

>Question #1

A 43-year-old lady with a history of schizophrenia presents to the emergency department with palpitations, headaches and dizziness for 3 days. She says that she can feel her heart pounding after which she becomes dizzy and feels faint. She has noted these bouts about three to four times per day. She does not suffer from diabetes and is not hypertensive.

She was diagnosed as having a UTI 4 days ago which is currently being treated with ciprofloxacin. Her medication history includes olanzapine and occasional paracetamol for her headaches.

On examination, her pulse was 135 bpm with an irregular rhythm. Blood pressure was 90/60mmHg. Attachment to a cardiac monitor revealed runs of ill sustained polymorphic tachycardia. She was immediately given IV lidocaine to which there was no response.

Which is the most appropriate next step in management?

- a) Immediate DC cardioversion
- b) IV amiodarone
- c) IV flecainide
- d) IV magnesium sulphate
- e) IV labetalol

Correct answer is d.

The scenario above is an acquired long QT syndrome secondary to both ciprofloxacin and olanzapine. Patients with long QT syndromes develop syncope and palpitations as a result of polymorphic ventricular tachycardia (torsades de pointes). Episodes may terminate spontaneously which is usual, but may also evolve into fatal ventricular fibrillation. The corrected QT interval in between the arrhythmia is usually >0.5s.

>Question #2

A 71-year-old patient presents to the Emergency Department with a 30 minute history of crushing central chest pain. ECG shows tall R waves in V1-2. Which coronary territory is likely to be affected?

- a) Lateral
- b) Posterior
- c) Anteroseptal
- d) Anterolateral
- e) Inferior

Correct answer is b.

>Question #3

A 54-year-old male business executive is referred to you after a heart murmur is detected at a medical examination he received after he transferred to a new company. He has no known past medical history or family history. He is well and leads an active lifestyle. On examination, you note a pansystolic murmur in the apex. Chest auscultation is unremarkable. An ECG demonstrates sinus rhythm at 64 beats/minute with no changes suggestive of ventricular hypertrophy. A transthoracic echocardiogram demonstrated good views with severe mitral regurgitation, preserved left ventricular function (EF 85%) and pulmonary arterial systolic pressure of 15 mmHg. Which of the following is appropriate management?

- a) Mitral valve replacement
- b) Mitral valve repair
- c) Percutaneous mitral valve repair (Mitraclip)
- d) Infective endocarditis prophylaxis and 6 monthly echocardiogram
- e) 6 monthly echocardiogram

Correc answer is e.

The patient has severe chronic non-ischaemic mitral regurgitation that has not decompensated. The aetiology is unclear: primary causes include mitral valve

prolapse, flail leaflet or rheumatic heart disease. Secondary causes include cardiomyopathy or coronary artery disease.

The patient does not meet **indications for intervention on the mitral valve**, which include

1. symptoms,
2. left ventricular dysfunction,
3. pulmonary hypertension,
4. new atrial fibrillation and
5. Dilated left ventricle.

It is uncommon for intervention to take place without any of the indications mentioned above. Instead, serial monitoring with 6 monthly echocardiograms is appropriate. **Infective endocarditis prophylaxis is no longer indicated for patients in the absence of a prosthetic valve repair or replacement.**

Percutaneous mitral valve repair is an emerging technique that is available to patients considered too high risk for mitral valve surgery, which uses a device to individually approximate the regurgitant leaflet. Comparing Mitraclip with mitral valve replacement or repair in severe mitral regurgitation patients with NYHA III/IV heart failure symptoms, one-year and four-year survival were similar with similar improvements in symptoms, LV size and function. However, Mitraclip patients demonstrated significantly higher incidence of requiring further surgery but significantly lower risks of major adverse events post-procedure.

>Question #4

A 60 year old man with a known history of congestive cardiac failure and asthma is reviewed in a cardiology clinic. He is noted to have a blood pressure of 95/63mmHg and a heart rate of 98bpm. An ECG confirms sinus rhythm. He has previously developed symptoms of wheeze with beta blockade. He is commenced on ivabradine 5mg twice daily by his cardiologist.

Which of the following should the patient be warned of as a recognised side effect of ivabradine?

- a) Neutrophilia
- b) Hypotension
- c) Phosphenes
- d) Diaphoresis
- e) Renal failure

Correct answer is c.

Ivabradine use may be associated with visual disturbances including phosphenes and green luminescence

Ivabradine is a second line agent for rate control in the treatment of chronic heart failure in those in whom beta-blockade is contraindicated, not tolerated or therapy fails. Other rate limiting drugs such as the calcium channel blockers diltiazem or verapamil may sometimes be used but hypotension often limits their use. Digoxin too can sometimes be used, even in sinus rhythm although the rate limiting effect it has is relatively modest; **often digoxin and ivabradine are ultimately used in conjunction.**

NICE guidelines on the use of ivabradine give clear criteria on its use; the patient must have at least a moderate degree of heart failure with a left ventricular ejection fraction of <35%, they must be stable on optimal doses of other heart failure medications including angiotensin system modulators, an aldosterone receptor antagonist and a beta blocker if not contraindicated. It is important the patient is regularly monitored by a community heart failure team to titrate the dose. Ivabradine can also only be used in patients in sinus rhythm. It works by selectively inhibiting the If [ionic funny] channel which is a sodium/potassium symporter channel largely expressed within the sinoatrial node. Inhibition of this channel slows the intrinsic rhythmicity of the hearts pacemaker function. Pharmaceutical bradycardia aids in the management of cardiac failure as it allows improved diastolic filling and reduced myocardial oxygen usage.

Side effects of ivabradine include bradycardia, ventricular escape rhythms, dizziness, headache, muscle cramps and eosinophilia. Neutrophilia is rarely seen

with ivabradine. Hypotension is also not seen with ivabradine use which makes it an important drug in the arsenal when treating patients with cardiac failure in whom other rate limiting drugs may be relatively contraindicated.

An important side effect with ivabradine however is that of visual disturbance including the phenomenon of phosphenes, or 'flashing lights'. Other visual symptoms described include green discolouration of visual field, blurring of vision and scintillating scotomata. These visual symptoms are usually transient and mild. They arise due to inhibition of similar ionic funny channels in the retina to the sinoatrial If channels. Approximately 20% of patients taking the drug develop some form of visual disturbance but only 1% need to discontinue therapy because of it.

The use of ivabradine in renal dysfunction is not well established however manufacturers recommend it may be used with estimated glomerular filtration rates above 15ml/min. There is no evidence its use causes renal dysfunction although mild rises in plasma creatinine levels may be seen.

>Question #5

A 54-year-old woman presents to her general practitioner complaining of a rash on her chest, face and upper arms. The rash is transient and seems to mostly occur in the summertime. She has a past medical history of hypertension, palindromic rheumatism and heart failure with preserved ejection fraction. She takes amlodipine, furosemide, indapamide and hydroxychloroquine.

On examination, there is a maculopapular rash on her chest and distal forearms. It is not itchy.

Bloods tests:

Antinuclear antibody	negative
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What is the likely explanation for her symptoms?

- a) Furosemide
- b) Hydroxychloroquine
- c) Indapamide
- d) Solar urticaria
- e) Systemic lupus erythematosus

Correct answer is c.

Thiazides may cause photosensitivity

Indapamide is correct. Thiazide diuretics are a well-recognised cause of a photosensitive rash. The patient's rash occurs mostly in the summertime and on sun-exposed areas and is therefore consistent with this reaction.

Furosemide is incorrect. This is mainly associated with bullous skin reactions.

Solar urticaria is incorrect. The rash, in this case, is maculopapular and non-pruritic and therefore is not consistent with a diagnosis of urticaria.

Hydroxychloroquine is incorrect. This medication is associated with rashes but is actually used to treat conditions associated with photosensitive rashes and is therefore not the likely cause.

SLE is incorrect. Photosensitive rashes are common in lupus. However, the ANA is negative, which makes a diagnosis of the systemic form of lupus, overwhelmingly unlikely.

>Question #6

A 52-year-old is brought from the renal outpatient clinic to the emergency department (ED) after appearing very short of breathing having walked up the stair. His breathlessness was associated with some mild, generalised chest discomfort, but the patient puts this down to a recent chest infection he has just

recovered from.

When assessed in the ED the patient's breathlessness has improved and he denies any further chest discomfort. He reports he has been experiencing more frequent similar episodes, gradually increasing in severity over several months. He reports he recently had a productive cough with coryzal symptoms for the last 3-4 days but he feels it is improving. On questioning he confirms his symptoms of breathlessness are often worse during and post these chest infections.

He has a past medical history of chronic renal artery stenosis and resultant poorly controlled hypertension for which he is on multiple medications including an ACE inhibitor, beta-blocker and a diuretic.

An ECG is performed as seen below.

Shows S in V1 plus R in V6= 44mm



What is the patient's most likely diagnosis?

- a) Brugada syndrome
- b) Hyperkalaemia
- c) Left ventricular hypertrophy
- d) Pericarditis
- e) Wellen's syndrome

Correct answer is c.

This patient has presented with symptoms and typical ECG changes in keeping with left ventricular (LV) hypertrophy. The thickening of the cardiac muscle is most likely a result of chronic uncontrolled hypertension, hence the prolonged, progressive history. LV hypertrophy results in signs and symptoms of LV failure including breathlessness, angina and reduced exercise tolerance. The patient's ECG demonstrates left ventricular hypertrophy with ST elevation in V2-3, without

reciprocal lead changes and the combination of the S wave in V1 with the R wave in V5 or V6 clearly exceeding 40 mm.

Brugada syndrome is a genetic cardiac disorder resulting in electrical activity disorders and not structural issues. Features include syncope and sudden cardiac death. There are several types of Brugada syndrome with variation seen on ECG however the only potentially diagnostic ECG abnormality is of coved ST-segment elevation in 2 or more of V1-3 followed by a negative T wave. This is classical of Brugada type 1 syndrome and is commonly known as Brugada sign.

There is no clear reason why this patient would be hyperkalaemia. Although he has renal impairment it is not indicated that he has end-stage renal failure or that he is on renal replacement therapy therefore hyperkalaemia would not be expected. Common ECG changes noted in hyperkalaemia included peaked T waves, P wave widening/flattening, PR prolongation progressing to the bradyarrhythmias and conduction blocks. No clear features of hyperkalaemia can be seen on this patient's ECG.

Pericarditis is inflammation of the tissue surrounding the heart and can be due to infections. Patients normally present with pleuritic, constant chest pain and fevers. Although this patient has a very recent chest infection, pericarditis normally develops a few weeks post the resolution of infection as it is an immune response and not a direct result of the infection itself. ECG findings associated with pericarditis include widespread concave ST elevation and PR depression which are not present on this patient's ECG.

Wellen's syndrome is the ECG pattern of biphasic or deeply inverted T waves in the chest leads V2-3. It is highly specific for critical stenosis of the left anterior descending (LAD) artery and therefore is normally seen in patients presenting with ischaemic like symptoms. You would expect the patient's chest pain to be continuous and to be more severe if he was experiencing stenosis of the LAD.

>Question #7

A 45-year-old woman develops severe central chest pain. An ECG in the ambulance shows ST segment elevation in leads: I, aVL, V2-6. Shortly after she

arrives in hospital the pain resolves and a second ECG is entirely normal. She has had three similar episodes of chest pain in the past. All episodes of chest pain have come on at rest. Blood pressure is 140/80 mmHg, heart rate is 90 beats per minute and hearts sounds are normal. She underwent coronary angiography following a previous episode of chest pain three weeks ago, which showed no significant coronary artery disease. An echocardiogram is normal.

What is the likely diagnosis?

- a) Acute anterolateral myocardial infarction
- b) Crescendo angina
- c) Da Costas syndrome
- d) Prinzmetal's variant angina
- e) Takotsubo cardiomyopathy

Correct answer is d.

This woman is likely to be suffering from Prinzmetal's variant angina. Classically her pain occurs at rest and the ECG demonstrates ST segment elevation that disappears as the pain abates. Normal coronary angiography supports this diagnosis, however in many Prinzmetal's angina patients there is co-existing coronary artery disease. Symptoms and ECG changes are unlikely to be reproduced with exercise testing in Prinzmetal's angina.

Prinzmetal's angina is caused by coronary artery spasm, however the underlying pathophysiology causing spasm is not currently well understood. In some patients it is associated with other vasospastic disorders such as Raynaud's phenomenon.

The main stay of treatment is:

- Avoiding precipitants of spasm such as smoking.
- Calcium channel blockers, nitrates and/or nicorandil.

>**Question #8**

A 16-year-old female presents with a swollen knee. She states that she has had several similar episodes previously. She also complains of excessive bleeding after dental work. She states that her father has a bleeding disorder. On examination her right knee is hot and swollen. A knee aspirate reveals a bloody effusion.

Blood results are as follows:

Hb	110 g/l
Platelets	682 * 10 ⁹ /l
Prothrombin time (PT)	12 s (normal 10-13)
activated partial thromboplastin time (aPTT)	54 s (normal 25-36)
Factor VIII level	2% of normal (very low)
Factor IX level	Normal
von Willebrand Factor level	Normal

What disorder does she most likely have?

- a) Down syndrome
- b) Edwards syndrome
- c) Marfan syndrome
- d) Ehlers-Danlos syndrome
- e) Turner's syndrome

Correct answer is e.

Females with Turner's syndrome have just one X chromosome. Therefore they have the same probability of being affected by an X linked recessive disease as males

The patient clearly has haemophilia A as indicated by the prolonged aPTT and severely reduced factor VIII levels.

Rare cases of females with severe haemophilia can occur due to

1. **extreme lyonization,**
2. **homozygosity,**
3. **mosaicism, or**
4. **Turner syndrome.**

Females with Turner's syndrome have just one X chromosome. Thus they have the same probability of being affected by an X linked recessive disease as males. For the aforementioned reason, Turner's syndrome is the most likely underlying diagnosis out of the given options.

>Question #9

A 75-year-old man is an inpatient on the orthopaedic ward recovering from an elective knee replacement performed three days previously. The patient's immediate post-operative recovery had been unremarkable and he had begun to mobilise with the ward physiotherapist. Routine observations recorded on the ward had been unremarkable during the previous 24 hours.

During her drug round, the patient's nurse saw the patient suddenly become unable to breathe and clutch at his chest. After calling for help the nurse went to her patient and found him to be in cardiac arrest. Resuscitations attempts were initiated following advanced life-support protocol. Please see the below table for a summary of the patient's electrical rhythm and treatments administered during the initial phases of the resuscitation attempt.

The patient was noted to be in good physical health with his only comorbidity being hypertension, well controlled with medication. The patient's admission clerking recorded that he was a retired schoolteacher who lived independently at home with his wife. A review of the patient's drug chart indicated that he had

been receiving subcutaneous enoxaparin as prophylaxis against venous thromboembolism, but that the patient had refused to wear compression stockings during his admission as he found them uncomfortable.

Number of rhythm check	Result of rhythm check	Treatment administered
1	Pulseless electrical activity	IV adrenaline 1 mg
2	Ventricular fibrillation (coarse)	DC shock 150 J
3	Ventricular fibrillation	???

In addition to a further DC shock, what is the appropriate choice of IV drug treatment following the third rhythm check?

- a) IV adrenaline 1 mg
- b) IV atropine 400 micrograms & IV amiodarone 300 mg
- c) No IV drug treatment indicated
- d) IV adrenaline 1 mg & IV amiodarone 300 mg
- e) IV atropine 400 micrograms

Correct answer is a.

The advanced life support algorithm for pulseless electrical activity (PEA) and asystole requires adrenaline to be given immediately and then continued every 3 to 5 minutes (in practice, after every second rhythm check). By contrast, the algorithm for ventricular fibrillation and ventricular tachycardia (VF / VT) requires IV adrenaline to not be given until after the third shock and then continued every 3 to 5 minutes.

However, once adrenaline has been given during a resuscitation attempt it should be given every 3-5 minutes even if the type of rhythm changes from PEA/asystole to VF / VT, even if this is only the second cycle of the rhythm being VF / VT (as in

the example in the question).

As part of the VF / VT algorithm, IV amiodarone 300 mg should be given after three defibrillation attempts. A further dose of IV amiodarone 150 mg should be considered after a total of five defibrillation attempts. Atropine is not used as part of the advanced life-support algorithm.

>Question #10

A 64-year-old man presents to his general practitioner (GP) with several months of chest pain that occurs only on exertion. When he pauses to rest, the pain dissipates within a few minutes. He has a past medical history of high cholesterol, for which he takes atorvastatin. The GP commences him on bisoprolol.

Several weeks later, the patient re-attends, stating that the symptoms are still not under control.

What should be added next?

- a) Amlodipine
- b) Isosorbide mononitrate
- c) Ivabradine
- d) Nicorandil
- e) Verapamil

Correct answer is a.

If angina is not controlled with a beta-blocker, a longer-acting dihydropyridine calcium channel blocker should be added

This patient is experiencing stable angina - the chest pain is occurring on exertion, but not at rest. NICE recommend the use of a beta-blocker or calcium channel blocker (CCB) first-line. As this patient has been commenced on bisoprolol already, the next step is to add a CCB. A dihydropyridine CCB should be used, to avoid reducing the heart rate further, and so **amlodipine** is the most appropriate option here.

Isosorbide mononitrate would be appropriate to use as a third agent, if the combination of a beta-blocker and CCB was not adequate. However, at this stage, a CCB should be added.

Similarly, **ivabradine**, which modulates the 'funny current', would be appropriate as a third agent instead of isosorbide mononitrate. A CCB should be added first, as this patient is only on bisoprolol.

Nicorandil is another alternative agent to be added third, instead of isosorbide mononitrate or ivabradine. It would not be appropriate to add currently, as the patient is only on bisoprolol.

Verapamil is a non-dihydropyridine CCB. As such, it is negatively chronotropic and so should not be used alongside a beta-blocker; this may precipitate complete heart block. It would have been suitable as a first-line alternative to the beta-blocker.

>**Question #11**

An 89-year-old gentleman presented to the general medical clinic with complaints of palpitations. He was diagnosed with atrial fibrillation (AF) three months ago and started on bisoprolol. This was part of a plan to rate control, rather than rhythm control, his AF. The bisoprolol has been up-titrated as his symptoms did not come under control with his dose. Palpitations occur almost every day but are not associated with any other symptoms. He also has a history of ischaemic heart disease with a myocardial infarction five years ago and type 2 diabetes mellitus. He currently takes bisoprolol, metformin, aspirin and simvastatin. He has declined anticoagulation with warfarin or NOAC despite the risk as his wife died from an intracranial bleed whilst taking warfarin.

On examination, his heart rate is 94/min and irregular. His chest is clear on auscultation and there is no peripheral oedema. How should he be further managed?

- a) Amiodarone
- b) Dronedarone
- c) Amlodipine
- d) Diltiazem
- e) Left atrial ablation

Correct answer is d.

The correct answer is diltiazem. This is a gentleman with symptomatic, but not decompensating, permanent AF which is not responding to first-line treatment with a beta-blocker (bisoprolol). **NICE advises that first line treatment for a rate-control strategy in AF should be either a beta-blocker or a rate-limiting calcium channel blocker, but digoxin can be considered for non-paroxysmal AF in patients who are not very active. If the first-line treatment fails, either with continuing symptoms or poor response of ventricular rate then a combination therapy with any of the following two can be used: beta-blocker, diltiazem, digoxin.**

Amiodarone, dronedarone and left atrial ablation are all strategies for cardioversion. Amlodipine is a calcium channel blocker but is used in hypertension rather than in AF as it is not rate-limiting, and would therefore not help.

>Question #12

A 29-year-old man is admitted to the cardiology ward following a collapse whilst playing football. He has no recollection of events other than running on the pitch and waking up in the ambulance. In the last 2-3 months, he has been complaining of intermittent palpitations but put this down to work-related stress. There is no history of chest pain and he has no past medical history of note.

On auscultation, chest sounds are clear and heart sounds are normal. A bedside echocardiogram demonstrates a hypokinetic right ventricle.

Given the likely diagnosis, what is the most characteristic finding on this patient's

ECG?

- a) Left ventricular hypertrophy
- b) Positive deflection at the J point
- c) Right bundle branch block
- d) Slurred upstroke in QR
- e) Small positive deflection at the end of QRS complex

Correct answer: e

The most characteristic ECG finding in arrhythmogenic right ventricular dysplasia (ARVD) is the epsilon wave (a small positive deflection at the end of the QRS complex)

This patient has arrhythmogenic right ventricular dysplasia (ARVD), a type of inherited cardiovascular disease. ARVD can present as palpitations, syncope, or sudden cardiac death similar to hypertrophic obstructive cardiomyopathy (HOCM). Over time, the right ventricular myocardium is replaced by fibrofatty tissue and patients develop signs of right ventricular failure. This patient's echocardiogram finding of a hypokinetic right ventricular wall is suggestive of the diagnosis. The most characteristic ECG finding in ARVD is an **epsilon wave**. An epsilon wave is a positive deflection following the end of the QRS complex caused by post-excitation of right ventricular myocytes.

Left ventricular hypertrophy is a common ECG finding of HOCM. HOCM is the most common cause of sudden cardiac death and can also present with palpitations and syncope, as seen in this patient. However, this patient's echocardiogram findings are in keeping with ARVD, of which the epsilon wave is the most characteristic ECG finding.

An Osborn wave (or J wave) is a positive deflection at the J point. There are multiple causes, of which the most common is hypothermia. Other causes of J waves include left ventricular hypertrophy and Brugada syndrome. However, an Osborn wave is not associated with ARVD.

There are multiple causes of right bundle branch block, including right

ventricular hypertrophy. In ARVD, rather than hypertrophy of the right ventricle, the myocardium is replaced by fatty tissue. Right bundle branch block is not a sign of ARVD. Instead, the left bundle branch block is more likely to feature on an ECG of a patient with ARVD.

A **delta wave** is a slurred upstroke in the QRS complex that is commonly associated with pre-excitatory activity, such as that seen in Wolff-Parkinson White syndrome. It is not associated with ARVD.

>Question #13

A 72-year-old man is admitted to hospital with shortness-of-breath. On examination his pulse is 96/min, BP 100/64 mmHg, respiratory rate 20/min, temperature 37.5°C, oxygen saturations 96% on room air. A 12-lead ECG shows sinus rhythm, at a rate of 94/min with no diagnostic ST-T changes. The troponin I level is < 0.05 µg/L. A CT chest (with contrast) is ordered:



What is the most likely diagnosis?

- a) Superior vena cava obstruction
- b) Aortic dissection (Stanford type A)
- c) Infective endocarditis
- d) Aortic dissection (Stanford type B)
- e) Pulmonary embolism

Correct answer is e.

The CT shows a large saddle embolus where the pulmonary trunk splits to form the right and left pulmonary arteries.

Most image-based questions can be answered without looking at the image. If we therefore concentrate on the history a few clues point towards a pulmonary embolism (PE). The first is tachypnoea. Around 95% of patients with a PE have a respiratory rate $> 16/\text{min}$. Tachypnoea is not as common in any of the other diagnoses. A low-grade temperature is also an under appreciated sign of pulmonary embolism. This could of course be consistent with infective endocarditis but there are no other features to support this diagnosis.

>**Question #14**

You are the on-call medical doctor called to review a patient in the Emergency Department. A 54 year old male patient with a past history asthma, ischaemic heart disease and transient ischaemic attack has presented with palpitations. His admission ECG shows a regular, narrow complex tachycardia. Vagal manoeuvres have been tried in the department with no success. You decide to give intravenous adenosine in an attempt to chemically cardiovert. An initial dose of 6mg is given into a proximal vein with a large flush. The patient soon loses consciousness and an 11 second ventricular standstill is noted on the rhythm strip before slow return of sinus rhythm.

Which of the patient's medications is most likely to be responsible for this?

- a) Phallocontin

- b) Dipyridamole
- c) Bisoprolol
- d) Simvastatin
- e) Montelukast

Correct answer is b.

The effects of adenosine are enhanced by dipyridamole

Adenosine is a useful medication in the investigation and treatment of regular narrow complex tachycardias. With reference to the Resuscitation UK guidelines, it has a place as a second line measure to vagal manoeuvres when no 'adverse features' are present. Adenosine works by transiently blocking the AV node by effect on the A1 receptor, causing potassium efflux and hyperpolarisation.

Don't be side-tracked by the fact that this patient is asthmatic, yes this is a relative contraindication to the administration of adenosine, but that is not the question. Theophyllines have been known to antagonise adenosine necessitating higher dosages. **Dipyridamole blocks the cellular uptake of adenosine, increasing concentration at receptors and potentiating its effect.** Adenosine should therefore be used with caution, if at all, in patients taking dipyridamole, and certainly low starting doses should be considered.

>Question #15

A 78-year-old lady presents with syncope. She states that she has had several episodes of light headedness over the past couple of months. There are no obvious precipitants of the dizzy spells. She has a past medical history of angina, COPD and hypertension. Her regular medicines include GTN, aspirin, ramipril and furosemide.

ECG results are as follows:

Rate	68 beats per minute
Rhythm	Sinus rhythm

PR interval	220 ms
QRS duration	130 ms
QRS morphology	Right bundle branch block
Cardiac axis	Left axis deviation

What investigation will you order?

- a) 7-day ECG Holter monitor
- b) Transthoracic echocardiogram
- c) Transesophageal echocardiogram
- d) Brain natriuretic peptide (BNP)
- e) Tilt-table test

Correct answer is a.

Patients who present with syncope and have an ECG showing incomplete trifascicular block need a Holter test to assess for episodes of complete heart block

The right bundle branch block (RBBB), left axis deviation (LAD) and prolonged PR interval are suggestive of incomplete trifascicular block. These patients require a 7-day ECG Holter monitor to assess for episodes of complete (third degree) heart block. It is likely that a permanent cardiac pacemaker will be required to prevent further syncopal episodes.

A tilt-table test is useful for investigating vasovagal syncope.

The patient may also have underlying structural heart disease so a transthoracic echo would also be prudent to perform. However, the most immediate concern is of arrhythmia, making a 7 day holter the better answer.

>Question #16

A 78-year-old man who is usually fit and well and takes levothyroxine for hypothyroidism presents to the acute medical take after two episodes of syncope. He recalls sitting at the dinner table and passing out with no dizziness prior. He can usually walk 100 meters at a time.

On examination, he has a loud systolic murmur heard over the 2nd intercostal space on the left side radiating to the carotid. He has a slow rising pulse and his chest is clear. No JVP is seen and there is no ankle oedema.

Chest x-ray	enlarged heart with calcification of the aortic knuckle
ECG	sinus, 75 beats per minute
ECHO	Aortic valve cross sectional area of 0.8mm^2 with a pressure gradient of 42mmHg, cusps appear calcified and poorly mobile

What other investigation should be performed?

- a) Coronary angiography
- b) Thyroid function test
- c) CT chest
- d) Treadmill ECG testing
- e) HbA1c

Correct answer is a.

Aortic stenosis co-occurs with atherosclerotic disease -> perform angiogram prior to surgical intervention

This gentleman meets the ECHO criteria for severe aortic stenosis and should be listed for aortic valve replacement (AVR) immediately. He shows no signs of failure or haemodynamic compromise and is therefore fit. Given that AVR involves opening the chest, it is a perfect time to perform coronary artery bypass grafting

and since there is a correlation of aortic stenosis with atherosclerosis, it is beneficial to perform an angiogram prior to the AVR procedure. The patient effectively gets two procedures for one. CT chest would be important if he had respiratory disease. HbA1c and thyroid function tests are not essential unless there was a suspicion that these were abnormal. Treadmill testing is contraindicated in symptomatic and severe aortic stenosis where it can cause syncope.

>Question #16

A 72-year-old female presents with a 6-month history of gradual onset, progressive exertional dyspnoea associated with bilateral increasing lower limb pitting oedema. She has no previous cardiac history and prior to 6 months ago, had no limitations to exercise tolerance. Her past medical history includes B cell lymphoma diagnosed four years ago and in remission after one course of chemotherapy. On examination, the jugular venous pulse is raised at 6cm above the angle of Louis. A soft systolic murmur and bibasal inspiratory crackles can be heard on auscultation. Abdominal examination demonstrates a pulsatile 3cm liver edge. Her blood pressure is 125/77 mmHg, heart rate 68/min, saturations 92% on 2 Litre Oxygen, respiratory rate 22/min. ECG reveals sinus rhythm of low voltage. Chest radiography demonstrates bibasal alveolar shadowing with prominent upper lobe vasculature.

Urine dip is as follows: blood -ve, leucocytes -ve, nitrates -ve, protein 3+, pH 5.5. Blood tests are as follows:

Na ⁺	139 mmol/l
K ⁺	4.9 mmol/l
Urea	7.4 mmol/l
Creatinine	140 µmol/l

CRP	3 mg/l
Trop	<0.03 (normal range < 0.03)

Bilirubin	6 µmol/l
ALP	32 u/l
ALT	260 u/l

A transthoracic echocardiogram is performed, reported as significant increase in echogenicity, symmetrical left ventricular wall thickening, impaired bilateral diastolic ventricular dysfunction, bilaterally enlarged atria, pulmonary artery systolic pressure 45 mmHg, a 5mm pericardial effusion.

What is the most likely underlying diagnosis for this patient?

- a) Amyloid cardiomyopathy
- b) Non-ST elevation myocardial infarction (NSTEMI)
- c) Idiopathic dilated cardiomyopathy
- d) Chemotherapy induced cardiomyopathy
- e) Viral pericarditis leading to tamponade

Correct answer is a.

A clinical picture of decompensated congestive cardiac failure is described with subtle details of hepatomegaly (possibly congested, possibly related to the underlying diagnosis), associated heavy proteinuria, echogenic TTE and low voltage criteria ECG, on a background of previous B-cell lymphoma. The unifying diagnosis is amyloid cardiomyopathy, likely secondary to AL amyloid from B-cell dyscrasia, with cardiac, renal and possibly liver involvement. There is no evidence of a sudden onset cardiac event suggestive of an ischaemic cause. Echocardiogram does not demonstrate ventricular dilation, only impaired function. A 5mm pericardial effusion is not sufficiently significant to cause tamponade. Although

chemotherapy-induced cardiomyopathy is possible, it is difficult to tie in the delayed onset from the end of treatment with renal involvement. It also does not explain the echogenic pictures on transthoracic echo.

>Question #18

A 35-year-old woman presented to the emergency department with intermittent palpitations, breathlessness, and non-specific chest discomfort. The previous day she had found out that she was 14-weeks pregnant which had caused significant stress and anxiety. She has a long history of anxiety and depression, managed by cognitive behavioural therapy. She reported that she had intermittently had episodes of palpitations and shortness of breath for the past 10 years and had attributed this to her anxiety. These symptoms had worsened over the past couple of weeks and today her symptoms were intolerable. Her medications consisted of over-the-counter vitamin supplements.

She looked anxious, but otherwise, the examination was unremarkable.

An ECG was performed showed normal sinus rhythm, T wave inversion in V1 - V3.

Whilst in the emergency department, she complained of severe palpitations and dizziness.

Her ECG was repeated showed, wide complex regular tachycardia with left bundle branch block (LBBB) pattern. Heart rate 150 bpm. Fusion beats and capture beats present.

As she was being transferred into resus, she spontaneously cardioverted back to sinus rhythm.

An echocardiogram was performed:

The right ventricle is dilated with depressed right ventricular systolic function and localised apical aneurysm. Left ventricular size and function are normal.

What is the most likely cause of her presentation?

- a) Aortic dissection

- b) Takutsubo cardiomyopathy
- c) Peripartum cardiomyopathy
- d) Arrhythmogenic right ventricular cardiomyopathy
- e) Brugada syndrome

Correct Answer: d

Arrhythmogenic right ventricular cardiomyopathy - T wave inversion in V1-3.

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is characterised by the replacement of the myocardium of the right ventricle with fibrofatty tissue. The abnormal fibrofatty tissue predisposes to ventricular tachycardia (VT), right ventricular systolic dysfunction, and can cause sudden death. It is often inherited but can be sporadic. Presentation is often with nonspecific palpitations, chest discomfort, and syncope. This patient has a typical presentation: the pregnancy-associated increase in stroke volume is likely the trigger for her symptoms recently worsening. Her two ECGs are characteristic: typical changes include inverted T waves in the precordial leads V1-V3 (looking at the right ventricle) with episodes of ventricular tachycardia originating in the right ventricle (giving the ECG a LBBB morphology as the arrhythmia takes an abnormal route of conduction from right to left through slowly-conducting myocardium rather than conducting tissue). Epsilon waves may also be seen in ARVC. Her echocardiogram is also characteristic - infiltration and thinning of the right ventricular wall can occur to the point that systolic function is impaired and an aneurysm forms.

Note that the differential for a wide-complex regular tachycardia on an ECG includes SVT with aberrant conduction (as well as VT). However, the fusion beats and capture beats are more in keeping with VT in this case.

This patient's presentation is not typical for aortic dissection. An echo might see cardiac tamponade or new aortic regurgitation if this was the diagnosis but this is not the case here.

Takutsubo cardiomyopathy is caused by transient systolic dysfunction of the left ventricle (LV), presenting similarly to an acute coronary syndrome. Patients typically have chest pain, a raised troponin, and ECG findings consistent with ischaemia such as ST elevation/T wave inversion. This is typically triggered by

recent physical or emotional stress. Patients are initially managed as an ACS but cardiac angiography shows no significant coronary artery disease. Instead, the LV wall shows hypokinesis or akinesis which creates the shape of an 'octopus pot' during systole. It may be argued that this patient has experienced a recent stressor (the news of pregnancy) however her presentation, long history of symptoms, and echo findings are more in keeping with ARVC.

Peripartum cardiomyopathy (PPCM) is a rare type of cardiomyopathy that is often dilated and occurs in late pregnancy or early postpartum (typically one month before or after birth). The aetiology is unclear but is thought to be multifactorial. Criteria for diagnosis of PPCM include impairment of LV systolic function (LVEF <45%) which was not the case in this patient. Furthermore, this patient presented in the early second trimester - too early for PPCM but late enough for pregnancy-related haemodynamic changes to trigger her underlying ARVC.

Lastly, Brugada syndrome is another important differential to consider in young patients with palpitations, ECG changes in V1-V3, and episodes of VT. Brugada is caused by an inherited mutation in the cardiac sodium channel. However, echocardiography is typically normal and an ECG may show unusual ST elevation in V1-V3 with T wave inversion in these leads.

>**Question #19**

You are called to review a 65-year-old man who was admitted following a pre-syncopal episode. He has a history of previous NSTEMI 3 years ago. His nurse explains he is feeling lightheaded and feels his heart racing in his chest.

On arrival, he appears sweaty and unwell. He opens his eyes to pain, localises to pain, and makes incomprehensible sounds. His pulse is palpable at 180 bpm with BP 88/54 mmHg.

ECG demonstrates a wide complex monomorphic tachycardia with prolonged QRS 150msec.

What is the most appropriate next step?

- a) Adrenaline
- b) Amiodarone

- c) Magnesium sulphate
- d) Synchronised DC cardioversion
- e) Unsynchronised DC cardioversion

Correct answer is d.

A synchronised cardioversion is the treatment for an unstable patient in VT

Synchronised DC cardioversion is the preferred treatment for tachyarrhythmia in unstable patients displaying life-threatening adverse signs. In this situation, unstable VT with a pulse should be managed with synchronised DC cardioversion. To convert atrial or ventricular tachyarrhythmias, the shock must be synchronised to occur with the R wave of the ECG. An unsynchronised shock could coincide with a T wave and cause ventricular fibrillation (VF).

Adrenaline 1mg 1:10000 is used in the ALS algorithm in the treatment of unconscious patients with shockable and non-shockable rhythms. In the ALS algorithm, shockable rhythms include pulseless VT and VF. In this case, the patient is unconscious with VT and a pulse can be felt.

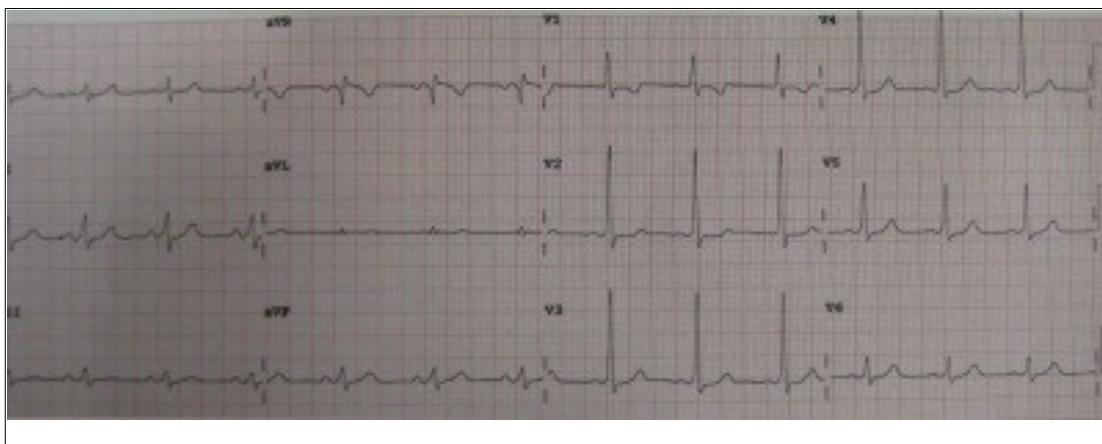
While **amiodarone** can be used to treat ventricular tachycardia, it would not be the most appropriate next step here, as this patient has signs of unstable VT, which must be managed with synchronised DC cardioversion in the first instance. Amiodarone is also used as part of the ALS algorithm for the treatment of patients with pulseless VT or VF. However, given the palpable pulse, synchronised DC cardioversion would be more appropriate here.

Magnesium sulphate is the treatment of choice in broad-complex polymorphic tachycardia. Given this patient is unstable with a monomorphic VT, synchronised DC cardioversion would be the most appropriate next step.

Unsynchronised DC cardioversion would be used in the treatment of patients with pulseless VT or VF. Given the palpable pulse, synchronised DC cardioversion would be more appropriate.

>Question #21

A 26-year-old female presents with a third episode of palpitations associated with shortness of breath and chest discomfort. She has no other past medical history, thyroid function tests unremarkable. She denies taking any recreational drugs and has no significant family history. Her ECG is as follows:



Which of the following drugs would be safe to administer immediately if she becomes tachycardic?

- a) Adenosine
- b) Digoxin
- c) Diltiazem
- d) Verapamil
- e) Procainamide

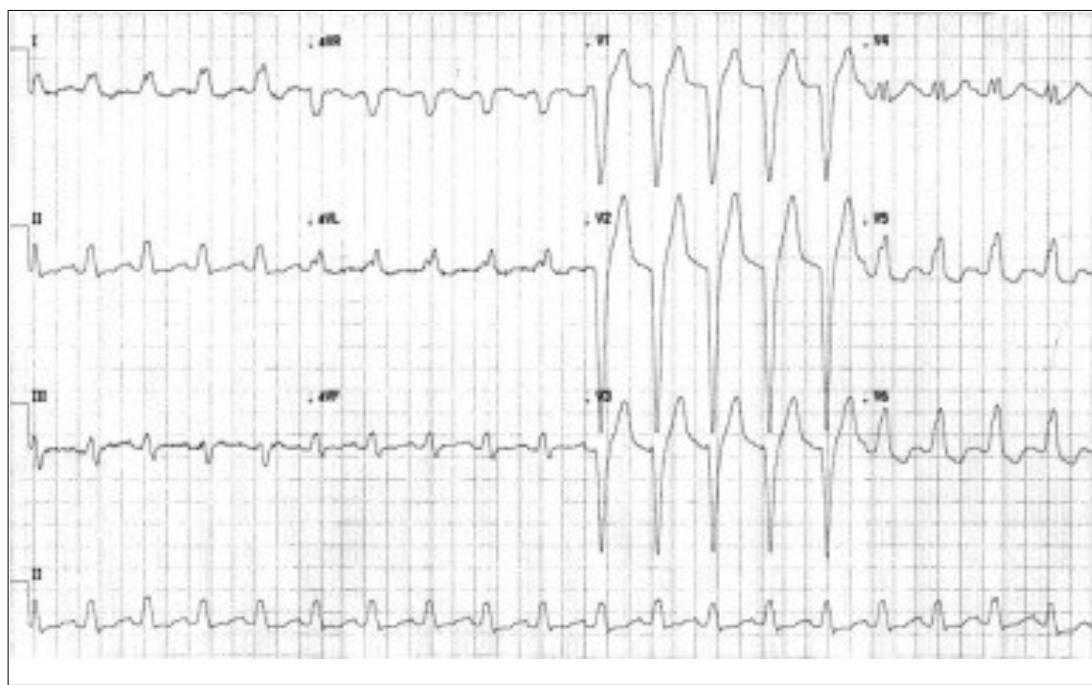
Correct answer is e.

The ECG demonstrates Wolff-Parkinson-White (WPW) syndrome, with a delta wave upstroke as a result of pre-excitation prior to the QRS complex. WPW is an atrioventricular re-entry tachycardia, with an accessory pathway allowing rapid conduction between the two channels, bypassing the slow-conducting AV node. Any drug that increases the delay of the normal conduction pathway via the AV node increases conduction via the accessory pathway, potentially inducing

ventricular fibrillation or tachycardias. The only drug that does NOT act on the AV node in this setting is procainamide.

>Question #22

A 72-year-old woman presents with palpitations. Her past medical history includes ischaemic heart disease and chronic obstructive pulmonary disease. Her pulse is 120/min, blood pressure 110/76 mmHg and the chest is clear on auscultation. The ECG is shown below:



What is shown on the ECG?

- a) Left bundle branch block
- b) Anterior ST elevation myocardial infarction
- c) Right bundle branch block
- d) Narrow complex tachycardia
- e) Atrial flutter

Correct answer is a.

The morphology of the QRS complexes is diagnostic for left bundle branch block

(LBBB). This finding can be associated with a wide variety of underlying problems but in an acutely unwell patient myocardial ischaemia needs to be excluded.

