and it accumulates in the thyroid, destroying the gland. The patient should be rendered euthyroid before treatment is given, with anti-thyroid medications
2. **Antithyroid drugs**, e.g. carbimazole, propylthiouracil, methimazole: if these are going to work, they will usually restore a euthyroid state in **4–12 weeks**. They are less likely to lead to permanent remission than either of the other main treatment options, and long-term therapy may be necessary. Side effects can be severe, and include hepatotoxicity, vasculitis and agranulocytosis
3. **Surgery:** the least favoured treatment option of non-

neoplastic causes of hyperthyroidism because of the morbidity, expense and free availability of alternative effective treatments. The procedure of choice is a sub-total thyroidectomy, and this should only be performed in patients rendered euthyroid with antithyroid drugs. The drugs should be stopped 2 weeks before surgery and **potassium iodide** given to reduce the vascularity of the gland

Whatever treatment option is chosen **β-blockers** are a useful adjunct to treatment because they block the sympathetic side effects that are so distressing in hyperthyroidism. Propranolol also has the ability to **block peripheral conversion of T<sub>4</sub> to T<sub>3</sub>**.

*Q. What is thyroid storm?*

**A.**

This is also known as thyrotoxic crisis, and is a condition

with a **20–50% mortality**, despite optimal management. It occurs as an exaggerated manifestation of hyperthyroidism, and is precipitated by **major stressors**, such as infection, trauma, major surgery, diabetic ketoacidosis, MI, CVA, PE and withdrawal of thyroid medications. It presents with an **overactivated sympathetic nervous system** (palpitations, tachycardia, tachyarrhythmia, cardiac failure, anxiety, agitation), and typically a **very high fever**. Jaundice is a late and ominous sign.

Diagnosis should be made clinically, as thyroid function tests often fail to be grossly deranged. Treatment is . . .

- ALS stabilization of airway, breathing and circulation
- Commencement of an **intravenous fluid infusion**
- **$\beta$ -blocker infusion** to block adrenergic effects
- **Cooling blankets**
- **Paracetamol**
- **Propylthiouracil** to block production of new  $T_4$
- **Iodine** to block release of pre-formed  $T_4$

**Do not give the iodine until the propylthiouracil has had chance to work** (90 min) otherwise there is a risk of making the thyroid storm worse. Treat any precipitating factors that may be present.

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## OSCE station 6

You arrive at an OSCE station and are asked to **examine a patient's breasts** (this will almost always be on a dummy; hence, you would be expected to both *show* what you *would* do and *describe* what you *would* do because the limitations of the model mean that you cannot physically demonstrate what you would like the patient to do).

- **Introduce** yourself to the patient, stating full name and grade
- **Explain** what it is that you propose to do, and that there will be a nurse chaperone present at all times
- Gain **consent**
- Give the patient time to get undressed in private if she is not already de-robed
- Ensure that you have a **female nurse chaperone**
- Once undressed, uncover the patient from lower ribs upward
- Begin with **inspection**: inspect in a number of ways . . .
  - With the patient sitting comfortably at rest with her arms **by her sides**
  - With the patient's hands in the air **above her head**
  - With the patient's hands **on her hips**; ask the patient to push in on her hips to tense the pectoralis musculature
- With the patient **leaning forward slightly**
- On inspection you are looking for a number of things . . .
  - Obvious lumps or masses
  - Scars (previous neoplastic, augmentative or reductive surgery)
  - Sinuses
  - Bruising (old fine needle aspiration site)
  - Inflammatory changes in skin (*peau d'orange*)
  - Colour of skin (if darkened, may suggest previous radiotherapy)
  - Irregular skin thickening
  - Retraction
  - Dimpling
  - Asymmetry
  - Nipple changes [e.g. indrawing, eczema (Paget's disease), differing directions, inversion]
  - Lactation
  - Fungation or ulceration
- Move on to **palpation** of the breast tissue itself. The best position to have the patient in is **supine, with the hand behind the head**.
- There are a number of ways to palpate the mass of the breast; which one you use does not matter **as long as you cover the whole of the breast tissue**. Expect the palpation of a single breast to take **3 minutes**. Either . . .
  - Split the breast into four quadrants and palpate each quadrant in turn using small circular movements with the pads of the fingers, about the size of a 50 pence piece; *or* . . .
  - Start just anterior to the axilla and circle around the periphery of the breast in an ever-decreasing circle; *or* . . .
  - Start just anterior to the axilla and palpate circularly up and down the breast tissue moving from lateral to medial
- If you find any abnormality you must describe its . . .
  - **Site** on the breast (and on which breast): often giving a time in the '*o'clock*' form helps; also site within the breast (superficial or deep)
  - **Size** in cm
  - **Shape** (circular, ovoid, elongated)
  - **Surface** (regular or irregular)
  - **Consistency** (soft or hard)
  - **Tender** or non-tender
  - Overlying **skin** (tethered or mobile)
  - Underlying **fascia** (tethered or mobile)

