# Comprehensive Core Clinical Cases Self-Assessment for Medical Students 

Andrew Sewart MBChB PhD MRCP MRCGP

GP Partner
Bellingham, Northumberland

Henriette van Ruiten MBChB MRCPCH
Paediatric Registrar
Northern Deanery

A Problem-based Learning Approach

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## CARDIOVASCULAR CASES: QUESTIONS

## CARDIOVASCULAR CASE 1

Tim, a 60-year-old solicitor, visits his GP complaining of recent episodes of central chest tightness on exertion, which settles on rest. There is nothing of note in his past medical history. Tim is referred to the rapidaccess chest pain clinic, where he is diagnosed with stable angina.

Q 1. What are the main risk factors for coronary heart disease (CHD)? 5 marks
$\qquad$
$\qquad$
$\qquad$
$\qquad$

Q 2. List four drugs that may be prescribed to control angina. 4 marks

Tim is also prescribed a statin and aspirin.

Q 3. What is the recommended upper limit for fasting total cholesterol (TC) and low-density lipoprotein (LDL) in secondary prevention?

2 marks

# Q 4. What blood test must you request before prescribing a statin, and what advice must you give to patients on statin therapy? 

Despite optimised medical treatment, Tim remains symptomatic, so he undergoes coronary angiography with a view to revascularisation.

Q 5. What two procedures are used for revascularisation? 2 marks

Total:
15 marks

## CARDIOVASCULAR CASE 2

John, a 72-year-old hypertensive, visits his GP complaining of several months' history of increasing breathlessness. He is now breathless even when doing simple tasks around the home, such as dressing.

Q 1. List four non-cardiac causes of gradually progressive dyspnoea.

John's GP suspects heart failure and organises a number of investigations.

Q 2. List four causes of heart failure.
4 marks
$\qquad$
$\qquad$
$\qquad$

Q 3. From the history above classify John's heart failure according to the New York Heart Association (NYHA) criteria.

Q 4. What hormone is significantly raised in heart failure? 2 marks Q 5. Which key investigation would you request to confirm heart failure? 1 mark

John is diagnosed with left ventricular heart failure.

Q 6. Which two drugs are the first-line treatment for heart failure? 2 marks

Total:
15 marks

## CARDIOVASCULAR CASE 3

David, a 51-year-old builder, is referred to his GP from the well-man clinic, as his blood pressure was recorded as $164 / 96 \mathrm{mmHg}$.

Q 1. Define the systolic/diastolic ranges for mild (phase 1), moderate (phase 2) and severe (phase 3) hypertension.

You request an electrocardiogram (ECG), shown in Figure 1.1 below:


Figure 1.1: David's ECG.
Q 2. What does David's ECG show?
1 mark


Figure 1.2: CVD risk prediction chart.

## Q 4. List two causes of secondary hypertension.

David smokes ten cigarettes a day, and his fasting bloods are measured:
total cholesterol (TC) 6.34 mmol/l, low-density lipoprotein (LDL)
$4.22 \mathrm{mmol} / \mathrm{l}$, high-density lipoprotein (HDL) $1.26 \mathrm{mmol} / \mathrm{l}$, triglycerides
$2.4 \mathrm{mmol} / \mathrm{l}$, glucose $4.6 \mathrm{mmol} / \mathrm{l}$.

Q 5. Calculate his 10-year cardiovascular disease (CVD) risk.
1 mark

You determine that David's blood pressure needs to be treated.

Q 6. What four lifestyle changes would you recommend? 2 marks
$\qquad$
$\qquad$
$\qquad$
$\qquad$

Q 7. What antihypertensive treatment would you offer first line?

Q 8. Name two electrolyte abnormalities caused by thiazide diuretics. 2 marks
$\qquad$
$\qquad$

# Q 9. Name four complications that might arise if David's hypertension is not treated. <br> 2 marks 

Total:
15 marks

## CARDIOVASCULAR CASE 4

A GP organises an electrocardiogram (ECG) (shown in Figure 1.3) for one of her patients, 78-year-old Sarah, after checking her pulse during a routine consultation for her hypertension.


Figure 1.3: Sarah's ECG.