>Question #23

A 46-year-old man is referred to the acute medical assessment unit with a fever of 38.9 °C. He reports a three-week history of general malaise, reduced appetite, intermittent sweating, and occasional rigors. The patient has no medical history and has had no previous operations or procedures. He denies any drug allergies. His observations are:

- Respiratory rate: 24/min
- Oxygen saturation: 95% on room air
- Heart rate: 105bpm
- BP: 101/62mmHg
- Temperature: 38.9 °C
- GCS: 15/15

On examination, the patient has a loud early diastolic murmur over the right second intercostal space. Splinter haemorrhages are noted in three of his fingernails.

What is the most appropriate antibiotic to start?

- a) Amoxicillin
- b) Benzylpenicillin
- c) Flucloxacillin
- d) Vancomycin + low-dose gentamicin
- e) Vancomycin + low-dose gentamicin + rifampicin

Correct answer is a.

IV amoxicillin is the empirical treatment of choice in native valve endocarditis

Fever + a new cardiac murmur raises suspicion of infective endocarditis. A loud early diastolic murmur over the right second intercostal space is suspicious for

aortic regurgitation secondary to endocarditis. This will cause the patient's shortness of breath.

Amoxicillin is the correct answer. Given the patient has no medical or surgical history, he should be treated for native valve endocarditis by starting high-dose intravenous amoxicillin. Ideally, six sets of blood cultures should be sent, with as many as possible before the initiation of treatment (although this should not delay treatment if the patient is unstable).

Benzylpenicillin is the incorrect answer. Benzylpenicillin is the treatment for infective endocarditis caused by a fully sensitive *Streptococcus* organism.

Flucloxacillin is the incorrect answer. Flucloxacillin is used to treat infective endocarditis caused by a fully sensitive *Staphylococcus* (usually *Staphylococcus aureus*.)

Vancomycin + low-dose gentamicin is the incorrect answer. Vancomycin + low-dose gentamicin can be used for infective endocarditis in penicillin-allergic patients or patients with methicillin-resistant *Staphylococcus aureus* (MRSA) infective endocarditis.

Vancomycin + low-dose gentamicin + rifampicin is the incorrect answer. Vancomycin + low-dose gentamicin + rifampicin is the treatment for infective endocarditis on a prosthetic valve. This patient has no history of valve replacement.

>Question #24

A 30-year-old man presents with a fever. He complains that he has had a fever, headache and lethargy for three weeks. On examination, his temperature is 38.5C. You notice a healed human bite mark on his right forearm, he tells you he was bitten in a pub brawl about a couple months ago. Diastolic and systolic murmurs are heard on auscultation. A trans-oesophageal echo demonstrates an oscillating mass on a bicuspid aortic valve, with aortic regurgitation. Three blood cultures are taken before administration of empirical antibiotics, two grow small colonies of

tiny pleomorphic gram-negative bacilli.

What is the likely causative organism?

- a) *Eikenella corrodens*
- b) *Escherichia coli*
- c) *Phoenicoparrus andinus*
- d) *Staphylococcus aureus*
- e) *Streptococcus viridans*

Correct answer is a.

This patient fits the Duke criteria for infective endocarditis as there is:

echocardiographic evidence of an intra-cardiac mass associated with a valve, a fever of >38 and two blood cultures of an endocarditis causing microorganism. Additionally, this patient's bicuspid aortic valve is a risk factor for developing endocarditis.

***Eikenella corrodens* is a gram-negative bacilli, which is a commensal of the human mouth.** It is a member of the HACEK group, an acronym for a group of organisms that can cause gram-negative endocarditis (Haemophilus species, *Actinobacillus actinomycetemcomitans*, *Cardiobacterium hominis*, *Eikenella corrodens*, and *Kingella* species.) **The human bite injury and gram-negative culture make *Eikenella corrodens* the most likely causative organism.**

Streptococcus viridans and *Staphylococcus aureus* are commoner causes of infective endocarditis, which are easily cultured and are gram-positive.

Escherichia coli could cause endocarditis and is a gram-negative bacilli, but is not the most likely in this case.

Phoenicoparrus andinus is the binomial nomenclature for the rare Andean Flamingo, which are not known to cause infective endocarditis.

>**Question #25**

A 67-year-old male presents with increasing chest pain on exertion over the past 3 weeks, stopping him from going to work. His past medical history includes hypertension, type 2 diabetes mellitus, sick sinus syndrome, recent abstinence from alcohol after a history of an episode of acute liver decompensation 9 months ago and stable angina. He previously tried isosorbide mononitrate but reported significant headaches and facial flushing. He has not previously suffered myocardial infarctions, with a recent echo demonstrating 70% ejection fraction. An ECG demonstrates first-degree heart block with normal QRS complexes at 50 beats/ minute, blood pressure is 140/76 mmHg. He currently takes bisoprolol 5mg OD alone and has been using his GTN spray with increasing frequency without effect.

What is the most appropriate next step in management?

- a) Reperfusion therapy with coronary artery bypass graft or percutaneous coronary intervention
- b) Ranolazine
- c) Ivabradine
- d) Diltiazem
- e) Nicorandil

Correct answer is e.

NICE recommends the routine prescription of a short-acting nitrate for stable angina, followed by either a beta blocker or calcium channel blocker as first-line therapy with insufficient symptomatic control. Next, NICE recommends combining both beta blockers and calcium channel blockers. Patients requiring more than 2 antianginals should be considered for reperfusion therapies, with the addition of a third drug only if the patient is not a candidate for PCI or CABG.

In this case, the patient has first-degree heart block with relative bradycardia and calcium channel blockers would be contraindicated. NICE recommends the addition of either ranolazine, nicorandil, ivabradine or a long-acting nitrate. **Sick sinus syndrome would be a contraindication to ivabradine and calcium channel blocker. In addition, alcohol excess and recent decompensation, despite current abstinence, is likely to have resulted in liver dysfunction, an absolute contraindication to ranolazine. Nicorandil is only contraindicated in LV failure**

and cardiogenic shock, acting as a potassium channel opener, and is thus the only appropriate anti-anginal in this scenario.

>Question #26

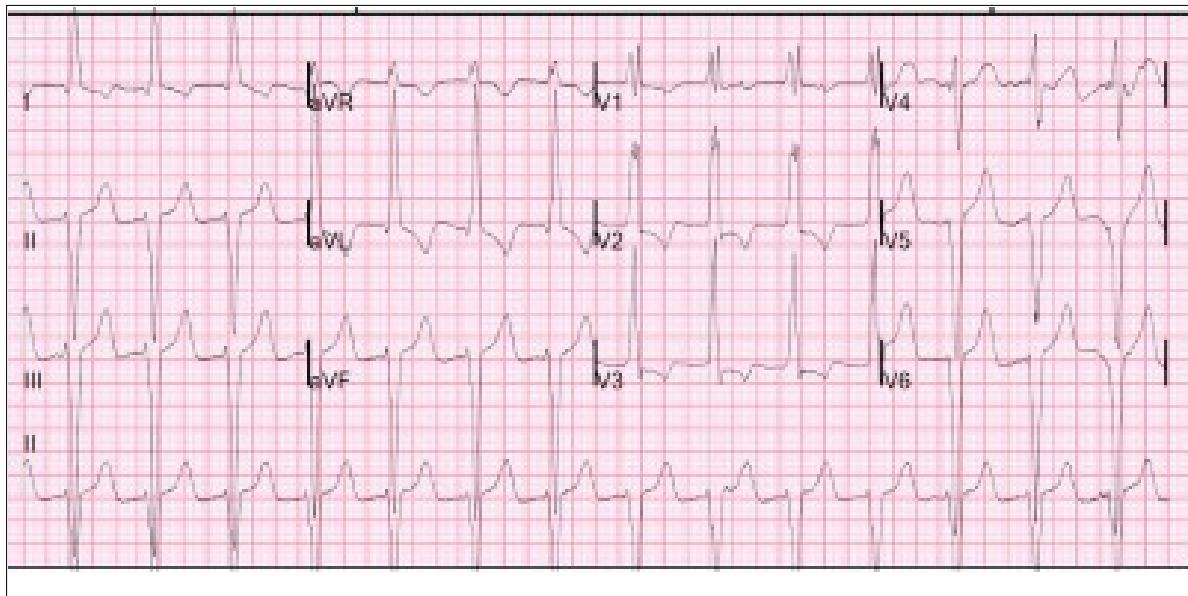
A 70-year-old presents to the outpatients' department having been referred by his GP with on-off palpitations for several months. The patient has a myocardial infarct over 20 years ago which was successfully managed via thrombolysis but otherwise, he has no significant past medical history.

The patient reports he has spontaneous episodes of palpitations approximately once every 2 weeks, and occasional feeling faint. He fainted once when walking quickly for the bus and so now takes it slowly when exerting himself and has no further similar episodes. He is otherwise very well and denies any episodes of chest pain etc.

He is on secondary preventive medication including:

- Aspirin low dose
- Bisoprolol 10mg twice daily
- Atorvastatin 40mgs once daily
- Ramipril 10mg once daily

His examination and observation are normal and an ECG performed is shown below.



What is the most appropriate management plan for the patient?

- a) Admit for urgent ablation workup
- b) Admit for urgent pacemaker workup
- c) No additional management is required
- d) Reduce the dose of his beta-blocker
- e) Arrange for urgent 24-hour Holter monitor

Correct answer is b.

The patient's ECG show trifascicular block with a right bundle branch block (RBBB), left anterior hemiblock and 1st-degree heart block. This with the history of pre-syncope and syncope is concerning and is an indication for admission and pacemaker workup as the patient is at risk of further significant arrhythmias or cardiac blocks.

Trifascicular block is a ventricular escape rhythm that usually arises from the left anterior or posterior fascicle regions following ischaemic heart disease or structural heart disease. Symptomatic trifascicular block is an indication for implantable cardiac pacemaker or permanent pacemaker work up.

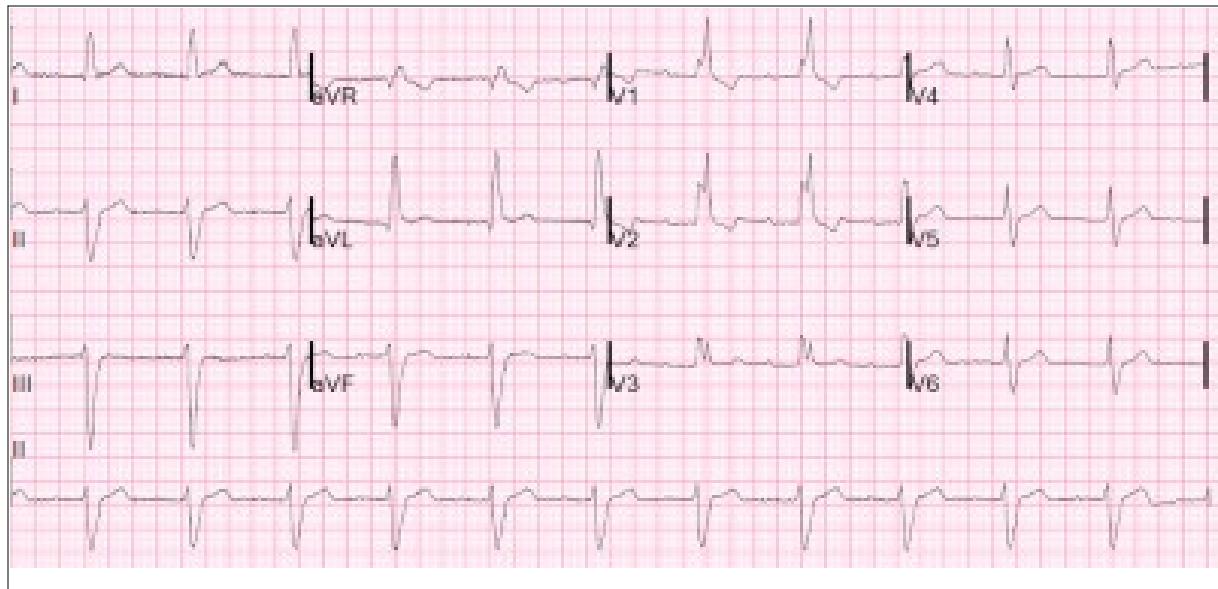
The patient has concerning features of symptomatic trifascicular block with pre-syncope and syncope on exertion which warrants urgent assessment for a pacemaker.

The patient's trifascicular block is very unlikely to be associated with his beta-blocker medication and therefore reducing the dose will not remove the risk of further significant arrhythmias or cardiac blocks.

Arranging for an urgent 24-hour Holter monitor is unnecessary as the patient's ECG is unlikely to change and the current risk of arrhythmias or heart block requires urgent intervention.

>Question #27

A 77-year-old man is reviewed in the cardiology. He presented to his GP with intermittent dizziness and reduced exercise tolerance. An ECG accompanies the referral letter:



What does the ECG show?

- a) Left bundle branch block
- b) Long QT syndrome
- c) ECG changes consistent with hypokalaemia
- d) Bifascicular block

- e) Trifascicular block

Correct answer is e.

RBBB +left anterior or posterior hemiblock + 1st-degree heart block = trifascicular block

The ECG demonstrates RBBB + left anterior hemiblock + 1st-degree heart i.e. trifascicular block.

>Question #28

A 58-year-old man attends cardiology clinic with retrosternal chest pain on exertion. He reports the onset of his symptoms when walking more than 50 meters on flat ground or when playing on the floor with his grandchildren. The symptoms have been present at the current level for at least the previous 9 months and the patient denied any episodes of pain at rest. The patient's GP has previously attempted treatment with bisoprolol and amlodipine but both medications were discontinued for unwanted effects (bradycardia and ankle swelling respectively). The patient has no other medical history except for hypercholesterolaemia. Regular medications are aspirin 75 mg daily and pravastatin 10 mg daily with nitrate spray and sildenafil used as required. He is formerly a heavy smoker but managed to quit the previous year.

Clinical examination is unremarkable with no evidence of cardiac failure. Blood pressure is 102 / 72 mmHg.

Please see below for the results of previous investigations.

Electrocardiogram: sinus rhythm at 58 beats per minute; borderline left axis deviation; normal QRS complex; non-specific lateral ST segment abnormalities; normal T waves.

Transthoracic echocardiogram: normal valvular function; no regional wall motion abnormality; ejection fraction 55-60 %.

Urea	6.7 mmol / L
Creatinine	80 micromol / L
Sodium	138 mmol / L
Potassium	4.1 mmol / L

Cardiac stress magnetic resonance imaging: significant evidence of ischaemia in the region of the lateral left ventricle; estimated 20 % of LV myocardium with significant evidence of ischaemia.

What is the most appropriate management of the patients chest pain?

- a) Ticagrelor
- b) Nicorandil
- c) Percutaneous coronary intervention
- d) Ivabradine
- e) Long-acting isosorbide mononitrate

Correct answer is c.

The role of percutaneous coronary intervention in stable angina is a controversial subject. Revascularisation has not been shown to reduce mortality or the rate of myocardial infarction in such patients. However, sub-group analysis of randomised controlled trials suggests some benefit for the above outcomes over medical therapy. Therefore, **ESC guidelines recommend offering revascularisation to patients with stable coronary artery disease and ischaemia in > 10 % of the left ventricle.** Also, the patient's age and lack of co-morbidities make this the most appropriate strategy in this case.

The other possible answers that are options for the medical management of stable angina are contraindicated in this patient. **Both nicorandil and ISMN are contra-indicated for this patient due to his borderline hypotension and use of sildenafil.** Ivabradine is contra-indicated due to the patient's history of

bradycardia. It is also not used in patients with moderate to severe angina symptoms as has been shown to increase the rate of cardiovascular events in such patients.

Ticagrelor is a P2Y12 adenosine diphosphate receptor blocker with utility as an oral antiplatelet agent.

>Question #29

A 36 year old woman presents to the medical assessment unit with a 2 day history of pleuritic right sided chest pain, and shortness of breath. She is 38 weeks pregnant with her first child; her pregnancy so far had been uncomplicated.

On examination, she is slightly tachypnoeic, with no added sounds on chest auscultation.

There is no clinical evidence of a deep vein thrombosis. Her oxygen saturations are 95% on room air, heart rate 98/min.

What is the next appropriate investigation?

- a) CTPA
- b) D Dimer
- c) Chest x-ray
- d) V/Q scan
- e) Bilateral leg dopplers

Correct answer is c.

Venous thromboembolic event (VTE) is an important cause of maternal death in the UK. Pregnant women are 10x more likely to have a VTE than non-pregnant women of the same age.

Where there is suspicion of a pulmonary embolism (PE), a chest x-ray should be performed initially. If this does not explain the patient's symptoms, compression duplex Dopplers should be performed. A diagnosis of deep vein thrombosis (DVT) can indirectly confirm a PE, reducing radiation doses for the mother.

If both tests are negative, and the clinical suspicion of PE remains high, further imaging should be organised; either CTPA or ventilation-perfusion scan (V/Q scan). British thoracic society guidelines (BTS) recommend a CTPA in non-pregnant women.

The decision as to which scan to perform should be ideally taken with the input from the patient. V/Q scanning carries a slightly increased risk of childhood cancer compared with CTPA but carries a lower risk of maternal breast cancer. The ventilation component of the V/Q scan may be able to be omitted, reducing the radiation dose.

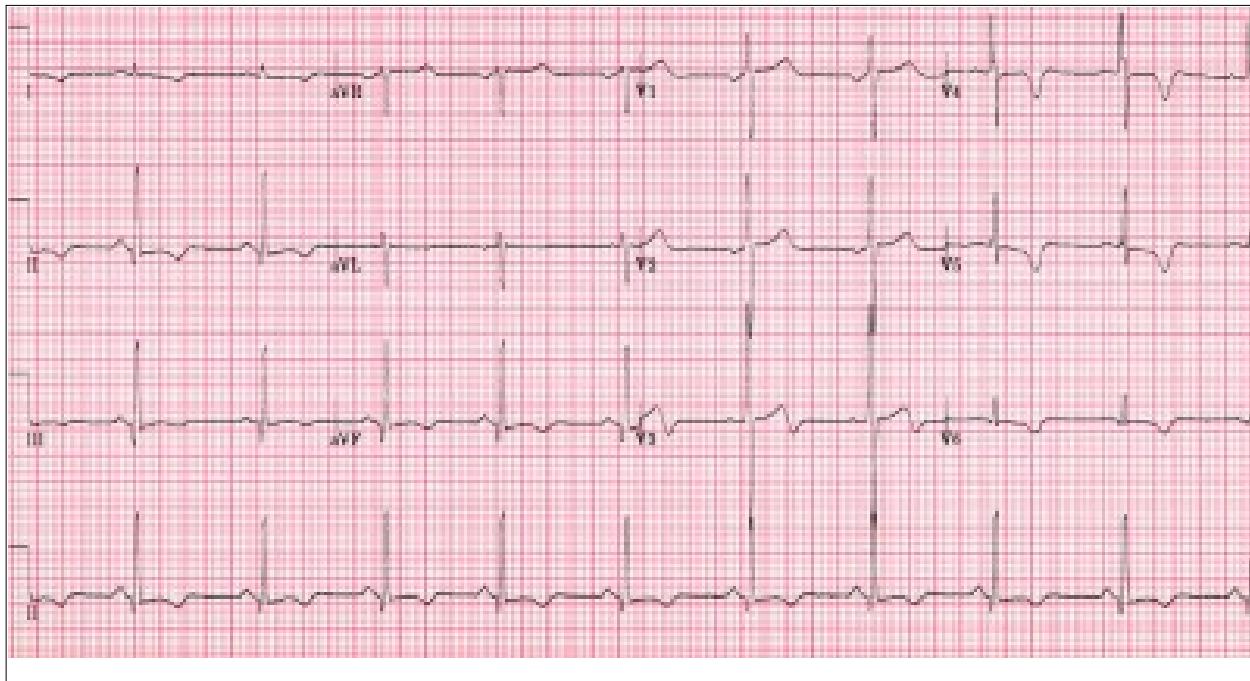
A D Dimer would not assist in the diagnosis, as it may be raised anyway in pregnancy due to disturbances in the coagulation system.

Guidelines: RCOG- The acute management of thrombosis and embolism during pregnancy and the puerperium.

>**Question #30**

A 34-year-old is brought into the emergency department following a syncope episode whilst playing tennis. The patient reports he is now feeling well with no symptoms. On further questioning, he only plays tennis occasionally and does not exercise regularly. He reports experienced shortness of breath and chest discomfort when playing and has had a few episodes of feeling light-headed but has never fainted before. He is otherwise well with no recent illness or significant past medical history.

Observations are all within normal range and an ECG performed is shown below.



What is the patient's most likely diagnosis?

- a) Brugada syndrome
- b) Dilated cardiomyopathy
- c) Hypertrophic obstructive cardiomyopathy
- d) Arrhythmogenic right ventricular dysplasia (ARVD)
- e) Wellen's syndrome

Correct answer is c.

This patient has presented with features in keeping with hypertrophic obstructive cardiomyopathy (HOCM). The condition is due to a genetic defect that causes a disorder of the cardiac muscle and, although patients can be asymptomatic, it is the commonest cause of sudden cardiac death in the young. Patients who do present with symptoms typically experience exertional dyspnoea, angina and syncopal episodes, commonly following or during exertion. **ECG findings are of left ventricular hypertrophy, non-specific ST-segment and T-wave abnormalities, progressive T wave inversion and deep Q waves. On this patient's ECG widespread T wave inversion can be seen, especially in lead I & II as well as non-specific ST-segment changes in V2 & 3.** The diagnosis can be confirmed via cardiac echo and management is dependant on the extent of obstruction and patient symptoms.

Brugada syndrome is another genetic cardiac disorder however unlike HOCM it results in electrical activity disorders and not structural issues. Features include syncope and sudden cardiac death however it is not always associated with exertion or exercise. There are several types of Brugada syndrome, each with variations seen on ECG. However, the only potentially diagnostic ECG abnormality is of coved ST-segment elevation in 2 or more of V1-3 followed by a negative T wave. This is classical of Brugada type 1 syndrome and is commonly known as the Brugada sign.

Dilated cardiomyopathy is a disease of the myocardial and characterised by progressive ventricular dilation and dysfunction. Presentation is normal with worsening biventricular failure including peripheral and pulmonary oedema. Syncope episodes are rare and symptoms are not fluctuant, as seen in this patient. ECG abnormalities are those associated with atrial and ventricular hypertrophy with conduction delays (e.g. LBBB), left axis deviation and poor R wave progression.

Arrhythmogenic right ventricular dysplasia (ARVD), also known as arrhythmogenic right ventricular cardiomyopathy (ARVC), is a result of a genetic defect affecting the desmosomes of the myocardium. ARVD results in non-ischemic cardiomyopathy, mainly affecting the right ventricle. The condition leads to hypokinetic areas of the ventricle wall and myocardium fibrofatty replacement with which results in associated arrhythmias. Patients typically present with palpitations, syncope, and potentially sudden cardiac death however ECG findings are of T wave inversion in leads V1 to V3 and of right bundle branch block which are not present in this case.

Wellen's syndrome is the ECG pattern of biphasic or deeply inverted T waves in the chest leads V2-3. It is highly specific for critical stenosis of the left anterior descending artery and therefore is normally seen in patients presenting with ischaemic-like symptoms.

>Question #31

A 72-year-old man presents to the emergency department with palpitations. An ECG confirms atrial fibrillation. His CHA₂DS₂-VASc score is 2 and he is commenced on bisoprolol and edoxaban.

He is referred and attends for catheter ablation, which he undergoes successfully. Post-procedure ECG confirms sinus rhythm.

He is followed up 4 weeks later in the clinic and a repeat ECG confirms the maintenance of sinus rhythm.

What is the best course of action at this stage?

- a) Continue edoxaban for a further 4 weeks then stop
- b) Continue edoxaban for a further 8 weeks then stop
- c) Continue edoxaban long-term
- d) Discontinue edoxaban
- e) Switch to aspirin

Patients who've had a catheter ablation for atrial fibrillation still require long-term anticoagulation as per their CHA₂DS₂-VASc score

The correct answer is **continue edoxaban long-term**. Although the ablation has been successful and rhythm control has been achieved, patients in this setting have the same stroke risk as those remaining in atrial fibrillation.

Anticoagulation should, therefore, be continued long-term.

>Question #32

A 22-year-old woman presents to the emergency department with a 1-day history of abdominal pain and vomiting. She denies fever or diarrhoea and disclosed that she went out to a party the night before and consumed 7 glasses of wine. She reports that there is a heart condition that runs in her family, but she cannot remember the name of the condition.

Her observations are within normal limits. On examination, there is central and epigastric tenderness but no guarding or peritonism. Her respiratory and

cardiovascular examinations were unremarkable.

Venous blood gas:

pH	7.35	(7.35 - 7.45)
Na ⁺	138 mmol/L	(135 - 145)
K ⁺	4.1 mmol/L	(3.5 - 5.0)
Bicarbonate	22 mmol/L	(22 - 29)
Lactate	3.5 mmol/L	(< 1.6)
Glucose	6.2 mmol/L	(4 - 8)

ECG: Sinus rhythm, normal axis, QRS duration 110ms, QTc 550ms.

What drug is it most important to avoid?

- a) Codeine
- b) Cyclizine
- c) Hyoscine butylbromide
- d) Omeprazole
- e) Ondansetron

5HT-3 receptor antagonists shouldn't be used as antiemetics in patients with long-QT syndrome

This patient has a prolonged QTc interval of 550ms. A normal QTc interval for women should lie between 350-460ms. Patients with a prolonged QTc interval are at particular risk of developing critical arrhythmias such as Torsades de pointes (a polymorphic ventricular tachycardia). It is therefore extremely important to avoid drugs that may prolong the QTc interval further. **Ondansetron is a type of 5HT-3 receptor antagonist that is associated with QTc interval prolongation and should**

therefore be avoided.

Codeine is a weak opioid analgesic with common side effects of nausea, drowsiness and constipation. It does not prolong the QTc interval.

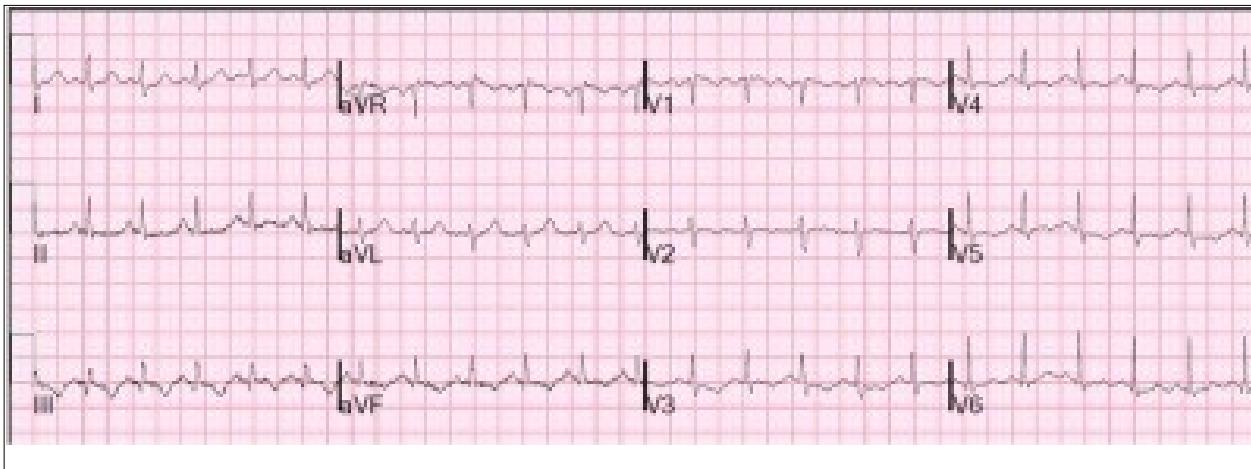
Cyclizine is a H1-receptor antagonist. Side effects of cyclizine include urinary retention, dry mouth, and blurred vision. Prolongation of the QTc interval is not a common feature of this medication.

Hyoscine butylbromide is used in the treatment of gastric spasm. It is an anticholinergic agent and is, therefore, contraindicated in myasthenia gravis, urinary retention and gastrointestinal obstruction. It does not prolong the QTc interval.

Omeprazole is a proton-pump inhibitor used in the treatment of gastritis and gastro-oesophageal bleeding. Complications include electrolyte disturbances (e.g. hypomagnesaemia) and increased risk of *Clostridium difficile* infections, acute interstitial nephritis, but not prolongation of the QTc interval.

>Question #33

A 19-year-old student is brought to the Emergency Department by her friends. Around one hour ago she 'collapsed' whilst playing hockey. Her friends describe her complaining that she felt light-headed and then 'fainting' to the ground. She lost consciousness for a few seconds before returning to normal quite quickly. There is no past medical history of note other than the use of Microgynon 30 (a combined oral contraceptive pill). For the past 4-5 days the patient has experienced shortness-of-breath and a central chest pain which is worse when she coughs. On examination her pulse is 120/min, blood pressure 96/60 mmHg and chest auscultation reveals scattered wheezes. An ECG done on admission is shown below:



What is the most likely diagnosis?

- a) Hypertrophic obstructive cardiomyopathy
- b) Vasovagal attack
- c) Acute coronary syndrome
- d) Pulmonary embolism
- e) Asthma attack

Correct answer is d.

It is usually taught that pulmonary embolism (PE) presents with pleuritic chest pain, dyspnoea and haemoptysis. This combination of symptoms is however only found in less than 20% of cases. As PE is a potentially life-threatening condition it is important to be aware of the wide variety of symptoms and signs that may accompany cases.

A lot of patients who develop a PE have risk factors. There is one present in this case - combined oral contraceptive pill use. Tachycardia is also a common sign.

It would be unusual to develop an asthma attack with no previous history of asthma. Occasional wheezes are a relatively common finding in patients following a pulmonary embolism.

The ECG shows a sinus tachycardia and a partial S1Q3T3 - the S wave is not particularly convincing.

>Question #34

A 22-year-old Afro-Caribbean man presents with chest pain. He states the pain is sharp in nature, worse on inspiration, and localised to the right lateral chest wall. He has no past medical history and takes no regular medicines.

An ECG is performed:

P waves	Normal morphology
PR interval	140ms
QRS	110ms
QTc	420ms
T waves	Inverted in V1-V6
ST segments	No elevation or depression

What is the most likely explanation of these ECG results?

- a) NSTEMI
- b) STEMI
- c) Normal variant
- d) Prinzmetal's angina (variant angina)
- e) Stable angina pectoris

Widespread T wave inversion in the chest leads can be a normal variant in patients with Afro-Caribbean ethnicity

Coronary artery disease would be very unlikely in a man of this age. The absence of ST elevation makes an acute STEMI even more unlikely. T-wave inversion can of course be a feature of an NSTEMI or stable angina, however due to the absence of risk factors, and the patients age, it would be a very unlikely diagnosis.

Prinzmetal's angina due to vasospasm would be a possibility. However this usually presents with ST elevation.

Widespread T-wave inversion in the chest leads can be a normal variant in patients with Afro-Caribbean ethnicity and this would be the most likely diagnosis in a young man with no cardiovascular risk factors.

The patient's pain is pleuritic in nature and an alternative diagnosis should therefore be considered (e.g. pneumothorax).

>Question #35

A 52-year-old female who you see regularly represents to your clinic after routine blood tests having recently been started on ramipril for her hypertension. Her blood pressure (BP) prior to initiation of ramipril in the clinic was 145/98mmHg and her baseline creatinine prior to treatment was 100umol/L. On review today her clinic BP appears to be well controlled at 132/84mmHg and her bloods show her creatinine has raised to 125umol/L.

What is the most appropriate step in the management of her hypertension?

- a) Reduce dose of ramipril
- b) Continue current dose of ramipril
- c) Stop ramipril and consider angiotensin receptor blocker
- d) Increase dose of ramipril
- e) Stop ramipril and consider calcium channel blocker

Correct answer is b.

The main consideration here is the renal function, which shows an increase in creatinine by 25%. Her BP appears to be well controlled. **The BNF recommends the angiotensin-converting enzyme inhibitors should only be stopped if the creatinine increases by 30% or greater or eGFR falls by 25% or greater.** This lady's results are within these limits and have shown good effect. It would therefore be pertinent to continue the ramipril at the current dose and monitor the renal function as per normal protocol

>Question #36

A patient attends a cardiology clinic following a recent myocardial infarct (MI) which was successfully managed via percutaneous coronary intervention (PCI). The patient has been taking his secondary prevention medications regularly and reports no issues or symptoms.

Their examination is unremarkable and the patient's observations are as follows:

- Heart rate 55 beats per minute
- Blood pressure 134/80 mmHg
- Respiratory rate 16 breaths per minute
- Oxygen saturations 98% on air
- Temperature 36.2 °C

An ECG performed has widened QRS complexes and atrioventricular dissociation in keeping with accelerated idioventricular rhythm (AIVR) with a rate of 55 beats per minute.

What management should be advised?

- a) Arrange for urgent repeat PCI
- b) Arrange for urgent pacemaker insertion
- c) No additional management is required
- d) Stop the patient's beta blocker and continue all other medications
- e) Urgent atropine

Correct answer is c.

Accelerated idioventricular rhythm is common and un concerning following recent MI.

AIVR is a benign ectopic rhythm of ventricular origin. It is common post-MI following the reperfusion of an ischaemic myocardium. AIVR is usually self-

limiting and therefore treatment is not necessary, especially as the patient is asymptomatic and haemodynamically stable with a normal heart rate and blood pressure.

Arranging for urgent repeat PCI is unnecessary as there is no indication of new/further ischaemic damage or an ischaemic event having occurred. AIVR is due to an ectopic rhythm and therefore PCI is of no value.

Pacemaker insertion is not required as AIVR is due to a benign ectopic rhythm and therefore pacemaker insertion would not remove the cause. As the patient is asymptomatic and haemodynamically stable no treatment is required.

Stopping the patient's beta-blocker is unnecessary as this will not eliminate the AIVR. Beta-blockers are an important part of secondary prevention post-MI and therefore should be continued.

Atropine could be considered to overcome AIVR via increase the sinus rate however this is rarely required. As the patient is symptom-free and haemodynamically stable the use of atropine is unnecessary.

Question #37

An 84-year-old man comes for review. Four weeks ago an opportunistic blood pressure reading was taken and recorded as 150/92 mmHg. You therefore arranged ambulatory blood pressure monitoring (ABPM) along with a standard hypertension work-up. You did not calculate his 10-year cardiovascular risk on account of his age. The following results were obtained:

Na ⁺	141 mmol/l
K ⁺	4.2 mmol/l

Urea	6.5 mmol/l
Creatinine	101 µmol/l
Total cholesterol	4.9 mmol/l
HDL cholesterol	1.2 mmol/l
Fasting glucose	5.5 mmol/l

Urine dipstick was normal. The ECG showed sinus rhythm, 72 bpm and first degree heart block.

The daytime average blood pressure reading was 145/80 mmHg. What is the most appropriate course of action?

- a) Diagnose stage 1 hypertension and advise about lifestyle changes
- b) Start treatment with an ACE inhibitor
- c) Start treatment with a calcium channel blocker
- d) Start treatment with a thiazide-like diuretic
- e) Repeat the ABPM

Stage 1 hypertension is defined by an ABPM reading of $\geq 135/85$ mmHg, with stage 2 hypertension having a cut-off of $\geq 150/95$ mmHg.

This patient therefore has stage 1 hypertension. As he is > 80 years he does not need treatment.

>Question #38

A 56-year-old woman presents to her GP with discolouration around her eyes which has been present for some months. She feels very conscious about its

appearance but denies any pain, itch or discomfort.

On examination, she appears well. The area in question appears as Xanthelasma around eyes.

Given the likely diagnosis, which of the following would be an appropriate management option?

- a) Topical emollient
- b) Topical hydrocortisone
- c) Topical ketoconazole
- d) Topical trichloroacetic acid
- e) Urgent referral to secondary care

Correct answer is d.

The photo above demonstrates xanthelasma - high lipid levels leading to soft yellow/orange plaques, periorbitally. They are not of clinical concern - except for the underlying lipid profile which should be investigated and treated accordingly. They can be left alone, but if patients are keen for treatment, a commonly used option is topical trichloroacetic acid.

Topical emollient is unnecessary. This will not help to alleviate the appearance of the xanthelasma.

Topical hydrocortisone is inappropriate here. As a steroid, this would not help to reduce the appearance of xanthelasma.

Topical ketoconazole is also inappropriate. This is an antifungal and may be used for seborrhoeic dermatitis, which would instead present with dry, flaky skin and erythema - not yellow/orange plaques.

An urgent referral to a hospital is also unwarranted. Xanthelasma is of no clinical concern and requires no further investigation.

>**Question #39**

A 68-year-old man presents with occasional lightheadedness over the past few weeks. During his morning walk today, he experienced a brief spell of dizziness. He denies experiencing chest pain, breathlessness, or any other significant complaints.

His medical history includes diabetes, hypertension, and myocardial infarction for which he underwent coronary artery bypass grafting (CABG) seven years ago. He is currently on medications including aspirin, bisoprolol, lisinopril, atorvastatin, and vildagliptin.

Observations are as below:

- BP 140/85 mmHg
- Heart rate 74 bpm
- Respiratory rate 16/min
- Oxygen saturation 98% on room air

An ECG was subsequently carried out shows Wenckebach phenomenon.

What is the most appropriate management option?

- a) Discharge him and recommend a follow up ECG
- b) Give atropine
- c) Intravenous infusion of isoproterenol
- d) Refer for cardiac resynchronisation therapy
- e) Review his medications

The correct answer is **review his medications**.

The ECG findings demonstrate a progressive prolongation of the PR interval until a beat is dropped. This is characteristic of a 2nd-degree Mobitz 1 or Wenckebach-type block. This is usually a relatively benign rhythm with a low risk of progression to higher-degree blocks. This patient has symptoms of lightheadedness and dizziness, coupled with stable vital signs. As long as there is no hemodynamic compromise, there is no need for urgent intervention. Searching for a reversible cause should be the first step. This patient is on bisoprolol, a known cause of AVN block, and should be reviewed.

Discharge him and recommend a follow up ECG is incorrect. It is indicated if the patient is asymptomatic with a first or 2nd-degree Mobitz 1 heart block. This patient has symptoms of light-headedness and dizziness, which require intervention.

Give atropine is incorrect. Atropine is generally indicated for acute symptomatic AVN block with bradycardia and adverse features (heart failure, shock, syncope, myocardial ischaemia). There are no adverse features in this case.

Intravenous infusion of isoproterenol is also an incorrect answer. Reviewing any potential causes should be the initial approach for symptomatic hemodynamically stable patients. IV isoproterenol is indicated for hemodynamically unstable patients who fail to respond to transcutaneous pacing. Given the patient's history of coronary artery disease, it is important to avoid it if possible, due to the potential development of ischemic chest pain.

Refer for cardiac resynchronisation therapy is incorrect. Cardiac resynchronisation can be beneficial if there is heart failure with an ejection fraction of less than or equal to 35% and a widened QRS (e.g. left bundle branch block) complex on the ECG. It has no role in the management of the AVN block.

>Question #40

A 34-year-old male is brought in from a nearby bar having collapsed. His friends who accompany him tell you that the patient had been moderately intoxicated but had not reported any symptoms prior to his collapse.

On arrival, the patient is conscious but reports feeling generally weak and light-headed. He has never had an episode like this before but has had episodes of palpitation with associated shortness of breath previously.

On examination, the patient is conscious and reports some lightheadedness and palpitation but feels otherwise ok when lying down. He has a fast, thready pulse between 130-150bpm and a blood pressure of 89/65mmHg. Heart sounds are normal and examination is otherwise unremarkable.

What is the most appropriate management?

- a) IV adenosine
- b) IV metoprolol
- c) Cardioversion
- d) Pacemaker insertion
- e) Percutaneous coronary intervention

Correct answer is c.

This patient has presented with a tachyarrhythmia and is haemodynamically unstable with an acute collapse, most likely syncope induced, and shock, demonstrated by the hypotension and cool extremities, etc. As seen on the ECG the patient's rhythm is fast and irregular with a varied rate of between 130 and 150 bpm. The QRS complexes are narrow and clear 'p' waves cannot be seen before each complex. This is in keeping with a tachyarrhythmia, most likely secondary to atrial fibrillation however the specific cause is not relevant as urgent synchronised cardioversion is required in any unstable patient presenting with a tachyarrhythmia. Cardioversion should be carried out in a resuscitation setting, with procedural sedation if possible. Other indications of instability in arrhythmias include evidence of myocardial ischaemia and heart failure. Following cardioversion, further management is based on the QRS complexes (narrow vs broad) and the rhythm (regular vs irregular).

Adenosine is the treatment of choice in patients presenting with supraventricular tachycardias (SVT), which may be the underlying diagnosis in this case, however, it should be used in hemodynamically stable patients only. As the patient is unstable cardioversion is required.

Metoprolol is an IV beta-blocker and, although it can be used in some tachyarrhythmias, this patient is unstable and requires urgent electrical cardioversion. IV beta-blockers can be used in stable patients with tachyarrhythmias (e.g. atrial fibrillation) for rate control as so long as they have an adequate systolic blood pressure and there are no other contraindications (e.g. asthma).

Pacemaker insertion is used in the management of bradycardia arrhythmias and does not have a role in acute arrhythmias causing compromise.

Percutaneous coronary intervention is used in the management of myocardial infarction (MI). Although the patient has some risk factors, and cocaine use can induce an MI, it is unlikely to be the underlying issue in this case with the patient not reporting chest pain symptoms and no clear evidence of ST-elevation seen on the ECG.

>Question #41

A 43-year-old gentleman who is admitted and found to have a STEMI has chest pain on the ward. He is currently recovering from his percutaneous coronary intervention the day before and has felt well until now. The pain is over the left side of the chest and severe if he breathes. He has no cough or fevers and his observations are within normal limits.

On examination, he has normal heart sounds and a clear chest. He is mildly tender over the chest wall. His femoral puncture sites are clean with no haematoma. He is in sinus rhythm on the cardiac monitor and has soft non-oedematous calves. No JVP is seen.