- **Temperature** (any evidence of inflammation)
- **Transillumability** (can you shine a light through it or not)
- Once you have completely described it, move on to palpate the rest of the breast tissue from where you left off; just because you have found one abnormality does not mean there are not more to find (there usually are in the OSCE)!
- Describe each abnormality in the same way
- Once you reach the **nipple**, you must bi-manually palpate it by placing two fingers either side and gently rocking the nipple, with the specific purpose of palpating any masses behind the nipple, and also of expressing any abnormal secretions or galactorrhoea from the nipple itself
- Move on to the **axilla** (it is best to sit the patient up again to do so) . . .
  - Begin with **inspection**, looking for signs of masses, infection (*hidradenitis suppuritiva*), rashes or unusual skin pigmentation (*acanthosis nigricans*)
  - Palpate any breast tissue you suspect to be deep in the axilla (do not forget that the **axillary tail of Spence** can reach far into the axilla in some women)
  - Then palpate for **lymph nodes**. Support the arm of the patient with your non-examining hand, and,

- starting as high in the axilla as you can, palpate the medial and lateral axillary walls, attempting to trap any nodes against the chest wall or medial humerus respectively as you do so. Warn the patient that this part of the examination may be the least comfortable
- Describe any abnormalities found in the same way as for a breast lump
- Complete your palpation by feeling for any enlarged **supraclavicular lymph nodes** *from behind*
- **Immediately** cover the patient up, thank them and turn to the examiner to present your findings

Be aware that you may get asked to examine a patient who has had a **previous mastectomy**. Do not panic! Begin by inspecting the mastectomy scar and axilla, looking for signs of masses or nodularity. Note especially the colour of the skin (radiation) and the presence of lymphoedema (impaired lymph drainage post-surgery). Move on to palpation: palpate *gently* along the scar (it is often exquisitely tender), and then move on to the rest of the chest wall in a similar way as if the breast was still present. Take special care in palpating the axilla, looking for lymphadenopathy.

The way to differentiate between the most common three breast masses is shown in the table below . . .

	<i>Breast cyst</i>	<i>Fibroadenoma (breast mouse)</i>	<i>Carcinoma</i>
Age of patient	Early middle age, aged 35–50 years; uncommon post-menopause except in women on HRT	Young, aged 15–30; rare after age 50 years	Older, aged 35–100+; usually patient is >50 years
Shape of lesion	Spherical	Spherical, discoid or lobular	Irregular
Consistency of lesion	Soft	Firm	Hard
Surface of lesion	Delineated from surrounds	Delineated from surrounds	NOT delineated from surrounds
Tender or not	Usually	Rarely	Rarely
Tethering	Moderately mobile	Very mobile	Commonly tethered to skin or fascia
Number of lesions at one time	Commonly multiple	Can be multiple, but usually single	Usually single

*Continued*