Q 1. What does the ECG show?
1 mark

Q 2. List six causes of this rhythm.
3 marks
$\qquad$
$\qquad$
$\qquad$

Q 3. What two drugs may be used for rate control?

Q 4. What two methods may be used to attempt cardioversion?
2 marks

Q 5. What two drugs may be used for rhythm control?
2 marks

Sarah opts for rate control and agrees to be anticoagulated to reduce her risk of stroke.

Q 6. Name four factors used to stratify stroke risk. 2 marks
$\qquad$
$\qquad$
$\qquad$
$\qquad$

Q 7. What drug is used for anticoagulation, how is it monitored and what is the
target range?
$\qquad$
$\qquad$

Total:
15 marks

# CARDIOVASCULAR CASES: ANSWERS 

## CARDIOVASCULAR CASE 1

Tim, a 60-year-old solicitor, visits his GP complaining of recent episodes of central chest tightness on exertion, which settles on rest. There is nothing of note in his past medical history. Tim is referred to the rapidaccess chest pain clinic, where he is diagnosed with stable angina.

## Q 1. What are the main risk factors for coronary heart disease (CHD)?

## A 1 mark each for any of the following:

## - Age

- Diabetes
- Smoking
- Hyperlipidaemia: total cholesterol $>6.5 \mathrm{mmol} / \mathrm{I}$
- Hypertension
- Sedentary lifestyle
- Diet high in saturated fats (and low in fruit and vegetables)
- Family history of CHD


## LEARNING POINTS

(1) Stable anginal pain is: (1) constricting discomfort in the front of the chest, neck, shoulder, jaw and/or arm; (2) precipitated by physical exertion; and (3) relieved by rest or glyceryl trinitrate (GTN). Continuous or very prolonged pain that is not related to activity is unlikely to be stable angina.
(1) The likelihood of suspected stable angina can be estimated from whether the patient has typical symptoms (1-3) and the presence of risk factors such as smoking and diabetes. Clinical assessment alone may be sufficient to diagnose stable angina, although if the diagnosis is less certain it may require further investigations such as angiography, stress echocardiogram (using either exercise or dobutamine), myocardial perfusion scans or CT calcium scoring (CT of coronary arteries for calcified plaques is indicative of CHD). Exercise ECG is not recommended for diagnosing stable angina.
(1) Resting ECG may be normal or may show ischaemia or previous infarction, for example abnormalities in Q waves, left bundle branch block (LBBB), ST-segment or T-wave abnormalities (eg flattening or inversion).

Q 2. List four drugs that may be prescribed to control angina.
A 1 mark each for any of the following:

- Beta-blocker
- Calcium-channel blocker: use a dihydropyridine calcium-channel blocker (eg amlodipine)
- Long-acting nitrate
- Ivabradine: slows down heart rate by inhibiting the sinus node
- Nicorandil: vasodilator
- Ranolazine


## LEARNING POINTS

(1) Optimal drug treatment consists of one or two anti-anginal drugs; first-line are $\beta$-blockers and/or calcium-channel blockers. Use other anti-anginal drugs if symptoms are not satisfactorily controlled or if $\beta$-blockers/calcium-channel blockers are not tolerated or are contraindicated.
(1) Also give a short-acting nitrate for preventing and treating episodes of angina. Advise patients on common side-effects (headache, flushing, light-headed); use it during episodes of angina and before exercise or exertion; if treating an episode of angina, repeat dose after five minutes if pain persists, and call ambulance if pain is still present five minutes after second dose.

Tim is also prescribed a statin and aspirin.

Q 3. What is the recommended upper limit for fasting total cholesterol (TC) and low-density lipoprotein (LDL) in secondary prevention?

2 marks
A 1 mark each for the following:

- TC $<4 \mathrm{mmol} / \mathrm{I}$
- LDL $<2 \mathrm{mmol} / \mathrm{I}$


## LEARNING POINT

(1) Patients with existing CHD should ideally have a fasting TC of $<4 \mathrm{mmol} / \mathrm{l}$ (or a $25 \%$ reduction from baseline, whichever is the greater) and LDL of $<2 \mathrm{mmol} / \mathrm{l}$ (or a $30 \%$ reduction, whichever is the greater).

Q 4. What blood test must you request before prescribing a statin, and what advice must you give to patients on statin therapy?