Na ⁺	156 mmol/l
K ⁺	3.4 mmol/l
Urea	5.6 mmol/l
Creatinine	78 µmol/l
CRP	45 mg/l

Hb	134 g/l
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Platelets	$398 * 10^9/l$
WBC	$13.0 * 10^9/l$

ECG	sinus rhythm, T wave inversion in V1-V3, PR 180ms and flat in most leads
Chest x-ray	clear lung fields, heart is at the upper limit of normal for size

What is the likely diagnosis?

- a) Hospital acquired pneumonia
- b) Pericarditis
- c) Pulmonary embolism
- d) Coronary artery dissection
- e) Pleurisy

Pleuritic chest pain at <48hrs after MI -> pericarditis.

This gentleman develops chest pain in the first 24-48hrs after an MI. A hospital-acquired pneumonia would usually take longer to develop. A coronary artery dissection can be a complication of PCI but there would most likely be ischaemic changes seen on the ECG. **Pleurisy is usually a post inflammatory condition after a lower respiratory tract infection. Pulmonary embolism is possible but patients with MI are anticoagulated and this is early for a PE to develop. It is recognised that a number of MI patients develop an acute pericarditis in the first 48hrs.**

Treatment is supportive and it is key to rule out other complications first.

>**Question #42**

A patient attends a cardiology clinic following a recent myocardial infarct (MI) which was successfully managed via percutaneous coronary intervention (PCI). The patient has been taking their secondary prevention medications regularly and reports no issues or symptoms.

Their examination is unremarkable and the patient's observations are as follows:

- Heart rate 55 beats per minute
- Blood pressure 134/80 mmHg
- Respiratory rate 16 breaths per minute
- Oxygen saturations 98% on air
- Temperature 36.2 °C

An ECG performed has widened QRS complexes and atrioventricular dissociation in keeping with accelerated idioventricular rhythm (AIVR) with a rate of 55 beats per minute.

What management should be advised?

- a) Arrange for urgent repeat PCI
- b) Arrange for urgent pacemaker insertion
- c) No additional management is required
- d) Stop the patient's beta blocker and continue all other medications
- e) Urgent atropine

Accelerated idioventricular rhythm is common and un concerning following recent MI.

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Stopping the patient's beta-blocker is unnecessary as this will not eliminate the AIVR. Beta-blockers are an important part of secondary prevention post-MI and therefore should be continued.

Atropine could be considered to overcome AIVR via increase the sinus rate however this is rarely required. As the patient is symptom-free and haemodynamically stable the use of atropine is unnecessary.

>Question #48

A 76-year-old man presents to the emergency department with complaints of shortness of breath on exertion, difficulty walking up stairs, and significant weight gain over the last 2 weeks. His daughter who is with him expresses concerns about him and says that he is needing to lay upright in his armchair in order to get any sleep.

His medical history includes 2 previous non-ST-segment elevation myocardial infarctions for which he was treated with coronary stenting. Physical examination shows a heart rate of 111 beats per minute and blood pressure of 113/76 mmHg. He is visibly short of breath at rest and is using accessory muscles. His JVP is 7cm above the angle of Louis, his apical impulse is laterally displaced, and heart sounds are audible with a holosystolic murmur heard loudest at the apex. Auscultation of his chest reveals audible crepitations bi-basally and he has 2+ peripheral oedema extending to his mid-thighs. An ECG shows Q-waves in his inferior leads, and his bloods show elevated troponins.

Which of the following is the most appropriate next step in the management of this patient?

- a) Obtain an urgent echocardiogram

- b) Prescribe oral furosemide and discharge from the emergency department with a view to follow up with his GP
- c) Load him with aspirin and clopidogrel
- d) Commence bisoprolol to control his heart rate
- e) Administer a bolus of intravenous furosemide and then start him on an infusion

Correct answer is e.

The most appropriate medication in patients with acute heart failure and a preserved ejection fraction who have signs of volume overload is addition/up-titration of a loop diuretic

This patient presents with acute decompensated congestive heart failure. He has evidence of significant volume overload on exam, with an elevated JVP and signs of pulmonary and peripheral oedema. The aetiology of his heart failure is likely to be ischaemic cardiomyopathy related to his history of myocardial infarctions and manifests with a displaced apical impulse and a holosystolic murmur heard loudest at the apical region, suggestive of significant ventricular dilatation. His symptoms are severe and he requires inpatient hospitalisation and treatment with intravenous loop diuretics to improve his symptoms and to optimise his volume status.

An echocardiogram would be useful in the evaluation of this patient but is not the most appropriate next step as he is currently significantly symptomatic and needs intravenous loop diuretics to improve his volume status.

Although this patient does need to be treated with loop diuretics, he is in significant distress and likely needs to be admitted as an inpatient and should be treated more aggressively with intravenous loop diuretics.

This patient does have a history of ischaemic heart disease and is at risk of having further ischemic events. However, the findings on his ECG represent an old inferior infarct, and there are no features to suggest an acute event. In addition, he may have elevated troponins simply as a result of having ischaemic cardiomyopathy. He does not necessarily need to be loaded on dual antiplatelet therapy at this

moment, but he should have his troponins trended to ensure there is no significant rise in their values.

Starting a beta-blocker in acute decompensated heart failure is contraindicated

>Question #49

A 58-year-old woman was admitted with a stroke following a month's history of recurrent fevers, anorexia and weight loss. On examination, she had a left-sided hemiparesis and facial droop. Cardiovascular examination revealed splinter haemorrhages in 5 of her fingers across both hands, and a soft diastolic murmur heard loudest in expiration over the aortic area.

A trans-thoracic echocardiogram showed an oscillating vegetation on an aortic leaflet, in the path of regurgitant jets. Two blood cultures were positive for Streptococci spp. She was diagnosed with infective endocarditis and started on intravenous benzylpenicillin 1.2g every 4 hours and gentamicin 1mg/kg twice daily.

She was reviewed after 5 days on antibiotic therapy with the following results.

Na+	139 mmol/l
K+	4.7 mmol/l
Urea	14.2 mmol/l
Creatinine	178 µmol/l
Serum bilirubin	16 µmol/l
Serum alkaline phosphatase	115 IU/l
Serum aspartate aminotransferase	18 IU/l

C Reactive protein	89 mg/l
Haemoglobin	138 g/l
White cell count	13.6 x 10^9/L
INR	1.1
Blood cultures	<i>Streptococcus Bovis</i>
Penicillin Minimum Inhibitory Concentration (MIC)	0.6 mg/L (high)
ECG	Prolonged PR interval (not present on admission ECG)

What is the next most important management?

- a) Increase antibiotics to intravenous benzylpenicillin 2.4g every 4 hours and gentamicin 1mg/kg twice daily
- b) Organise an urgent colonoscopy
- c) Switch antibiotics to ceftriaxone 2g once daily
- d) Refer to cardiothoracic surgeons
- e) Organise urgent trans-oesophageal echocardiogram

Correct answer is d.

This patient has infective endocarditis, as diagnosed by two positive major criteria from the Duke's criteria for infective endocarditis. In addition, she has a number of minor criteria, including fever, vascular events (stroke), immunological events (splinter haemorrhages, renal impairment secondary to glomerulonephritis).

Despite starting on appropriate empirical treatment she appears to have an ongoing infection from her blood tests, and her ECG demonstrates a prolonged PR interval which may be reflective of an enlarging aortic abscess disrupting the

atrioventricular node, which is an indication for referral for cardiothoracic surgery in infective endocarditis.

Other surgical indications include:

- Heart failure: valve obstruction resulting in pulmonary oedema or shock, severe acute regurgitation
- Uncontrolled infection: abscess, false aneurysm, persisting fever and positive blood cultures for greater than 10 days despite appropriate antibiotics, multiresistant microorganisms
- Prevention of embolism: large vegetations resulting in one or more embolic episodes despite appropriate antibiotic therapy, or other predictors of complications eg. heart failure.

She has a penicillin-resistant streptococci, according to the British Society of Antimicrobial Chemotherapy guidelines, should be treated with vancomycin and gentamicin, so options (a) and (c) are incorrect. ***Streptococcus Bovis* bacteraemia is associated with bowel malignancy, and a colonoscopy should be arranged to rule out malignancy, however, the worsening PR prolongation takes priority and should be managed first.**

An urgent trans-oesophageal echocardiogram may be useful to evaluate the size of the aortic abscess, however, it would not change management as this patient needs to be referred to the cardiothoracic surgeons as there is already evidence of enlarging vegetation.

>Question #50

A 54-year-old male previously treated for lymphoma is seen in clinic with a 6-month history of exertional dyspnoea which is progressive. He was treated four years ago and was told that there was no evidence of disease on the final CT scan. His GP treated him for a presumed lower respiratory tract infection two weeks ago. He returned from a business trip to Thailand six months ago, during which he consumed more alcohol than he usually does. He takes no medications and is

otherwise fit and well, and is a non-smoker. On examination there is mild pedal oedema, his chest is clear and jugular venous pressure(JVP) is raised on inspiration. Auscultation of his heart reveals an extra heart sound very soon after S2.

What is the likely cause of his symptoms?

- a) Superior vena cava obstruction
- b) Cardiac tamponade
- c) Left ventricular failure
- d) Chronic obstructive pulmonary disease
- e) Constrictive pericarditis

Correct answer is e.

Examination of this patient demonstrates Kussmaul's sign - paradoxical elevation of the JVP on inspiration. This sign is seen in constrictive pericarditis, cardiac tamponade and restrictive cardiomyopathy. This presentation gives no other features to suggest tamponade. His constrictive pericarditis here is likely cause by the previous radiotherapy for lymphoma. Other causes of constrictive pericarditis include TB and chronic pericarditis. Definitive management involves surgical pericardial stripping.

>Question #51

A 55-year-old with previous rheumatic heart disease aged 32 presents with an 18-month history of exertional dyspnoea. An initial echo demonstrated significant raised pulmonary arterial pressures of 77 mmHg, she undergoes a left and right heart catheter with results as follows:

Oxygen saturations		
Right atrium	8 mmHg	71%
Right ventricle	39/8 mmHg	71%

Pulmonary artery	45/12 mmHg	71%
Capillary wedge	20 mmHg	93%
Left ventricle	165/11 mmHg	93%
Aorta	90/58 mmHg	

What is the most likely diagnosis?

- a) Aortic stenosis
- b) Mitral stenosis
- c) Aortic stenosis and mitral stenosis
- d) Aortic stenosis and pulmonary hypertension
- e) Aortic stenosis, mitral stenosis, pulmonary hypertension

Correct answer is e.

The key to questions regarding saturations and cardiac catheters is to spot the 'step-up' in oxygen saturation and abnormalities in gradients across valves. There are no 'step-ups' in oxygen saturations, demonstrating no shunts. However, you will note that the pulmonary arterial pressure is greater than the normal one-fifth of systolic measurements; hence pulmonary hypertension is present. In addition, there is a greater than 25mmHg gradient across the aorta valve, demonstrating moderate aortic stenosis. Lastly, the capillary wedge pressure is equivalent to the left atrial pressure, which should also be the same as the left ventricular diastolic pressure. A normal mitral valve expects less than 5mmHg pressure difference. Using these inferences, the mitral valve gradient is calculated by the capillary wedge pressure of 20mmHg (same as the left atrial pressure) minus the diastolic left ventricular pressure of 11mmHg: the 9mmHg difference thus also demonstrates mitral stenosis.

>**Question #52**

A 65-year-old man is reviewed in the outpatient cardiology clinic and reports palpitations and fatigue when carrying out normal household tasks. He has a history of heart failure (with an ejection fraction of 41%) and chronic obstructive pulmonary disease. He takes bisoprolol 10mg once daily, ramipril 10mg once daily and uses a tiotropium bromide inhaler.

Observations:

- Heart rate 88 beats per minute
- Blood pressure 145/82 mmHg
- Respiratory rate 18/minute
- Oxygen saturations 96% on room air (95% on exertion)

On examination, the pulse is regular.

What is the most appropriate next step in the patient's management?

- a) Add a long-acting beta agonist and inhaled corticosteroid
- b) Add digoxin
- c) Add ivabradine
- d) Add spironolactone
- e) Increase the dose of bisoprolol

Correct answer is d.

Offer a mineralcorticoid receptor antagonist, in addition to an ACE inhibitor (or ARB) and beta-blocker, to people who have heart failure with reduced ejection fraction if they continue to have symptoms of heart failure

Add spironolactone is correct. The patient reports palpitations and fatigue on minimal exertion. These symptoms suggest symptomatic heart failure rather than worsening COPD. This is reaffirmed by the normal SpO₂ results following exertion. In a patient that is on maximally tolerated doses of beta-blocker and an ACE inhibitor, the correct next step is to add spironolactone.

Add a long-acting beta-agonist and inhaled corticosteroid is incorrect. Symptoms of palpitations and fatigue are more suggestive of symptomatic heart failure rather than COPD where wheezing and shortness of breath would be more typical. Therefore adjusting his COPD inhaler regimen is incorrect.

Add digoxin is incorrect. Digoxin is indicated in the management of symptomatic heart failure (especially in the context of co-existent atrial fibrillation) but only after other medications such as spironolactone have been initially trialled. This patient is not in AF.

Add ivabradine is incorrect. This is a 3rd line option for those patients with an ejection fraction < 35% and a heart rate > 75.

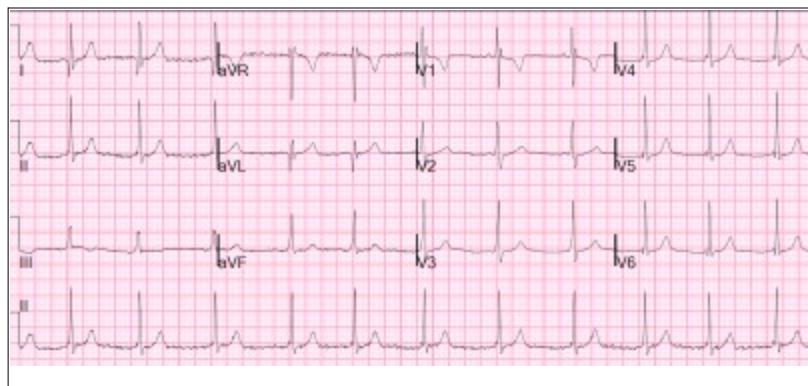
Increase the dose of bisoprolol is incorrect. 10mg is the maximum dose of bisoprolol that is recommended for heart failure.

>Question #53

A 40-year-old woman presents to the emergency department with a 10-day history of intermittent palpitations. She reports that her symptoms have worsened over the last 8 hours and her palpitations are now persistent and are associated with lightheadedness. She has no past medical history of note and takes no regular medications.

On examination, she appears clammy and warm to the touch. Her heart rate is 72 beats/min with a blood pressure of 125/83mmHg.

An ECG is taken (as shown below):



What is the most likely diagnosis?

- a) Accelerated junctional rhythm
- b) Atrial flutter
- c) Normal sinus rhythm
- d) Supraventricular tachycardia
- e) Wolff-Parkinson white syndrome

This patient has **Wolff-Parkinson White syndrome** a pre-excitation syndrome that causes clinical features of palpitations, shortness of breath, chest pain and in severe cases collapse and shock, including cardiac arrest. Wolff-Parkinson White is caused by an accessory pathway conducting impulses from the sinoatrial node to the ventricles giving rise to an atrioventricular re-entry tachycardia. ECG features include a shortened PR interval, left axis deviation or right axis deviation (depending on the location of the accessory pathway) and a widened QRS complex from the 'delta' wave (a slurred upstroke of the QRS complex). The delta wave can be seen in this patient's ECG, along with a shortened PR interval.

Accelerated junctional rhythm is incorrect. Accelerated junctional rhythm is when the atrioventricular node conducts at a faster rate than the sinoatrial node. This gives rise to an ECG with appearances of a narrow complex rhythm, with a ventricular rate between 60-100 beats/min. Retrograde P waves can be seen. Causes include digoxin toxicity, beta-blockers and myocardial ischaemia.

Atrial flutter is incorrect. Atrial flutter is an irregular supraventricular arrhythmia. Although it can cause similar symptoms to those experienced by the patient, a characteristic ECG finding is a saw-tooth appearance of the baseline of the ECG which is not seen here.

Normal sinus rhythm is incorrect. Although the patient's rhythm is sinus with QRS complexes following a P wave. There are clear delta waves preceding the QRS complexes that are consistent with Wolff-Parkinson White syndrome.

Supraventricular tachycardia (SVT) is incorrect. SVT is caused by a re-entry circuit within the atrioventricular node. This is in contrast to atrioventricular re-entry tachycardia which is caused by an accessory conducting pathway (e.g. Wolff-

Parkinson White syndrome). Symptoms are similar to this patient's presentation with palpitations, shortness of breath and chest pain that may be exacerbated by caffeine or alcohol. However, the presence of a delta wave is more suggestive of an accessory conducting pathway, making supraventricular tachycardia.

>Question #54

A 45-year-old male is referred to the rapid access chest pain clinic with symptoms typical of angina. He is a non-smoker and has no past medical history. The pain is located in the centre of his chest and made worse by exertion. According to the NICE guidelines, which is the most appropriate investigation?

- a) Treadmill ECG
- b) Trans-thoracic echocardiogram
- c) CT-coronary angiography
- d) Nuclear perfusion stress test
- e) Diagnostic coronary angiography

The correct answer is **CT-coronary angiography**. According to the NICE guidelines (CG95), a CT coronary angiogram should be offered as the first-line investigation for patients with typical or atypical angina symptoms and who are in the intermediate risk group (10-29% 10-year risk of developing cardiovascular disease). This patient fits into this category due to his age, sex, and lack of other risk factors. The CT coronary angiogram will help visualise the coronary arteries and identify any significant stenosis.

Treadmill ECG was once a common initial test for angina but it has now been superseded by more accurate tests such as CT coronary angiography. Treadmill ECG can have false positives and negatives, hence it is not recommended as a first-line investigation by NICE.

A **Trans-thoracic echocardiogram** is useful in assessing cardiac function but it does not directly visualise the coronary arteries so it would not be appropriate in this case. It may be used in conjunction with other tests if there are signs of heart failure or valvular heart disease.

Nuclear perfusion stress test involves injecting a radioactive tracer and then imaging the heart under stress and at rest. While this test can identify areas of myocardium that are under-perfused due to significant stenosis, it exposes patients to radiation, is more expensive than CT angiography and does not directly image the coronaries. Hence, it is usually reserved for cases where other investigations are contraindicated or inconclusive.

Finally, **Diagnostic coronary angiography**, while considered the gold standard for diagnosing coronary artery disease, is invasive and carries risks such as bleeding, stroke or kidney damage from contrast dye. As per NICE guidelines, it should only be used when non-invasive tests are inconclusive or contraindicated or when immediate revascularisation is being considered.

>**Question #55**

A 60-year-old man is admitted to the Emergency Department with acute dyspnoea. He is unable to give a full history and his notes are not yet available. His chest x-ray is shown below:



What is the most likely explanation for these changes seen over the heart?

- a) Left ventricular aneurysm
- b) Sarcoidosis
- c) Atrial myxoma
- d) Primary hyperparathyroidism
- e) Previous episodes of uraemia

Correct answer is e.

Pericardial calcification most commonly develops following repeated episodes of

acute pericarditis. Calcification of the pericardium often results in constrictive pericarditis.

>Question #56

A 54-year-old female presents with heart palpitations. She has no past medical history and takes no regular medicines. Her cardiorespiratory exam is normal. An ECG is performed:

Rate and rhythm	145 beats per minute. Regular rhythm
P waves	Not visible
QRS	145ms. RBBB pattern
QTc	420ms
Axis	Right axis deviation

What is the most likely explanation of these ECG results?

- a) Ventricular tachycardia
- b) Supraventricular tachycardia with bundle branch block
- c) Torsades de pointes (TdP)
- d) Atrial fibrillation with bundle branch block
- e) Supraventricular tachycardia

Correct answer is b.

The ECG results confirm a broad complex tachycardia with a regular rhythm. The differential is therefore between supraventricular tachycardia (SVT) with bundle branch block (BBB) and ventricular tachycardia (VT). Distinguishing between VT and SVT with BBB can be challenging. The presence of RBBB and RAD favours a diagnosis of SVT with BBB. If you are unsure of the diagnosis, it is always safer to

assume the patient has VT and treat accordingly.

The absence of P waves should make you consider a diagnosis of atrial fibrillation (AF) with BBB, however the regular rhythm precludes this diagnosis. In SVT, the P waves are often buried within the QRS complex.

The normal QTc and regular rhythm precludes the diagnosis of Torsades de pointes (TdP).

>Question #57

A 54-year-old man attends with recurrent episodes of syncope.

Respiratory rate 18 breaths per minute, SpO₂ 98% on room air, heart rate 75 beats per minute, BP 114/67 mmHg.

An ECG shows some non-conducted p-waves.

What would be the most appropriate intervention?

- a) AAI pacemaker
- b) Ablation
- c) DDD pacemaker
- d) Temporary pacing
- e) VVI pacemaker

Correct answer is c.

In patients with Mobitz type II AV block, or complete heart block, a DDD or DDDR pacemaker is indicated

The stem describes a stable patient with Mobitz type II AV block. Pacemaker insertion is the most appropriate option.

The most common pacing mode is DDD. This means that there is **dual** pacing and sensing of both the atrial and ventricles.

DDD pacemaker is the correct option. 'D' stands for dual and means that the pacemaker senses and paces both atrial and ventricular activity. Mobitz type II is a high-degree atrioventricular block, this means that the pacemaker needs to sense

both atrial and ventricular activity e.g. if there is a non-conducted p-wave (as in Mobitz type II) the pacemaker senses atrial activity, senses ventricular non-activity, and subsequently stimulates a ventricular response.

VVI pacemaker is incorrect. A VVI pacemaker is a single lead pacemaker that senses and paces the ventricles only. The 'I' stands for 'inhibition' and indicates that if the pacemaker senses ventricular activity it inhibits its pacing output. VVI pacemakers, also known as ventricular demand pacemakers, are commonly used for ventricular bradycardia such as atrial fibrillation with a slow ventricular response.

AAI pacemaker is incorrect. An AAI pacemaker is a single lead pacemaker that senses and paces the atria only. The 'I' stands for 'inhibition' and indicates that if the pacemaker senses atrial activity it inhibits its pacing output. It is used only for patients with sinus node dysfunction.

Transcutaneous pacing would be used for patients who were unstable as a stop-gap measure whilst they awaited pacemaker insertion. This would not be appropriate in this case as we have a stable and well patient.

Ablation is used to prevent aberrant electrical signalling from causing arrhythmias. In the case of Mobitz type II, the issue is non-conduction rather than aberrant conduction, ablation is therefore not appropriate.

>Question #58

A 63-year-old female patient attends the Emergency Department with crushing central chest pain and 3mm ST segment elevation in leads II, III and aVF. She is taken to the cardiac catheter laboratory where she has a primary PCI with a satisfactory angiographic result.

Six hours later, whilst on CCU, she develops complete heart block. The patient is asymptomatic and her haemodynamic parameters are as follows:

Pulse 44bpm, regular

Blood pressure - 123/75mmHg

What is the most appropriate course of action?

- a) Synchronised direct current cardioversion (DCCV)
- b) Continue close monitoring and observation of the patient
- c) Start an infusion of isoprenaline
- d) Insertion of a permanent pacemaker
- e) Insertion of a temporary pacing wire

Correct answer is b.

In the description of the patient's presentation, it is stated that her ECG showed ST elevation in leads II, III and aVF, indicating an inferior STEMI. Complete heart block soon after an inferior MI is not uncommon, and usually resolves without the need for intervention. The key to this question is the fact that the patient is asymptomatic and haemodynamically stable. In view of this fact, it is sensible to closely monitor and observe on the assumption that given enough time post-reperfusion she will return to sinus rhythm. If she was haemodynamically unstable, a temporary pacing wire would be the best option in the first instance, with an upgrade to a permanent system if she did not recover to sinus rhythm in due course.

>Question #59

A 64-year-old man with mitral regurgitation attends the cardiology clinic for review. An ECG reveals new atrial fibrillation.

He is well and denies any shortness of breath or chest pain. His blood pressure is 115/98 mmHg and his heart rate is 64 beats per minute. On examination, he has an audible S3 and a 3/6 pansystolic murmur heard loudest in the mitral area, his apex beat is displaced.

A recent echo showed a regurgitant mitral valve with evidence of left atrial and ventricular dilatation but a preserved ejection fraction.

His current medications include naproxen for osteoarthritis and over-the-counter vitamin D supplements.

From the following options, what would be the most appropriate next step?

- a) Adopt a 'watch and wait' approach with serial yearly echocardiography
- b) Commence bisoprolol
- c) Commence sacubitril-valsartan
- d) Refer for cardioversion
- e) Refer for mitral valve replacement

New AF in mitral regurgitation >> refer for mitral valve replacement

Refer for mitral valve replacement is the correct answer. This patient has evidence of asymptomatic severe mitral regurgitation as indicated by narrow pulse pressure, audible S3 and evidence of left ventricular dilatation. Given he has now developed atrial fibrillation on a background of severe mitral regurgitation he should be referred for consideration of a valve repair or replacement.

Commence bisoprolol is incorrect. The patient in this scenario has a preserved ejection fraction as indicated by the echo findings. If there was evidence of reduced ejection fraction current NICE guidelines advises considering starting a beta-blocker and ACE inhibitor.

Commence sacubitril-valsartan is incorrect. This would be considered a second-line treatment for heart failure with reduced ejection fraction. In this case, the patient has a preserved ejection fraction.

Adopt a 'watch and wait' approach with serial yearly echocardiography is incorrect. This could be considered as per patient preference, but in the case of severe mitral regurgitation with the development of new atrial fibrillation patients should be offered a referral for mitral valve replacement.

Refer for cardioversion is incorrect. In this case, the patient has known mitral regurgitation. Mitral disease is associated with the development of atrial fibrillation due to the associated structural changes in the left atrium. There is a much higher chance of maintaining sinus rhythm with mitral valve replacement compared to cardioversion. Mitral valve replacement is therefore the preferred option.

>Question #60

A 80-year-old man with a past medical history of gout, reflux and ischaemic heart disease is admitted to the emergency department with a atrial fibrillation with fast ventricular response. He is managed according to ALS protocol and is stabilised. A full set of bloods are sent and are displayed below:

Hb	135 g/l
Platelets	$260 * 10^9/l$
WBC	$6 * 10^9/l$

Mg	0.34 $\mu\text{mol}/l$
Ca (adj)	2.1 u/l
PO4	0.8 u/l

This is discussed with the cardiology registrar, who advises correction of the magnesium.

What medication is the most likely cause of hypomagnesaemia in this case?

- a) Aspirin
- b) Omeprazole
- c) Ranitidine
- d) Colchicine
- e) Ramipril

Correct answer is b.

Careful electrolyte balance is important in the management of arrhythmias.

During the generation of the action potential in cardiac pacemaker cells, phase 4 (inflow of potassium) is dependant on magnesium channels, and, although the exact effect *in vivo* of magnesium administration is unclear, restoring normomagnesaemia is important in patient presenting with dysrhythmias.

PPI use is associated with hypomagnesaemia - the exact mechanism of this is not known, but may be related to poorer intestinal absorption from dietary sources in patients on PPIs.

>Question #61

A 38-year-old female of Asian descent, with no significant past medical history, presents after a syncopal event while pruning hedges. She has had a 6-month history of fever, arthralgia and for the past few weeks has had multiple episodes of vertigo and one syncopal event. She denies headaches or visual complaints. Her examination reveals a diminished radial pulse in the left arm and a systolic blood pressure difference in the upper extremities of 14 mmHg. A bruit is auscultated along the left upper extremity. Dopplers of the upper extremities indicate a stenotic area along the subclavian that is later confirmed by Magnetic Resonance Angiography (MRA). She is diagnosed with subclavian steal syndrome. Laboratory tests reveal normocytic normochromic anaemia, elevated CRP and ESR, negative ANA and ANCA, and all other laboratory tests are within normal range.

Of the following, what is the most likely diagnosis?

- a) Fibromuscular dysplasia
- b) Ehlers-Danlos syndrome
- c) Takayasu arteritis
- d) Giant cell arteritis
- e) Wegener's granulomatosis

Correct answer is c.

This patient likely has Takayasu arteritis which is essentially a chronic vasculitis primarily of the aorta and its branches. These patients can present in a variety of

different ways depending on the vessels affected, however, they all typically have a prodrome of systemic symptoms including fatigue, weight loss and low-grade fevers prior to developing any vascular complaints. From a pathology standpoint biopsies of vessels are very similar to giant cell and are typically not performed. There are 6 criteria for the diagnosis of Takayasu.

Presence of 3 of the 6 has 90% sensitivity and specificity for diagnosis:

- 1. Age onset <=40 years
- 2. Claudication of the extremities
- 3. Decreased pulsation of one or both brachial arteries
- 4. Difference of at least 10 mm Hg in systolic blood pressure between the arms
- 5. Bruit over one or both subclavian arteries or the abdominal aorta
- 6. Arteriographic narrowing or occlusion of the entire aorta, its primary branches, or large arteries in the proximal upper or lower extremities, not due to arteriosclerosis, fibromuscular dysplasia, or other causes.

Fibromuscular dysplasia typically affects the renal arteries leading to renal artery stenosis and hypertension, and it not accompanied by other systemic manifestations like fever and malaise. Ehlers-Danlos syndrome is a genetic defect in type III collagen and can lead to aneurysms along with hyperelasticity of the skin and hypermobile joints, but other systemic manifestations are typically not present. Giant cell arteritis is most similar to Takayasu in pathology, however typically affects older patients and usually presents with headaches and tenderness over the temporal artery. Lastly, Wegener's is actually a small vessel vasculitis and its most common presenting symptoms include persistent rhinorrhoea, purulent/bloody nasal discharge, oral and/or nasal ulcers, polyarthralgias, myalgias, or sinus pain. Most with Wegener's are ANCA positive.

>Question #62

A 78-year-old gentleman is seen in clinic with long-standing heart failure with reduced ejection fraction (32%). He has had numerous admissions this year with

heart failure decompensation and is wondering if there is anything else that you can do for him. You review his ECG, which is in sinus rhythm with a heart rate of 64/min with a QRS of 136 msec and left bundle branch block. His blood pressure is: 98/55 mmHg. You also review his bloods:

Na ⁺	136 mmol/L	(135 - 145)
K ⁺	4.7 mmol/L	(3.5 - 5.0)
Urea	6.8 mmol/L	(2.0 - 7.0)
Creatinine	126 µmol/L	(55 - 120)

He is currently on carvedilol 25 mg BD, enalapril 10 mg BD, bumetanide 2 mg BD, aspirin 75 mg, ivabradine 2.5 mg BD.

What alteration to this gentleman's management could potentially decrease this gentleman's probability of being re-admitted?

- a) Arrange cardiac resynchronisation therapy-pacemaker (CRT-P) implantation
- b) Increase ivabradine
- c) Increase bumetanide
- d) Increase spironolactone
- e) Initiate sacubitril/valsartan

Correct answer is a.

Cardiac resynchronisation therapy can be used in patients with a QRS duration of >130 msec and LBBB morphology to improve symptomatology.

The European societies of cardiology (ESC) guidelines have highlighted the importance of cardiac resynchronisation in patients with heart failure. Evidence exists indicating the importance of implantation of such devices in patients with intraventricular delay with most studies focusing on the impact of left bundle branch block. The stronger evidence related CRT-P in such patients include left

bundle branch block (LBBB) and QRS >150 msec; however the presence of LBBB on its own provided that the QRS is longer than 120 msec is associated with improved mortality/morbidity. There is no evidence of benefit from CRT for patients with QRS <120 msec, while the presence of right bundle branch block (RBBB) implies a worse disease state where CRT may not necessarily benefit such patients.

The possibility of altering medications in this case remains attractive however one should be aware of the potential of introducing detriment to the patient in doing so.

This patient is already tolerating a relatively slow heart rate therefore ivabradine could potentially cause further slowing.

He is borderline hypotensive with raised potassium (making the possibility of increasing spironolactone less favourable).

>**Question #63**

A medical opinion was sought from the obstetrics team regarding a 38-year-old 28 weeks pregnant lady. A routine blood pressure check revealed a blood pressure of 158/98 mmHg. Other than suffering from hyperemesis gravidarum, her pregnancy had proceeded without complication. She specifically denied the presence of headaches, vomiting, any change in vision, abdominal pain, seizures or bleeding per vagina. She had noticed no change in the frequency of foetal movements, and her 20-week antenatal scan revealed the presence of a healthy foetus with a rate of growth within the expected range. Her past medical history was unremarkable; she was a non-smoker and did not consume alcohol. Her blood pressure at the booking antenatal appointment was 148/88 mmHg. Her sister suffered from pre-eclampsia during her pregnancy necessitating delivery by caesarean section. Examination of the cardiovascular system revealed normal heart sounds, a JVP of 3cm and the absence of pedal oedema. Examination of the neurological system was unremarkable with normal reflexes, cranial nerve function and peripheral

motor and sensory function. Urinalysis revealed no abnormality.

What is the next best management step?

- a) Commence ramipril
- b) Commence labetalol
- c) Commence indapamide
- d) Commence magnesium sulphate
- e) Transfer to high dependency unit to observe for signs of pre eclampsia

Correct answer is b.

This lady has pre-existing hypertension; her blood pressure at the antenatal booking clinic was elevated and continues to be elevated throughout (as opposed to gestational hypertension in which hypertension develops after 20 weeks). If untreated hypertension is associated with adverse maternal and foetal outcomes including intrauterine growth restriction, placental abruption, cerebrovascular accidents and prematurity. There are no clinical features of preeclampsia, including notably the presence of proteinuria and peripheral oedema, and there is, therefore, no indication for admission to a high dependency unit or to commence magnesium sulphate. Of the remaining options, labetalol is the safest antihypertensive to use in pregnancy; methyldopa is an alternative. The usual first line ACE inhibitors are absolutely contraindicated in pregnancy.

>>Question #64

A 52-year-old man is advised by his GP to attend hospital following a routine blood test demonstrating an increase in creatinine two weeks following a dose increase in his lisinopril. His lisinopril was increased from 5mg daily to 5mg twice a day.

He has a letter which demonstrates investigation results, shown below. He reports that home monitoring of blood pressure has shown his control to be better.

	21/11/2016	6/11/2016
Na ⁺	140 mmol/l	138 mmol/l

K ⁺	4.5 mmol/l	4.1 mmol/l
Urea	5.5 mmol/l	5.4 mmol/l
Creatinine	110 µmol/l	92 µmol/l

How should his lisinopril be managed and monitored?

- a) Continue lisinopril and repeat U&Es in 1-2 weeks
- b) Reduced the dose of lisinopril and repeat U&Es in 1-2 weeks
- c) Stop lisinopril and repeat U&Es in 1-2 weeks
- d) Stop lisinopril and arrange for an urgent out-patient imaging to exclude renal artery stenosis
- e) Continue lisinopril and repeat U&Es within 48 hours

Correct answer is a.

The correct answer is to continue lisinopril and repeat U&Es in 1-2 weeks. Up to an increase in creatinine of 30% can be tolerated following the start or increase in an ACE-inhibitor such as lisinopril, but U&Es should be repeated in 1-2 weeks. If the increase is greater than 30% then the ACE-inhibitor should be stopped.

>>Question #65

A 28-year-old male presents to the emergency department with chest tightness worse on inspiration. He informs you that over the past 3 days he has had a low-grade temperature and a degree of myalgia. Prior to all this he had never experienced any chest tightness. He was given glyceryl trinitrate spray and aspirin in the ambulance, neither of which has made any difference.

Examination is unremarkable; however you note that his temperature is 37.4°C. He is a smoker and he informs you that his father, mother and paternal uncle all have had heart attacks around the age of 60. His bloods are the following:

Creatinine	95 µmol/L	(55 - 120)
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CRP	35 mg/L	(< 5)
Troponin T (0hrs)	580 ng/L	(<14)
Troponin T (3hrs)	625 ng/L	(<14)
D-dimer	Negative	

An ECG is performed indicating sinus rhythm, no intraventricular delay and widespread ST elevations; you also notice was subtle PR depression.

What treatment should be initiated in the case of this gentleman?

- a) Arrange urgent angioplasty and potential revascularization
- b) Load patient with aspirin and clopidogrel
- c) Load patient with aspirin and ticagrelor
- d) Prescribe ibuprofen TDS
- e) Prescribe ibuprofen TDS and colchicine BD

Correct answer is e.

First line management of acute pericarditis involves combination of NSAID and colchicine.

The history, examination and blood investigations are consistent with acute pericarditis/myopericarditis. The high troponin results indicate myocardial injury which in this case can be attributed to myopericarditis. Troponin is a marker of myocardial injury which can result due to primary ischaemia (plaque rupture or intraluminal coronary artery thrombus), ischaemia secondary to misbalance of supply and demand (tachy/bradyarrhythmias, aortic dissection or severe aortic valve disease, sepsis, severe anaemia or respiratory failure etc.) and myocardial injury not necessarily associated to ischaemia or multifactorial (such as myopericarditis, severe pulmonary embolism with right heart strain, cardiac stunning, cardiac surgery or intervention etc.) There is no strong, dynamic rise in the already high troponin level.

The fact that he is a smoker undeniably puts this gentleman at a higher risk of coronary event, however his blood tests, ECG and history point further to the

direction of pericarditis. Given this, options 1, 2 and 3 which are associated with the management of acute coronary syndrome (ACS) are wrong.

The current guidelines as per the European Society of cardiology state that the treatment of choice in such patients is a combination of non-steroidal anti-inflammatory drugs (e.g. ibuprofen) and colchicine (option 5) and do not advocate the use of a single agent, such as ibuprofen (option 4).

>>Question #66

A 27-year-old female presents with palpitations and dizziness. She states that she has had a few episodes similar to this in the past and each time they have spontaneously resolved. On examination she has a blood pressure of 87/63 mmHg and a heart rate of 182bpm. An ECG shows an irregularly irregular rhythm, no identifiable P waves, and a QRS of 136ms.

Your senior decides to electrically cardiovert her. While the cardioversion is being prepared you instruct the patient to perform a vagal manoeuvre. Shortly after this she loses consciousness and goes into cardiac arrest.

What is the most likely underlying disorder?

- a) Torsades de Pointes
- b) Atrial fibrillation
- c) Atrioventricular nodal re-entry tachycardia (AVNRT)
- d) Monomorphic ventricular tachycardi
- e) Wolff-Parkinson-White syndrome (WPW)

Correct answer is e.

AV blocking drugs and vagal manoeuvres are absolutely contraindicated in patients with AF and pre-excitation

There are few arrhythmias, which can cause an irregularly irregular broad complex tachycardia. These include irregular atrial arrhythmias with bundle branch block (e.g. atrial fibrillation), and polymorphic ventricular tachycardia (including Torsades de Pointes). Another important group of conditions to be aware of include the pre-excitation syndromes.

Pre-excitation refers to early activation of the ventricles due to impulses bypassing the AV node via an accessory pathway. This results in a broad QRS complex e.g. the slurred upstroke of QRS (delta wave) as is seen in WPW. When present with atrial fibrillation, the accessory pathway allows for rapid conduction directly to the ventricles bypassing the AV node. This results in an irregularly irregular broad complex tachycardia.

The Valsalva manoeuvre increases the vagal tone resulting in AV node block. This promotes conduction of the atrial signals through the accessory pathway with a resultant increase in ventricular rate and possible degeneration into VT or VF, as is seen in this case.

AV blocking drugs and vagal manoeuvres are absolutely contraindicated in patients with AF and pre-excitation. In a haemodynamically unstable patient, urgent synchronised DC cardioversion is required. Medical treatment options in a stable patient include procainamide, although DC cardioversion may be preferred. Procainamide is a class 1 anti-arrhythmic which preferentially blocks the accessory pathway.

>>Question #67

A 47 year old woman presents to the emergency department with shortness of breath. She has been progressively getting worse over two weeks and has now started to feel short of breath on rest. She is known to have mitral valve prolapse and is awaiting surgery but has so far not been given a date. She also has type 2 diabetes and depression. She takes metformin, sertraline and furosemide.

On examination, she looks unwell. She has bilateral crepitations with no wheeze on auscultation of her chest, a raised JVP, and a systolic murmur. A chest X-ray shows pulmonary oedema. She is treated with IV diuretics but remains breathless and hypoxic. Which type of ventilatory support would be most appropriate?

- a) No ventilator support is appropriate
- b) Negative pressure ventilation

- c) Intubation
- d) Bilevel positive airway pressure (BIPAP)
- e) Continuous positive airway pressure (CPAP)

Correct answer is e.

Acute heart failure not responding to treatment - consider CPAP

The correct answer is continuous positive airway pressure (CPAP). This is a patient with a known mitral valve prolapse presenting with decompensation. It can be assumed that the lesion is significant as she is due to have surgery to repair it. She has developed signs of pulmonary oedema and she is not responding to treatment with diuretics. Vasodilators and opiates could be a plan of further medical management, but CPAP could help reduce pulmonary oedema. BIPAP would be appropriate in ventilatory failure in COPD, whilst negative pressure ventilation is more of historical treatment for polio.

Mitral valve prolapse has a prevalence of 2-3% in the population and the majority of cases will be asymptomatic. Risk factors for progression the severe disease include:

- Comorbidities: atrial fibrillation, left sided cardiac failure, hypertension and obesity.
- Echocardiogram findings: mitral leaflet thickness >5mm, prolapse of the posterior leaflet, moderate or severe regurgitation.
- Stress echocardiogram findings: regurgitation during exercise but not at rest.

>>Question #68

A 69 year-old male presents to the emergency department with central crushing chest pain which has been ongoing for 4 hours. It is associated with shortness of breath.

Blood results are as follows:

Urea	7.8 mmol/L	(2.0 - 7.0)
Creatinine	134 μmol/L	(55 - 120)
Troponin	8422 ng/L	(< 15)

An ECG is performed:



What is the most likely diagnosis?

- a) Anterolateral STEMI
- b) Inferolateral STEMI
- c) Myocarditis
- d) Pericarditis
- e) Posterior STEMI

Correct answer is b.

The ECG demonstrates ST elevation in the inferior (II, III, and aVF) and lateral leads (V5-6) confirming a diagnosis of an **inferolateral STEMI**. ST depression in V1-2 is suggestive of an associated posterior infarction. This constellation of ECG abnormalities is typically produced by occlusion of the proximal circumflex artery.

Anterolateral STEMI would present with ST elevation in the anterior (V1-V4) and lateral leads.

Posterior STEMI presents with reciprocal changes in the anterior leads including

horizontal ST depression and a dominant R wave in V2.

Both **myocarditis** and **pericarditis** present with global concave ST elevation. The troponin is usually normal in pericarditis and raised in myocarditis.

DiscussImprove

>>Question #69

A 59 year-old woman is complaining of a six-hour history of chest pain and dizziness in the high-dependency unit. She underwent a mitral valve replacement five days ago and had some temporary trans-venous pacing wires removed earlier today.

On examination, she has oxygen saturations of 93% on 2 litres via nasal cannula, has a heart rate of 110/min, a blood pressure of 76/43mmHg, has a temperature of 37.9°C, and is responsive to voice. She feels cool peripherally. An ECG is performed at the bedside which shows sinus rhythm with QRS complexes of alternating amplitude.

What is the most appropriate management given the likely diagnosis?

- a) Electrolyte replacement
- b) Emergency percutaneous coronary intervention
- c) Pericardiocentesis
- d) Prolonged course of antibiotics
- e) Urgent temporary pacing wire insertion

Correct answer is c.

Electrical alternans is suggestive of cardiac tamponade

This woman has clinical signs of cardiac tamponade which is confirmed by electrical alternans on ECG. Electrical alternans occurs due to the movement or 'swinging' of the heart within the pericardium as a result of the fluid present. This results in a variable amplitude of the QRS complex depending on the hearts

proximity to the ECG leads. In this case, this was likely precipitated by a traumatic removal of trans-venous pacing wires causing bleeding into the pericardial space. As a result, she requires an urgent echocardiogram to confirm the diagnosis and subsequent **pericardiocentesis** or re-sternotomy to drain the blood. It is worth noting that her acute kidney injury is the result of poor cardiac output as a result of her tamponade.

Although there are electrolyte abnormalities on this patient's blood tests that should be replaced, this is not likely to be the cause of her acute presentation. An arrhythmia post-operatively could cause a reduction in cardiac output however the ECG performed here demonstrates sinus rhythm and therefore **electrolyte replacement** would not reverse her current condition.

An acute coronary syndrome must be considered in cardiac post-operative patients complaining of chest pain however the electrical alternans on ECG with no remarkable ST changes to speak of is more consistent with a diagnosis of cardiac tamponade. Therefore, **emergency percutaneous coronary intervention** is not the most appropriate option here.

A **prolonged course of antibiotics** may be employed if a diagnosis of infective endocarditis is suspected however this is not the case here for a variety of reasons. Firstly, infective endocarditis post-operatively is unlikely to present within 5 days. Furthermore, the electrical alternans on ECG is indicative of cardiac tamponade rather than infective endocarditis. Finally, although the patient has a fever and a raised CRP, these are common findings post-operatively due to the systemic inflammatory response to surgery and are not specific for infective endocarditis.

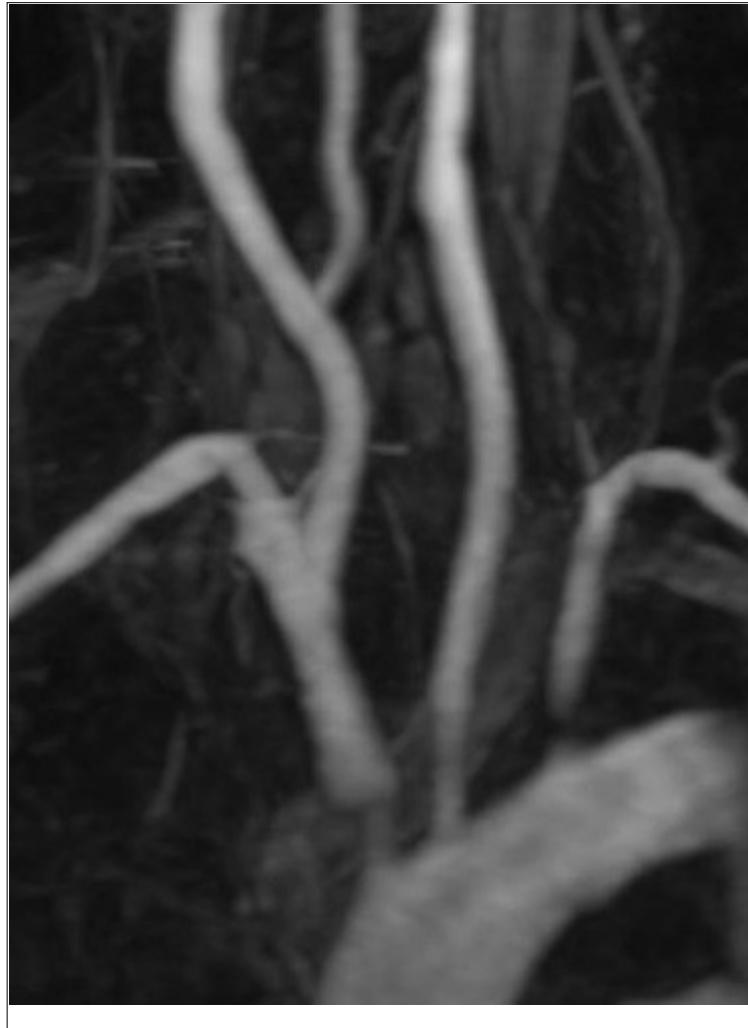
Arrhythmias are common in the post-operative period for patients who have undergone cardiac surgery, particularly valve surgery, however, this patient is in sinus rhythm at an acceptable rate. Therefore, as this patient's pathology is mechanical rather than electrical, **urgent temporary pacing wire insertion** will not help with her current issue.

>>Question #70

A 32 year-old woman is investigated for persistent malaise, fever, headaches and raised inflammatory markers. She was diagnosed with Raynaud's syndrome two years ago after complaining of cold hands. Her CRP level 64 mg/l.

On examination blood pressure is 82/65 mmHg in the right arm and 70/52 mmHg in the left arm. The heart rate is 74/min and a diastolic murmur is noted on auscultation of the heart.

Magnetic resonance angiography is requested:



What is the most appropriate management?

- a) Plasma exchange
- b) Rituximab
- c) Prednisolone
- d) Cardiothoracic surgery referral
- e) Intravenous immunoglobulin

Correct answer is c.

The magnetic resonance angiography (MRA) demonstrates stenoses of the supra-aortic arteries, especially the brachiocephalic trunk and the left subclavian artery with near-occlusion of the left vertebral artery. These findings are consistent with a diagnosis of Takayasu's arteritis.

>>Question #71

A 64 year old man with a past medical history of type 2 diabetes mellitus and hypertension attends the Emergency Department complaining of general malaise and feeling unwell. He had undergone successful primary coronary intervention (PCI) post myocardial infarction 5 weeks previously. He has a temperature of 38.2 degrees Celsius, blood pressure 166/88 mmHg and the doctor notes a lacy reticular rash extending over his legs.

Hb	14.2g/dl
Eosinophils	$1.3 * 10^9/l$
WBC	$13.5 * 10^9/l$
Urea	8mmol/l
Creatinine	144 μ mol/l

What is the most likely cause of his symptoms?

- a) Allergic rash to contrast
- b) Infected femoral site
- c) Cholesterol embolus
- d) Infective endocarditis
- e) Polyarteritis nodosa

Correct answer is c.

This is a classic story for a cholesterol embolus post an invasive arterial procedure, whereby a plaque has been ruptured and there is lodging of debris in small/medium arteries causing mechanical occlusion, inflammation and end-organ damage. Patients may have the lacy reticular rash of livedo reticularis and this, together with acute renal failure and an eosinophilia should lead the clinician to consider this diagnosis.

Patients tend to present with non-specific symptoms such as fever, weight loss and myalgia for weeks to months before developing end-organ damage or stroke.

The current mainstay of treatment is supportive.

>>Question #72

A 33 year-old woman is brought into the Emergency Department by her husband. He tells you that she is abnormally drowsy and he has difficulty rousing her. She has a background of depression which is medicated by her General Practitioner, but is otherwise well.

On examination she is afebrile but has dry, warm skin. Her heart rate is 144bpm with a blood pressure of 108/90mmHg. She has a respiratory rate of 8 breaths per minute and saturating at 92% on air. Her Glasgow coma scale (GCS) is 12 (E3V4M5) and she appears agitated. Her pupils are dilated and she has hyperreflexia. Whilst examining her abdomen it is noted that she has a palpable

bladder and bowel sounds are absent.

Investigations:

Arterial blood gas (ABG):

pH	7.28	(7.35-7.45)
PCO ₂	7.3 kPa	(4.9-6.1 kPa)
PO ₂	10.1 kPa	(10-13.1 kPa)
Bicarbonate (HCO ₃ -)	28 mmol/L	(22-28 mmol/L)
Base Excess (BE)	3.1 mmol/L	(-2 to 2 mmol/L)
Sodium	134 mmol/L	(135-145 mmol/L)
Potassium	4.4 mmol/L	(3.5-5.5 mmol/L)
Chloride	105 mmol/L	(95-110 mmol/L)
Lactate	2.2 mmol/L	(0.2-1.6 mmol/L)
Glucose	5.3 mmol/L	(4-7 mmol/L)

ECG - Polymorphic ventricular tachycardia, QTc 510ms.

What is the most important next step in the management of this patient?

- a) Synchronised DC cardioversion
- b) Intravenous amiodarone
- c) Intravenous magnesium sulphate

- d) Intravenous lidocaine
- e) Intravenous sodium bicarbonate

Correct answer is c.

IV magnesium sulfate is used to treat torsades de pointes

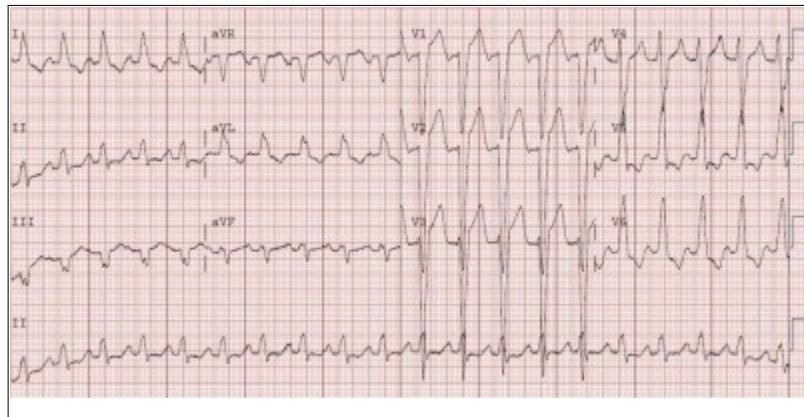
This woman has had a tricyclic antidepressant (TCA) overdose. In excess, TCAs can cause QT interval prolongation which can predispose to ventricular arrhythmias. A polymorphic ventricular arrhythmia with a prolonged QT interval is characteristic of torsades de pointes. The treatment for this is intravenous magnesium sulphate.

As she currently has a stable blood pressure (systolic > 90mmHg), DC cardioversion would not be appropriate at this stage. Intravenous amiodarone and lidocaine can be used to medically cardiovert a monomorphic ventricular tachycardia, but are not the treatment of choice in torsades de pointes.

Sodium bicarbonate is the first-line treatment of TCA overdose. However, in the presence of torsades de pointes, intravenous magnesium sulphate is the more appropriate choice to give initially.

>>Question #73

A 64 year-old man presents with intermittent chest pains for the past three days. Whilst you are taking the history he complains of worsening pain and you arrange an ECG immediately:



He is tachycardic and sweaty with a blood pressure of 124/86mmHg. What does the ECG show?

- a) Narrow complex tachycardia with ST elevation
- b) Right bundle branch block with ST elevation
- c) Left bundle branch block with ST elevation
- d) Ventricular tachycardia with ST elevation
- e) Posterior myocardial infarction

Correct answer is c.

The ECG shows left bundle branch block (LBBB). The relationship between new LBBB and acute coronary syndrome (ACS) is complicated with many cardiologists ignoring the standard guidelines (both UK and US) to initiate reperfusion therapy due to the poor correlation seen in clinical practice between coronary lesions and standard LBBB. The Sgarbossa criteria have therefore been developed to evaluate whether new LBBB represents an ACS. In this particular ECG there is more ST elevation than is normally expected with LBBB indicating a likely ACS.

>>Question #74

A 71-year-old patient presents to the Emergency Department with a two hour history of crushing central chest pain. He is known to have a history of ischaemic heart disease. The ECG shows the following:

- ST elevation greater in lead II than in lead III with abnormal Q waves in II, III, and aVF
- ST depression, tall, broad R waves and upright T waves in V1-3. Dominant R wave in V2
- ST elevation in V5-V6

Where is the lesion most likely to be?

- a) Left anterior descending
- b) Left circumflex
- c) Right coronary artery
- d) Left main stem
- e) Posterior interventricular

Ischaemic changes in leads I, aVL +/- V5-6 - left circumflex

These are classical findings of a circumflex occlusion. The table below shows how the changes correspond to the cardiac anatomy:

ECG changes	Component of infarction
ST elevation greater in lead II than in lead III with abnormal Q waves in II, III, and aVF	Inferior component of infarction
ST depression, tall, broad R waves and upright T waves in V1-3. Dominant R wave in V2	Posterior component of infarction
ST elevation in V5-V6	Lateral component of infarction

Please see the link for an example ECG with a description of the changes.

Question #75

A 24-year-old female is brought into the emergency department by ambulance. She has a family history of Wolff-Parkinson-White syndrome and was binge drinking last night. She is complaining of palpitations which she says started suddenly 1 hour ago.

Her observations are as follows

- Heart rate 180 bpm
- Blood pressure 100/60 mmHg
- Saturations 98% on air
- Respiratory rate 26/min
- Temperature 36.8°C

An ECG (electrocardiogram) is done which shows pre-excited atrial fibrillation (AF)

Which of the following is an appropriate treatment?

- a) Adenosine
- b) Atenolol
- c) Flecainide
- d) Diltiazem
- e) Digoxin

Correct answer is c.

In pre-excited AF don't give anything that blocks conduction at AV node (including calcium channel blockers, adenosine or digoxin) as this can cause ventricular tachycardia

Flecainide is the only appropriate treatment on this list

All of the other options are drugs which block transmission through the AV (atrioventricular) node. Giving these drugs in pre-excited AF (atrial fibrillation) can precipitate VT (ventricular tachycardia) or VF (ventricular fibrillation).

Note that flecainide is contraindicated in patients with structural heart abnormalities. In this situation then amiodarone can be given instead.

If any of the following are present: hypotension, signs of shock, altered mental status, chest pain or acute heart failure then synchronised cardioversion should be used rather than medication.

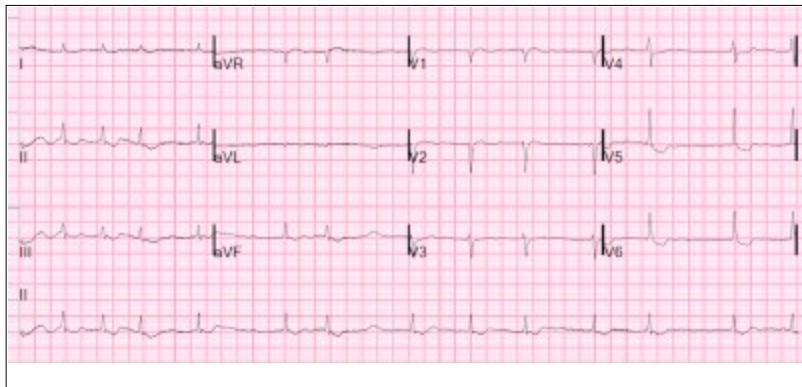
Discuss (6)Improve

Question #76

A 77-year-old woman is seen in a heart failure clinic. Her past medical history includes congestive cardiac failure, atrial fibrillation, hypertension, and depression. Her regular medications include bisoprolol, digoxin, amlodipine, furosemide, and citalopram.

Repeat digoxin levels are displayed below:

Digoxin level	1.1 ng/mL	(0.7-2.0)
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Which agent is responsible for the abnormalities displayed in her ECG?

- a) Amlodipine
- b) Bisoprolol
- c) Citalopram
- d) Digoxin
- e) Furosemide

Correct answer is d.

This woman's ECG displays 'scooped' ST depression in leads II, III, aVF, V5, and V6 in the context of atrial fibrillation. This is a classical sign seen in the digoxin effect, the ECG changes that arise with digoxin within normal therapeutic levels. It is important to note that in digoxin toxicity, the most common dysrhythmias are supraventricular tachycardia with a slow ventricular response due to blocking of the AV node.

Whilst calcium channel blockers can significantly disrupt the ECG, this is associated primarily with non-dihydropyridines such as verapamil or diltiazem, rather than dihydropyridines such as amlodipine which are less cardioselective.

Bisoprolol can give rise to bradycardia and PR prolongation on ECG but the ST changes observed here are consistent with the digoxin effect. Therefore, bisoprolol is incorrect.

Citalopram is a selective serotonin reuptake inhibitor and therefore can result in QT prolongation. The ST changes as described here are not consistent with this

syndrome and therefore this is an incorrect answer.

Furosemide is a loop diuretic that can result in hypokalemia and therefore flattened and inverted T waves. The ST segment in this ECG shows the classical 'scooped' appearance and therefore is more consistent with the digoxin effect rather than hypokalemia. Furosemide is therefore incorrect.

Question #77

A 62-year-old man sees his GP with complaints of leg pain. Over the last few months, he notices a crampy uncomfortable feeling in the back of both of his calves when he walks to the shops. The discomfort can be so bad that he has to stop and rest for a few minutes after which he notices that his symptoms are improved. On occasions, he also gets the symptoms when he is shopping in the supermarket. He attributes this to overexerting himself and tries to rest on his shopping trolley while walking but does not seem to help. He has a 40-pack year smoking history and takes amlodipine for his blood pressure, and paracetamol and ibuprofen for lower back pain that has troubled him for years.

Physical examination reveals mild atrophy of his thigh and calf muscles bilaterally, in addition to shiny pale skin with significant hair loss throughout his lower limbs. His pedal pulses are bilaterally impalpable, and popliteal pulses are faint. Power in both lower limbs is normal throughout all movements, and he has normal patellar reflexes bilaterally and absent ankle reflexes. His Babinski reflex is downgoing on the left side and equivocal on the right side. A recently obtained ankle brachial pressure index test yielded a result of 0.70 on the right side and 0.95 on the left side. X-rays of his lumbar spine show evidence of joint space narrowing and osteophytes.

Which one of the following is the next best step in the management of this patient?

- a) MRI scan of lumbar spine
- b) Check his HbA1c to screen for diabetes

- c) Refer to vascular surgery for consideration of peripheral arterial stenting or bypass surgery
- d) Starting him on duloxetine to manage his pain
- e) Screen for coronary artery disease with ECG stress testing

Correct answer is c.

In patients with cardiovascular risk factors and symptoms suggestive of claudication with an equivocal/borderline ankle brachial pressure index study result, the next best study is an ankle brachial pressure index after exercise

This gentleman has symptoms of claudication, in particular, vascular claudication. Although he does have a history of chronic back pain for which he takes analgesia, he is less likely to have neurogenic claudication compared to vascular claudication. An important discriminating factor between neurogenic claudication and vascular claudication is that patients with neurogenic claudication have symptoms on exertion that improve with manoeuvres such as leaning forward, and sitting down, whereas vascular claudication does not change with these manoeuvres and only improves with rest. In addition, his physical exam shows evidence of peripheral arterial disease characterised with muscle atrophy, shiny skin with hair loss and impalpable pedal pulses. Absent ankle jerk reflexes and equivocal Babinski reflexes can be normal variants and do not always indicate neurological pathology. This patient has rightly already undergone an ankle brachial pressure index assessment, and the results indicate a diagnosis of peripheral arterial disease. He is functionally impaired by his symptoms and so the next best step in his management would be to refer him to vascular surgery for consideration of treatment strategies which may include percutaneous interventions with stenting and/or surgical bypass.

Although this patient has evidence of degenerative joint disease on his lumbar spine x-ray, his clinical presentation is not consistent with neurogenic claudication and so an MRI scan of his lumbar spine is not indicated.

Although it is important to screen for and aggressively manage cardiovascular risk factors in patients with peripheral arterial disease, this in itself would not address the patient's symptoms or the disease course.

This patient's pain is most consistent with vascular claudication as opposed to neuropathic pain, for which an agent like duloxetine would be appropriate.

Although individuals with peripheral arterial disease have a significant likelihood of having concomitant coronary disease, this patient is not complaining of angina or symptoms suggestive of coronary disease and so screening for this is not indicated. The next best step in managing this patient is to address and treat his symptoms related to his peripheral arterial disease.

Question #78

A 77-year-old man with known atrial fibrillation is admitted following an upper gastrointestinal haemorrhage. His atrial fibrillation is managed using bisoprolol and warfarin. Since his admission, he has had four large episodes of haematemesis. You, the emergency department doctor, request the patient's INR to be checked as one of a series of investigations. The haematology laboratory phone through and inform you his INR is 8.5. He is currently hypotensive (90/45 mmHg) and tachycardic (120 beats per minute). You begin resuscitation using 0.9% saline, and send a cross match, group and save. What is the most appropriate treatment of this patients INR?

- a) Fresh frozen plasma + stop warfarin
- b) Vitamin K + stop warfarin
- c) Prothrombin complex concentrates
- d) Prothrombin complex concentrates + vitamin K + stop warfarin
- e) Stop warfarin

Correct answer is d.

Major bleeding - stop warfarin, give intravenous vitamin K 5mg, prothrombin complex concentrate

The nub of this question is the emergency management of haemorrhage in patients on warfarin. This patient has an INR greater than 8 and is actively bleeding. Therefore the answer is 4.

Patients on warfarin have reduced levels of Factor X, IX, VII and II. Rapid correction is most effectively achieved through administration of prothrombin complex concentrates.

The British Journal of Haematology states that: 'Emergency anticoagulation reversal in patients with major bleeding should be with 2550 u/kg four-factor prothrombin complex concentrate and 5 mg intravenous vitamin K'

Question #79

An 89-year-old woman presents to the emergency room with increased shortness of breath and decreased mobility of 1-week duration. Her shortness of breath is worse at night and she sometimes wakes up gasping for breath. She has fallen over twice in the last week which isn't normal for her. She has a past medical history of diabetes mellitus type 2 and hypertension. She has had no previous surgeries. She lives independently with her husband and her daughter lives close by and helps with the shopping.

On examination, she is found to have coarse crackles bi-basally on auscultation of her chest. She has a regular heart rate with a pan-systolic murmur loudest over the apex.

Investigations show:

Haemoglobin	11g/dl
WCC	$6 \times 10^9/l$
Platelets	$178 \times 10^9/l$
Sodium	139 mmol/l
Potassium	4.2 mmol/l

Urea	8 mmol/l
Creatinine	92 µmol/l
Blood cultures	<i>Methicillin-sensitive Staphylococcus aureus</i>

Echocardiogram severe mitral regurgitation with large mobile structure on valve leaflet

Chest X-ray: bilateral blunting of the costophrenic angles and upper lobe diversion

What is the best treatment for this lady?

- a) Flucloxacillin orally
- b) Flucloxacillin intravenously
- c) Amoxicillin orally and vancomycin intravenously
- d) Ceftriaxone intravenously
- e) Amoxicillin intravenously and vancomycin intravenously

Correct answer is b.

This lady has a native mitral valve endocarditis with a bacteraemia caused by a methicillin-sensitive *Staphylococcus aureus*. Once cultures have grown the causative bacteria and it is found to be sensitive to methicillin guidelines suggest flucloxacillin intravenously to be the treatment of choice. Flucloxacillin orally would not provide sufficient antimicrobial cover nor will ceftriaxone, amoxicillin and vancomycin.

There is some debate about the optimal length of treatment, but 6 weeks of intravenous therapy is generally accepted as the length of treatment needed. Shorter therapeutic regimens may be effective in selected patients with right-sided endocarditis and with endocarditis due to highly susceptible *Streptococcus viridans* treated with synergistic antimicrobials.

Question #80

A 28-year-old lady pregnant lady of 37 weeks gestation presented to the Emergency Department a few hours earlier with new onset chest pain. The pain was sharp and made worse with inspiration. She also complained of rapid onset progressively increasing shortness of breath, affecting her ability to complete sentences. She denied the presence of a cough or sputum production and did not suffer from haemoptysis or calf pain. Other than a successful external cephalic version for a breech presentation five days ago and a placenta praevia which spontaneously resolved, her pregnancy was unremarkable. She was in good health with an unremarkable past medical history and normal routine investigations throughout her pregnancy. She smoked 15 cigarettes per day and did not consume alcohol. Her mother suffered from an unexplained deep vein thrombosis when she was 42 years old but otherwise her family history was unremarkable.

Initial examination revealed a heart rate of 122bpm, respiratory rate 24/min, oxygen saturations of 98% on air, a temperature of 37.6°C and a blood pressure of 112/72 mmHg. She was struggling to complete full sentences. Examination of her cardiorespiratory system revealed good air entry in both bases, a JVP of 3cm, the absence of pedal oedema and soft and non-tender calves. Examination of her gastrointestinal system was unremarkable and ultrasound auscultation revealed the presence of a fetal heartbeat.

As she was being tended to for initial investigations, her condition rapidly deteriorated. Her oxygen saturation dropped to 88% on air, and her blood pressure was recorded as 88/66mmHg. She appeared cool and clammy, and her respiratory rate increased to 32/min. The doctor tending to venepuncture noted the presence of oozing of blood from the wound. She was promptly transferred to the intensive care unit and the following investigations were conducted:

Hb	101 g/l
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Platelets	$75 * 10^9/l$
WBC	$12.2 * 10^9/l$

Na ⁺	136 mmol/l
K ⁺	4.8 mmol/l
Urea	14.1 mmol/l
Creatinine	158 μmol/l

INR	3.9
APTT	84 s
D-Dimer	2920 ng/ml

Urinalysis: ketones ++, leucocytes/nit/prot/blood/glucose negative

Portable chest x-ray: normal appearance of heart and lung fields

ECG: sinus tachycardia 148bpm, T wave inversion leads V3-V6

Arterial blood gases on air:

pH	7.48
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PaO ₂	5.9 kPa
PaCO ₂	2.2 kPa
BE	1
HCO ₃	24 mmol/l

She was commenced on 15 litres/min oxygen via non-rebreathe mask, and an arterial line and central venous line were sited, as well as the presence of two large bore peripheral cannulae. She was immediately commenced on 2 litres of Hartmann's solution stat.

What is the most likely diagnosis?

- a) Pulmonary embolus
- b) Septic shock
- c) Peripartum cardiomyopathy
- d) Aortic dissection
- e) Amniotic fluid embolus

Correct answer is e.

In this instance, pulmonary embolism would not account for the presence of DIC and there is no evidence of deep vein thrombosis clinically. Septic shock can follow a similar path to amniotic fluid embolus, but in this instance, there is little evidence for a focus of sepsis

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- d) Aortic dissection
- e) Amniotic fluid embolus

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Question #83

A 77-year-old man has attended the Emergency Department as he is very concerned about a purple lacy rash that has appeared on his arms and legs over the past few days. It is not painful and does not itch. His past medical history includes hypertension, type 2 diabetes, diverticulitis and atrial fibrillation. He takes amlodipine, ramipril and metformin and was started on warfarin one week ago by his GP. Examination confirms a widespread purple mottled rash across all four limbs and the abdomen. His chest is clear, heart sounds are normal and the abdomen is soft, non-tender.

Blood results on admission show:

Hb	119 g/l
MCV	85 fl
Platelets	142 * 10 ⁹ /l
WBC	10.1 * 10 ⁹ /l
Neuts	4.5 * 10 ⁹ /l

Lymphs	$1.9 * 10^9/l$
Eosinophils	$1.5 * 10^9/l$

Na ⁺	142 mmol/l
K ⁺	4.6 mmol/l
Urea	14.6 mmol/l
Creatinine	169 µmol/l

What is the most likely diagnosis?

- a) Amyloidosis
- b) Antiphospholipid syndrome
- c) Cholesterol atheroemboli
- d) Churg-Strauss syndrome
- e) Focal segmental glomerulosclerosis

Correct answer is c.

The purple, lacy, non-itchy rash described here is livedo reticularis.

Antiphospholipid syndrome, Churg-Strauss syndrome and cholesterol atheroemboli can all cause livedo reticularis

Churg-Strauss and cholesterol atheroemboli could both result in raised eosinophils as seen here. However, this patient was started on warfarin a week ago by his GP which is a recognised precipitant of cholesterol atheroemboli which makes it the most likely diagnosis here.

Question #83

A 70-year-old man is admitted with fever, chills and dysuria. He has a medical history of ischaemic heart disease, hypertension, hyperlipidaemia and diabetes. His current medications include aspirin, ramipril, bisoprolol, atorvastatin, metformin and dapagliflozin. He is an ex-smoker and requires assistance with personal care. He is started on cefuroxime for urosepsis. His admission ECG shows:



What is the most likely diagnosis?

- a) Atrial fibrillation
- b) Atrial flutter
- c) Sinus tachycardia
- d) Ventricular tachycardia
- e) Wolff Parkinson White syndrome

The ECG shows an atrial rate of 300 bpm and ventricular rate of 150 bpm suggesting atrial flutter with a 2:1 AV block (regular p waves seen as notched t waves can be better appreciated in lead III, V1 and V2).

Atrial flutter is the correct answer. There are regular saw-toothed P waves at 300 bpm. There are regular QRS complexes at 150 bpm. Thus, this is atrial flutter with a rapid ventricular response and 2:1 AV block.

Atrial fibrillation is incorrect. Atrial fibrillation, unlike atrial flutter, is irregular with fibrillatory p waves caused by disorganised and chaotic atrial electrical activity. Contrary to atrial fibrillation, atrial flutter is more amenable to cardioversion than

medications.

Sinus tachycardia is incorrect. In sinus tachycardia, every P wave is followed by a QRS complex and the PR interval is <200 ms. However, in this patient's ECG, not every p wave is followed by a QRS complex suggesting a 2:1 AV block. Moreover, the saw-toothed P waves at 300 bpm and prolonged PR interval is suggestive of atrial flutter.

Ventricular tachycardia is a broad complex tachycardia. The QRS complex here is narrow measuring < 120 ms and regular at 150 bpm suggesting an Atrial Flutter with 2:1 block.

Wolff Parkinson White syndrome is incorrect. WPW is a pre-excitation syndrome characterised by PR interval <120 ms, delta wave and QRS >110 ms which are absent in this patient's ECG.

Discuss (1)Improve

Question #84

A 66-year-old gentleman requests to see his general practitioner, concerned regarding one of the medications that he was started on following a myocardial infarction. He tells you that the cardiology team has started him on atorvastatin, which he does not wish to take unless there is no other alternative, as he has had relatives that had bad experience with simvastatin, including muscle pain and disturbed sleep.

He has no family history of hypercholesterolaemia at a young age, but a number of his relatives are on statins following cardiac events.

What options exist with regards to his secondary prevention?

- a) Ezetimibe 10 mg OD
- b) Continue with atorvastatin at the current dose

- c) Fenofibrate 160 mg OD
- d) Lomitapide 5 mg OD
- e) Niacin (as per product literature)

Correct answer is b.

Statins are the only lipid-regulating drugs that are used in secondary prevention of cardiovascular disease (with the exception of ezetimibe in cases of primary hypercholesterolaemia)

With regards to secondary prevention the only NICE approved medication at the moment is statins. None of the other drugs mentioned above are licensed for secondary prevention with the exception of ezetimibe in primary hypercholesterolaemia. There is no indication, in a 66 year old gentleman that he is suffering from primary hypercholesterolaemia. As no dose is given in the question, one should assume that continuing at the current dose provided that the patient understands that no alternative exists is the right answer. Indeed if a dose was given, reducing the dose or switching to an even more hydrophilic agent (such as pravastatin) would be an appropriate option.

Question #85

A 22-year-old male presents to clinic complaining of several episodes of heart palpitations. He states that during the attacks his heart beats extremely fast and he feels dizzy.

You arrange a 7 day cardiac holter monitor. The results are as follows:

PR interval	95ms
QRS duration	105ms
Events	One episode of tachycardia of 145 bpm with normal QRS morphology and QRS 110ms

What is the most likely diagnosis?

- a) Lown-Ganong-Levine syndrome
- b) Wolff-Parkinson-White syndrome type A
- c) Wolff-Parkinson-White syndrome type B
- d) Monomorphic ventricular tachycardia
- e) Polymorphic ventricular tachycardia

Correct answer is a.

Lown-Ganong-Levine syndrome (LGL) is a pre-excitation syndrome of the heart characterised by a short PR interval and normal QRS width

The key thing to note is the short PR interval. The normal PR interval is 120-200ms. A short PR interval is suggestive of a pre-excitation syndrome. Pre-excitation syndromes occur due to an accessory pathway between the atria with the ventricles, resulting in early depolarisation of the ventricles and a tendency for tachyarrhythmias. Pre-excitation through the accessory pathway results in a short PR interval.

The normal QRS width (normal width <120ms) precludes the diagnosis of Wolff-Parkinson-White syndrome which is characterised by a wide QRS with a slurred upstroke (delta wave). The normal QRS also rules out ventricular tachycardia.

The diagnosis is therefore Lown-Ganong-Levine syndrome (LGL) which is a pre-excitation syndrome of the heart characterised by a short PR interval and normal QRS width.

Discuss (6) Improve

Question #86

A 65-year-old Caucasian man attends the dermatology clinic with a 3-week history of a new rash. He complains of a discolouration of his forearms and hands that initially appeared whilst on holiday in the south of France. It is mildly itchy but not

painful. There is no history of a change in diet or detergents and he has no known allergies. His past medical history includes hypertension, type 2 diabetes, ischaemic heart disease and atrial fibrillation.

On examination, there is a purplish discolouration of his hands up to his elbows bilaterally. His face and scalp are mildly erythematous. There is no blistering or crusting.

Which of the following is most likely to be responsible for this patient's presentation?

- a) Aspirin
- b) Digoxin
- c) Indapamide
- d) Metformin
- e) Ramipril

Correct answer is c.

Thiazides may cause photosensitivity

This patient has drug-induced photosensitivity, a skin reaction secondary to UV radiation causing expected burns or dermatitis in patients taking photosensitising medications. Drugs which cause phototoxicity include: antibiotics (tetracyclines, sulfonamides, fluoroquinolones), NSAIDs, diuretics, sulfonylureas, antipsychotics as well as amiodarone, quinine and hydroxychloroquine. The clinical features of photosensitivity can vary according to the medication taken as well as the type of reaction. Some reactions may be phototoxic whilst others are photoallergic. A phototoxic reaction results from direct damage mediated by UV activation of the photosensitising agent. These reactions can appear within minutes to hours after exposure and appears as a sunburn reaction with reddening and swelling. Rarely, the skin may change pigmentation (e.g. blue-green discolouration seen with amiodarone). In severe reactions, blisters and vesicles may be seen. Other symptoms may include itch, as seen in this scenario. Thiazides typically cause a phototoxic reaction. In contrast, a photoallergic reaction is a cell-mediated response. These are less common and present as an eczematous, itchy skin

reaction within 24-72 hours after exposure to sunlight. Unlike phototoxic reactions, photoallergic reactions are capable of spreading to areas that have not been exposed to sunlight. The treatment of drug-induced photosensitivity is to avoid the trigger, if possible. If the medication is essential to be taken then protective measures should be taken including sunscreen or protective clothing.

In toxic doses, digoxin is capable of causing side effects including arrhythmias, dizziness, yellow or green visual changes and skin reactions. However, they do not commonly cause photosensitivity. If there are any concerns regarding digoxin toxicity, a digoxin level can be taken with the therapeutic window ranging between 0.7ng/mL and 2.0ng/mL.

Aspirin is a common cause of acute or chronic urticaria. However, it does not cause photosensitivity.

Metformin and ramipril do not commonly cause a rash or skin discolouration. In rare cases, patients may have an allergic reaction to these medications which results in the eruption of urticaria and, in extreme cases, lip and tongue swelling and airway compromise. However, they do not cause photosensitivity.

Question #87

A 28-year-old man presented with heart palpitations. He states that he has had a couple of episodes each week for the past 3 months. He describes the palpitations as a rapid beating of his heart. He has no past medical history and takes no regular medicines.

An ECG is performed:

P waves	Normal morphology. Inverted in lead I
PR interval	130ms
QRS	110ms. Loss of R wave progression in chest leads

QTc	410ms
Axis	Right axis deviation

What is the most likely explanation of the ECG results?

- a) Torsades de pointes
- b) Wolff-Parkinson-White (WPW) syndrome
- c) Misplacement of the limb lead
- d) Dextrocardia
- e) AV nodal reentrant tachycardia (AVNRT)

Correct answer is d.

Dextrocardia is associated with an inverted P wave in lead I, right axis deviation, and loss of R wave progression

The inverted P wave in lead I, right axis deviation, and loss of R wave progression should alert you to dextrocardia. Misplacement of the limb leads can cause a similar picture with inverted P wave in lead I and right axis deviation (RAD), however we would not expect loss of R wave progression, making dextrocardia the best answer in this question.

The PR interval is not short, and there is no delta waves present, making Wolff-Parkinson-White (WPW) unlikely.

The QTc is normal making Torsades de pointes unlikely.

Question #88

A 70-year-old woman with a history of type 2 diabetes mellitus and hypertension is reviewed in clinic. You can see from the records their is no evidence of diabetic retinopathy, chronic kidney disease or cardiovascular disease.

Her current medication is as follows:

- simvastatin 40mg on
- ramipril 10mg od
- amlodipine 5mg od
- metformin 1g bd

Recent blood results are shown below:

Na ⁺	142 mmol/l
K ⁺	4.4 mmol/l
Urea	7.2 mmol/l
Creatinine	86 µmol/l
HbA1c	45 mmol/mol (6.3%)

Urine dipstick shows no proteinuria. Her blood pressure today in clinic is 134/76 mmHg.

What is the most appropriate course of action?

- Add gliclazide
- Increase amlodipine
- Increase ramipril
- Add losartan
- No changes to medication required

Correct answer is e.

Newly diagnosed patient of black African or African-Caribbean origin with hypertension - add a calcium channel blocker

Her diabetic control is good - NICE do not advocate changing treatment at this stage unless the HbA1c is $\geq 6.5\%$.

As she has no complications from her diabetes the blood pressure target is $< 140/80 \text{ mmHg}$. No changes are therefore required to her antihypertensive regime.

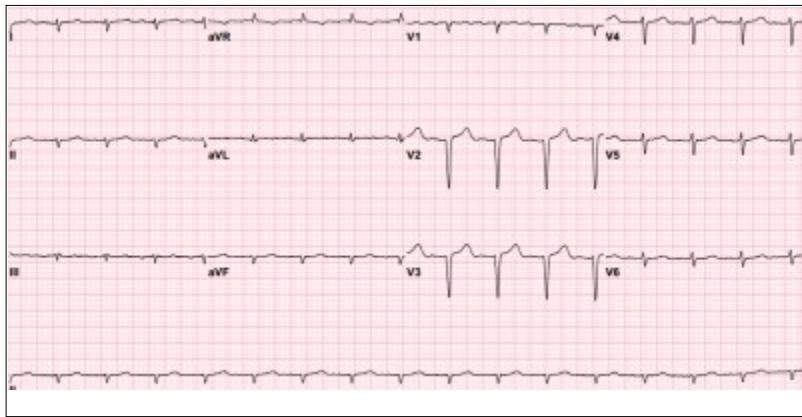
Discuss (7)Improve

Question #89

An 81-year-old man is referred to the cardiology clinic due to worsening heart failure. Over the past 12 months he has become increasingly short-of-breath, particularly upon exertion or when lying flat. He has also suffered from ankle swelling which his GP has treated with large doses of furosemide. Around 6 months ago a clinical diagnosis of heart failure was made and he is already taking bisoprolol, ramipril and atorvastatin. He was diagnosed with multiple myeloma around 3 years and has been treated with melphalan to date.

On examination his pulse is 72/min and blood pressure 98/62 mmHg. The JVP is elevated at 6cm above the angle of Louis. There are crackles in both bases and pitting oedema to his knees.

An ECG accompanies the referral:



- a) Constrictive pericarditis
- b) Infective endocarditis
- c) Chronic pulmonary embolism
- d) Cardiac amyloidosis
- e) Hypercalcaemia-induced heart failure

Correct answer is d.

This patient is likely to have AL amyloidosis secondary to his myeloma. This has resulted in cardiac amyloidosis as evidenced by the low voltage QRS complexes associated with poor R wave progression in the chest leads (a pseudoinfarction pattern).

Question #90

An 18-year-old man attends the clinic with blurred vision. This has progressively worsened over the past few years. He has a past medical history of recurrent deep vein thromboses and mild learning difficulty. On examination, you note an increased arm span to body height ratio, and the presence of scoliosis. Ophthalmological assessment reveals a downward lens dislocation.

What is the most likely diagnosis?

- a) Ectopia lentis syndrome
- b) Ehlers danlos syndrome
- c) Homocystinuria
- d) Marfan's syndrome
- e) Multiple endocrine neoplasia type 2B

Correct answer is c.

Tall, long fingered, downward lens dislocation, learning difficulties, DVT - homocystinuria

The patient has several clinical features of a Marfanoid body habitus. The differential for a Marfanoid body habitus is wide and includes Marfan's syndrome and homocystinuria.

Homocystinuria is correct. The patient has several clinical features associated with homocystinuria (e.g. Marfanoid body habitus, recurrent DVTs, learning disability and ectopia lentis). It is important to remember that the ectopia lentis in homocystinuria is inferonasal, in contrast to the superior-temporally dislocation associated with Marfan's syndrome.

Ectopia lentis syndrome is incorrect. Ectopia lentis syndrome is an inherited connective tissue condition that shares some of the features of Marfan's syndrome - particularly lens dislocation of the eye. However, patients with ectopia lentis syndrome do not have any vascular complications associated with Marfan's syndrome. Patients with ectopia lentis syndrome do not have any of the other features demonstrated in this case.