A 1 mark each for the following:

- Liver function tests (LFTs)


## LEARNING POINT

(1) Statins are potentially hepatotoxic (as evidenced by an increase in aminotransferases) and are contraindicated in active liver disease. LFTs should be checked at baseline, at three months and at one year. Discontinue if ALT/ AST rise to three times the upper limit (normal range is $3-35 \mathrm{IU} / \mathrm{I}$ ).

- Patients should report any unexplained muscle pains, tenderness or weakness.

LEARNING POINT
(1) Although rare, statins may give rise to myositis (diagnosed by raised creatine kinase (CK)), causing muscle pain, weakness and tenderness. In severe cases this can lead to rhabdomyolysis and acute renal failure. Risk is increased in presence of co-morbidities such as renal impairment or if statins are used in combination with other drugs (eg macrolide antibiotics, fibrates).

Despite optimised medical treatment, Tim remains symptomatic, so he undergoes coronary angiography with a view to revascularisation.

Q 5. What two procedures are used for revascularisation?
2 marks
A 1 mark each for the following:

- Percutaneous coronary intervention ( PCI ): angioplasty (balloon dilatation) and stenting. It improves symptoms but not prognosis.
- Coronary artery bypass graft (CABG) (eg using saphenous veins or internal thoracic arteries). It improves prognosis in a subset of patients with CHD (> 65 years old, multi-vessel disease, diabetic).


## LEARNING POINT

(1) Stents are either bare-metal or drug-eluting. The latter have a reduced risk of restenosis, although an increased risk of stent thrombosis, so patients are put on both aspirin and clopidogrel (for one year).

## CARDIOVASCULAR CASE 2

John, a 72-year-old hypertensive, visits his GP complaining of several months' history of increasing breathlessness. He is now breathless even when doing simple tasks around the home, such as dressing.

Q 1. List four non-cardiac causes of gradually progressive dyspnoea.
4 marks
A 1 mark each for any of the following:

- Chronic obstructive pulmonary disease (COPD)
- Fibrotic lung disease
- Lung cancer
- Pleural effusion
- Multiple pulmonary emboli
- Anaemia


## LEARNING POINT

(1) Initial investigations in progressive dyspnoea may include: electrocardiogram (ECG), chest X-ray, spirometry and blood tests, including full blood count, to exclude anaemia.

John's GP suspects heart failure and organises a number of investigations.

Q 2. List four causes of heart failure.
4 marks
A 1 mark each for any of the causes listed below:
Heart failure is caused by structural or functional abnormalities of the heart:

- Ischaemic heart disease
- Hypertension
- Cardiomyopathies, eg dilated cardiomyopathy
- Valvular heart disease, eg mitral regurgitation (volume overload), aortic stenosis (obstruction to outflow)
- High output, eg anaemia, hyperthyroidism
- Arrhythmia, eg atrial fibrillation
- Cor pulmonale: right heart failure secondary to pulmonary disease, eg COPD


## LEARNING POINTS

(1) Heart failure itself can be classified in a number of ways, including:

- Left versus right heart failure; when occurring together (most commonly) it is termed congestive heart failure. This is useful for understanding clinical symptoms and signs. Symptoms of left heart failure include dyspnoea, orthopnoea, paroxysmal nocturnal dyspnoea (PND) and cardiac wheeze. Symptoms of right-sided failure include peripheral oedema, anorexia and nausea (caused by bowel oedema), and abdominal distension due to ascites.
- Systolic dysfunction versus diastolic dysfunction - or left ventricular systolic dysfunction (LVSD) versus heart failure with preserved ejection fraction (HFPEF) - owing to insufficient contraction or relaxation, respectively, of the ventricle, and both causing a reduced stroke volume. The reduced stroke volume in HFPEF is due to a stiff ventricle (eg secondary to hypertension).

Q 3. From the history above classify John's heart failure according to the New York Heart Classification (NYHA) criteria.

## A Grade III heart failure, ie breathlessness on minimal exertion.

LEARNING POINT
NYHA heart failure classification

| Grade | Extent of breathlessness |
| :--- | :--- |
| I | No undue breathlessness |
| II | Breathlessness on moderate exertion |
| III | Breathlessness on minimal exertion |
| IV | Breathlessness at rest |

Q 4. What hormone is significantly raised in heart failure?