Ehlers danlos syndrome is incorrect. The Ehlers-Danlos syndromes (EDS) are generally characterised by joint hypermobility, joint instability and dislocations, scoliosis, and other joint deformities. In the rarer types of EDS, there is also a weakness of specific tissues that can lead, for example, to major gum and dental disease, eye disease, cardiac valve and aortic root disorders, and life-threatening abdominal organ, uterine, or blood vessel rupture. Thrombotic complications and lens dislocation are rare in this condition making it a less likely diagnosis.

Marfan's syndrome is incorrect. Although the patient has a Marfanoid body habitus, the presence of downward lens dislocation, learning difficulties, and recurrent DVTs favour the diagnosis of homocystinuria.

Multiple endocrine neoplasia type 2B is incorrect. This is a rare, genetic disorder that affects the endocrine glands and causes medullary thyroid cancer,

pheochromocytoma, and parathyroid gland cancer. Although it can be associated with a Marfanoid body habitus, there are no other clinical features to suggest that this is the diagnosis.

Question #91

An 84-year-old male was admitted to the coronary care unit (CCU) after being admitted as a primary percutaneous coronary intervention (PPCI) call for sudden onset chest pain and ST elevation on anterior leads of his ECG. Angiography demonstrated a mid-left anterior descending artery acute occlusion likely secondary to plaque rupture that was stented with a drug-eluting stent. Two hours after being admitted to CCU, he complained of nausea and vomited twice, which settled after being prescribed cyclizine by a passing medical senior house officer. Thirty minutes afterwards, you are asked to see the patient as the patient has become increasingly short of breath. He complains of no new chest pain, nausea, vomiting, sweating or palpitations. Interrogation of his cardiac telemetry reveals a new sinus tachycardia only without other arrhythmias. On examination, the patient is in respiratory distress, with bibasal inspiratory crackles and raised jugular venous pulse.

His observations are as follows: blood pressure is 186/80 mmHg, heart rate 120 beats per minute and regular, Sats 92% on 3 litres, respiratory rate 33/min.

A repeat 12-lead ECG reveals no new ST elevation changes or other ischaemic changes. A portable chest radiograph demonstrates bibasal alveolar oedema and bilateral small pleural effusions. What is the cause of the patient's deterioration?

- a) In-stent thrombosis
- b) Pulmonary embolus
- c) New independent cardiac ischaemic occlusion
- d) Cyclizine induced heart failure
- e) Cardiac tamponade secondary to cardiac instrumentation

Correct answer is d.

This patient has entered acute pulmonary oedema, associated with systemic

hypertension and tachycardia following administration of cyclizine in the context of an acute STEMI, typical of cyclizine-induced heart failure. Cyclizine is a H1 receptor antagonist and anticholinergic, resulting in systemic hypertension and inducing tachycardia. It can aggravate a fragile myocardium into acute failure and is hence not recommended by NICE in patients with ACS or severe heart failure¹. In this patient, the lack of chest pain or ST elevation on ECG makes acute in-stent thrombosis or a further ACS episode unlikely. The patient is hypertensive instead of hypotensive, with no mention of low voltage on ECG, making cardiac tamponade an unlikely cause.

1. NICE evidence 2013 'Can cyclizine be given to a patient who has an MI?'

Question #92

A 71-year-old gentleman is reviewed in hospital prior to discharge after having received treatment for an exacerbation of COPD. His systolic blood pressure has been recorded as measuring between 138mmHg to 156mmHg. He has a background of ischaemic heart disease, gout, gallstones and a fractured neck of femur which was repaired with a dynamic hip screw. He is found to have ACR of 31mg/mmol. He normally takes allopurinol, aspirin, tiotropium, Symbicort, salbutamol.

What is the most appropriate pharmacological management for his hypertension?

- a) ACE-inhibitor
- b) Calcium channel blocker
- c) Thiazide-like diuretic
- d) Beta-blocker
- e) Alpha-blocker

Correct answer is a.

The correct answer is ACE-inhibitor. This is an elderly man with hypertension and proteinuria. A calcium channel blocker would have been appropriate if he did not have proteinuria, whilst a thiazide-like diuretic and alpha-blocker feature at later stages in the NICE guidelines. Beta-blockers can provoke bronchospasms in COPD.

Question #93

A 44-year-old woman presents to the emergency department with dyspnoea. She has been feeling intermittently dizzy and short-of-breath for the past 2 weeks. On examination her pulse is 180/min, blood pressure 100/66 mmHg, oxygen saturations 98% on room air. Her chest is clear and she appears well perfused. An ECG is obtained:



What is the most appropriate treatment? ,

- a) Intravenous amiodarone
- b) Intravenous adenosine
- c) Intravenous magnesium
- d) Intravenous labetalol
- e) Unsynchronised DC shock

Correct answer is c.

IV magnesium sulfate is used to treat torsades de pointes

The ECG shows an irregular wide-complex tachycardia, a form of polymorphic ventricular tachycardia or more specifically torsades de pointes. This patient had an underlying long QT interval secondary to a combination of medications. The acute treatment for this is intravenous magnesium.

Precipitating medications should, of course, be reviewed and electrolyte

abnormalities corrected.

If the patient was in shock or periarrest then the ALS tachycardia should be followed, i.e. SYNCHRONISED DC shocks. There are, however, no 'adverse' signs in this patient.

Question #94

A 19-year-old female presents to the medical outpatient with a history of palpitations. She suffers from bouts of anxiety and dizziness associated with these palpitations and has had one episode of syncope. She has had bouts of atrial fibrillation in the past, although documentary evidence is not available.

On examination, her blood pressure is 125/85 mmHg and her pulse is 140 bpm.

Her ECG reveals a broad complex regular tachycardia with a short PR interval and a slurred upstroke of the QRS complex. There is additionally a tall R wave in V1.

Which of the following would be the most appropriate initial step in medical management?

- a) IV adenosine
- b) IV verapamil
- c) IV digoxin
- d) IV propranolol
- e) IV procainamide

Correct answer is e.

The description of the ECG given is that of WPW syndrome, a form of Atrio-Ventricular Reciprocating Tachycardia that results from conduction over an accessory pathway.

There are two types of WPW syndrome, Type A and Type B.

In case of type A (left atrioventricular connections), a positive R wave will be seen in V1 ('positive delta') on the precordial leads of the electrocardiogram,

In type B (right atrioventricular connections), a predominantly negative delta wave will be seen in lead V1 ('negative delta').

The treatment of choice in the long term management is radiofrequency catheter ablation of the accessory pathway.

Care must be taken in the use of AV nodal blocking agents, especially when the tachycardia is broad complex, since these may paradoxically increase the conduction over the accessory pathway and lead to a 1:1 atrioventricular conduction which may result in ventricular fibrillation.

A simple mnemonic to remember for drugs to avoid in WPW syndrome is ABCD (Adenosine, -Blockers, Calcium Channel Blockers, Digoxin).

Question #95

A 72-year-old man attends cardiology clinic for routine follow-up of ischaemic heart disease. The patient had suffered a non-ST elevation myocardial infarction the previous year treated with a drug eluting stent (see below angiogram report). The patient reported being generally well over previous months although did experience retro-sternal chest pain on exertion, typically on walking up hills or climbing stairs. The patient denied any episodes of pain at rest and did not feel his symptoms were worsening. The patients regular medications were aspirin 75 mg daily, clopidogrel 75 mg daily, atorvastatin 80 mg daily and ramipril 5 mg daily. As required medications were sildenafil and a nitrate spray (which he reported as giving effective relief from his chest pain episodes).

Clinical examination did not demonstrate any evidence of cardiac failure. Blood pressure in clinic was 125 / 75 mmHg.

Results of investigations conducted 1 year previously following presentation with NSTEMI are included below.

Angiography: 90 % stenosis to left anterior descending artery treated with drug-eluting stent; 60 % stenosis to mid-right coronary artery; minor disease to other

vessels.

Transthoracic echocardiogram: valvular function unremarkable; mild hypokinesis to anterior myocardium; overall ejection fraction approximately 40-45 %.

Electrocardiogram: sinus rhythm at 75 beats per minute; normal axis; normal QRS; no significant ST changes; inverted T waves in V3-V5.

What is most appropriate next line therapy for the patients chest pain?

- a) Amlodipine
- b) Bisoprolol
- c) Percutaneous coronary intervention
- d) Long-acting isosorbide mononitrate
- e) Nicorandil

Correct answer is b.

The patients reported symptoms are consistent with a diagnosis of stable angina secondary to known ischaemic heart disease. Medical treatment for symptomatic relief is the appropriate next line therapy. In this patient with a history of NSTEMI and controlled blood pressure a beta-blocker is preferred over a calcium-channel blocker such as amlodipine.

Both ISMN and nicorandil are second-line options for medical therapy after intolerance or failure of beta-blocker and calcium-channel blocker therapy. Such both medications induce systemic vasodilatation they would be contraindicated in this patient while he still was taking sildenafil.

Revascularisation techniques such as PCI have not been shown to reduce mortality or rate of MI in stable coronary artery disease. Therefore, medical therapy should be attempted before consideration of invasive treatment.

Al-Lamee R, Davies J, Malik I. What is the role of coronary angioplasty and stenting in stable angina? BMJ 2016;352:i205.

Question #96

A 67-year-old with a history of ischaemic heart disease (primary percutaneous intervention for a STEMI three years ago) is admitted with a pyrexia of unknown origin. On examination his pulse is 96/min, temperature 38.2°C and blood pressure 104/66 mmHg. A systolic murmur is noted but auscultation of the chest is unremarkable. His post-myocardial infarction echocardiogram three years ago showed no valvular disease. Chest x-ray is normal and urine dipstick shows blood ++. A petechial rash is noted on his hands and legs. A presumptive diagnosis of infective endocarditis is made and empirical treatment with IV amoxicillin and gentamicin given. Two days later blood cultures show a coagulase-negative staphylococcus. What is the most appropriate action with respect to antibiotic therapy?

- a) Switch to flucloxacillin
- b) Switch to flucloxacillin + vancomycin + rifampicin
- c) Make no changes to treatment
- d) Switch to flucloxacillin + vancomycin
- e) Switch to flucloxacillin + rifampicin

Correct answer is a.

The BNF now recommend flucloxacillin monotherapy for native-valve endocarditis caused by staphylococci.

Question #97

A 64-year-old caucasian male presented to heart failure clinic with shortness of breath on exertion. He has a history of heart failure, initially diagnosed three years previously.

His current medications included ramipril, aspirin, bisoprolol, simvastatin and spironolactone. He is concerned that despite all of these medications his breathlessness is worsening. In addition to this he is now using four pillows to sleep at night.

On examination he is comfortable at rest with fine crackles at both lung bases and

pitting oedema reaching up to both knees. His observations reveal a respiratory rate of 16 breaths per minute, oxygen saturation 96% in room air, blood pressure 110/85 mmHg, heart rate 70 beats per minute, temperature 37.2°C.

His ECG revealed a normal sinus rhythm with narrow QRS complexes. A recent echocardiogram completed two weeks previously demonstrated an ejection fraction of 30%. His current medications were reviewed and he was found to be on the maximum dose for each of these.

His baseline U&E results are as follows:

Na ⁺	136 mmol/L	(135 - 145)
K ⁺	4.5 mmol/L	(3.5 - 5.0)
Bicarbonat e	26 mmol/L	(22 - 29)
Urea	6.7 mmol/L	(2.0 - 7.0)
Creatinine	110 µmol/L	(55 - 120)

What is the optimum treatment option for this patient?

- a) Cardiac Resynchronisation Therapy
- b) Digoxin
- c) Hydralazine combined with a nitrate
- d) Ivabradine
- e) Sacubitril-Valsartan (after stopping ACE inhibitor)

Correct answer is d.

Sacubitril-valsartan is considered in heart failure patients with a LVEF < 35% who are still symptomatic on ACE-inhibitors & beta-blockers

This patient has heart failure, he is symptomatic despite maximum medical therapy with an ACE inhibitor, beta blocker and aldosterone antagonist. His heart rate is 70 bpm, therefore ivabradine cannot be used at this time. After a period of ACE inhibitor washout sacubitril-valsartan is the optimal therapeutic option.

Digoxin has been found to not have any improvement in long-term outcome and therefore is not the correct answer in this case.

Hydralazine with a nitrate is indicated in the afro-caribbean population; we are told this patient is caucasian, hence this answer is incorrect.

Ivabradine is contraindicated as the patients heart rate is 70 bpm, the guidelines state it should be > 75 bpm for ivabradine to be prescribed.

Question #98

A 54-year-old gentleman is investigated following complaints of frothy urine. Investigations show proteinuria and mild kidney injury, but normal full blood count, liver function tests, inflammatory markers and an ultrasound scan of the kidneys. During examination, he was found to have a systolic murmur on the left sternal edge. A transthoracic echocardiogram demonstrates a left ventricular ejection fraction of 55%, normal valves, and increased thickness of the left ventricle to 16mm. In addition, there is a 2mm pericardial effusion and ground-glass changes of the left ventricle. He has no chest pain or shortness of breath. What is the most likely explanation of these echocardiogram findings?

- a) Cor pulmonale
- b) Myocarditis
- c) Mitochondrial disease
- d) Type 2 diabetes mellitus
- e) Amyloidosis

The correct answer is amyloidosis. The symptoms and signs described indicate amyloid deposition in the kidneys and heart. The echocardiogram is particularly telling and the images can be described as a global speckled pattern. Cor pulmonale is unlikely without evidence of respiratory disease, myocarditis would not explain the proteinuria, whilst diabetes would not explain the echocardiogram changes. Mitochondrial disease is more likely with ventricular dilatation but would not show as a ground-glass pattern.

Question #99

A 75-year-old male presents with increasing chest pain on exertion. He was first diagnosed with stable angina 7 years ago. His past medical history includes hypercholesterolaemia and hypertension. His drugs history includes bisoprolol and nifedipine, which he has been taking for the past 3 years. A recent angiogram was arranged as an outpatient by his GP, demonstrating stenoses in his left circumflex artery, distal right coronary artery and mid-left anterior descending artery. What is the most appropriate long-term treatment of choice for his angina?

- a) Coronary artery bypass graft
- b) Percutaneous coronary intervention with 3 stents
- c) Addition of ivabradine
- d) Addition of nicorandil
- e) Addition of ranolazine

Correct answer is a.

NICE guidelines recommend the use of no more than 2 antianginals prior to consideration of reperfusion therapies. In the case of this gentleman, the consideration would be whether CABG or PCI would offer greater benefits: although the risks of recurrent MIs and stroke are similar for both PCI and CABG at one year, patients with complex anatomy, triple vessel disease or proximal left mainstem disease report better long-term survival and freedom from MI is greater with CABG. CABG is thus favoured to PCI in this patient according to American Heart Association and European Society of Cardiology guidelines.

Discuss (3)Improve

Question #100

A 40-year-old male who is otherwise fit and well presents to clinic with dyspnoea on exertion. This has progressed over the last year to the point where he can only walk 0.5 miles before stopping. He denies having a cough. He has no chest pain. He has smoked 15 cigarettes per day for 25 years. He reports that his father had a myocardial infarction (MI) aged 54 years, but otherwise reports no cardiac-related family history. His body mass index (BMI) is 24 kg/m^2 , heart rate 80/min, blood pressure 130/77mmHg, respiratory rate 18/min and he is saturating at 97% on air. Chest auscultation reveals occasional expiratory wheeze and he has no pedal oedema. Auscultation of the heart reveals a fixed split S2. His jugular venous pressure is not elevated.

Chest X-ray: dilated pulmonary vessels.

ECG: sinus rhythm, 78/min, right bundle branch block (RBBB)

What is the likely cause of his exertional dyspnoea?

- a) Atrial septal defect
- b) Ischaemic heart disease
- c) Bronchial malignancy
- d) Chronic obstructive pulmonary disease
- e) Chronic pulmonary embolism

Correcv answer is a.

The underlying cause here is likely to be an atrial septal defect. Presentation often occurs late in life with an incidental finding of RBBB. It may not be picked up in infancy as a murmur may not be audible. Presentation with symptoms occurs later in life as the right atrium becomes dilated and hence the cardiac efficiency decreases. A fixed-split S2 is a common finding in such patients.

Question #101

A 53-year-old man with a history of diabetes presents to the Emergency Department with crushing central chest pain, vomiting, and diaphoresis.

Observations :

- Pulse: 32 beats per minute.
- Blood pressure: 70/40 mmHg.
- Oxygen saturation: 88%

Examination reveals pale and cool skin. An ECG shows ST-elevation in leads II, III, and aVF, along with complete heart block, and his troponin test is positive.

Following initial management, including percutaneous coronary intervention (PCI) with stenting, he is admitted to the intensive care unit.

Three days post admission, he develops severe dyspnoea, cold extremities, and diaphoresis. Auscultation reveals a holosystolic murmur on the left sternal edge. His observations:

- Pulse: 123 per minute
- Blood pressure: 65/45 mmHg.
- Oxygen saturation: 80%

What is the most appropriate next step in management?

- a) CT coronary angiography
- b) Cardiac MRI
- c) Immediate surgical consultation
- d) Re-transfer to the catheter lab
- e) Transthoracic echocardiography

Correct answer is e.

Acute heart failure: echocardiography is indicated in new-onset heart failure, cardiogenic shock, suspected valvular or post-MI problems

Transthoracic echocardiography is the correct answer. In this scenario, the patient developed cardiogenic shock (hypotension, tachycardia and cool skin) with a holosystolic murmur following a complicated inferior wall STEMI (as denoted by complete heart block on ECG). The differential diagnoses include acute mitral regurgitation (MR) and acute ventricular septal rupture (VSR). A holosystolic murmur heard over the left sternal edge suggests an acute VSR. An urgent transthoracic echocardiogram is warranted to delineate the site and extent of the defect. This diagnostic step should be performed concurrently with medical therapy, preceding an immediate surgical consultation.

CT coronary angiography is not appropriate in this context. Although CT coronary angiography can be useful for patients with suspected stable angina, in this case, where acute VSR is likely following a well-established inferior wall STEMI, echocardiography will provide rapid and precise information about the lesion that will inform surgical management decisions.

Cardiac MRI would not be suitable in this setting. Although it can sometimes provide superior detail compared to echocardiography, its longer scan times, limited availability, and inadequacy for haemodynamically unstable patients render it a less favourable choice for imaging in this emergency.

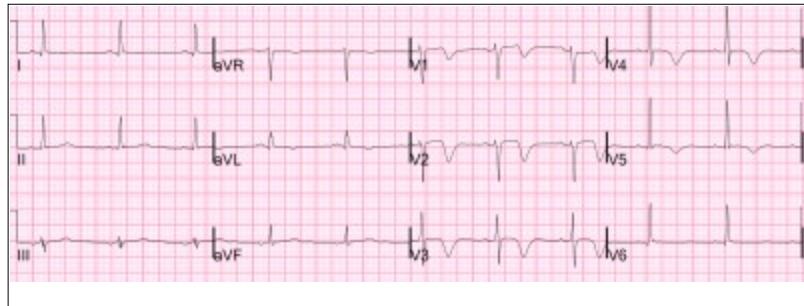
Immediate surgical consultation plays a pivotal role in managing both acute MR and VSR; however, it should be preceded by a detailed lesion assessment using echocardiography to inform subsequent surgical intervention.

Re-transfer to the catheter lab would not benefit this patient. Such a step may be considered if re-infarction is suspected. Given that the clinical picture points towards an acute VSR post-myocardial infarction, echocardiographic evaluation remains crucial for diagnosis, and coronary angiography will not diagnose such a complication.

Question #102

A 55-year-old man presents to the emergency department with a 2-hour history of central chest pain and dizziness. He has tried taking paracetamol and ibuprofen but has seen little improvement. His past medical history includes hypertension, for which he takes amlodipine 10mg once daily. He smokes 20 cigarettes/day and drinks 12 units of alcohol per week.

An ECG is taken (as shown below):



What is the most likely diagnosis?

- a) Brugada syndrome
- b) Hypertrophic obstructive cardiomyopathy
- c) Posterior myocardial infarction
- d) Pulmonary embolus with right heart strain
- e) Wellen's syndrome

This patient has **Wellen's syndrome**. Wellen's syndrome is the critical ischaemia of the left anterior descending artery. Patients typically have a history of chest pain and ECG findings include biphasic T waves in the anterior leads or deep symmetrical T wave inversion in leads I and aVL associated with 1mm ST elevation in the chest leads. These can be seen in this patient's ECG.

Brugada syndrome is incorrect. Brugada syndrome is an autosomal dominant inherited cardiovascular disease. It can be asymptomatic and lead to sudden

cardiac death. ECG changes consistent with Brugada syndrome include convex ST-segment elevation > 2mm in at least 1 of V1-V3 that is followed by T wave inversion. A partial right bundle branch block may also be seen. Sometimes, a patient may have an appearance of a normal electrocardiogram (ECG). However, following the administration of flecainide, these ST-segment changes may appear.

Hypertrophic obstructive cardiomyopathy (HOCM) is incorrect. HOCM is an autosomal dominant inherited condition resulting in left ventricular hypertrophy with decreased cardiac compliance and ultimately decreased cardiac output. Most cases can be asymptomatic. However, symptoms include exertional dyspnoea or chest pain, syncope and sudden cardiac death. Given the family history of sudden cardiac death, HOCM is a reasonable differential. However, ECG features of HOCM include features of left ventricular hypertrophy (eg increased R wave height), deep Q waves and non-specific ST segment changes that are not seen in this patient's ECG.

Posterior myocardial infarction is incorrect. A posterior myocardial infarction causes ECG findings such as a dominant R wave in V2, and horizontal ST depression and broad R waves in V1-V3. Although the patient has cardiac-sounding chest pain, their ECG findings do not match those that we see here, making this answer incorrect.

Pulmonary embolus with right heart strain is incorrect. Classic findings of a pulmonary embolus include sinus tachycardia and the less common S1Q3T3 finding. However, in more significant disease, patients may exhibit features of **right heart strain** secondary to pulmonary hypertension. Right heart strain appears as ischaemic changes (including deep T wave inversion) in the right-sided leads of the heart. The history here is not suggestive of a pulmonary emboli and does not explain the biphasic T waves seen in V1 and V2.

Question #103

A 83 year old lady presents to heart failure follow up clinic. She has a history of NSTEMI and gallstones. Her symptoms are reasonably poorly controlled. She is able to walk around her bungalow, but struggles to walk to her local shops 100m away due to breathlessness.

She had recently seen her GP who added spironolactone to her regular medications due to persistent hypokalaemia. Her potassium has since normalised.

Her latest echo reveals an ejection fraction of 25%. Her ECG is sinus rhythm Her last BNP was 1000 pg/ml.

She is currently taking senna, ramipril 10mg, aspirin 75m, frusemide 40mg bd, simvastatin 40mg, and spironolactone 50mg.

Her observations at clinic are:

- oxygen saturations: 94% on room air
- blood pressure: 126/66 mmHg
- heart rate: 84/min

Which additional medication would be beneficial?

- a) Ivabradine
- b) Bisoprolol
- c) Digoxin
- d) Diltiazem
- e) Atenolol

Correct answer is b.

This patient is already taking some prognostically beneficial medications; ACE inhibitor and aldosterone antagonist. The addition of a beta blocker would be a beneficial medication; both from a preventative of re-modelling and a reduction in heart rate.

Heart rate is a well established modifiable risk factor, which when appropriately controlled can improve morbidity and mortality in heart failure. This patient's heart rate is not well controlled.

Bisoprolol, carvedilol, nebivolol and metoprolol are the only evidence-based cardioselective beta blockers for heart failure patients.

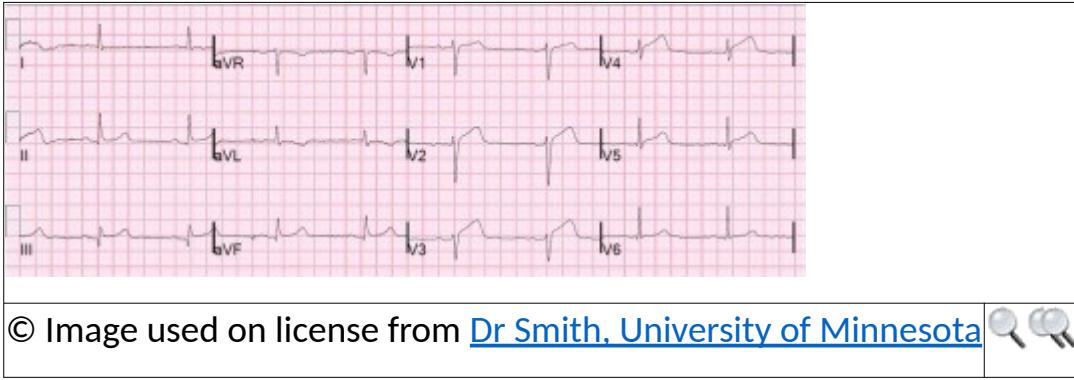
Ivabradine is an If (funny) channel blocker, which reduces heart rate by blocking the If current in the sinoatrial node. NICE guidelines advocate the use of ivabradine for heart failure in a select group of patients:

- Ejection fraction <35%
- Heart rate >75/min
- Sinus rhythm
- NYHA class 2-4
- Maximally titrated beta blocker therapy.

Since this patient is not on maximal beta blocker therapy, it would not be appropriate to commence ivabradine. Diltiazem may be used as an anti-anginal medication, but this patient does not have angina. Digoxin may be of benefit in heart rate, especially if the patient has atrial fibrillation, but should only be considered following failure of first and second line therapies.

Question #104

A 79-year-old man presents with severe central chest pain which started around 90 minutes ago. He is known to have ischaemic heart disease and had a coronary artery bypass graft (CABG) five years ago. On arrival in the Emergency Department he is clammy and vomiting. An ECG is taken:



What is the most accurate description of what is shown on this ECG?

(MI = myocardial infarction, STEMI = ST elevation myocardial infarction, NSTEMI = non-ST elevation myocardial infarction)

- a) Normal ECG
- b) Current inferior NSTEMI
- c) Current anterior NSTEMI
- d) Current inferior STEMI
- e) Current anterior STEMI

Correct answer is e.

Ischaemic changes in leads V1-V4 - left anterior descending

In the anterior leads (V2-4) ST elevation can clearly be seen indicating a STEMI. Angiogram revealed an acutely occluded saphenous vein graft to the left anterior descending which was opened with percutaneous coronary intervention.

In the inferior leads there is a pathological Q wave in lead III and non-diagnostic Q waves in leads II and aVF (see below for definitions). These changes suggest that this patient has previously had an inferior MI.

The older and simpler definition of a pathological Q wave:

- Q-wave of ≥ 0.04 s and an amplitude $\geq 25\%$ of the R-wave in that lead

The new (and much more complicated) Joint European Society of Cardiology/American College of Cardiology definition of a pathologic Q wave is:

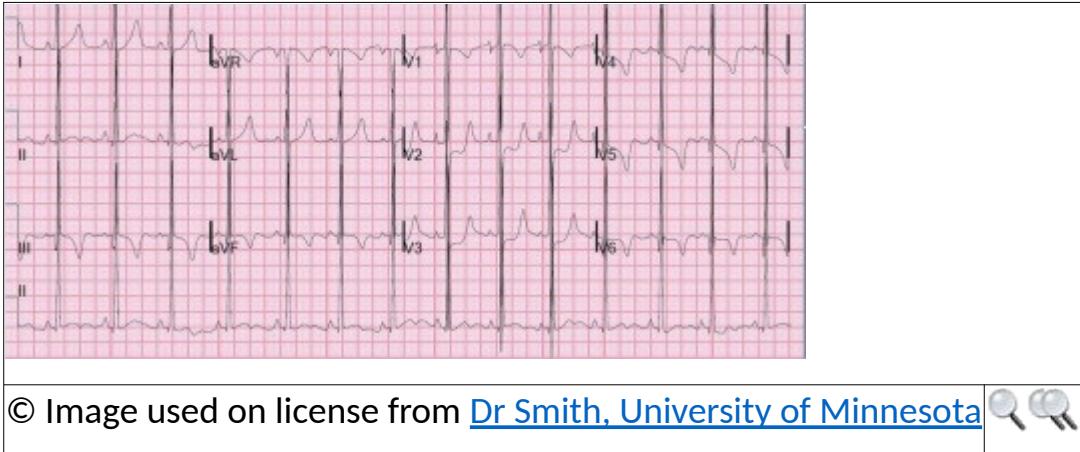
- any Q-wave in leads V2 - V3 ≥ 0.02 s or QS complex in leads V2 and V3
- Q-wave ≥ 0.03 s and > 0.1 mV deep or QS complex in leads I, II, aVL, aVF, or V4 - V6 in any two leads of a contiguous lead grouping (I, aVL,V6; V4 - V6; II, III, and aVF)
- R-wave ≥ 0.04 s in V1 - V2 and R/S ≥ 1 with a concordant positive T-wave in the absence of a conduction defect

Question #105

A 32-year-old female presents following a collapse whilst out jogging. The patient reports she experienced a subacute onset of chest discomfort that was quickly followed by symptoms of breathlessness and feeling faint. She thinks she 'blacked out' for a few seconds before recovering. Now she is feeling well with no symptoms.

On further questioning, she does not exercise regularly as she has experienced shortness of breath and chest discomfort in the past. She believes she has had a few episodes of feeling light-headed but has never fainted before. She is otherwise well with no recent illness or significant past medical history.

Observations are all within normal range and an ECG performed is shown below.



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What is the patient's most likely diagnosis?

- a) Brugada syndrome
- b) Dilated cardiomyopathy
- c) Hypertrophic obstructive cardiomyopathy
- d) Wellen's syndrome
- e) Arrhythmogenic right ventricular dysplasia

Correct answer is c.

This patient has presented with features in keeping with hypertrophic obstructive cardiomyopathy (HOCM). The condition is due to a genetic defect causing a disorder of the cardiac muscle and, although patients can be asymptomatic, it is the commonest cause of sudden cardiac death in the young. Patients who do present with symptoms typically experience exertional dyspnoea, angina and syncope episodes, commonly during or following exertion. ECG findings, as seen in this case, are of left ventricular hypertrophy, non-specific ST-segment and T-wave abnormalities, progressive T wave inversion and deep Q waves. The diagnosis can be confirmed via cardiac echo and management is dependent on the extent of obstruction and patient symptoms.

Brugada syndrome is another genetic cardiac disorder however unlike HOCM it results in electrical activity disorders and not structural issues. Features include syncope and sudden cardiac death however it is not always associated with exertion or exercise. There are several types of Brugada syndrome with variation seen on ECG however the only potentially diagnostic ECG abnormality is of coved

ST-segment elevation in 2 or more of V1-3, followed by a negative T wave. This is classical of Brugada type 1 syndrome and is commonly known as Brugada sign.

Dilated cardiomyopathy is a disease of the myocardial and is characterised by progressive ventricular dilation and dysfunction. Presentation is normal with worsening biventricular failure including peripheral and pulmonary oedema. Syncope episodes are rare and symptoms are not fluctuant, as seen in this patient. ECG abnormalities are those associated with atrial and ventricular hypertrophy with conduction delays (e.g. LBBB), left axis deviation and poor R wave progression.

Wellen's syndrome is the ECG pattern of biphasic or deeply inverted T waves in the chest leads V2 and 3. It is highly specific for critical stenosis of the left anterior descending artery and therefore is normally seen in patients presenting with ischaemic-like symptoms.

Arrhythmogenic right ventricular dysplasia (ARVD), is due to a genetic defect affecting the desmosomes of the myocardium. ARVD results in a non-ischemic cardiomyopathy mainly affecting the right ventricle leading to areas of hypokinesia and myocardium replacement with fibrofat. These changes can result in associated arrhythmias. Patients typically present with palpitations, syncope, and potentially sudden cardiac death however ECG findings are of T wave inversion in leads V1 to V3 and of right bundle branch block which are not present in this case.

Question #106

A 73-year-old man presents to the medical take with a 3-month history of thoracic back pain. Over the last week, he had been having episodes of sweats and shivers, particularly at night.

He was admitted and blood tests were taken.

Haemoglobin	87 g/L
-------------	--------

White cells	$11.6 \times 10^9/L$
Platelets	$214 \times 10^9/L$
MCV	70 fl
MCH	20 pg
Blood cultures	<i>Streptococcus gallolyticus</i>

MRI showed a discitis at thoracic disks 8/9.

ECHO: No vegetation seen

What is the next investigation for this patient?

- a) Colonoscopy
- b) CT chest/abdomen/pelvis
- c) Ultrasound abdomen/pelvi
- d) Repeat blood cultures
- e) PET scan

Correct answer is a.

Streptococcus bovis endocarditis is associated with colorectal cancer

Streptococcus gallolyticus is a subtype of *Streptococcus bovis*. *Streptococcus bovis* bacteraemia is associated with underlying colonic malignancies in approximately 10 to 25 percent of patients, therefore, all patients presenting with this should be investigated for rectal cancer. The gold standard investigation is a colonoscopy.

If a malignancy was found on colonoscopy then a CT chest/abdomen/pelvis would be warranted to look for metastases, but it would not be as sensitive as a

colonoscopy to look for colonic malignancies. An ultrasound of the abdomen would also be less sensitive to diagnose colonic malignancies than a colonoscopy.

A CT-PET scan may well show a malignancy in the colon, but the patient must be exposed to radioactive dye and radiation, therefore is not the first line investigation.

Repeat blood cultures would be useful when determining the length of treatment needed for this patient, however, they would not aid in the discovery of the underlying diagnosis.

Question #107

A 45-year-old woman presents to the emergency department after a road traffic accident where she sustains multiple injuries including an open fracture of her left tibia and fibula. The following day she has an open reduction and internal fixation of the left tibia and fibula and remains in hospital for physiotherapy. She is quite immobile during this period and then develops subsequent painful swelling and erythema of the left calf. Subsequent ultrasonography confirms a left-sided above knee deep vein thrombosis.

Before treatment starts, she develops sudden onset weakness in her right leg and right arm, dysarthric speech and a reduction in conscious level. Subsequent CT scanning confirms the presence of a left-sided infarct in the middle cerebral artery territory. Doppler investigation of the carotids shows a 20% stenosis on the left side and 10% on the right side. The 24-hour tape shows average heart rate 52bpm with 1.5s pauses maximum, sinus bradycardia

Which of the following features from further investigations would best explain this woman's presentation?

- a) Anti phospholipid antibody positive
- b) Protein C deficiency
- c) Patent foramen ovale (PFO)
- d) Hypercholesterolaemia

- e) Dilated cardiomyopathy on ECHO

Correct answer is c.

This unfortunate lady has developed a thrombus as a result of her recent operation and subsequent immobility. Part of the thrombus has embolised into her left middle cerebral artery.

One would normally expect the source of the embolus to be from the carotids or from the heart. However, the minimal carotid stenosis and lack of history of atrial fibrillation make this less likely. The finding of a PFO on an ECHO would explain how a thrombus from the leg would reach the brain rather than the lungs.

PFOs are extremely common and have been found in 25-30% of people at autopsy. PFOs have been linked to an increased risk of stroke. There is also some evidence that subclinical DVTs in the presence of a PFO may be one cause for cryptogenic stroke <http://content.onlinejacc.org/article.aspx?articleid=1120253>

The evidence is however limited that PFO closure will reduce the risk of stroke.

The other options except E are all risk factors for stroke but cannot explain the mechanism of embolism with near-normal carotids and sinus rhythm. E is not associated with an increased risk of embolic stroke.

Question #108

A 65-year-old gentleman is reviewed in cardiology clinic with known cardiomyopathy. He has started to develop symptoms of progressive exertional shortness of breath and is concerned about having this treated. He has not had any other symptoms and on examination, there is no peripheral oedema or chest signs. A recent echocardiogram demonstrates a provoked left ventricular outflow gradient of 64mmHg. What would be the most appropriate medical therapy?

- a) Phosphodiesterase type 5 inhibitor
- b) Beta-blocker
- c) Nitrate
- d) Digoxin

e) Ace-inhibitor

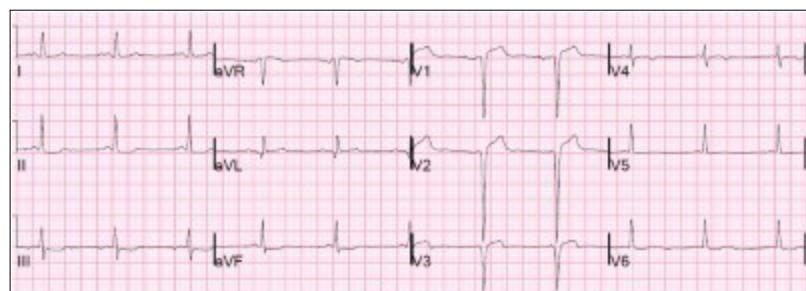
The correct answer is beta-blocker. Beta-blockers and verapamil can help with symptom management as they control the heart rate to the point where ventricular outflow obstruction is unlikely to occur. Nitrates and ace-inhibitors are contra-indicated as they lower blood pressure which can be very dangerous when combined with hypotension when the outflow is obstructed, whilst phosphodiesterase type 5 inhibitors and digoxin would offer no benefit. Phosphodiesterase type 5 inhibitors have a greater role in pulmonary hypertension whilst digoxin can be used to control the heart rate in AF when the patient is sedentary or there is coexisting heart failure.

Question #109

A 55-year-old man presents to the emergency department with a 1-day history of central chest pain. He also complains of progressive shortness of breath on exertion over the last 2 weeks. He was discharged from the cardiology ward 21 days ago after having a drug-eluting stent inserted following an anterolateral myocardial infarction. His past medical history includes diet-controlled type 2 diabetes mellitus and hypertension.

On examination, he is afebrile with a temperature of 36.4°C. His heart rate is 88 beats/min and regular with a blood pressure of 139/82mmHg. On auscultation, there are bibasal crepitations with saturations of 90% on air. There is pitting oedema to his mid-shins with soft and non-tender calves.

An ECG is taken, as shown below:



What is the most likely diagnosis?

- a) Anterior myocardial infarction
- b) Left ventricular aneurysm
- c) Papillary muscle rupture
- d) Pericarditis
- e) Pulmonary embolism

This patient has a diagnosis of a **left ventricular aneurysm**, a rare but serious complication of an anterior myocardial infarction. The complication tends to present more than 2 weeks after a myocardial infarction and symptoms may include chest pain or congestive heart failure (e.g. shortness of breath and fluid overload). This patient's ECG shows ST elevation in V1-V3 with QS waves and upright T waves. Persistent ST elevation after a myocardial infarction is suggestive of a left ventricular aneurysm. However, in a patient with a recent history of ischaemia, it is also important to exclude another ischaemic event. A left ventricular aneurysm is much more likely if there are absent reciprocal ST changes, dynamic ST changes and well-formed Q or QS waves.

Acute coronary syndrome is an important diagnosis to consider following a myocardial infarction. However, ECG findings that are more typical of acute coronary ischaemia include new ST changes with dynamic and reciprocal changes, which are not seen here. Furthermore, the more gradual onset of symptoms is more suggestive of an alternative diagnosis such as a left ventricular aneurysm.

Papillary muscle rupture is incorrect. An anterolateral myocardial infarction causes rupture of the anterolateral papillary muscle, producing symptoms of mitral regurgitation. Although symptoms of congestive cardiac failure are evident in such cases, the onset of symptoms is usually more acute compared to a left ventricular aneurysm. Furthermore, it does not explain this patient's ECG changes or normal heart sounds (where a pansystolic murmur of mitral regurgitation may

be seen).

Pericarditis is incorrect. Unlike Dressler's syndrome, post-MI pericarditis is an acute complication within the first 48 hours. The presentation is also similar to acute pericarditis with pleuritic chest pain worse on lying flat.

Pulmonary embolism is incorrect. ECG findings consistent with a pulmonary embolism include sinus tachycardia, RAD, RBBB or S1Q3T3. It does not explain this patient's ST elevation.

Question #110

A 22-year-old man is brought to the emergency department after being granted asylum in the UK. Immigration officials had been concerned about the patient's well-being immediately on his arrival in the country. The patient reported feeling progressively more unwell over the previous 3 months after he had fled from his village in Syria. The primary symptoms reported by the patient were fevers, severe night sweats and profound fatigue. In addition, the patient had found that his exercise tolerance has greatly reduced in the previous four weeks and he became markedly breathless on minimal exertion.

The patient explained that he had experienced a prolonged period of inadequate food and shelter due to the war in his home country and while travelling across Europe. The patient had previously lived in a small village in Syria and had worked as a sheep farmer. Prior to fleeing his home the patient had regularly consumed food products made with unpasteurised sheep's milk. The patient had previously been in good physical health and had suffered no significant medical problems during his life.

On general examination, the patient appeared unwell and diaphoretic. The patient's sweat was noted to have an unusually unpleasant odour. Examination of the cardiovascular system identified multiple splinter haemorrhages under the patient's fingernails and toenails. Jugular venous pressure was elevated by 3-4 cm and pitting oedema was present to the patient's knees. A harsh systolic murmur was heard over the aortic area on auscultation. Bilateral basal crackles were heard on auscultation of the patient's lungs. The patient's spleen was palpable on

examination of the abdominal system. No palpable lymphadenopathy was identified.

Please see the below table for results of initial investigations.

Haemoglobin	123 g / dL
Mean cell volume	80.3 fl
Lymphocytes	4.9 x 10 ³ / microlitre (reference 1.5-4.0)
Platelets	109 x 10 ³ / microlitre
Urea	8.9 mmol / L
Creatinine	126 micromols / L
Sodium	137 mmol / L
Potassium	4.1 mmol / L
Erythrocyte sedimentation rate	102 mm / h (reference 0-35)
C-reactive protein	99 mg / L (reference < 7)
Urinalysis	Positive for blood +++
Chest x-ray	Upper lobe blood diversion; generalised patchy airspace shadowing; bilateral shallow pleural effusions
ECG	Sinus rhythm; borderline right bundle branch

	block
Focused bedside transthoracic echocardiogram	0.6 cm vegetation on aortic valve; evidence of moderate-severe aortic stenosis; ejection fraction 30 %

Which of the following organisms is the most likely cause of the patient's presentation?

- a) *Clostridium burnetti*
- b) *Bartonella quintana*
- c) *Haemophilus parainfluenzae*
- d) *Kingella kingae*
- e) *Brucella melitensis*

Correct answer is e.

The patient presents with clear symptoms, signs and investigation results consistent with aortic valve endocarditis and secondary congestive cardiac failure. It is likely that the patient's prolonged exposure to inadequate living conditions has weakened his immune system allowing the development of an opportunistic infection.

Of the available options, *Brucella melitensis* is the most likely to be the causative microorganism. Endocarditis - most commonly of the aortic valve - is a rare complication of Brucella; however, it is responsible for the majority of mortality from this disease. Of the different *Brucella* species known to cause endocarditis *Brucella melitensis* is known to be associated with the most severe complications.

Brucellosis is the most likely diagnosis in this patient due to his history of consumption of unpasteurised milk products and close contact with livestock from an endemic region. The clinical sign of malodorous sweat is considered to be nearly pathognomonic for Brucellosis. In addition, the patient's blood results are typical for the condition featuring lymphocytosis and thrombocytopenia.

The patient requires blood cultures and serological testing to confirm the diagnosis. Given the severity of the patient's condition, he would require treatment with empirical antibiotics with consideration of aortic valve replacement at an appropriate point in the future.

The other available answers are all organisms known to be rare causes of endocarditis that usually will not be identified without specialist microbiological investigations. There is no information in the case presentation that makes any of these microorganisms a more likely causative agent of endocarditis than *Brucella melitensis*.

Ramin B, MacPherson P. Human brucellosis. *BMJ* 2010;341:c4545.

Question #111

A 50-year-old woman with a past medical history of type 1 diabetes, angina, and hypertension presents with vomiting and epigastric discomfort that came on suddenly earlier this morning.

Her blood glucose level is 10.4 mmol/L and her urine dip is '+' for ketones.

Observations are as follows:

- Respiratory rate: 19/min
- Oxygen saturations: 96% on room air
- Heart rate 96bpm
- Blood pressure 105/67 mmHg

Her blood results are awaited, and her ECG is shown below.



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What diagnostic option is most likely to confirm the diagnosis?

- a) Abdominal x-ray
- b) CT scan of the abdomen
- c) Repeat ECG with posterior chest leads
- d) Serum cortisol
- e) Venous blood gas

Correct answer is c

The ECG here shows sinus rhythm with a rate of roughly 85bpm. There is ST elevation in leads I, aVL, and v6. There is ST depression in lead v3 and v4.

Conduction appears to be normal. There are two premature ventricular complexes (PVCs).

The above findings can be explained by an acute coronary syndrome involving the posterior and lateral coronary territories. Posterior chest leads would help to confirm a posterior myocardial infarction, so **repeat ECG with posterior chest leads** is the correct answer.

A **venous blood gas** would be the option of choice if aiming to confirm diabetic ketoacidosis, but given the blood glucose is <11.0 mmol/L and ketonuria is less than 2+, this diagnosis is less likely.

CT scan of the abdomen would be correct if her symptoms were thought to be coming from an intraabdominal cause, but given the ECG evidence of an acute

coronary syndrome which would explain her epigastric discomfort and vomiting, this seems less likely.

Serum cortisol would be useful in confirming a diagnosis of adrenal crisis, which could present as epigastric discomfort, hypotension and vomiting. The ECG findings make the diagnosis of acute coronary syndrome more likely.

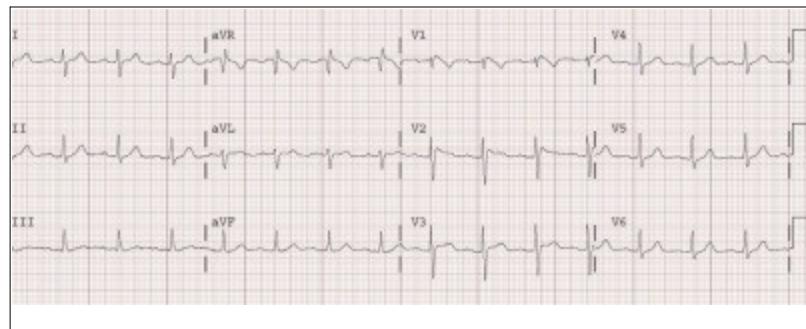
Abdominal x-ray would be useful in looking for evidence to suggest bowel obstruction or perforation. Obstruction would not explain the territorial ST-segment changes and so is not correct in this instance.

Question #112

A 30-year-old man is admitted to the Emergency Department after suffering a 'blackout' whilst at work. His colleagues report him collapsing without warning whilst waiting at the water machine. There has never happened before and he is normally fit and well.

On examination blood pressure is 102/68 mmHg, pulse 88/min, oxygen saturations 99% on room air and respiratory rate 16/min.

An ECG is taken:



What is the most likely diagnosis?

- a) Hypertrophic obstructive cardiomyopathy

- b) Anterior myocardial infarction
- c) Long QT syndrome type
- d) Arrhythmogenic right ventricular dysplasia
- e) Brugada syndrome

Correct answer is e.

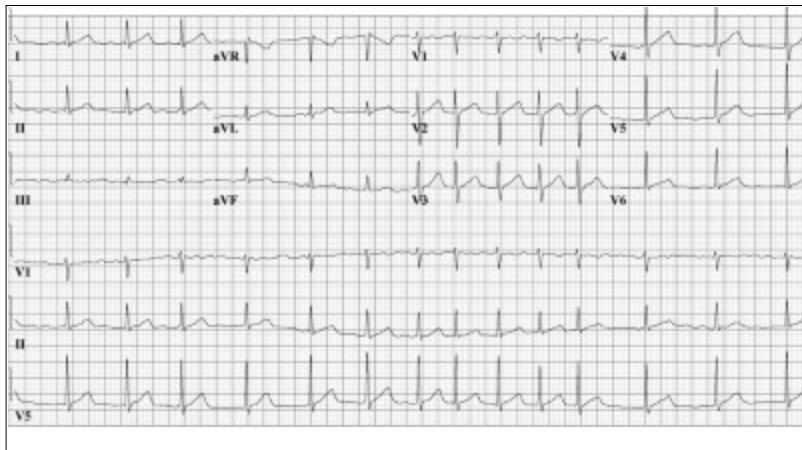
The ECG shows a Brugada pattern, most marked in V1, which has an incomplete RBBB, a downsloping ST segment and an inverted T wave.

Question #113

A 68-year-old man presents to hospital with left-sided upper and lower limb weakness that was present on waking that morning. The symptoms continue to persist in hospital. On examination, he has homonymous hemianopia. He has no significant medical history and is not on any regular medications.

A CT head is performed which is unremarkable.

An ECG is performed, which is shown below.



How would you manage this patient's anti-thrombotic medication?

- a) Apixaban for life
- b) Aspirin 300mg once daily for 14 days, then apixaban for life

- c) Aspirin 300mg once daily for 14 days, then clopidogrel 75mg once daily for life
- d) Clopidogrel 75mg once daily for life
- e) Thrombolysis with alteplase, then apixaban for life

Correct answer is b.

The ECG shows atrial fibrillation, as evidenced by the irregularly irregular rhythm and the lack of p-waves.

Aspirin 300mg once daily for 14 days, then apixaban for life is the correct answer. This patient has symptoms in keeping with a partial anterior circulation stroke. The CT head excludes an intracranial haemorrhage. Note, a normal scan is not sufficient to exclude a stroke, and stroke diagnosis should be based on clinical signs. The ECG shows atrial fibrillation which is the likely cause of the stroke. His CHA₂DS₂-VASc score is 3 due to age (+1) and stroke (+2). As such, he should receive two weeks of single anti-platelet treatment (aspirin 300mg once daily) as with all ischaemic strokes, followed by lifelong anticoagulation with a direct-acting oral anticoagulant (DOAC) such as apixaban, to prevent further strokes secondary to atrial fibrillation. Initiation of apixaban is delayed by two weeks post-stroke to prevent haemorrhagic transformation.

Apixaban for life is the incorrect answer. Therapeutic anticoagulation with a DOAC should not be initiated until after two weeks to avoid hemorrhagic transformation of an ischaemic stroke.

Aspirin 300mg once daily for 14 days, then clopidogrel 75mg once daily for life is the incorrect answer. This would be the correct answer if this patient did not have atrial fibrillation. Clopidogrel is insufficiently anti-thrombotic to prevent strokes secondary to atrial fibrillation and therefore a lifelong DOAC is required.

Clopidogrel 75mg once daily for life is the incorrect answer. A higher dose of antiplatelet is required to initially treat an ischaemic stroke (aspirin 300mg) and a DOAC is required long-term to reduce the atrial fibrillation-associated stroke risk.

Thrombolysis with alteplase, then apixaban for life is the incorrect answer. This

patient does not meet the criteria for thrombolysis as he awoke with the symptoms - thrombolysis requires symptom onset within the past four hours.

Question #114

A 59 year-old man with history of ischaemic heart disease and type 2 diabetes mellitus is eight hours post right curative hemicolectomy for bowel malignancy. In the surgical high-dependency unit he is noted to be tachycardic on the monitor

On examination the patient appears comfortable. The pulse rate is 200bpm and the blood pressure is 148/79mmHg. Oxygen saturations are 98% on 2L/min nasal oxygen. Capillary refill is 2 seconds.

The chest is clear to auscultation.

A 12-lead ECG reveals a regular broad complex tachycardia with a monomorphic waveform at a rate of 200bpm.

Post-operative blood tests reveal:

Hb	131 g/l
Platelets	563 * 10 ⁹ /l
WBC	13.4 * 10 ⁹ /l
Na ⁺	141 mmol/l
K ⁺	4.1 mmol/l
Mg ⁺⁺	0.87 mmol/l
Urea	4.2 mmol/l
Creatinin	121 µmol/l

e	
Bilirubin	23 µmol/l
ALP	109 u/l
ALT	34 u/l
Albumin	33 g/l

What is the most appropriate initial management?

- a) Magnesium sulphate 2g IV
- b) Amiodarone 300mg IV6
- c) Synchronised DC shock
- d) Metoprolol 5mg IV
- e) Adenosine 6mg IV5

Correct answer is b.

This case outlines a description of ventricular tachycardia.

Following the resuscitation council guidelines, in the absence of shock, syncope, myocardial ischaemia or heart failure (which would necessitate DC cardioversion), the most appropriate initial management is 300mg amiodarone intravenously.

Question #115

A 62-year-old woman is reviewed prior to discharge by the medical team. She was admitted with a lower respiratory tract infection. She now feels well and is ready for discharge. She has a background of type two diabetes mellitus, hypertension, hypothyroidism, osteoarthritis and dementia. Her medication history includes metformin 500mg BD, ramipril 2.5mg OD, levothyroxine 75 mcg OD and paracetamol 500mg QDS PRN. On the first two days in hospital, her metformin was stopped, but her other medications continued as in the community during her admission.

It is noted that she has persistently high blood pressure despite being confirmed as taking ramipril. What is the most appropriate plan to control her hypertension?

- a) Increase the dose of ramipril and check U&Es within 1-2 weeks
- b) Increase the dose of ramipril and check U&Es within 3-4 weeks
- c) Add amlodipine, increase the dose of ramipril and check U&Es within 3-4 weeks
- d) Add on amlodipine at a low dose
- e) Stop ramipril and start amlodipine instead

The correct answer is to increase the dose of ramipril and to check U&Es within 1-2 weeks. The patient is on a low dose of ramipril and there is room to increase it, and this should be done prior to adding on a second anti-hypertensive. It is important to test renal function within two weeks of either starting or increasing the dose of an ACE inhibitor to exclude deteriorating renal function, which would need to be investigated promptly. Amlodipine would have been a more appropriate anti-hypertensive choice if she did not have diabetes.

Question #116

A 58-year-old female reports having symptomatic episodes of palpitations for the last six months. She is normally fit and drinks the occasional small glass of red wine when she goes out for a meal. The last episode was four days ago when she was cooking in her kitchen. She describes that she felt light headed with central chest discomfort that eased after twenty minutes. In clinic, her observations are all stable and an initial twelve lead electrocardiogram shows sinus rhythm.

What would be the investigation of choice?

- a) 24 hour ambulatory electrocardiogram
- b) Electrocardiogram treadmill test
- c) 24 hour blood pressure
- d) 72 hour ambulatory electrocardiogram

e) Event recorder electrocardiogram

Correct answer is e.

Atrial fibrillation is a common arrhythmia that may be persistent or paroxysmal. It is more common in women compared to men.

All patients with suspected atrial fibrillation should have a manual pulse check followed by a twelve-lead electrocardiogram. Those who have episodes less than 24 hours apart should have a 24-hour ambulatory electrocardiogram. In patients who experience episodes more than 24 hours apart, an event recorder electrocardiogram would be the most suitable investigation of choice.

24-hour blood pressure monitoring would be considered in a patient with suspected hypertension.

Electrocardiogram treadmill test is useful for revealing abnormal heart rhythms induced by exercise and ischaemic changes suggestive of coronary heart disease.

A 72-hour ambulatory electrocardiogram is used in much the same way as a 24 and 48-hour test. However, current NICE guidelines suggest for atrial fibrillation of episodes more than 24 hours apart an event recorder device is most useful.

Question #117

A 72-year-old gentleman is admitted with syncope and palpitations. He is known to have atrial fibrillation and is on flecainide and warfarin. His past medical history also includes benign prostatic hypertrophy and essential thrombocytosis.

His other medications include tiotropium 18 micrograms OD, tamsulosin 400 micrograms OD, ramipril 2.5mg and simvastatin 40mg. He has recently stopped a tablet due to shortness of breath but cannot remember what it was.

On arrival, he is tachycardic with a heart rate of 215 /min. He is alert. He denies chest pain but is aware of some shortness of breath. He is attached to a 3-lead cardiac monitor which shows a narrow complex tachycardia of 210-220 beats per minute. It is difficult to tell on the monitor however it appears regular. A 12-lead

ECG confirms a regular narrow complex tachycardia with a ventricular rate of 215/min.

What is the most likely diagnosis?

- a) Atrial fibrillation
- b) Atrial flutter with 1:1 conduction
- c) Atrial flutter with variable conduction
- d) Atrio-ventricular re-entry tachycardia (AVRT)
- e) Atrio-ventricular nodal re-entry tachycardia (AVNRT)

Correct answer is b.

Flecainide is a pure sodium channel blocker which prolongs the cardiac action potential to slow conduction. It is recognised to increase the risk of AF transforming into flutter. Flutter with 1:1 conduction would result in a ventricular rate of 300 /min which is impossible to conduct via the AV node. However, because flecainide slows the rate of cardiac conduction it means that if a patient develops atrial flutter the atrial rate will not be 300 /min, but more likely around 200 /min which then could conduct via the AV node at a 1:1 ratio. For this reason, patients should always be co-prescribed a beta blocker to reduce risk if this happens, and you wonder if this was the tablet that had recently been stopped.

The key to this question is recognising the increased risk of atrial fibrillation transforming into atrial flutter in patients on flecainide. The regular rhythm on ECG makes atrial fibrillation and atrial flutter with variable conduction less likely. He has no specific risk factors for AVRT or AVNRT, however, his flecainide use increases the chance of atrial flutter and therefore this is the correct answer.

Question #118

A 28-year-old woman attended the emergency department and was subsequently referred to the cardiology team due to concerns about the risk of potential cardiac arrhythmia. The patient presented to the emergency department due to ongoing nausea and vomiting secondary to hyperemesis gravidarum. The patient stated she was 10 weeks pregnant with her first child and had been suffering from these symptoms for the past 6 weeks. Clinical records indicated the patient had also

attended the emergency department 3 weeks previously with the same presenting complaint.

On the initial occasion the patient had presented to the emergency department with hyperemesis gravidarum, she had been rehydrated with intravenous fluids and prescribed vitamin B6 and ginger capsules four times daily on the advice of the obstetric team. The patient reported that this treatment had given her only a modest improvement in her symptoms and she had stopped taking these medications after a few days. Subsequently, she had attended an out-of-hours medical centre and been prescribed ondansetron (4 mg three times daily). The patient had found a more significant benefit from ondansetron and had been taking it regularly over the past 2 weeks. In addition to her symptoms of nausea and vomiting, the patient reported feeling tired, lethargic and dehydrated. However, she denied experiencing any other symptoms, including palpitations or pre-syncopal episodes.

The patient's past medical history included a period of intravenous drug use in her early twenties. Three years previously the patient had successfully managed to stop her use of illicit drugs after being enrolled in a methadone treatment program. The patient was now on a consistent methadone dose of 80 mg once daily. There was no other significant past medical history and the patient took no other regular medications.

During the patient's assessment after attending the emergency department, concern was raised over a prolonged corrected QT interval on an ECG performed due to borderline hypokalaemia. The patient's ECG was otherwise unremarkable and was compared to previous ECGs the patient had undergone on her previous visits to the emergency department. Please see below for blood results and data from the patient's current and previous ECG.

Haemoglobin	123 g / dL
Mean cell volume	84.9 fl

White cell count	$11.7 \times 10^3 / \text{microlitre}$
Platelets	$187 \times 10^3 / \text{microlitre}$
Urea	$9.0 \text{ mmol} / \text{L}$
Creatinine	$82 \text{ micromol} / \text{L}$
Sodium	$139 \text{ mmol} / \text{L}$
Potassium	$3.6 \text{ mmol} / \text{L}$

Presentation	Relative date of ECG	Corrected QT interval / ms
2nd ED attendance for hyperemesis	Today	490
1st ED attendance for hyperemesis	3 weeks previously	430
Unrelated ED attendance	4 years previously	415

With regard to the patient's prolonged corrected QT interval, what is the most appropriate strategy for managing her medications?

- a) Continue both methadone and ondansetron and admit for cardiac monitoring
- b) Stop both methadone and ondansetron
- c) Continue both methadone and ondansetron and arrange outpatient ambulatory ECG
- d) Continue methadone, stop ondansetron

e) Continue ondansetron, stop methadone

Correct answer is d.

The upper limit of normal for corrected QT interval is considered to be 450 ms in men and 460 ms in women. QT prolongation is associated with torsades de pointes (TdP) and sudden cardiac death. However, the presence of a prolonged QT interval does not inevitably lead to TdP and TdP can occur in the presence of a normal QT interval. Each 10 ms increase in QT interval increases the risk of TdP by around 5-7 % and once corrected QT interval exceeds 500 ms, the risk of TdP is markedly increased and intervention is necessary.

The patient is taking 2 medications that are known to prolong the QT interval and these can be seen to have had an additive effect in prolonging the patient's QT interval. Specifically, the corrected QT interval increased from 430 ms when the patient was taking methadone to 490 ms when the patient was taking methadone and ondansetron. The normal baseline ECG suggests that the patient does not have a congenital condition causing QT prolongation. At the time of presentation, the patient's borderline hypokalaemia is a factor increasing the risk of TdP in the setting of her drug-induced prolonged QT interval.

Few recommendations exist for managing the risk of drug-induced prolonged QT interval. Expert advice suggests that when a QT prolonging drug is associated with a corrected QT interval of 480-500 ms in a woman (470-500 ms in a man), or when an increase of 60 ms or greater occurs, dose reduction or discontinuation is advised. If the corrected QT interval exceeds 500 ms then the drug should be discontinued and expert advice sought.

The use of methadone alone does not appear to cause a prolonged QT interval in this patient, whereas her QT interval increased by 60 ms following the addition of ondansetron. Given the importance to the patient's long-term health of adequate opiate replacement therapy, the risks associated with opiate withdrawal to a foetus in early pregnancy and the availability of other anti-emetic medications the most sensible course of action out of the available options is to discontinue ondansetron and continue methadone treatment.

Advice from the cardiology team could be sought to determine if the patient should be admitted for cardiac monitoring. The only available answer to include admission as an option also requires the patient to continue with both medications that prolong the QT interval and so is incorrect. Continuing both medications and discharging the patient for an outpatient ambulatory ECG (presumably to assess for TdP) is not an advisable management plan.

QT interval and drug therapy. BMJ 2016;353:i2732.

Question #119

A 65-year-old man is admitted with central chest pain that has been ongoing for two hours. It radiates to the left arm and is associated with nausea and vomiting. He has a past medical history of hypertension and diabetes which is diet controlled. He takes amlodipine and atorvastatin.

On examination his blood pressure is 147/89 mmHg and his heart rate is 110 beats per minute. His saturations are 96% on room air and he is afebrile. His cardiovascular and respiratory examinations are unremarkable.

An ECG shows T wave inversion in leads I, V4, V5 and V6.

Blood tests are as follows:

Hb	131 g/l	Na ⁺	136 mmol/l
Platelets	430 * 10 ⁹ /l	K ⁺	4 mmol/l
WBC	8 * 10 ⁹ /l	Urea	4 mmol/l
Neuts	6 * 10 ⁹ /l	Creatinine	84 µmol/l

Lymphs	$1 * 10^9/l$	CRP	41 mg/l
Eosin	$0.3 * 10^9/l$	Trop	130 ng/l

He is diagnosed with non-ST elevation myocardial infarction and commenced on aspirin, clopidogrel and low molecular weight heparin. His pain settles with morphine and glyceryl trinitrate spray within an hour. The following day he is commenced on ramipril and bisoprolol. 3 days later he undergoes an uncomplicated angiogram with stenting of the left circumflex artery.

On systems reviews prior to discharge he comments that he has noticed a rash on his legs and has not been passing much urine despite drinking plenty of water. On examination he has a bluish lacey discolouration over his legs. He has no palpable bladder.

Repeat bloods are as follows:

Hb	12.1 g/l	Na ⁺	137 mmol/l
Platelets	$560 * 10^9/l$	K ⁺	5 mmol/l
WBC	$10 * 10^9/l$	Urea	12 mmol/l
Neuts	$7 * 10^9/l$	Creatinine	184 µmol/l
Lymphs	$1.2 * 10^9/l$	CRP	60 mg/l
Eosin	$1.5 * 10^9/l$		

What is the most likely cause of his symptoms and deranged blood tests?

- a) Cholesterol embolism

- b) Contrast nephropathy
- c) Endocarditis
- d) Hypovolaemia during NSTEMI
- e) Newly started ACE inhibitor

Correct answer is a.

This gentleman has developed an acute kidney injury following angiography. The combination of a lacy rash (livedo reticularis) and raised eosinophils points to a likely diagnosis of cholesterol embolism. Contrast induced nephropathy is also possible following angiography but is not associated with rash or eosinophilia. Although renal function may deteriorate after starting an ACE inhibitor, it is unlikely to cause this degree of derangement so quickly. As the angiogram was uncomplicated, hypovolaemia is unlikely. Endocarditis would be unusual in the absence of fevers and other systemic signs.

Question #120

A 74-year-old man presents to the emergency department with a collapse. He has a past medical history of hypertension and atrial fibrillation. His medications include amlodipine and apixaban. A recent 24-hour tape arranged because of episodes of pre-syncope demonstrates sustained atrial fibrillation with episodes of bradycardia (minimum heart rate 20 beats per minute) associated with symptoms of pre-syncope.

The admission ECG demonstrates slow atrial fibrillation with a heart rate of 24 beats per minute.

The patient has recovered by the time he has been assessed by the medical team and his Glasgow coma scale is 15/15 and his heart rate is 82 beats per minute.

A decision is made to insert a permanent pacemaker.

What is the appropriate mode to programme the pacing system?

- a) AAI
- b) AOO

- c) OOO
- d) VAT
- e) VVI

VVI or VVR pacemakers are useful for pure sustained AF. Since the atria are not functioning they do not require sensing or pacing

VVI is the correct pacing mode. In this mode, the ventricle is sensed and the ventricle is paced. If a QRS complex is detected, it doesn't fire. If it is not detected i.e. there has been no ventricular electrical activity within the interval specified by its set rate, then it will fire. This is suitable for atrial fibrillation as the atria are not working correctly and there will be no detectable P wave and therefore the atria do not need to be sensed or paced.

AAI is not the correct pacing mode to choose. AAI pacing mode means atrial paced, atrial sensed, and inhibition of pacing output in response to an atrial sensed event (P wave). Since the atria are not functioning in AF, they do not require sensing or pacing. AAI pacing has better hemodynamic features than dual-chamber pacing and is the best mode for patients with sick sinus syndrome without a significant AV conduction disorder. This mode essentially relies on the AV node to fire and guarantees a certain atrial rate.

AOO is incorrect. This is asynchronous pacing. This refers to the continuous pacing of the atria irrespective of the presence or absence of an intrinsic rhythm. This pacing mode is limited to rare cases, such as when pacemaker-dependent patients (who do not have a ventricular paced setting) are subject to artefact (e.g., electrocautery during surgery), which could result in asystole due to oversensing and pacing inhibition if the pacemaker has been set to a non-asynchronous mode. It is never a permanent mode.

OOO is incorrect. This essentially means there is no sensing, no pacing and no response to sensing. It means the pacemaker is turned off.

VAT is incorrect. This mode means the atria are sensed and the ventricles are

paced. This is suitable for a patient with a dysfunctional AV node but working atria. For example, in someone with a complete heart block and a normal atrial rate, a VAT pacemaker will sense the atrium and pace the ventricle in response.

Question #121

A 60-year-old woman has a heart rate of 180bpm on the medical admission unit. An ECG performed demonstrates a regular narrow-complex tachycardia. The patient complains of palpitations but no chest pain and blood pressure remains 140/95. Initial vagal maneuvers are performed including carotid massage with no success. Intravenous adenosine 6mg is given followed by a further two doses of adenosine 12mg. Unfortunately, this failed to cardiovert or reveal an interpretable underlying rhythm.

What is the next management step?

- a) Further adenosine
- b) Synchronised DC cardioversion
- c) Verapamil
- d) Digoxin
- e) Amiodarone

The resuscitation council recommends:

Adenosine 6mg for supraventricular tachycardias followed by a further two doses of adenosine 12mg if no response. If adenosine is contra-indicated, or fails to terminate a regular narrow-complex tachycardia without demonstrating atrial flutter, consider giving a calcium-channel blocker, for example intravenous verapamil 2.5 - 5mg over 2 min.

The combination of vagal maneuvers and 6mg/12mg/12mg will terminate most regular narrow-complex tachycardias. Further adenosine is not thought to be of benefit.

Digoxin and amiodarone are used in the treatment of atrial fibrillation but not indicated in regular narrow-complex tachycardias. However, remember to consider alternative diagnoses such as atrial flutter/fibrillation, especially when

the above rhythm fails to terminate after adenosine.

The patient is stable; with no adverse features to suggest DC cardioversion is indicated

Question #122

A 24-year-old primigravida presents to the emergency department with a 72-hour history of nausea and vomiting with associated right upper quadrant pain. She has no past medical history of note and takes only pregnancy vitamins. During her 32-week midwife appointment 1 week ago, it was noted that she had gained weight and had borderline hypertension. The decision at that appointment was to monitor.

On examination, she is clinically dehydrated and appears unwell. Respiratory and cardiovascular examinations are unremarkable and abdominal examination demonstrates a gravid uterus and right upper quadrant tenderness. There is pitting oedema to the mid-shin. Observations are all within normal limits aside from a blood pressure of 145/90mmHg. She is alert and orientated, reports no headaches and has no rashes.

Blood tests are performed, results are as demonstrated:

Hb	100 g/L	Male: (135-180) Female: (115 - 160)
Platelets	$97 * 10^9/L$	(150 - 400)
WBC	$7.3 * 10^9/L$	(4.0 - 11.0)
PT	13.0 seconds	(9.5-13.5)
APTT	39.0 seconds	(30-40)

Na ⁺	132 mmol/L	(135 - 145)
K ⁺	3.4 mmol/L	(3.5 - 5.0)
Bicarbonate	22 mmol/L	(22 - 29)
Urea	7.5 mmol/L	(2.0 - 7.0)
Creatinine	100 µmol/L	(55 - 120)
Bilirubin	45 µmol/L	(3 - 17)
ALP	150 u/L	(30 - 100)
ALT	350 u/L	(3 - 40)
Albumin	34 g/L	(35 - 50)

An ultrasound abdomen shows patchy areas of hepatic enhanced echogenicity. Fetal monitoring is satisfactory, but the baby is in the breech position.

Given the likely diagnosis, what is the most appropriate management?

- a) Commence IV antibiotics
- b) Give steroids and organise an induction of labour
- c) Organise a plasma exchange with fresh frozen plasma
- d) Rehydrate with IV fluids and monitor
- e) Give steroids and organise a caesarean section within 48 hours

Delivery of the baby is the treatment of HELLP syndrome

This patient has HELLP syndrome as shown by her anaemia (secondary to haemolysis), thrombocytopenia and deranged LFTs. She is 33 weeks pregnant and

thus steroids would be recommended prior to delivery if possible to aid the maturation of her child's lungs. She does not have any evidence of a liver haematoma, disseminated intravascular coagulation or haemodynamic instability and thus she does not need an immediate delivery at this stage. Induction of labour would not be appropriate as her child is breech.

Differentials would include cholecystitis and thrombotic thrombocytopenic purpura (TTP).

IV antibiotics would be appropriate in the treatment of cholecystitis, but this patient has no features of infection, going against a diagnosis of cholecystitis.

Plasma exchange with fresh frozen plasma would be the recommended treatment for TTP, however, TTP would typically present with neurological symptoms such as confusion, headaches, and seizures in severe cases, which our patient does not have.

Question #123

A 24-year-old male presents to the emergency department with a collapse. The previous night he had been out drinking heavily with his friends in a nightclub. A friend attends with him and states that some of the lads had taken MCAT (Mephedrone) but he is unsure whether Michael also took MCAT. Michael felt well when he woke up but realised that he was late for his football match so rushed out of the house without any breakfast. He had just scored the first goal of the match when he collapsed. When he came around he complained of palpitations.

His past medical history includes asthma and hay fever for which he takes regular antihistamines. During the ambulance ride to hospital the paramedic performed some observations and an ECG. He was found to have a blood glucose of 3.6 and was given some GlucoGel and a biscuit. Other observations included a blood pressure of 90/60 mmHg, heart rate of 135/min which was irregular, irregular in character, respiratory rate of 16/min, afebrile.

The emergency department doctor reviewed the ECG and diagnosed atrial fibrillation with a fast ventricular response and prescribed digoxin in view of his

asthma history and blood pressure readings. The patient had a normal QTc. Five minutes later the cardiac monitor showed a broad complex tachycardia and Michael became unresponsive.

What was the cause of the patient's collapse?

- a) Hypertrophic cardiomyopathy (HCM)
- b) MCAT related arrhythmia
- c) Wolff-Parkinson-White syndrome
- d) Alcohol induced atrial fibrillation
- e) Torsades de pointes

Correct answer is c.

Wolff-Parkinson-White syndrome (WPW) is a disorder of the conduction system also known as a pre-excitation syndrome. The incidence of WPW is between 0.1% and 0.3% in the general population. Sudden cardiac death in people with WPW is rare (incidence of less than 0.6%).

In WPW there is an abnormal accessory pathway called the 'Bundle of Kent' found between the atria and ventricles. If this pathway is used signals may stimulate the ventricles to contract prematurely, resulting in atrioventricular re-entrant tachycardia.

Normally the AV node acts as a gatekeeper limiting the amount of electrical activity that can reach the ventricles. If a patient has an arrhythmia whereby the atria generate an excessively fast rhythm such as atrial flutter or fibrillation the AV node is able to reduce the amount of electrical activity that reaches the ventricles. However, the Bundle of Kent does not possess the ability to limit the electrical signals to the ventricles. If the person has an atrial arrhythmia the signals can pass down the bundle and cause the ventricles to contract at the same rate. In the extreme heart rates and when the accessory pathway is used the patient may develop dangerous cardiac arrhythmias which can trigger ventricular fibrillation and lead to death.

In WPW certain medications that can block the AV node or those that can

enhance conduction down the accessory pathway by increasing the refractory period in the AV node should be avoided. These include digoxin, adenosine, diltiazem, verapamil, other calcium channel blockers and beta blockers. In this case, the patient's WPW was worsened by administration of digoxin and her arrhythmia developed into ventricular fibrillation.

People with WPW are usually asymptomatic. Symptomatic patients may complain of shortness of breath, palpitations, dizziness and syncope. Diagnosis is usually made by ECG as patients may have the characteristic 'delta wave'. Patients may also be found to be in atrial fibrillation or have SVT. Treatment can include cardioversion in emergencies otherwise amiodarone. Definitive treatment would be ablation.

Question #124

A 65-year-old with exertional dyspnoea over the past 3 months is referred to the pulmonary hypertension team. An initial echocardiogram demonstrated 65% ejection fraction, preserved left ventricular function with a pulmonary arterial pressure of 72 mmHg. She undergoes a right and left heart catheter, revealing the following saturations:

Right atrium high	60%
Right atrium mid	89%
Right atrium low	70%
Right ventricle high	70%
Right ventricle mid	73%
Right ventricle low	72%

Pulmonary artery	71%
Capillary wedge	96%

What is the most likely diagnosis?

- a) Aortic stenosis
- b) Atrial septal primum defect
- c) Atrial septal secundum defect
- d) Pulmonary stenosis
- e) Mitral stenosis

The key to questions regarding saturations and cardiac catheters is to spot the 'step-up' in oxygen saturation. It is present between the high and mid right atrium, demonstrating the presence of a left-to-right shunt. Primum ASD defects typically occur lower in the septum than secundum defects, which typically can be found in the mid-atrial region.

Question #125

A 48-year-old man is brought in by ambulance to the Emergency Department after experiencing sudden onset crushing central chest pain radiating down his arm. His ECG showed an inferior STEMI and he undergoes successful PCI with 2 stents inserted into his right coronary artery. He is transferred to CCU and started on secondary prevention medication.

24 hours after his presentation he becomes bradycardic at 34bpm and his blood pressure falls to 80/47 mmHg. He denies chest pain but feels dizzy and light headed. His cardiac monitor shows 3rd-degree heart block. He is given atropine but his heart rate does not raise and his blood pressure remains low.

What is the most appropriate next step in his management?

- a) Isoprenaline infusion
- b) Temporary pacing wire
- c) Permanent pacemaker
- d) Repeat angiography
- e) Adrenaline

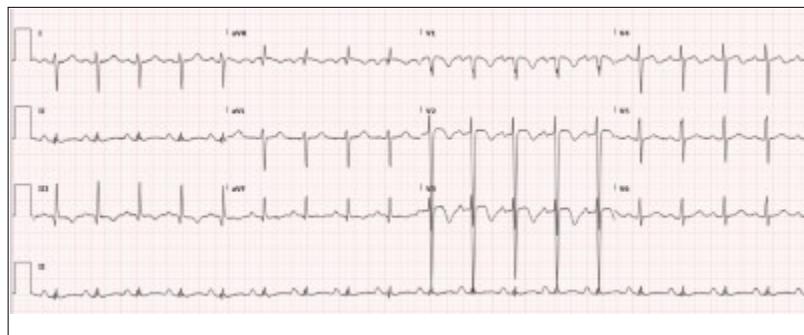
Correct answer is .b

This gentleman has suffered an inferior STEMI. Complete heart block is a recognised complication of both inferior and anterior STEMI. Inferior STEMI CHB is usually more transient and they only require treatment if they become symptomatic. Anterior STEMI CHB is a more serious complication and they are more likely to need a permanent pacing system. In this case, as he has not responded to atropine then isoprenaline infusion is unlikely to work. There is no suggestion of repeat infarction so angiography is not indicated. He will require a temporary pacing wire to be placed.

Question #126

A man in his 50's is brought to the emergency department by ambulance and was found collapsed in the street. His vital signs are as follows: Blood pressure 90/60 mmHg, pulse 120 bpm, respirations 25 bpm and temperature 37.9 °C.

The 12 lead ECG showed:



What is the most likely cause of the patient's presentation?

- a) Non-ST elevation myocardial infarction
- b) Pulmonary embolism

- c) ST elevation myocardial infarction
- d) Severe hypokalaemia
- e) Takotsubo cardiomyopathy

The ECG shows sinus tachycardia with right axis deviation, P pulmonale, Q waves in lead III and inverted T waves in the right precordial leads (V1-V3) and lead III.

Pulmonary embolism is the correct answer. Although the ECG is neither sensitive nor specific for pulmonary embolism, the classical findings of S1Q3T3 highly suggest right ventricular strain. Hence, the clinical picture is highly suspicious for pulmonary embolism, and the patient should promptly have an urgent CT pulmonary angiogram and thrombolysis if indicated. Other acute causes for right ventricular strain, like pneumothorax, upper airway obstruction and exacerbation of asthma/COPD, should also be considered in the differentials.

Non ST elevation myocardial infarction is incorrect. ECG changes like ST depression or T wave inversion in NSTEMI are often non-specific. If new, the T-wave inversions in this patient may suggest acute coronary syndrome, particularly right ventricular infarction, given the right ventricular strain pattern; however, NSTEMI is unlikely to cause collapse. The classical S1Q3T3 pattern in ECG, along with the clinical findings of tachycardia, tachypnoea and low-grade temperature, is more suggestive of pulmonary embolism in this patient.

ST elevation myocardial infarction is incorrect. Classically, STEMI is diagnosed if there is > 1-2 mm ST elevation in 2 contiguous leads or a new left bundle branch block (LBBB). Both of these are not found in this patient's ECG.

Severe hypokalaemia is incorrect. Although hypokalaemia may present with T-wave inversions in the precordial leads, there are no ST depression or U waves commonly seen in hypokalaemia. While hypokalaemia can cause muscle tetany, paralysis and respiratory failure, it is unlikely to cause collapse.

Takotsubo cardiomyopathy is incorrect. ECG changes like T wave inversion in Takotsubo cardiomyopathy are often non-specific. Moreover, the classical S1Q3T3

pattern in ECG, along with the clinical findings of tachycardia, tachypnoea and low-grade temperature, is more suggestive of pulmonary embolism in this patient.

Question #127

A 58 year-old man with a background of ischaemic heart disease and Crohn's disease has developed colonic enterocutaneous fistulae. He is admitted to hospital under the surgical team and a temporary ileostomy is formed to defunction the bowel and promote healing.

Two days post-operatively he develops palpitations and the surgical team request your assistance.

On examination the pulse rate is 220bpm and the blood pressure is 135/90mmHg. Oxygen saturations are 96% on 2L nasal oxygen.

The chest is clear to auscultation.

A 12-lead ECG reveals a wide-complex tachycardia with a polymorphic waveform.

Blood tests from the morning reveal:

Hb	129 g/l
Platelets	643 * 10 ⁹ /l
WBC	13.8 * 10 ⁹ /l
Na ⁺	129 mmol/l
K ⁺	3.3 mmol/l
Phosphate	0.63 mmol/l

Mg ⁺⁺	0.59 mmol/l
Urea	8.1 mmol/l
Creatinine	97 µmol/l
Bilirubin	15 µmol/l
ALP	143 u/l
ALT	53 u/l
Albumin	31 g/l

What is the most appropriate initial management?

- a) Adenosine 6mg Iv
- b) Magnesium sulphate 2g
- c) Metoprolol 5mg IV
- d) Synchronised DC shock
- e) Amiodarone 300mg IV

Correct answer is b.

In this scenario this patient has developed polymorphic ventricular tachycardia with no adverse features. This is likely to be secondary to electrolyte derangement in this case.

Initial management steps include stopping all drugs known to prolong the QT interval. Correct electrolyte abnormalities, especially hypokalaemia. Give magnesium sulphate 2 g IV over 10 min (= 8 mmol).

If adverse features are present (shock, syncope, myocardial ischaemia or heart failure) arrange immediate synchronised cardioversion.

Discuss (6) Improve

Question #1281

A 57-year-old man attends his GP surgery for review. For the past year, he has experienced exertional chest pain, which initially only came on whilst walking uphill, but has now started to affect him when walking on the flat. The pain is alleviated by the use of glyceryl trinitrate spray. He denies ever having had chest pain at rest and does not report any symptoms of postural hypotension. His other medications include aspirin, atorvastatin and atenolol, which has been maximally titrated.

What is the most appropriate next step in management for this patient?

- a) Refer for assessment for percutaneous coronary intervention (PCI) or CABG
- b) Start amlodipine
- c) Start isosorbide mononitrate
- d) Start ranolazine
- e) Start verapamil

If angina is not controlled with a beta-blocker, a longer-acting dihydropyridine calcium channel blocker should be added

The correct answer is **start amlodipine**. This patient has a likely diagnosis of stable angina, as he suffers from exertional chest pain which is alleviated by the use of GTN spray. As he is currently taking a beta-blocker at the maximum permissible dose, the next most appropriate step in management is to add in a calcium channel blocker. In this scenario, dihydropyridine calcium channel blockers are preferred to non-dihydropyridine calcium channel blockers, due to the increased risk of the latter causing complete heart block in combination with beta-blockers.

Refer for assessment for percutaneous coronary intervention (PCI) or CABG is incorrect. As the patient has stable angina, with no symptoms at rest, he does not warrant urgent referral for PCI. Non-urgent referral for PCI or CABG would be

appropriate if his symptoms were not improved by the addition of amlodipine.

Start isosorbide mononitrate is incorrect. This would be an appropriate third-line management option for stable angina in this patient if amlodipine failed to alleviate his symptoms. However, it should only be started whilst awaiting assessment for revascularization with PCI or CABG.

Start ranolazine is incorrect. This would also be more appropriate as a third-line management option whilst awaiting specialist assessment for PCI or CABG.

Start verapamil is incorrect. Although it would be reasonable to start a calcium channel blocker in this patient, starting a non-dihydropyridine calcium channel blocker such as verapamil in a patient already on a beta-blocker would run the risk of precipitating complete heart block.

Discuss (2)Improve

Question #129

An 80-year-old lady is brought in by ambulance having been found unresponsive by her granddaughter.

She has a history of vascular dementia, hypertension, atrial fibrillation and mild congestive cardiac failure. She lives alone and is visited by her granddaughter, who is her main carer, once per day. Her granddaughter states she has been more confused recently, finding it hard to know the time of day, or whether she had had meals or medications, but has been otherwise well.