## A B-type (or brain) natriuretic peptide (BNP).

## LEARNING POINT

(1) BNP is secreted by the ventricular myocardium in response to distension and acts to reduce circulating volume by inhibiting renin, antidiuretic hormone (ADH) and aldosterone secretion (similar actions to atrial natriuretic peptide (ANP)). BNP levels are raised in heart failure and are used as a screening test for patients with suspected untreated heart failure. BNP levels $<100 \mathrm{pg} /$ ml make heart failure unlikely, while high levels ( $>400 \mathrm{pg} / \mathrm{ml}$ ) carry a poor prognosis.

Q 5. Which key investigation would you request to confirm heart failure? 1 mark
A Echocardiogram: this is the key investigation in heart failure. It will confirm the diagnosis and its severity and may indicate the cause.

## LEARNING POINT

(1) Parameters assessed by echocardiography include left ventricular systolic and diastolic function, regional/localised hypokinesis (as a result of underlying coronary heart disease) and valvular function.

John is diagnosed with left ventricular heart failure.

Q 6. Which two drugs are the first-line treatment for heart failure? 2 marks

A 1 mark each for any of the following:

- Angiotensin-converting-enzyme inhibitors (ACEi); angiotensin II receptor blockers (ARBs) are an alternative first-line treatment, but may also be used in combination with ACEi as second-line treatment
- Beta-blockers (eg bisoprolol, carvedilol)


## LEARNING POINTS

(1) Drug treatment of heart failure is shown in the figure opposite.* ACE inhibitors, $\beta$-blockers and aldosterone antagonists improve prognosis. Diuretics and digoxin improve symptoms but do not reduce mortality.
(1) Cardiac resynchronisation therapy (CRT) involves implantation of a
bi-ventricular pacemaker. By ensuring the heart contracts in synchrony, cardiac function is maximised, thus improving both symptoms and prognosis. CRT is indicated in patients with NYHA grade III-IV symptoms on optimal medical management, an ejection fraction $\leq 35 \%$ and a QRS duration $>150 \mathrm{~ms}$ (indicating cardiac dys-synchrony).


## Drug treatment for heart failure due to left ventricular systolic dysfunction

*The treatment flow diagram is for patients with heart failure due to left ventricular systolic dysfunction (LVSD). Management of patients with heart failure with preserved ejection fraction (HFPEF) is primarily aimed at optimising treatment of underlying conditions such as hypertension, diabetes and coronary heart disease.

## Total:

## CARDIOVASCULAR CASE 3

David, a 51-year-old builder, is referred to his GP from the well-man clinic, as his blood pressure was recorded as $164 / 96 \mathrm{mmHg}$.

Q 1. Define the systolic/diastolic ranges for mild (phase 1), moderate (phase 2) and severe (phase 3) hypertension.

3 marks
A 1 mark for each of the following:

- Phase 1: 140-159/90-99 mmHg
- Phase 2: 160-179/100-109 mmHg
- Phase $3: \geq \mathbf{1 8 0} / \mathbf{1 1 0} \mathbf{m m H g}$


## LEARNING POINTS

(i) It is recommended to confirm a clinic diagnosis of hypertension with either ambulatory or home blood pressure monitoring, to exclude 'white coat hypertension', defined as a discrepancy of more than $20 / 10 \mathrm{mmHg}$ between clinic and ambulatory/home readings.
(1) Treat phase 2 and 3 hypertension; treat phase 1 hypertension if there are target organ damage, existing cardiovascular disease, renal disease, diabetes or a ten-year cardiovascular risk $\geq 20 \%$.

You request an ECG, shown opposite.

Q 2. What does David's ECG show?
1 mark
A Left ventricular hypertrophy (LVH).

## LEARNING POINT

(i) LVH causes tall $R$ waves in V5-6 and deep $S$ waves in V1-2 (consider LVH if $R$ wave in $\mathrm{V} 5-6>25 \mathrm{~mm}$ or combined R wave in V 6 and S wave in $\mathrm{V} 1>35 \mathrm{~mm}$ ). May also get T-wave inversion in the lateral leads (ie I, AvL, V5-6) and left axis deviation (ie positive in I, negative in II and III).


Figure 1.1: David's ECG.

Q 3. Why is this important with regard to hypertension?
1 mark

## A LVH indicates hypertensive end-organ damage and is an indication for antihypertensive treatment, even in patients with phase 1 hypertension.

## LEARNING POINTS

(1) In the presence of LVH, request an echo to assess LV size and function.
(i) Hypertension may also damage the kidneys, as evidenced by reduced estimated glomerular filtration rate (eGFR) and/or microalbuminuria (ie an albumin to creatinine ratio of $\geq 30 \mathrm{mg} / \mathrm{mmol}$; in diabetics the ratio is lower at $\geq 2.5$ in men and $\geq 3.5$ in women).
(1) Examination should also include fundoscopy, looking for hypertensive retinopathy (see next page).

| Grade | Hypertensive retinopathy |
| :--- | :--- |
| I | Tortuous retinal arteries with thick shiny walls (silver wiring) |
| II | Arteriovenous (AV) nipping (narrowing where arteries cross veins) |
| III | Flame haemorrhages and cotton-wool spots (small infarcts) |
| IV | Papilloedema |

Q 4. List two causes of secondary hypertension.
A 1 mark for each of the following:

- Renal or renovascular disease (eg renal artery stenosis, glomerulonephritis)
- Endocrine disorders: Cushing syndrome (corticosteroid excess), Conn syndrome (hyperaldosteronism), phaeochromocytoma (norepinephrine and epinephrine excess), acromegaly (human growth hormone excess)
- Medications (eg combined oral contraceptive, corticosteroids)
- Coarctation of the aorta
- Pregnancy


## LEARNING POINTS

(1) $95 \%$ of patients with hypertension have essential or primary hypertension; $5 \%$ of patients have secondary hypertension.
(1) Younger hypertensive patients should have a lower threshold for evaluating secondary causes of hypertension.

David smokes ten cigarettes a day and his fasting bloods are measured: total cholesterol (TC) $6.34 \mathrm{mmol} / \mathrm{I}$, low-densisty lipoprotein (LDL) $4.22 \mathrm{mmol} / \mathrm{l}$, high-density lipoprotein (HDL) $1.26 \mathrm{mmol} / \mathrm{l}$, triglycerides $2.4 \mathrm{mmol} / \mathrm{l}$, glucose $4.6 \mathrm{mmol} / \mathrm{l}$.

Q 5. Calculate his 10-year cardiovascular disease risk.
$A \geq \mathbf{3 0} \%$ : male, smoker, aged between 50 and 59 years, systolic BP of $164 \mathbf{m m H g}$, TC:HDL ratio of 5 .

## LEARNING POINT

(1) When investigating hypertension also assess cardiovascular disease (CVD) risk. CVD risk prediction charts (as shown in Figure 1.2) are found at the back of the British National Formulary and are used to estimate the risk of CVD (ie fatal and non-fatal myocardial infarction, stroke and angina) in patients with no previous history of CVD or diabetes. High-risk patients are defined as 10-year risk $\geq 20 \%$ and should be offered lipid-lowering medication (eg simvastatin 40 mg nocte). Aim to reduce TC to $<4 \mathrm{mmol} / \mathrm{l}$ and LDL to $<2 \mathrm{mmol} / \mathrm{l}$, or a $25 \%$ reduction in TC and a 30\% reduction in LDL from baseline, whichever provides the lowest value.


Figure 1.2: CVD risk prediction chart.

You determine that David's blood pressure needs to be treated.

## Q 6. What four lifestyle changes would you recommend?

A $1 / 2$ mark for each of the following:

- Lose weight: aim for body mass index (BMI) 20-25
- Stop smoking
- Take regular exercise
- Reduce salt consumption
- Reduce alcohol consumption to $\leq 21$ units/week
- Consume five portions of fruit and vegetables/day
- Reduce consumption of total and saturated fat


## LEARNING POINT

(1) Offer lifestyle advice to patients with hypertension at initial diagnosis and periodically thereafter.

Q 7. What antihypertensive treatment would you offer first line? 1 mark
A An angiotensin-converting enzyme inhibitor (ACEi) or, if not tolerated, an angiotensin II receptor blocker (ARB).