She takes aspirin 75mg once daily, amlodipine 5mg once daily, bisoprolol 5mg once daily, ramipril 2.5mg once daily, and simvastatin 40mg at night.

On examination in the Emergency Department she has a Glasgow coma scale of 10. Her heart rate is 30/min and her blood pressure is 68/36 mmHg. Her saturations are 95% on 60% oxygen with a respiratory rate of 30 breaths per minute.

She has a regular heart rhythm and normal first and second heart sounds with an S3 gallop. Her jugular venous pressure is raised at 6 cm and she has pitting oedema to the knees. Her capillary refill is 4 seconds and she is peripherally cool. She has fine bibasal crepitations in both lungs. Her abdomen is soft and non-tender. Her temperature is 36.1°C.

Her ECG shows complete heart block with a ventricular rate of 30/min. Her QRS complexes are broad with a right bundle branch block pattern. There are signs of left ventricular hypertrophy but no T wave or ST segment abnormalities.

She is given 3mg of IV atropine in 500 mcg boluses but the heart rate remains 30/min with a blood pressure of 67/40 mmHg.

Which further medication is most likely to result in a sustained improvement in her heart rate?

- a) Aminophylline
- b) Dopamine
- c) Glucagon
- d) Isoprenaline
- e) Noradrenaline

This lady has severe bradycardia and is haemodynamically unstable with signs of acute heart failure.

Resus Council Advanced Life Support Guidelines advocate atropine, up to 3mg, as the initial therapy for this. However, this lady has not responded to this initial measure. The next step would be a choice of a number of drugs or transcutaneous pacing.

This lady had a history of dementia with a recent cognitive decline, lives alone and is on both a beta blocker and a calcium channel blocker. This combined with new complete heart block on her ECG, without signs of new ischaemia, is suggestive of

an accidental overdose. This makes glucagon the most logical choice to further improve heart rate as it bypasses the beta blockade.

Aminophylline, isoprenaline and dopamine are all other recommended second-line drugs in unstable bradycardia, along with adrenaline (rather than noradrenaline) and could be administered if there is an unsatisfactory response to glucagon.

In practice, it is worth preparing to transcutaneously pace while further drugs are given, in case the bradycardia proves refractory to all medication or the clinical condition worsens further.

Question #130

A 65-year-old man presents with loss of consciousness. He has a past medical history of two myocardial infarctions, one of which was four weeks ago. His wife states that he felt dizzy, complained of his vision going dark, and then fell to the floor.

An ECG shows ST elevation in V1-V4 without reciprocal depression.

What is the most likely diagnosis?

1. Thromboembolic stroke
2. Ventricular tachycardia
3. Anterior STEMI
4. Subarachnoid haemorrhage
5. Subdural haematoma

The presence of ST elevation without reciprocal depression shortly following a myocardial infarction is suggestive of a left ventricle aneurysm. Left ventricle aneurysms predispose to both ventricular arrhythmias and cardiac thromboembolisms.

The presence of ST elevation without reciprocal depression shortly following a myocardial infarction is very suggestive of a left ventricle aneurysm. Left ventricle aneurysms predispose to both ventricular arrhythmias and cardiac thromboembolisms, both of which are high up the differential in this case. The patient has presented with classic features of syncope, which makes an arrhythmia much more likely than an embolic stroke which rarely presents with syncope.

Subarachnoid haemorrhage can present with both loss of consciousness and ST elevation, and would be an important differential to consider, albeit less likely.

Question #131

A 68-year-old man attends the emergency department with palpitations and dizziness. This had a sudden onset approximately one hour ago. He denies chest pain. His observations are as follows: heart rate 170 beats per minute, BP 100/70 mmHg, respiratory rate 18 breaths per minute, and temperature 37.2 °C.

An ECG is performed in the department:

What is the most likely diagnosis?

1. Atrial fibrillation with aberrant conduction
2. Monomorphic ventricular tachycardia (VT)
3. Polymorphic ventricular tachycardia (VT)
4. Supraventricular tachycardia with aberrant conduction
5. Ventricular flutter

The ECG demonstrates a broad-complex tachycardia. It can be difficult to distinguish between ventricular tachycardia and supraventricular tachycardia with aberrant conduction (e.g. LBBB). Therefore it is always safest to assume that the arrhythmia is ventricular in origin until proven otherwise.

Electrocardiographic features that increase the likelihood of VT include:

- Absence of typical RBBB or LBBB morphology
- Extreme axis deviation ('northwest axis'): QRS positive in aVR and negative in I and aVF
- Very broad complexes > 160ms
- AV dissociation
- Capture beats
- Fusion beats
- Positive concordance throughout the precordial leads
- Negative concordance throughout the precordial leads
- RSR' complexes with an R>R'

Monomorphic ventricular tachycardia is the correct option. The ECG demonstrates RSR' complexes with an R>R' which is the most specific finding in favour of VT. Since the morphology of the QRS waves is similar beat to beat then the diagnosis is monomorphic VT rather than polymorphic VT.

Atrial fibrillation with aberrant conduction is incorrect. The presence of P waves (seen best in lead II) and the regular rhythm make this an unlikely diagnosis.

Polymorphic ventricular tachycardia is incorrect. This would also present with a broad complex tachycardia with similar criteria as discussed above. However, in this condition, the morphology of the ventricular complex would vary from beat to beat.

Supraventricular tachycardia with aberrant conduction is incorrect. Although this is a possibility, the extremely broad QRS duration and the presence of RSR' complexes with R>R' favour the diagnosis of monomorphic ventricular

tachycardia.

Ventricular flutter is incorrect. This condition is an extreme form of ventricular tachycardia (VT) with loss of organised electrical activity. The ECG would demonstrate a continuous sine wave with no identifiable P waves, QRS complexes, or T waves.

Question #132

A 76-year-old woman is brought into the Emergency Department following an episode of loss of consciousness. On more detailed review, she reveals that she has been suffering increasingly with shortness of breath on exertion, with great difficulty in managing her shopping and cleaning.

Her past medical history include right hip fracture 6 years ago after a fall walking down the stairs; this required a total hip replacement.

She takes no regular medication and has no drug allergies.

On examination, her pulse was 80 beats per minute, blood pressure 104/89 mmHg, and respiratory rate 16 breaths per minute. She had a slow rising pulse, and on auscultation of the chest there was an ejection systolic murmur heard loudest at the aortic area, and radiating to the carotid arteries.

Electrocardiogram (ECG) shows sinus rhythm and criteria for left ventricular hypertrophy.

Chest x-ray shows clear lung fields.

Transthoracic echocardiogram:

Aortic valve area	0.8 cm ² (3-4)
Transvalvular gradient	55 mmHg (0)

The diagnosis of aortic stenosis is explained to the patient, and she opts for open surgical valve replacement.

What other investigation is warranted before proceeding to aortic valve replacement?

1. Exercise tolerance testing
2. Dopamine stress echocardiogram
3. 24 hour holter ECG
4. Transoesophageal echocardiogram
5. Coronary angiography

The valve area <1 cm² and transvalvular gradient >50 mmHg on transthoracic echocardiogram confirm the diagnosis of severe aortic stenosis, and the patient has opted for open surgical valve replacement.

Patients undergoing open surgical valve replacement should first undergo coronary angiography to exclude any coronary stenosis that could simultaneously be treated with bypass grafting.

This patient has undergone an 12 lead ECG and transthoracic echocardiogram. Further ECG or echocardiogram studies are not usually necessary.

Question #133

A 13 year-old girl presents with chest pain and fever.

On examination the temperature is 38.1°C, heart rate is 90 beats/minute and respiratory rate is 18 breaths/minute. The chest is clear to auscultation. There is an early diastolic murmur at the left sternal edge.

ECG reveals sinus rhythm, PR interval 210ms.

Blood tests reveal:

Hb	121 g/l
Platelets	420 * 10 ⁹ /l
WBC	9.3 * 10 ⁹ /l
Na ⁺	136 mmol/l
K ⁺	3.7 mmol/l
Urea	3.8 mmol/l
Creatinine	72 µmol/l
Bilirubin	22 µmol/l
ALP	110 u/l
ALT	53 u/l
Albumin	36 g/l
C-reactive protein	36
Antistreptolysin O antibody titre	320 units/ml

Which other sign may also be present in this patient?

1. Dermatitis herpetiformis
2. Erythema marginatum

3. Pyoderma gangrenosum
4. Vitiligo
5. Livedo reticularis

Acute rheumatic fever (ARF) is caused by a reaction to a bacterial infection with particular strains of group A streptococcus.

Symptoms of ARF generally develop several weeks after an episode of streptococcal pharyngitis

ARF causes a variety of characteristic clinical features:

- Polyarthritis
- Carditis
- Sydenham's chorea
- Erythema marginatum (A characteristic skin rash that occurs in about 10% of first attacks of ARF in children. It is rare in adults). The rash appears as pink or red macules (flat spots) or papules (small lumps), which spread outwards in a circular shape. As the lesions advance, the edges become raised and red, and the centre clears.

Question #134

A 67-year-old woman is reviewed in cardiology clinic following a GP referral. A recent transthoracic echocardiogram demonstrated severe aortic stenosis with an aortic valve at 0.7cm^2 and a mean pressure gradient of 62mmHg and a left ventricular ejection fraction of 43%. Pulmonary arterial pressure is normal. She has a chest X-ray two months ago which was normal. She denies any symptoms of shortness of breath, chest pain or lightheadedness.

She is normally fit and well with a past medical history of hypothyroidism, two caesarian sections, osteoarthritis and migraines. She is a non-smoker. What is the

most appropriate management plan?

1. Review in six months
2. Cardiac MRI
3. Exercise test
4. Aortic valve replacement
5. Transcatheter aortic valve implantation (TAVI)

The correct answer is aortic valve replacement. This is an asymptomatic patient with severe stenosis but she has an ejection fraction of less than 50%. This means that she should be referred for aortic valve replacement or TAVI if unsuitable. Exercise testing would be recommended if her ejection fraction was greater than 50%. Cardiac MRI would not be helpful in this instance. If she had an ejection fraction greater than 50% and passed exercise testing then she could be reviewed in six months.

Source:

Vahanian A., Alfieri O., Andreotti F., et al. Guidelines on the management of valvular heart disease (version 2012). The Joint Task Force on the Management of Valvular Heart Disease of the European Society of Cardiology and the European Association for Cardio-Thoracic Surgery. European Heart Journal. 2012;33:24512496. doi: 10.1093/eurheartj/ehs109

Discuss (14) Improve

Question #135

A patient with a severe rheumatic heart disease is about to attend a gastroscopy with oesophageal dilatation. He has no current active gastrointestinal infection. What endocarditis prophylaxis is recommended?

1. Ciprofloxacin PO 30 min pre-procedure
2. None8

3. IV ampicillin at time of surgery
4. IV cefazolin at time of surgery
5. Erythromycin PO 30 min pre-procedure

Prophylaxis is only indicated when one of the following are met:

Prosthetic cardiac valve or prosthetic material used for cardiac valve repair

Previous infective endocarditis

Cardiac transplantation with the subsequent development of cardiac valvulopathy

Congenital heart disease but only if it involves:

Unrepaired cyanotic defects, including palliative shunts and conduits

Completely repaired defects with prosthetic material or devices whether placed by surgery or catheter intervention, during the first 6 months after the procedure (after which the prosthetic material is likely to have been endothelialised)

Repaired defects with residual defects at or adjacent to the site of a prosthetic patch or device (which inhibit endothelialisation)

Congenital heart disease but only if it involves:

- Unrepaired cyanotic defects, including palliative shunts and conduits
- Completely repaired defects with prosthetic material or devices whether placed by surgery or catheter intervention, during the first 6 months after the procedure (after which the prosthetic material is likely to have been endothelialised)
- Repaired defects with residual defects at or adjacent to the site of a prosthetic patch or device (which inhibit endothelialisation)

Active or not active gastrointestinal infection does not alter your management of prophylaxis, but would be advisable to defer the procedure if possible.

Discuss (1)Improve

Question #136

A 72-year-old woman with a 30 year history of type 2 diabetes mellitus comes for review. She was diagnosed with chronic kidney disease (secondary to diabetes) 8 years ago and has seen declining renal function since. Her current medication includes ramipril 10mg od, amlodipine 10mg od, simvastatin 40mg on and Novomix 30 insulin bd.

Her most recent renal function tests show the following:

Na ⁺	139 mmol/l
K ⁺	5.3 mmol/l
Urea	10.2 mmol/l
Creatinine	123 µmol/l
eGFR	40 ml/min/1.73m ²

Blood pressure in clinic is 156/88 mmHg and this is confirmed on a second reading. What should be done regarding her blood pressure medication?

1. Add bisoprolol
2. Add indapamide
3. Add doxazosin

4. Add spironolactone
5. Add an angiotensin II receptor blocker

Poorly controlled hypertension, already taking an ACE inhibitor and a calcium channel blocker - add a thiazide-like diuretic

Tight blood pressure remains a key management aim in patients with diabetic nephropathy. ACE inhibitors are clearly the most evidence based management in this arena but her blood pressure is persistently high. If we look at the NICE guidelines the next step would be the addition of a thiazide based diuretic (e.g. indapamide) and there is no reason not to follow these recommendations in this situation.

If the eGFR is less than 30 ml/min/1.73m² then thiazides should be avoided as the BNF states:

Thiazides and related diuretics are ineffective if eGFR is less than 30 mL/minute/1.73 m² and should be avoided; metolazone remains effective but with a risk of excessive diuresis

Spironolactone and angiotensin II receptor blockers may risk precipitating hyperkalaemia.

Question #137

A 78-year-old woman attends the cardiology outpatient department after being referred by her general practitioner with angina. The patient reports consistent chest tightness after walking around 200 metres on flat ground but denies any episodes of pain at rest. Her symptoms have been stable at the current level for at least six months. Her GP has previously attempted treatment with both metoprolol and amlodipine but both were stopped after the patient reported dizziness and was found to be hypotensive.

The patient was formerly a heavy smoker but had stopped 15 years previously after suffering a non-ST elevation myocardial infarction (managed with medical

therapy only). The patient had been diagnosed with type 2 diabetes mellitus 10 years previously and was known to have chronic kidney disease.

The patients current medications included aspirin 75 mg daily, simvastatin 20 mg daily, ramipril 1.25 mg daily and gliclazide 60 mg twice daily. She sometimes used a nitrate spray to relieve episodes of angina but found that it normally made her very dizzy.

Clinical examination was unremarkable except for a blood pressure of 98 / 65 mmHg.

Please see below for results of recent investigations.

Transthoracic echocardiogram: normal valvular function; moderately impaired left ventricular; ejection fraction 35 %.

Electrocardiogram: sinus rhythm at 85 beats per minute; normal axis; normal QRS complex, normal ST segments and T waves.

Urea	15.6 mmol / L
Creatinine	234 micromol / L
Sodium	135 mmol / L
Potassium	4.9 mmol / L

Cardiac stress MRI: evidence of mild myocardial ischaemia in left anterior descending territory, estimated < 10 % myocardium ischaemic.

What is the correct next line therapy for the patients angina?

1. Percutaneous coronary intervention
2. Nicorandil

3. Ranolazine
4. Ivabradine
5. Long-acting isosorbide mononitrate

The patient has stable angina and has been intolerant of first line medical therapy. Due to her co-morbidities several of the second-line medical therapy options are contra-indicated. Ranolazine acts a late inward calcium channel antagonist but is contra-indicated in severe renal disease. Both nicorandil and ISMN are contra-indicated in this patient due to her history of hypotension.

Therefore, ivabradine is the most appropriate option. The drug acts as an If channel antagonist to reduce heart rate and is appropriate in this patient without a history of bradycardia. It should not be used in patients with moderate to severe angina as it has been shown to increase the incidence of cardiovascular events in these patients.

Revascularisation techniques such as PCI have not been shown to reduce mortality or rate of MI in stable coronary artery disease. Therefore, medical therapy options should be exhausted before consideration of invasive treatment (particularly in this patient with renal failure making contrast administration very undesirable).

Al-Lamee R, Davies J, Malik I. What is the role of coronary angioplasty and stenting in stable angina? BMJ 2016;352:i205.

Discuss (9) Improve

Question #138

A 43-year-old man presents with palpitations to the emergency department. His ECG demonstrates a broad complex tachycardia at 180 beats/min, suggestive of ventricular tachycardia. His blood pressure is 134/80 mmHg, he is uncomfortable but alert. The emergency department doctor has administered a 300mg bolus of amiodarone and the consultant subsequently inserted a central venous line to continue amiodarone treatment with a 900mg infusion over 24 hours. He

transiently settles but on the second day of admission, his cardiac monitor demonstrates persistent VT at 180 beats per minute again. His blood pressure is 140/75 mmHg. What is your treatment?

1. No treatment
2. Repeat bolus of amiodarone 300mg
3. DC cardioversion
4. Intravenous lidocaine infusion
5. Emergency VT ablation

The patient has entered refractory VT, continuing despite a therapeutic dose of amiodarone and sufficient loading. It is unlikely further boluses of amiodarone will produce additional therapeutic effects. Similarly, no treatment is not an option, his myocardium is unlikely to continue tolerating such a persistent tachycardia without soon reducing left ventricular output and arrest. He is not haemodynamically unstable and hence does not justify DC cardioversion as an emergency procedure. Similarly, emergency VT ablation is not appropriate after only 1st line treatment. Intravenous lidocaine is the second line treatment: first as a bolus, followed by an infusion regime over 6.5 hours.

Question #139

An 87-year-old gentleman is reviewed in the cardiology clinic. He has unstable angina with multiple confluent lesions in all three main coronary vessels and in major tributaries on angiography. The lesions are not amenable to stenting or bypass. On assessment in the clinic, the blood pressure is 104/43mmHg, heart rate is 57bpm. His ECG confirms atrial fibrillation but no new ischaemic changes. There is no clinical sign of heart failure. He is currently taking aspirin, bisoprolol, ramipril, isosorbide mononitrate, nicorandil and simvastatin. His main symptom is chest pain on minimal exertion.

Which of the following is the most appropriate drug to control this patients angina?

- a) Diltiazem
- b) Digoxin
- c) Ivabradine
- d) Omega
- e) Ranolazine

National guidelines were published by the Scottish Intercollegiate Guideline Network (SIGN) on the management of stable angina in 2007 with updates by NICE in 2011. Stable angina is defined as chest pain of cardiac aetiology which is precipitated by exertion with a demonstrable flow limiting lesion on angiography. Many patients who experience angina will ultimately go on to have revascularisation procedures to improve or restore coronary flow which will improve their symptoms of angina, however while waiting for interventional procedures optimisation of drug therapy is the aim. Drug therapy also is considered first-line treatment for non-critical lesions.

First line therapy in the treatment of angina is beta-blockade. Beta-blockers reduce myocardial oxygen consumption by acting as a negative inotrope and also by reducing heart rate. Some patients are unable to take beta-blockers such as asthmatics, or find that side effects such as tiredness, eczema or sexual dysfunction mean they are not tolerable. In these instances, use of calcium channel blockers such as diltiazem are recommended as first-line treatment. Since both of these classes of drug cause a reduction in heart rate and blood pressure their use must be monitored carefully, especially in the elderly. Concomitant use of beta-blockers and calcium channel blockers should be avoided where possible due to the risk of high degree conduction block and cardiac arrest.

Long-acting nitrates, such as isosorbide mononitrate or dinitrate are considered second-line therapy in stable angina. They can be used as monotherapy or as an adjunct to beta-blocker or calcium channel blocker therapy. They work by causing vasodilatation; both drugs possess a nitric oxide moiety which causes endothelial relaxation in blood vessels. This causes coronary vasodilation and improved blood flow to the myocardium with improved symptoms. A consequence of vasodilatation is hypotension and this should be considered when prescribing this

drug to patients.

Another second-line option drug for stable angina in those patients who cannot tolerate nitrates or they are ineffective is nicorandil. This drug has a similar ultimate effect in that it causes dilatation of peripheral and coronary vessels to reduce symptoms of angina. It works by activating potassium channels on endovascular smooth muscle cells reducing intracellular calcium and hence relaxing the blood vessel. It too can cause profound hypotension and severe headaches.

Ivabradine is a drug which can be used for both angina and heart failure. It works by reducing heart rate to reduce myocardial oxygen demand and improve diastolic function. In this case, the heart rate is adequately controlled so reduction is not necessary. Ivabradine also works by inhibiting the hearts natural pacemaker potential in the sinoatrial node, hence it only works when the patient is in sinus rhythm, unlike here.

Ranolazine is a viable option in the management of angina where other avenues have been exhausted. It works by inhibiting the delayed sodium influx channel in the myocardium, reducing the intracellular calcium concentrations in the heart muscle. This, in turn, leads to a negative inotropic effect and reduction in symptoms of angina. Ranolazine does not cause a profound hypotensive effect and is the best choice in this scenario.

Digoxin is not commented upon as a specific treatment for heart failure in NICE guidance although it does have a modest positive inotropic effect. The relative bradycardia of the patient in the vignette makes this an unwise choice here.

Omega 3 and other fatty fish oils are not recommended to be prescribed to heart failure patients in NICE guidance due to lack of evidence of any effectiveness.

Question #140

A 73-year-old gentleman sees his GP with progressive shortness of breath. He can now walk 10 yards before stopping to catch his breath and he is unable to lie flat. He sleeps upright in his armchair and has stopped sleeping in his bed over the last

three months.

He has known hypertension and has had two previous myocardial infarctions. On examination, his apex beat is displaced to the 7th intercostal space in the left mid-axillary line and there are crepitations in the two lung bases. His JVP is raised at 5cm and there is bilateral leg oedema.

Troponin	<20 ng/ml
B-natriuretic peptide	14,000 ng/L (normal range 0-200)
Chest X-ray	cardiomegaly and increased alveolar shadowing in the perihilar areas
ECG	QRS 110ms, large volume complexes in chest leads

Which intervention is proven to reduce mortality in this case?

- a) Cardiac Resynchronisation Therapy (CRT)
- b) Digoxin
- c) Furosemide
- d) Ramipril
- e) Coronary artery bypass graft

ACE inhibitors offer prognostic benefit in chronic heart failure

Of the above options, ramipril is proven to reduce mortality in congestive cardiac failure through adjusting remodelling of the heart. Digoxin and furosemide can help symptoms but no trial shows a benefit in mortality. CRT can be helpful in cases of ventricular dyssynchrony but the QRS is narrow and this gentleman would

not benefit from it. Coronary artery bypass grafting may be useful if this gentleman had symptoms of angina but he does not.

Question #141

A 14-year-old female presented to her GP having collapsed at school. Her friends stated that whilst she was playing hockey she inexplicably collapsed and had lost consciousness for a few seconds. This was her first episode of collapse. No seizure activity was observed, and she made a full and spontaneous recovery a few moments later. She denied the presence of prodromal symptoms, and other than occasional palpitations upon exertion she was otherwise well. She had a cochlear implant inserted in early childhood for congenital hearing impairment but otherwise had no past medical history of note, and was not taking any medications. There was no family history of note.

Examination revealed the presence of a healthy athletic 14-year-old female. Her heart rate was 58bpm and regular, and her blood pressure 108/78 mmHg. Cardiovascular examination revealed a JVP of 3cm and was otherwise unremarkable with normal heart sounds. Gastrointestinal examination and neurological examinations were likewise unremarkable.

Initial investigations revealed the following results:

ECG: 57bpm normal, sinus rhythm QRS 112 ms, PR 122ms, QTc 502ms, normal ST and T wave morphology.

What is the most likely diagnosis?

- a) Brugada syndrome
- b) Romano Ward syndrome
- c) Wolff Parkinson White syndrome
- d) Jervell & Lange-Nielsen syndrome
- e) Hypertrophic obstructive cardiomyopathy

Inherited long QT syndrome, sensorineural deafness - Jervell and Lange-Nielsen syndrome

This female has long QT syndrome (LQTS), as per the findings of her ECG in combination with a history of collapse on exertion. The main differential diagnosis here is Romano-Ward syndrome, which is also a LQTS but is not associated with deafness, as opposed to Jervell & Lange-Nielsen syndrome, which is associated with deafness. A normal cardiac examination with no evidence of hypertrophy on ECG would make the diagnosis of hypertrophic obstructive cardiomyopathy less likely.

Question #142

A 10-year-old girl is reviewed. Her mother describes her as being 'generally unwell'. For the past week she has been having joint pains and fever. Her mother also describes episodes of her making jerky, irregular movements. She recently went on holiday to the Lake District and had a severe sore throat around 4 weeks ago.

On examination her temperature is 37.9°C, pulse 90/min, blood pressure 100/62 mmHg. Auscultation of the heart is unremarkable. A rash is noted on her torso:



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What does the rash most likely represent?

- a) Erythema marginatum
- b) Erythema multiforme
- c) Erythema chronicum migrans
- d) Janeway lesion
- e) Erythema toxicum

The underlying diagnosis is rheumatic fever. This is supported by the recent sore throat, chorea (jerk, irregular movements) and polyarthralgia.

Erythema toxicum is a non-specific rash seen in newborns.

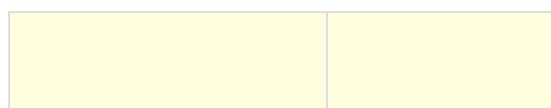
Discuss (5) Improve

Question #143

A 53-year-old woman has been admitted to the hospital with progressively worsening dyspnoea. She denies a cough but describes orthopnoea and the feeling of palpitations.

Observations are shown below:

Heart rate	112 bpm
Blood pressure	86/42 mmHg
Respiratory rate	28 /min
Oxygen saturations	99%
Temperature	36.8 °C



On examination, she is overweight and appears dyspnoeic. Her airway is self-maintained and her chest appears clear on auscultations. Heart sounds are difficult to auscultate, her JVP is raised and her capillary refill time is normal. There is no evidence of pedal oedema.

Given the likely diagnosis, what might you see on the ECG?

- a) Deep lateral Q waves with left ventricular hypertrophy
- b) Electrical alternans
- c) Global ST elevation
- d) S1 Q3 T31
- e) Trifascicular block

Electrical alternans is suggestive of cardiac tamponade

A history of dyspnoea, orthopnoea, and palpitations, associated with hypotension and tachycardia are classical symptoms of many cardiorespiratory illnesses. However, quiet or absent heart sounds with a raised JVP should raise concerns for cardiac tamponade. This can be confirmed with the ECG findings of sinus tachycardia, low QRS voltage, or electrical alternans.

Electrical alternans is a diagnostic feature of large pericardial effusion and cardiac tamponade. The alternate QRS amplitudes are due to the 'swinging' effect of the heart whilst contracting which subsequently gives rise to increased and decreased electrical impedance.

Deep lateral Q waves with left ventricular hypertrophy is a classical ECG finding of hypertrophic cardiomyopathy (HOCM). HOCM is the most common cause of sudden death in young people and is due to left ventricular hypertrophy without an identifiable cause. Patients can display all of the above symptoms. Dyspnoea is common due to left ventricular stiffness, resistance, and impaired filling. Patients

may have a jerky pulse and may also present in arrhythmia, most commonly, atrial fibrillation. However, compared to the above scenario, patients have audible heart sounds, and may have a loud S1 and sometimes a 4th heart sound. There may be an ejection systolic murmur radiating to the apex which changes in intensity with a change in posture.

Pericarditis is associated with **global ST elevation**. Patients typically present with many of the above symptoms. Pericarditis is a common cause of pericardial effusion due to the accumulation of fluid in the pericardial sac due to inflammation. The distinguishing feature between pericarditis and pericardial effusion is the presence or absence of heart sounds, with pericarditis having audible heart sounds.

Symptoms of dyspnoea, palpitations, and orthopnoea, along with hypotension and tachycardia are common signs and symptoms associated with a pulmonary embolism (PE). The most common ECG finding in PE is sinus tachycardia, however, patients may have evidence of right axis deviation, atrial arrhythmia, right bundle branch block, P pulmonale, and **S1 Q3 T3**. S1 Q3 T3 is the presence of deep S waves in lead I along with Q waves and T wave inversion in lead 3.

Trifascicular block is the combination of third-degree atrioventricular block, right bundle branch block, and either left anterior fascicular block or left posterior fascicular block. These patients generally required pacemaker insertion. The most common causes are structural heart disease, particular fibrosis of the myocardium, and ischaemic heart disease. It may also occur in anterior myocardial infarction and aortic stenosis. Many of the pathological causes of trifascicular block may cause the symptoms described in the above scenario, however, heart sounds would still be audible. Trifascicular block is not associated with pericardial effusion.

Question #144

A 24-year-old female presents to clinic complaining of several episodes of heart palpitations. She states that during the attacks it feels like her heart skips a bit. She denies pre-syncope or syncope. She states that she has one episode every few days. She drinks approximately 28 units of alcohol per week and several cups of

coffee per day.

You arrange a 7 day cardiac holter monitor. The results are as follows:

PR interval	100ms
QRS duration	110ms
Events	24 single ectopics with normal P wave morphology and a QRS of 110ms

What is the most appropriate initial management plan?

- a) Bisoprolol
- b) Ablation
- c) Reassurance and lifestyle modifications
- d) Implantable Cardioverter Defibrillator (ICD)
- e) Verapamil

Patients with ventricular ectopics can usually be managed by reassurance and lifestyle modifications. If pharmacological management is required, then beta blockers are first line

The ectopics captured have normal P wave and QRS duration suggestive of supraventricular ectopics of sinus or atrial origin. The first line management of supraventricular ectopics is generally reassurance and lifestyle modifications. The patient should be advised to reduce alcohol and caffeine intake. If symptoms persisted then a beta blocker would be first line.

Question #145

A 45-year-old man is being worked up for chest pain.

He has a background of bipolar disorder for which he takes olanzapine but is otherwise fit and well.

He has had a cardiac MIBI scan that is reported as follows:

Images display a moderate perfusion abnormality in the right ventricle on the stress images with associated dyskinesis. This defect is also present at rest.

Given the above information, what is the most likely cause of these imaging findings?

- a) Atherosclerosis
- b) Hypertrophic cardiomyopathy
- c) Kawasaki disease
- d) Soft tissue attenuation defect
- e) Transmural scarring

If a cardiac MIBI scan shows that the defect is present on both stress and rest, this is suggestive of a fixed defect such as myocardial necrosis and fibrosis secondary to infarction

Here we have a patient with a perfusion defect in the right ventricle at rest and during stress. This indicated that the lesion was not reversible.

Transmural scarring is correct. When scar tissue is formed within the myocardium, there will be a fixed perfusion defect as blood supply to this area never normalises.

Atherosclerosis is incorrect. Atherosclerosis commonly causes reversible defects that normalise at rest.

Soft tissue attenuation defect is incorrect. Although soft tissue attenuation

defects can cause the appearance of fixed defects, there would not be associated wall motion abnormalities.

Kawasaki disease is incorrect. Although this vasculitis can be a cause of myocardial infarction it is very rare. It usually affects children under the age of five and is associated with fever, strawberry tongue, rash, and lymphadenopathy, none of which are seen in this case.

Hypertrophic cardiomyopathy is incorrect. This would be visible on MIBI scan as ventricular hypertrophy, if a defect is present, it is likely to be present only during stress.

Question #146

A 66-year-old man was admitted from the community with an anterior STEMI to the local district general hospital. He complained of severe chest pain before his wife rang for help. She noted he was clammy and sweaty. About five minutes after being transferred into the emergency department a nurse observed that the patient looked unwell, appeared grey and poorly responsive, . The nurse put out a peri-arrest call. The cardiac monitor showed a broad complex tachycardia. His femoral pulse was faintly palpable and his blood pressure was 76/36 mmHg.

What should be carried out immediately ?

- a) Start CPR
- b) IV fluids
- c) DC cardioversion
- d) IV amiodarone
- e) Transfer to tertiary center for immediate PCI

DC cardioversion should be given to a patient presenting with ventricular tachycardia with haemodynamic compromise

The answer is immediate DC cardioversion for a patient with ventricular tachycardia presenting with haemodynamic compromise. If the patient was stable, amiodarone and magnesium could be considered. It is important to check the electrolytes. Ideally, the patient needs PCI but must be stable before transfer can take place. There is no role for CPR as this is not a cardiac arrest situation.

Question #147

You are asked to review a 22-year-old female with a heart murmur. Her height is 4ft 7 inches and she reports having always been short for her year. She has been diagnosed with autoimmune hypothyroidism, for which she is taking levothyroxine replacement and is also taking 'hormone replacement to protect her bones' after her GP noted she had not started her periods by 19 years old.

On examination, you note short 4th metacarpals on both hands and lymphoedematous in her hands and feet. Auscultation of her precordium reveals an ejection systolic murmur at the left infraclavicular region. Her body mass index is 22.4 kg/m². The patient's blood tests are as follows:

Hb	123 g/l
Platelets	380 * 10 ⁹ /l
WBC	6.5 * 10 ⁹ /l

Na ⁺	144 mmol/l
K ⁺	4.8 mmol/l
Urea	7.2 mmol/l
Creatinine	110 µmol/l

Adjusted calcium	2.40 mmol/l
Phosphate	1.1 mmol/l
PTH	9 mg/dl (8.5-10.2mg/dl)

What is the underlying unifying diagnosis?

- a) Turner's syndrome
- b) Pseudohypoparathyroidism
- c) Pseudopseudohypoparathyroidis
- d) Noonan syndrome
- e) Down's syndrome

A syndromic presentation appears most likely in this patient. An acute infective endocarditis is unlikely for someone who is systemically well, a regurgitant murmur would also be more likely. The symptoms cannot be attributed to delayed puberty, defined as onset of puberty outside 95% of the normal population and typically before 18 years old. The combination of primary amenorrhoea, short stature, lymphoedema, shortened 4th metacarpal, autoimmune predisposition and an ejection systolic murmur suggestive of aortic coarctation should infer a diagnosis of Turner's syndrome, a disorder caused by loss of an X chromosome.

Noonan syndrome, an autosomal dominant disorder, is a reasonable differential in a patient with short stature, lymphoedema and cardiac problems. However, patients with Noonan syndrome typically presents with learning difficulties, facial dysmorphisms and webbed neck. While valvular abnormalities are less common, hypertrophic cardiomyopathy is more commonly present.

Similar to Noonan syndrome, Down's syndrome, caused by trisomy 21, also

classically presents with facial dysmorphisms such as upslanting palpebral fissures, low-set ears and epicanthic folds. Cognitive impairment and learning difficulties are also common. However, similar to our patient, short stature, thyroid dysfunction and cardiac problems, predominantly ASD, VSD and PDA are frequently associated.

Pseudohypoparathyroidism is caused by normal parathyroid hormone release but end organ resistance to hormonal effects, resulting classically in round facies and shortened fourth and fifth metacarpals. The patient is commonly obese with possible learning difficulties.

Pseudopseudohypoparathyroidism presents with the phenotype of pseudohypoparathyroidism but with normal calcium chemistry.

Question #148

An 8-year-old boy presents to the emergency department with seven days of fever. He originally attended six days previously and was discharged home with advice to return if the fever persisted. There has been no response to paracetamol treatment. He has not past medical history.

Observations:

- Heart rate 84 beats per minute
- Blood pressure 100/70 mmHg
- Temperature 39.4C

On examination, his conjunctivae are erythematous. He has red, cracked lips and a 'strawberry tongue'. There is unilateral cervical lymphadenopathy. The palms of his hands and soles of his feet are red. The examination is otherwise normal.

Urinalysis and a chest x-ray are unremarkable.

Blood tests:

Hb	136 g/L	Male: (135-180) Female: (115 - 160)
Platelets	$189 * 10^9/L$	(150 - 400)
WBC	$8.9 * 10^9/L$	(4.0 - 11.0)
Na ⁺	140 mmol/L	(135 - 145)
K ⁺	4.2 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	77 µmol/L	(55 - 120)
CRP	35 mg/L	(< 5)
Bilirubin	14 µmol/L	(3 - 17)
ALP	88 u/L	(30 - 100)
ALT	64 u/L	(3 - 40)
γ ³ GT	44 u/L	(8 - 60)
Albumin	36 g/L	(35 - 50)

In addition to aspirin, what other treatment has the most evidence for benefit in treating this condition?

- a) Antibiotic treatment
- b) Cyclophosphamide
- c) Intravenous immunoglobulin
- d) Prednisolone
- e) Rituximab

Intravenous immunoglobulin , uses include: Kawasaki disease

Intravenous immunoglobulin is correct. The child has Kawasaki disease as evidenced by a fever lasting > 5 days, bilateral conjunctival congestion, cervical lymphadenopathy and changes of the lips and extremities. This is a medium-vessel vasculitis that occurs in children. In addition to aspirin, intravenous immunoglobulin should be administered to prevent the formation of coronary artery aneurysms, which is a feared complication of this condition.

Antibiotic treatment is incorrect. The clinical syndrome, in this case, suggests Kawasaki disease as the cause of fever, rather than infection. This is an inflammatory rather than infective condition and does not require antibiotic treatment.

Cyclophosphamide is incorrect. This has been used occasionally in refractory Kawasaki disease but it has less evidence than IVIG. Additionally, there is a risk of infertility associated with this treatment and therefore it should be avoided in people of child-bearing age unless absolutely necessary.

Prednisolone is incorrect. There is more limited evidence for the benefit of corticosteroids in this condition compared with IVIG. Adjunctive treatment with corticosteroids could be considered in particular high risk groups (Age < 12 months, Asian ethnicity) and in the setting if particular blood test abnormalities (ALT >100 IU/L, albumin ≤30 g/L, sodium ≤133 mmol/L, platelets ≤30 x 10⁹/L, CRP >100 mg/L, anemia) or cardiac or coronary artery involvement on echo at presentation.

Rituximab is incorrect. This is an anti-CD20 monoclonal antibody that is used in

the treatment of some vasculitis. There is limited evidence for its use in Kawasaki disease but it has been used occasionally in IVIG refractory disease.

Discuss (3) Improve

Question #149

A 30-year-old pregnant woman (24 weeks) is admitted to the acute medical unit with sudden onset shortness of breath and pleuritic chest pain. She has no past medical history other than eczema and this is her first pregnancy.

On examination, auscultation of her chest reveals only a mild wheeze with oxygen saturations of 94% on room air and a respiratory rate of 25/min. Her heart sounds are normal, with a heart rate of 97bpm and blood pressure is 105/60 mmHg. An ECG shows sinus rhythm.

The FY1 doctor on the ward has requested a D-dimer to try and 'speed up the diagnosis', which comes back positive.

What is the next appropriate investigation?

- a) Ventilation-Perfusion (V/Q) scan
- b) Chest x-ray
- c) Peak flow measurement
- d) CT Pulmonary Angiogram (CTPA)
- e) Ultrasound doppler of the lower legs

It is likely that this woman has had a pulmonary embolism given the acute nature of the shortness of breath and pleuritic chest pain, together with tachycardia and reduced oxygen saturations.

The correct way to investigate a pregnant woman with a possible pulmonary embolism is to first perform a chest x-ray. This is vital to ensure other diagnoses such as pneumonia and pneumothorax are identified

Ultrasound doppler of the lower legs is the next step as this can demonstrate a venous thromboembolism of the lower limb vasculature without radiation. If a thrombus is found, appropriate management with low molecular weight heparin can be initiated.

If the chest x-ray and the ultrasound doppler are normal, a ventilation-perfusion scan should be performed. The presence of a normal chest x-ray can essentially function as the ventilation part of the V/Q scan. This allows for a half-dose radiation V/Q scan.

The radiation dose to the foetus of a V/Q scan is slightly higher than that of a CTPA, however both are well below the threshold of specific harm to the foetus. Please note that a CTPA should only be used in the presence of lung disease including asthma or an abnormal chest x-ray, as the radiation dose to the proliferating maternal breasts may be significant in the development of carcinogenesis later in life.

A d-dimer is not appropriate as it may be raised in pregnancy and may lead to further inappropriate tests based on the positive result alone.

Question #150

A 44-year-old gentleman is admitted to hospital with a 2 week history of flu-like symptoms, wheeze, shortness of breath on lying flat and palpitations. There is no background history of note. His observations include a heart rate of 98/min, blood pressure of 96/50 mmHg and saturations of 96% while on 2L of oxygen. He is apyrexial.

On examination you note a low volume pulse with a raised JVP and a raised Y descent. A third heart sound is audible. There are no murmurs. There are bilateral crepitations and pitting oedema of the legs.

Blood investigations indicate:

Hb	158 g/L	Male: (135-180) Female: (115 - 160)
Platelets	$335 * 10^9/L$	(150 - 400)
WBC	$11.7 * 10^9/L$	(4.0 - 11.0)
Neuts	$8.0 * 10^9/L$	(2.0 - 7.0)
Lymphs	$2.3 * 10^9/L$	(1.0 - 3.5)
Mono	$0.7 * 10^9/L$	(0.2 - 0.8)
Eosin	$0.69 * 10^9/L$	(0.0 - 0.4)

Na ⁺	138 mmol/L	(135 - 145)
K ⁺	4.2 mmol/L	(3.5 - 5.0)
Urea	5.3 mmol/L	(2.0 - 7.0)
Creatinine	78 µmol/L	(55 - 120)
CRP	12 mg/L	(< 5)

Three blood cultures have been sent and the preliminary report is negative.

The ECG on admission shows sinus rhythm with left ventricular hypertrophy pattern but no features of ischaemia; the PR interval is normal.

An echocardiogram is performed and notes shows preserved systolic function with

moderate concentric left ventricular hypertrophy. Both atria are dilated and there is evidence of diastolic dysfunction. There is no valvular pathology and no suspicious lesions surrounding the valves.

Given this scenario, which imaging modality would be most appropriate in identifying the diagnosis of this patient?

- a) Transoesophageal echocardiography
- b) Right and left cardiac catheterization
- c) Myocardial perfusion scan
- d) Cardiac MRI
- e) High resolution CT thorax

Cardiac MRI is the investigation of choice alongside echocardiography in order to identify the potential cause of cardiomyopathy

The echocardiography and examination findings suggest the potential of restrictive cardiomyopathy. In addition this gentleman has significant eosinophilia which could alert us to the possibility of Loffler's endocarditis.

The options provided cover a wide spectrum of cardiac imaging modalities, however cardiac MRI yields the greatest detail with regards to function and structure of the heart in such scenarios.