## LEARNING POINTS

(i) For patients aged $<55$ years offer ACEi or ARB (though these should not be combined to treat hypertension).
(i) For patients aged > 55 years or black Afro-Caribbean patients, offer a calciumchannel blocker (eg amlodipine). If not tolerated or unsuitable, offer a thiazide-like diuretic (eg chlortalidone or indapamide); bendroflumethiazide is no longer a first-line thiaizide diuretic, although in patients who are already taking it and whose blood pressure is stable and well controlled continue treatment.
(1) If further antihypertensive treatment is required, combine ACEi (or ARB) initially with either a calcium-channel blocker or a thiazide-like diuretic, although some patients may require all three.
(i) If additional treatment is required, consider addition of spironolacatone ( 25 mg once daily) or higher-dose thiazide-like diuretic; if further diuretic treatment is not tolerated or contraindicated or ineffective, consider an $\alpha$ - (eg doxazosin) or $\beta$ - (eg atenolol) blocker.

Q 8. Name two electrolyte abnormalities caused by thiazide diuretics. 2 marks
A 1 mark for each of the following:

- Hyponatraemia
- Hypokalaemia: often mild, requiring no correction
- Hyperuricaemia: can precipitate gout
- Hypercalcaemia: thiazide diuretics lower urinary calcium excretion and may be used to prevent formation of calcium-containing kidney stones
- Hyperglycaemia: may impair diabetic control

Q 9. Name four complications if David's hypertension is not treated. 2 marks
A $1 / 2$ mark for each of the following:

- Stroke: individuals with hypertension have a six-fold increased risk of stroke compared with normotensive individuals
- CVD: individuals with hypertension have a three-fold increased risk of CVD compared with normotensive individuals
- Heart failure
- Peripheral vascular disease (PVD)
- Chronic renal failure: hypertensive nephropathy
- Visual impairment: hypertensive retinopathy

Total:
15 marks

## CARDIOVASCULAR CASE 4

A GP organises an electrocardiogram (ECG) (shown in Figure 1.3) for one of her patients, 78-year-old Sarah, after checking her pulse during a routine consultation for her hypertension.


Figure 1.3: Sarah's ECG.

Q 1. What does the ECG show?
1 mark

## A Atrial fibrillation.

## LEARNING POINTS

(1) Atrial fibrillation (AF) is diagnosed by absent $P$ waves and irregular QRS complexes. AF results from a chaotic irregular atrial rhythm (300-600 bpm, of which only a proportion are sufficient to generate an action potential that is conducted to the ventricles, causing an irregular ventricular rate).
(1) Atrial fibrillation may present with symptoms of palpitations, breathlessness and chest pain. However, the majority of presentations are clinically silent, with the first presentation as a cerebrovascular accident, heart failure or incidental ECG finding, or following detection of an irregular pulse on examination.

Q 2. List six causes of this rhythm.
A $1 / 2$ mark each for any of the following:

- Hypertension
- Coronary heart disease
- Myocardial infarction
- Hyperthyroidism
- Valvular heart disease (particularly mitral valve disease)
- Pneumonia
- Pulmonary embolism
- Alcohol excess
- Heart failure
- Lone atrial fibrillation (ie no identifiable cause)
- Cardiomyopathy


## LEARNING POINT

(1) Atrial fibrillation can be classified as acute, paroxysmal (ie episodes of AF that terminate spontaneously), persistent (terminated either pharmacologically or electrically - see below) or permanent (fails to respond to attempts to cardiovert or when cardioversion is deemed inappropriate).

Q 3. What two drugs may be used for rate control?
A 1 mark each for any of the following:

- Beta-blockers
- (Rate-limiting) calcium-channel blocker: diltiazem or verapamil
- Digoxin


## LEARNING POINTS

(1) The treatment of AF involves either rhythm or rate control. First-line treatment in paroxysmal AF is rhythm control; in permanent AF it is rate control. The decision whether to rate- or rhythm-control in persistent AF depends on age (aim for rhythm control in younger patients), on whether AF is symptomatic, if there is evidence of heart failure (cardioversion improves left ventricular function) and on suitability for cardioversion (eg anticoagulation is contraindicated).
(i) If patients are rate-controlled, first-line treatment is either a standard $\beta$-blocker or rate-limiting calcium-channel blocker. If further rate control is needed, consider the addition of digoxin.

## Q 4. What two methods may be used to attempt cardioversion?

## A 1 mark for each of the following:

- Chemical cardioversion: anti-arrhythmic drugs
- DC cardioversion


## LEARNING POINT

(1) If AF is acute (< 48 hours), there is no need to anticoagulate before chemical or DC cardioconversion (haemodynamically compromised patients require urgent DC cardioversion); otherwise, anticoagulate three weeks before and four weeks after cardioconversion (DC cardioversion is first line in persistent AF). If the patient is at high risk of attempted DC cardioversion being unsuccessful (eg previous failure), pre-treatment with anti-arrhythmic drugs before DC cardioversion increases the likelihood of restoring and maintaining sinus rhythm.

Q 5. What two drugs may be used for rhythm control?

## A 1 mark for any 2 of the following:

- Standard $\beta$-blockers
- Class 1 anti-arrhythmic drugs (ie flecainide, propafenone)
- Class 3 anti-arrhythmic drugs (ie sotalol, amiodarone)


## LEARNING POINTS

(i) Several anti-arrhythmic drugs can be used to maintain sinus rhythm in patients with paroxysmal or persistent AF who have been successfully cardioverted. First-line treatment is a standard $\beta$-blocker; where a standard $\beta$-blocker is ineffective, contraindicated or not tolerated, use amiodarone with underlying structural heart disease, or a class 1c agent (eg flecainide) or sotalol without underlying structural heart disease.
(i) Pill-in-the-pocket therapy involves self-administration of an anti-arrhythmic drug in patients with paroxysmal AF to terminate a new episode.
(i) Anti-arrhythmic drugs are classified according to the changes they cause in the action potential or AP (see Figure opposite).


0 - rapid depolarisation due to opening of fast sodium channels
1 - early repolarisation due to closure of sodium channels and opening of potassium channels
2 - plateau phase where potassium outflux equals calcium influx due to opening of slow calcium channels
3 - late repolarisation following closure of calcum channels
4 - return to resting membrane potential by $\mathrm{Na} / \mathrm{K}$ ATPase

Class 1 (further divided into $1 \mathrm{a}, \mathrm{b}, \mathrm{c}$ ): slow phase 0 (eg flecainide (1c))
Class 2: slow phase 4 (ie standard $\beta$-blockers)
Class 3: slow phase 3 (eg amiodarone, sotalol)
Class 4: calcium-channel blockers. More effective in supraventricular tachycardia, where the AP in the AV (and SA) node is generated by calcium influx as opposed to calcium influx in the myocardium

Sarah opts for rate control and agrees to be anticoagulated to reduce her risk of stroke.

Q 6. Name four factors used to stratify stroke risk.

## A $1 / 2$ mark each for any of the following:

- Congestive heart failure
- Hypertension
- Age $\geq 75$ years old
- Diabetes
- Stroke or previous transient ischaemic attack (TIA)


## LEARNING POINTS

(i) The decision whether to anticoagulate a patient in paroxysmal, persistent or permanent AF depends on their risk of stroke, assessed on the basis of the risk factors above. This is termed the $\mathbf{C H A D S}_{2}$ score: each factor scores 1 point (2 points if previous stroke/TIA). Low risk is score 0 , intermediate risk is score 1 and high risk is score $\geq 2$.
(1) AF carries a risk of embolic stroke of $2-4 \%$ per year. Warfarin therapy can reduce this by around $60 \%$, compared to $30 \%$ reduction with aspirin alone. Those at high risk should ideally be anticoagulated with warfarin; those at low risk can simply be treated with aspirin (75-300 mg), while intermediate-risk patients can be offered the choice.

Q 7. What drug is used for anticoagulation, how is it monitored and what is the target range?

A 1 mark each for the following:

- Warfarin
- International normalised ratio (INR) (normal range is $\mathbf{0 . 9 - 1 . 2}$ ). It is calculated by comparing the prothrombin time (PT) of the patient with a standard value
- 2-3

Total:
15 marks