The possibility of infective endocarditis remains low and a transoesophageal echocardiogram would not necessarily add anything further with regards to the diagnosis.

A catheterisation or myocardial perfusion scan will provide excellent information regarding ischaemia however the information regarding the cause of this restrictive cardiomyopathy will not be revealed.

There is no indication for a high resolution CT thorax given the high grade of suspicion regarding a cardiac primary problem.

Question #151

You are reviewing in clinic a 67-year-old man who has type 2 diabetes. His glycaemic control is reasonable with metformin therapy; the latest HbA1c is 54 mmol/mol (7.1%). A few weeks ago he was noted to have a clinic blood pressure reading of 152/90 mmHg. A 24 hour blood pressure monitor was requested. The report shows his average blood pressure was 142/88 mmHg. What is the most appropriate course of action?

- a) Do nothing for now, monitor his blood pressure regularly
- b) Start an ACE inhibitor
- c) Start a calcium channel blocker¹
- d) Repeat the 24 hour blood pressure monitor in 4-8 weeks time
- e) Request an ultrasound of his kidneys

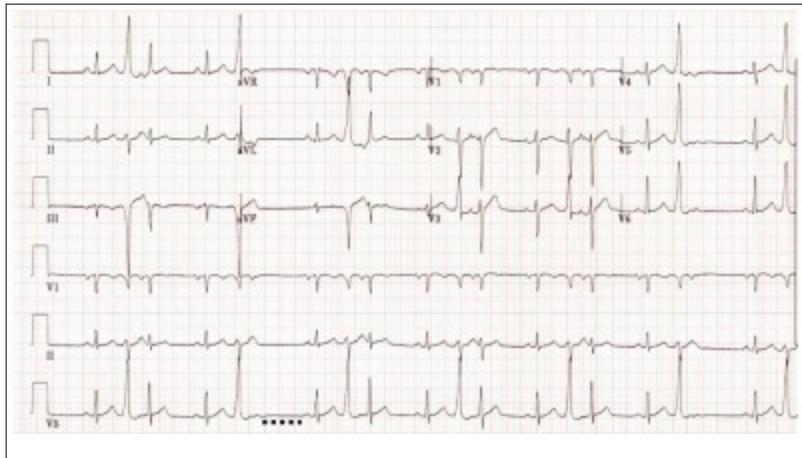
Hypertension in diabetics - ACE inhibitors/A2RBs are first-line regardless of age

This patient has stage 1 hypertension as defined by NICE. He should however be treated because he has underlying diabetes.

The first-line treatment for a patient aged > 55 years is a calcium channel blocker. However, in patients with diabetes ACE inhibitors are used first-line due to their renoprotective effect.

Question #152

A 58-year-old woman presents to the emergency department complaining of palpitations for half an hour. She has been diagnosed with diabetes, heart failure and depression and is on metformin, dapagliflozin, losartan, digoxin and sertraline. She reports experiencing abdominal cramps and loose stools over the last two days, which began following dinner at a restaurant. Her heart rate is 103 beats per minute, her BP is 100/70 mmHg, and respiratory rate is 20 breaths/min. An ECG was taken:



What is the most likely cause of the findings on the ECG?

- a) Anxiety
- b) Dapagliflozin
- c) Digoxin
- d) Metformin
- e) Sertraline

The correct answer is **digoxin**. The electrocardiogram (ECG) presented demonstrates premature ventricular complexes (PVCs), which are characterised by abnormally large QRS complexes that disrupt the underlying sinus rhythm. A typical PVC is identifiable on an ECG as a broad QRS complex, exceeding 120 milliseconds in duration, with an abnormal morphology. It presents prematurely and is associated with discordant ST segment and T wave changes, followed by a compensatory pause prior to the next normal sinus impulse. Digoxin toxicity is recognised for inducing a spectrum of arrhythmias that may be life-threatening. Arrhythmias associated with digoxin toxicity include paroxysmal atrial tachycardia, atrioventricular (AV) blocks, junctional rhythms, and ventricular arrhythmias such as PVCs and ventricular tachycardia. Notably, PVCs are often the initial indicator of digoxin toxicity and represent the most frequent arrhythmia linked to elevated serum digoxin levels. In this case, it is probable that hypokalaemia induced by

diarrhoea has precipitated the digoxin toxicity.

Anxiety can lead to the emergence of PVCs; however, it is not deemed the primary cause in this clinical scenario. Anxiety-related PVCs are thought to originate from sympathetic nervous system overactivity. Nonetheless, given the circumstances described above, digoxin toxicity remains a more plausible explanation for this patient's presentation.

Metformin is not associated with arrhythmias when used within therapeutic dosages. Metformin toxicity might arise in contexts such as renal impairment or intentional overdose and could result in severe lactic acidosis that may precipitate arrhythmias. Despite this possibility, hypokalaemia secondary to diarrhoea resulting in digoxin toxicity appears to be a more convincing cause for the observed PVCs.

Sertraline, a selective serotonin reuptake inhibitor (SSRI), is utilised primarily in managing depression and does not align with this clinical picture as a causative agent for arrhythmia. Although sertraline is among the preferred antidepressants following myocardial infarction (MI), there exists no direct association between sertraline use and arrhythmic events. It should be noted that certain psychiatric medications such as clozapine, chlorpromazine and thioridazine have been implicated in arrhythmic side effects.

Dapagliflozin stands incorrect for this question. As an SGLT-2 inhibitor prescribed for heart failure and diabetes management, dapagliflozin has been reported to reduce incidents of significant ventricular arrhythmias including cardiac arrest or sudden death; thus it would be improbable for dapagliflozin to account for the clinical findings described here.

Question #153

A 72-year-old man is reviewed in the cardiology outpatient department with worsening shortness of breath, which now occurs at rest. He has a past medical history of heart failure with reduced ejection fraction and ischaemic heart disease. His medications include bisoprolol, ramipril, and spironolactone.

On examination, he is clinically euvolemic.

An ECG demonstrates a QRS of 140ms and a left bundle branch block.

What intervention is most likely to improve the patient's symptoms?

- a) Amiodarone
- b) Biventricular pacemaker
- c) Dual chamber pacemaker
- d) Implantable cardiac defibrillator
- e) Single chamber atrial pacemaker

Cardiac resynchronization therapy can be used in patients with a QRS duration of >130 msec and LBBB morphology to improve symptomatology

Biventricular pacemaker is correct. Cardiac synchronization therapy (CRT) with a biventricular pacemaker has been shown to improve heart failure symptoms in those patients with a QRS duration > 130ms and LBBB morphology, inadequately controlled on optimal medical therapy. He has NYHA class IV heart failure as he has symptoms at rest. Typically, leads of this pacemaker are placed in both ventricles and often one in the right atrium in addition. They are designed to help the heart contract more efficiently.

Amiodarone is incorrect. This is typically used to control abnormal tachyarrhythmias. It has not been shown to improve symptoms in patients with chronic heart failure.

Dual chamber pacemaker is incorrect. This type of pacemaker paces both the right atrium and right ventricle. It is indicated in conditions such as high-grade atrioventricular block rather than advanced heart failure as in this patient.

Implantable cardiac defibrillator is incorrect. This is typically indicated in patients with serious ventricular arrhythmias at risk of sudden death. It can be used for some patients with NYHA class I-III heart failure and a prolonged QRS 120-149ms

but without LBBB. However, if there is an LBBB morphology as in this case then a CRT is preferred to improve the symptoms of heart failure.

Single chamber atrial pacemaker is incorrect. This is a pacemaker that usually paces just the right atrium. This type of pacemaker has largely fallen out of favour as they require intact atrioventricular conduction and do not safeguard against this failure in the future. Typically, this type of pacing would only be used for severe symptomatic sinus bradycardia or sick sinus syndrome. It would not result in the required synchronization of ventricular contraction which would result in improved symptoms of heart failure.

Discuss (2)Improve

Question #154

A 68 year-old man with a history of ischaemic heart disease and type 2 diabetes mellitus is 2 days post right curative hemicolectomy for bowel malignancy.

He has developed chest pain and shortness of breath. The surgical team have requested an urgent medical review.

On examination the patient appears distressed and is complaining of chest pain and breathlessness. Capillary refill time is 5 seconds centrally. The pulse rate is 200bpm and the blood pressure is 87/45mmHg. Oxygen saturations are 92% on fIO₂ 0.4 via Venturi mask.

There are crackles to the midzones bilaterally on chest auscultation.

A 12-lead ECG reveals a regular broad complex tachycardia with a monomorphic waveform.

Blood results from the morning reveal:

Hb	129 g/l
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Platelets	564 * 10 ⁹ /l
WBC	15.8 * 10 ⁹ /l
Na ⁺	128 mmol/l
K ⁺	3.3 mmol/l
Urea	12.9 mmol/l
Creatinine	101 µmol/l
Bilirubin	27 µmol/l
ALP	125 u/l
ALT	34 u/l
Albumin	36 g/l

What is the most appropriate initial management?

- a) Adenosine 6mg iv
- b) Amiodarone 300mg IV
- c) Magnesium sulphate 2g IV
- d) Synchronised DC shock
- e) Metoprolol 5mg IV

In the context of a tachyarrhythmia, a systolic BP < 90 mmHg → DC cardioversion

This question outlines a case of ventricular tachycardia with adverse features including shock and heart failure.

According to the Resuscitation Council guidelines, if a patient with a tachyarrhythmia is unstable (i.e. has adverse features likely to be caused or made worse by the tachycardia - shock, syncope, heart failure or myocardial ischaemia) synchronised cardioversion is the treatment of choice.

If cardioversion fails to restore sinus rhythm, and the patient remains unstable, the next step is to give amiodarone 300 mg IV over 10-20 min and re-attempt electrical cardioversion. The loading dose of amiodarone may be followed by an infusion of 900 mg over 24 h.

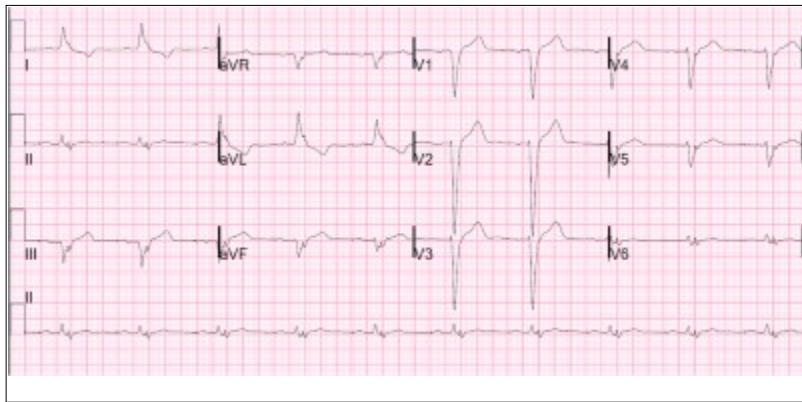
If the patient is conscious, cardioversion should be carried out under sedation or general anaesthesia in the presence of an anaesthetist.

Question #155

A 49-year-old man presents to the emergency department of a tertiary centre with a 4-hour history of central chest pain. There is no radiation of pain, but he reports to be feeling breathless and light-headed. Other than a recent viral upper respiratory tract infection, he has been well in himself recently. His past medical history includes hypertension, hyperlipidaemia and a post-operative deep vein thrombosis 2 years ago. Current medications include ramipril, amlodipine and atorvastatin and he denies any allergies.

On examination, he appears clammy to touch. His chest is clear on auscultation, although he is mildly tachypnoeic with saturations of 94%. Heart sounds are normal and his peripheral pulses are intact. He has a soft and non-tender abdomen.

An ECG is recorded, as shown below:



Given the likely diagnosis, what is the most appropriate next step in the management of this patient?

- a) Discharge with NSAIDs
- b) Fibrinolysis
- c) Insulin and dextrose infusion
- d) Percutaneous coronary intervention
- e) Rivaroxaban

This patient's ECG shows a left bundle branch block (LBBB) with broadened QRS complexes (> 120ms), a dominant S wave in V1 (creating 'W' impression) and broad monophasic R waves in leads I, aVL, V5 and V6 (creating 'M' impression). In patients with an ECG showing LBBB, it is important to establish whether or not this is a new finding as a new presentation of LBBB is concerning for myocardial ischaemia. In combination with a history of chest pain, shortness of breath, and being clammy to touch, it is likely that this patient is having a myocardial infarction. For patients presenting to the hospital within 12 hours of a STEMI with the availability of primary percutaneous coronary intervention (PCI) within 2 hours, PCI is the most appropriate and definitive management of this patient where the obstructed artery can be stented. As this patient has presented within 4 hours to a tertiary centre, it is likely that PCI can be achieved and is, therefore, the correct answer.

It would be inappropriate to discharge this patient with analgesia as a new finding of LBBB and a clinical history of chest pain is highly suggestive of myocardial

ischaemia and needs urgent treatment.

Fibrinolysis is an alternative option for managing acute coronary syndrome. However, if PCI is available, it should be prioritised as the most appropriate treatment.

Insulin and dextrose are used in the management of hyperkalaemia. ECG changes in hyperkalaemia commonly include peaked T waves. However, in severe hyperkalaemia, ECG changes can include bundle branch blocks including LBBB. This is much rarer than myocardial ischaemia and given the history of chest pain, shortness of breath and sweating, an acute coronary syndrome is the more likely cause for which PCI is indicated.

Rivaroxaban is used in the treatment of a pulmonary embolism (PE). Although this patient has a higher risk of venous thromboembolism with a previous DVT, it is very unlikely for a PE to cause LBBB.

Question #156

A 26-year-old patient comes into the Emergency Department in cardiac arrest. The paramedics tell you that she was pulled out of a lake by friends after she had suddenly become unresponsive while swimming. Her friends started cardiopulmonary resuscitation (CPR) at the scene. On arrival, the paramedics noted that the patient was in Ventricular Fibrillation (VF). The patient was defibrillated, given IV amiodarone and adrenaline. Her friends tell you she has no significant past medical history and was always fit and well until this point.

Despite continued attempts to resuscitate the patient, she dies in the Emergency Department. A post-mortem is carried out which is normal.

What is the most likely diagnosis?

- a) Hypertrophic Obstructive Cardiomyopathy (HOCM)
- b) Long QT1 Syndrome
- c) Long QT3 Syndrome
- d) Right Arrhythmogenic Ventricular Dysplasia (RAVD)
- e) Wolf-Parkinson White Syndrome

All of these differentials can result in sudden cardiac death in a young patient. However, you have been told here that the patient's postmortem was normal which makes HOCM and RAVD unlikely.

This leaves Long QT1 Syndrome, Long QT3 Syndrome and Wolf-Parkinson-White as possible differentials. The big clue here is that the patient became unresponsive when swimming which is typical of Long QT1 Syndrome.

Long QT syndromes can result in sudden cardiac death when patients are exposed to an adrenergic surge which can put the patient into VF or VT.

Long QT1: adrenergic surge due to intense physical activity such as swimming

Long QT2: adrenergic surge due to intense emotion such as excitement or fear

Long QT3: death during sleep

Question #157

An 84-year-old presents to the emergency department with and acute deterioration in shortness of breath on a background of 9 months of progressively reduced exercise tolerance. Her past medical history includes hypertension, previous myocardial infarctions 7 months ago, chronic kidney disease and COPD. She has a 50 pack year smoking history giving up 2 years ago. Her exercise tolerance is 150 yards, limited by shortness of breath.

On examination, an ejection systolic murmur can be heard in the aortic area with bibasal crackles on chest auscultation. An admission chest x-ray is consistent with pulmonary oedema and intravenous diuresis is commenced. A transthoracic

echocardiogram demonstrates ejection fraction of 33% with impaired left ventricular function, no vegetations, a bicuspid heavily calcified aortic valve with an area of 0.7cm² and a peak gradient of 32mmHg. Angiography demonstrates non-flow limiting stenosis of 65% in the left anterior descending artery but no lesions requiring revascularisation. Lung function testing following successful diuresis reveals forced vital capacity at 55% of predicted and forced expiratory volume in 1 second at 48% of predicted.

The patient is keen for a definitive intervention if appropriate. What is the appropriate next action?

- a) No action
- b) Balloon aortic valvuloplasty
- c) Aortic valve replacement
- d) Transcatheter aortic valve implantation
- e) Coronary artery bypass graft and aortic valve replacement

The patient has presented with pulmonary oedema on a background of severe aortic stenosis, secondary to calcifications and a bicuspid valve. Interestingly, this is a patient who presents with low-gradient low-area aortic stenosis not uncommonly encountered in more elderly patients. In the context of poor LV function, the aortic valve gradient may be normal or only mildly raised in the presence of a severely narrowed aortic valve area.

A second consideration is the suitability of this patient for surgery. She has underlying lung disease secondary to COPD and functional impairment on pulmonary testing, renal impairment and recent myocardial infarction. She is a high-risk surgical candidate and unlikely suitable for aortic valve replacement. Inaction is not an option, the patient has presented with pulmonary oedema and underlying exercise-induced dyspnoea. Balloon aortic valvuloplasty is a palliative procedure prone to restenosis for patients unsuitable for other interventions. She does not require a CABG for her coronary artery disease, which worryingly seems inconsistent with the degree of left ventricular impairment, suggesting aortic stenosis to likely be responsible for the majority of symptoms. A transcatheter

aortic valve implant (TAVI) is the appropriate management in this situation. The catheter-delivered device produces similar one-year survival as aortic valve replacement but a higher risk of stroke, TIAs and vascular complications. Compared to medical management alone, one-year survival is reduced with TAVI from 50.7% to 30.7%¹.

1. Thomas M, Schymik G, Walther T et al. One-year outcomes of cohort 1 in the Edwards SAPIEN Aortic Bioprostheses European Outcome (SOURCE) registry: the European registry of transcatheter aortic valve implantation using the Edwards SAPIEN valve. *Circulation*. 2011 Jul;124(4):425-33. Epub 2011 Jul 11.

Question #158

A 52-year-old lady was admitted with sudden onset central chest pain and breathlessness. Her past medical history included hypertension, atrial fibrillation and she was a smoker of 20 cigarettes a day. She lived alone after having recently split up with her husband which had been a difficult few weeks. Her friend had been staying with her occasionally during this difficult period.

She had a family history of ischaemic heart disease. She had recently complained to her friend of a cold that was now passing. Medications included aspirin 75mg OD and bisoprolol 5mg OD. Her friend called 999 and she was admitted directly to the angiogram suite as the paramedics noticed that her ECG showed ST elevation in the anterior chest leads.

Her angiogram showed mild coronary atherosclerosis but an akinetic left ventricle. Her troponin T was significantly elevated at 7800ng/L (normal < 14).

What is the most likely diagnosis?

- a) Pericarditis
- b) Myocardial infarction
- c) Embolic event
- d) Takotsubo cardiomyopathy
- e) Pneumothorax

Takotsubo cardiomyopathy (also known as broken heart syndrome, apical ballooning syndrome, acute stress cardiomyopathy) is non-ischaemic cardiomyopathy where there is a temporary weakening of the heart muscle. This is often triggered by emotional stress or physical stress. Takotsubo cardiomyopathy is more commonly seen in post-menopausal women. Patients most commonly present with chest pain and shortness of breath. Sudden onset congestive cardiac failure may also occur.

On initial presentation diagnosis of Takotsubo cardiomyopathy may be difficult and patients are often treated as an acute myocardial infarction. ECGs classically mimics ST-segment elevation myocardial infarction. Patients often proceed acutely to an angiogram which shows the absence of significant heart disease. Cardiac enzymes are significantly raised.

An echocardiogram may demonstrate pathognomonic wall motion abnormalities, in which the base of the left ventricle is contracting normally or is hyperkinetic while the remainder of the left ventricle is akinetic or dyskinetic. This coupled with the lack of significant coronary artery disease that would explain the wall motion abnormalities allows the diagnosis to be made.

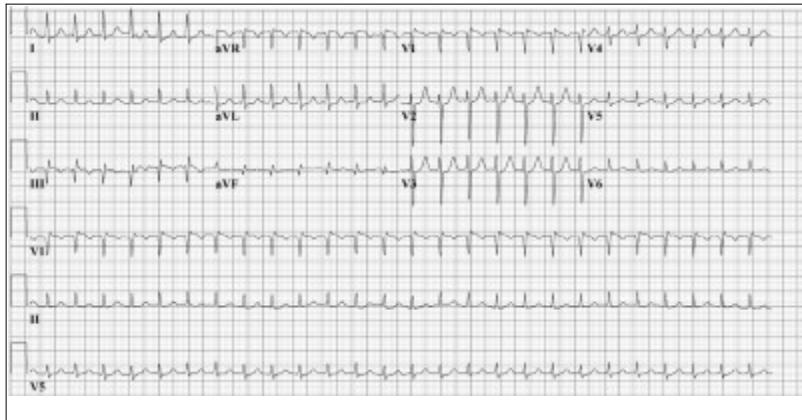
Treatment of Takotsubo cardiomyopathy is generally supportive. Patients may be hypotensive, however, treatment with inotropes often worsens the disease. Patients may require intra-aortic balloon pump, fluids, beta blockers or calcium channel blockers. Aspirin is also commonly used in the management. Once over the initial event outcomes are favourable. Even when ventricular systolic function is heavily compromised at presentation, it typically improves within the first few days and normalises within the first few months.

Question #159

A 41-year-old woman attends the emergency department with a 6-hour history of palpitations and breathlessness. She denies chest pain or recent illness. Her past medical history includes type 1 diabetes and she smokes 15 cigarettes/day.

On examination, she has a heart rate of 140bpm with a blood pressure of 122/77mmHg. Peripheral pulses are present bilaterally with a capillary refill of < 2 seconds. On auscultation, her heart sounds are normal with vesicular breath sounds throughout.

An ECG is taken, as shown below:



What is the most likely diagnosis?

- a) Atrial flutter
- b) Sinus tachycardia
- c) Supraventricular tachycardia
- d) Ventricular tachycardia
- e) Wolff-Parkinson White syndrome

This patient's ECG shows a regular, narrow-complex tachycardia in keeping with supraventricular tachycardia (SVT). Given the absence of clear P waves, it is likely that the cause of this SVT is an atrioventricular nodal re-entry tachycardia (AVNRT) secondary to a re-entry circuit causing premature ventricular contraction. SVT is

more common in women with a heart rate varying from between 140-280bpm. Symptoms include palpitations and breathlessness, as seen here, and may occur spontaneously or following provocation with caffeine, drugs or alcohol.

Atrial flutter is a form of supraventricular tachycardia caused by a re-entry circuit into the right atrium. This typically causes an atrial rate of 300bpm. The ventricular rate is determined by the degree of the atrioventricular block at the atrioventricular node e.g. a 2:1 block would result in a heart rate of 150bpm, whilst a 3:1 block would give a rate of 100bpm. Symptoms include palpitations or shortness of breath, whilst common ECG findings include a narrow complex tachycardia with inverted flutter waves in leads II, III and aVF, giving rise to a saw-tooth appearance on the baseline of the ECG. These are not seen here, making it a less likely diagnosis.

Sinus tachycardia is a common ECG finding in patients presenting with palpitations. However, the absence of discernable P waves makes sinus tachycardia less likely.

Ventricular tachycardia presents as a broad-complex tachycardia with widened QRS complexes. This ECG shows a narrow-complex tachycardia, making ventricular tachycardia unlikely.

Wolff-Parkinson White (WPW) syndrome causes a tachyarrhythmia secondary to an accessory excitatory pathway stimulating premature ventricular contraction. WPW syndrome may present as palpitations, as seen in this patient. However, the ECG findings in Wolff-Parkinson White include a shortened PR interval and a slurred upstroke of the QRS complex (delta wave) causing QRS interval prolongation which is not seen here.

Question #160

A 47-year-old woman presents to an endocrinology clinic with a history of hypertension. She was first diagnosed with hypertension following a private health assessment 8 years ago and at that time underwent testing to exclude any secondary causes of her hypertension. She was subsequently commenced on ramipril, which was up titrated to 10mg. When her blood pressure remained

above 150/100mmHg, her GP added amlodipine 10mg and then bendroflumethiazide 2.5mg. During a recent medication review, the patient's blood pressure was noted to be 170/100mmHg. Her GP thus referred her to the endocrine team for a reconsideration of secondary hypertension. On questioning, she denies any chest pain, palpitations, change in vision, or symptoms consistent with postural hypotension. She has no family history of hypertension, does not smoke and drinks minimal alcohol.

On clinical examination, heart sounds are normal and chest clear, abdomen soft and non-tender with no organomegaly.

Her test results are as follows:

Na ⁺	137 mmol/L	(135 - 145)
K ⁺	4.6 mmol/L	(3.5 - 5.0)
Bicarbonate	25 mmol/L	(22 - 29)
Urea	3.0 mmol/L	(2.0 - 7.0)
Creatinine	68 µmol/L	(55 - 120)
Hba1c	48 mmol/mol	(42-47)

CT chest / abdomen / pelvis shows no abnormalities.

Echocardiogram demonstrates left ventricular hypertrophy only.

Urine dip:

Blood	-
-------	---

Protein	-
Glucose	Trace

Given the diagnosis, which of the following agents would be most appropriate?

- a) Doxazosin
- b) Spironolactone
- c) Furosemide
- d) No indication for further anti-hypertensive agents
- e) Candesartan

Poorly controlled hypertension, already taking an ACE inhibitor, calcium channel blocker and a standard-dose thiazide diuretic. $K^+ > 4.5\text{mmol/l}$ - add an alpha- or beta-blocker

This patient has resistant hypertension, that is to say, hypertension which has not responded to three appropriately dosed anti-hypertensive agents. Effective treatment is needed given her evidence of end-organ damage (left ventricular hypertrophy).

Given that her potassium is > 4.5 , spironolactone would not be recommended.

Furosemide would not be recommended in a patient without evidence of heart failure prior to the trial of alpha blockade.

Candesartan should not be co-prescribed with ramipril.

Doxazosin (an alpha-blocker) or alternatively a beta-blocker, would be the most appropriate 4th line antihypertensive agent.

Question #161

A 39-year-old male is admitted with sharp central chest pain which is worse on inspiration. He states that the pain is partially relieved by leaning forward. He has no past medical history and takes no regular medicines.

ECG results are as follows:

PR segment	Depression
ST segment	Global concave ST elevation

Blood results are as follows:

Troponin	9.82 ug/l (normal range 0.015 - 0.045)
----------	--

What investigation is most useful diagnostically?

- a) Cardiac magnetic resonance imaging
- b) Serial troponins
- c) Transesophageal echo
- d) Myocardial Perfusion Imaging (MPI)
- e) Serial ECGs

Cardiac magnetic resonance imaging has been shown to be very useful in diagnosing myocarditis by visualising markers for inflammation of the myocardium

The patient has clinical and electrocardiographic features of pericarditis. Although a small rise in troponin can be expected in pericarditis, a significant rise should alert you to the possibility of myopericarditis.

Cardiac magnetic resonance imaging has been shown to be very useful in diagnosing myocarditis by visualising markers for inflammation of the myocardium

e.g. regional myocardial oedema, hyperaemia and inflammatory necrosis.

All the other options may be useful, however the most sensitive investigation for diagnosing myopericarditis is cardiac MRI.

Question #162

A 56-year-old lady with previously diagnosed rheumatic heart disease with mitral stenosis attends for her annual review. She remains fit and well and can walk 100 metres before she develops breathlessness. She gets occasional burning pains in her chest that come on after rich meals and last 20-30 minutes.

She currently takes aspirin, ramipril and a statin. She does not smoke and is taking regular walks each day. On examination, she has a loud S1 heart sound with a mid-diastolic murmur heard in full expiration when in the left lateral position. Her face has a red flush in the cheeks and there is no JVP visible. Her chest is clear and there is no oedema.

Hb	104 g/l	Na ⁺	139 mmol/l
Platelets	450 * 10 ⁹ /l	K ⁺	5.2 mmol/l
WBC	5.6 * 10 ⁹ /l	Urea	4.5 mmol/l
Neuts	4.5 * 10 ⁹ /l	Creatinine	78 µmol/l
Lymphs	0.3 * 10 ⁹ /l	CRP	14 mg/l
Troponin	negative		

ECG	sinus rhythm, left ventricular hypertrophy
-----	--

Chest X-ray	enlarged cardiac shadow with loss of the aorto-pulmonary window
ECHO (today)	mitral valve cross sectional area 0.8cm^2 , LV ejection fraction 60%
ECHO (1yr ago)	mitral valve cross sectional area 1.1cm^2 , LV ejection fraction 62%

What is the most appropriate intervention?

- a) Refer to cardiothoracic surgery
- b) Furosemide
- c) Routine follow up with ECHO in 1 year
- d) Repeat ECHO in 6 months
- e) Coronary angiogram

Mitral stenosis with cross sectional area of $<1\text{cm}^2$ warrants surgical intervention

This lady has known mitral stenosis. She has a valve cross sectional area of less than 1cm^2 which is an indication for mitral valve surgery and should be referred immediately. Furosemide would not benefit her as there is no sign of overload. She could continue ECHO surveillance but this will not change her situation. The burning chest pains do not sound cardiac in nature and it would probably be worth trialling omeprazole.

Question #163

A 73-year-old gentleman was shown to have left ventricular dysfunction and hypertrophy on an echocardiogram post acute myocardial infarction (MI). Prior to discharge, he was started on ramipril 2.5mg, bisoprolol 2.5mg, atorvastatin 10mg and aspirin 75mg. His medical history included osteoporosis for which he took Calcichew D3 forte and alendronic acid. He did not smoke or drink alcohol. On admission his kidney function showed the following:

Sodium	136 mmol/L
Potassium	3.7 mmol/L
Urea	7.0 mmol/L
Creatinine	120 micromol/L
eGFR	64 ml/min/1.73m ²

His GP checked his bloods again ten days after the initiation of his new medication to titrate the dose of ramipril as requested on the discharge letter from the cardiology team. The results are below:

Sodium	134 mmol/L
Potassium	4.2 mmol/L
Urea	8.0 mmol/L
Creatinine	156 micromol/L
eGFR	50 ml/min/1.73m ²

How would you manage this gentleman's deteriorating renal function?

- a) Continue the ramipril and repeat the bloods in one week
- b) Decrease the dose of ramipril to 1.25mg daily
- c) Stop the ramipril
- d) Switch the ramipril to losartan

- e) Request a renal ultrasound scan

NICE guidelines are useful for everyday practice like managing chronic kidney disease (CKD). Knowing the major points about recent guidelines will also help you in your revision for exams. Guideline 73 is about the diagnosis and management of CKD, the link is below.

It is useful for staging CKD and guiding typical frequency of blood monitoring. In our gentleman's case, his renal function deteriorates due to starting ramipril. NICE guidance says it is acceptable to have a 25% reduction in eGFR OR a 30% rise in creatinine after starting an ACE inhibitor or angiotensin receptor blocker.

Question #164

A 43 year old man has been referred to endocrinology clinic for blood pressure investigation and management. Over the past year the patient has had a persistently raised blood pressure between 170/100mmHg and 180/110mmHg. Despite starting the patient on ramipril 5mg once daily four weeks ago the patient's blood pressure on this visit remained raised at 164/93mmHg.

During the history taking part of the consultation the patient mentions that he has been troubled with headaches for the past year, and has noticed that his stool frequency has increased and his stools have become looser. He tells you that he's rather embarrassed to admit that he's been having flushing episodes, and feels that his clothes are much looser than they were 1 year ago.

On discussing his family history he mentions that both his mother and his father had to have cancerous lumps removed in their middle ages. His mother had a breast lump removed, and his father had some form of pancreatic mass removed.

On examination the patient is tall with a wide arm span. Examination of the cardiovascular system reveals only a minor tachycardia of 95bpm and a quiet systolic flow murmur not radiating to the carotids. Abdominal examination reveals no palpable masses, and examination of his lungs is completely normal.

The GP had previously arranged a 24h urinary catecholamine test; results are as follows:

Total urine catecholamines 210mcg/24hr. This is raised.

As a follow up to this test the GP had arranged a CT of the patient's abdomen and pelvis which is reported as normal apart from a few incidental simple renal cysts.

Urinalysis in clinic today shows:

Leucocytes	-
Blood	-
Glucose	+
Ketones	-

Which test is most likely to elucidate the cause of the patient's hypertension?

- a) Pentagastrin stimulation test
- b) 24h urinary HIAA
- c) MRI renal angiography
- d) GH, IGF-1 and 'gut hormones'
- e) MIBG (metaiodobenzylguanidine) scan

This question tests the candidates knowledge of causes of secondary hypertension. The commonly listed causes of secondary hypertension are listed below:

Endocrine	Adrenal	Renal	Cardiovascular
Cushing's Syndrome	Conn's Syndrome	Diabetic nephropathy	Aortic

Endocrine	Adrenal	Renal	Cardiovascular
			dissection
Acromegaly	Adrenal hyperplasia	Chronic glomerulonephritis/tubulointerstitial nephritis	-
Thyroid disease	Phaeochromocytoma	Adult polycystic kidney disease	-
Hyperparathyroidism	-	Renovascular disease	-
-	-	-	-

The best way of narrowing down to the correct answer in this question is to actually look at what the answers are suggesting:

Pentagastrin stimulation test	Test for medullary carcinoma of the thyroid (associated with MEN2 - multiple endocrine neoplasia)
24h Urinary HIAA	Screening for carcinoid
MRI renal angiography	Test for renal artery stenosis
GH, IGF-1 and 'gut hormones'	Test for pancreatic malignancy associated with MEN1
MIBG scan	Test for phaeochromocytoma

While this patient might well have MEN2 (as suggested by the patients body habitus and features consistent with phaeochromocytoma), medullary thyroid

cancer would be unlikely to cause his blood pressure issues. Therefore the pentagastrin stimulation test, where one injects the patient with pentagastrin and looks for an abnormally high release of calcitonin as a response, is not a suitable first test. It may well be considered if the patient is diagnosed with a phaeochromocytoma to rule out medullary thyroid cancer as part of MEN 2.

The patient does have a few features consistent with carcinoid syndrome, for example the episodic flushing and increased stool frequency. However carcinoid syndrome doesn't present with these symptoms until it has metastasised to the liver, and this patient has had a normal CT scan of his abdomen making this a less likely diagnosis.

MRI renal angiography is used to diagnose renal artery stenosis; this patient has far too many other endocrine features for this to be renal artery stenosis. In addition the fact his blood pressure has not got worse with angiotensin converting enzyme inhibitors makes the diagnosis less likely.

Testing for gut hormones would be done in a patient in whom you were considering a diagnosis of pancreatic neoplasm as part of MEN1.

This leaves the MIBG (metaiodobenzylguanidine) scan. The patient has raised levels of total urinary catecholamines, suggesting a diagnosis of phaeochromocytoma, however, his CT scan shows no evidence. As you may be aware there is a rule of 10 that is commonly used to describe phaeochromocytoma, in that:

- 10% are malignant
- 10% are bilateral
- 10% are familial
- 10% are in children
- 10% are extra-adrenal

The other sites that phaeochromocytomas can present include:
Anywhere along the sympathetic chain which runs alongside the spinal cord, the distal aorta, ureters and urinary bladder.

The MIBG scan uses radioactive iodine as a tracer for phaeochromocytoma tumour cells, which is detected through the use of a gamma camera., making it the next test that should be used in diagnosing this patient's phaeochromocytoma, i.e. the cause of his hypertension.

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Question #165

A 48-year-old female presents with chest pain. She describes the pain as central, dull and tight. She has associated nausea. She has no past medical history and takes no regular medicines.

An ECG is performed:

P waves	Normal morphology
PR interval	130ms
QRS	115ms
ST segments	ST elevation in V1-V3

Blood results are as follows:

Troponin (on admission)	< 0.015 µg/l (normal < 0.015)
Troponin (6 hours later)	< 0.015 µg/l (normal < 0.015)

What is the most likely diagnosis?

- a) Acute STEMI
- b) Acute NSTEMI
- c) Unstable angina
- d) Stable angina
- e) Prinzmetal's angina (variant angina)

In coronary vasospasm (Prinzmetal's angina, or variant angina), the ECG shows ST elevation that is very similar to an acute STEMI. However, unlike acute STEMI the ECG changes are transient, reversible with vasodilators and not associated with myocardial necrosis

The presence of ST elevation and chest pain should alert you to the diagnosis of acute STEMI. However importantly the serial troponins are negative effectively ruling out myocardial infarction. In coronary vasospasm (Prinzmetal's angina, or variant angina), the ECG shows ST elevation that is very similar to an acute STEMI. However, unlike acute STEMI the ECG changes are transient, reversible with vasodilators and not associated with myocardial necrosis. Prinzmetal's angina is therefore the most likely diagnosis.

Acute NSTEMI is excluded by the normal serial troponins.

Angina pectoris (stable or unstable) is of course a possibility. However the presence of ST elevation, the relatively young age of the patient, and the absence of cardiovascular risk factors, makes Prinzmetal's angina the more likely diagnosis.

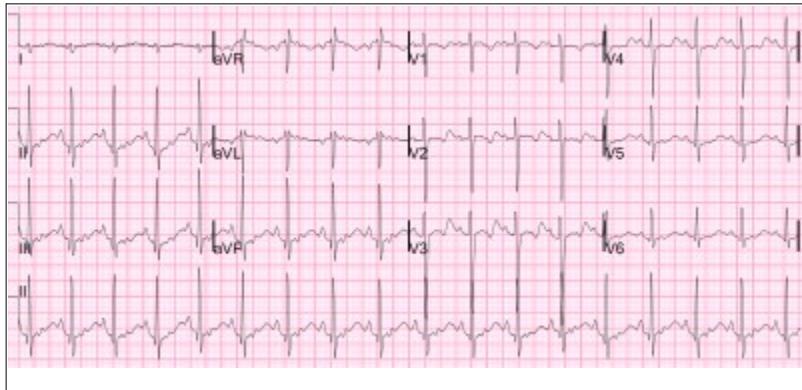
The patient will need coronary angiography. The gold standard test for diagnosing variant angina is to visualise the coronary arteries by angiography before and after injection of a provocative agent such as acetylcholine to precipitate an attack of vasospasm

Question #166

A 22-year-old woman presents to the emergency department with syncope. She also states that she has had profuse vomiting and diarrhoea for the last 3 days. Observations are as follows: heart rate 100 bpm, blood pressure 100/60 mmHg,

respiratory rate 18 breaths per minute, SpO₂ 98% on air, temperature 37.2 °C.

An ECG is performed in the department:



What is the likely cause of this ECG abnormality?

- a) Arrhythmogenic right ventricular dysplasia (ARVD)
- b) Hyperkalaemia
- c) Hypokalaemia
- d) Hypothermia
- e) Wolff-Parkinson-White syndrome

The ECG demonstrates sinus rhythm with a rate of approximately 100 beats per minute. There are prominent U waves following the T wave.

Hypokalaemia is correct. The electrocardiographic features are most in keeping with hypokalaemia. U waves are present and classic of hypokalemia, however, it is important to remember that they can be present in other conditions (e.g. bradycardia, hypocalcaemia, hypomagnesaemia, hypothermia and raised intracranial pressure). Other ECG features of hypokalaemia include small or absent T waves (occasionally inversion), prolonged PR interval, ST depression, and long QTc.

Arrhythmogenic right ventricular dysplasia (ARVD) is incorrect. ARVD is characterised by an epsilon wave which is a small deflection buried at the end of the QRS complex usually best seen in the ST segment of V1 and V2.

Hyperkalaemia is incorrect. ECG features of hyperkalaemia include peaked T waves, P wave widening/flattening, PR prolongation, bradyarrhythmias, and QRS widening with bizarre QRS morphology.

Hypothermia is incorrect. Hypothermia classically presents with sinus bradycardia and Osborn waves (J wave) defined as positive deflection seen at the J point in precordial and true limb leads. Osborn waves are characteristically seen in hypothermia, however, they are not pathognomonic and can be seen in other conditions such as hypercalcaemia and myocarditis.

Wolff-Parkinson-White syndrome is incorrect. This condition is associated with a short PR interval (< 120ms), broad QRS (> 100ms), and a slurred upstroke to the QRS complex (the delta wave).

Question #166

An 82-year-old man presents with loss of consciousness. His wife states that he complained of feeling dizzy, before going pale in colour and falling to the ground. He was unconscious for approximately one minute. On regaining consciousness he was not confused and recovered within minutes. He denies any chest pain, shortness of breath or palpitations. He states that over the past few months he has had several episodes of light headedness however he has never lost consciousness before.

A 7 day holter was performed:

Heart rate	3 episodes of bradycardia of 20-30 bpm which correlated with episodes of pre-syncope
------------	--

Other events	Absence of P waves for 3.5 seconds
--------------	------------------------------------

How will you manage this patient?

- a) VVI pacemaker
- b) Biventricular pacemaker
- c) AAIR pacemaker
- d) Reassurance
- e) Bisoprolol

In pure sinus node dysfunction without AF or evidence of AV block, an AAIR or DDDR pacemaker can be used. Most cardiologists would choose a DDDR pacemaker since many of these patients go onto develop AV block

The 7 day holter confirms symptomatic sinus bradycardia and an episode of sinus arrest (absence of P wave for > 3 seconds). This confirms the diagnosis of sick sinus syndrome.

Sick sinus syndrome can be difficult to manage, as it can present with bradyarrhythmias, tachyarrhythmias, or a mixture of both. Bradyarrhythmias can be well controlled with pacemakers, while tachyarrhythmias respond well to rate limiting drugs such as calcium channel blockers, digoxin and beta blockers. However drugs to control tachyarrhythmia may exacerbate bradyarrhythmias. Therefore, a pacemaker is often implanted before drug therapy is begun for the tachyarrhythmia.

This patient has been having symptomatic bradycardia episodes secondary to sinoatrial node disease. Therefore an AAIR pacemaker is indicated. AAI pacemakers both sense and pace the atria. They are useful for isolated sinoatrial node disease. These are not suitable in patients with atrial fibrillation.

VVI pacemakers will both sense and pace the ventricle. VVI or VVR pacemakers

are useful for pure sustained slow atrial fibrillation.

Biventricular pacemakers are used in heart failure with left bundle branch block.

Bisoprolol is a rate limiting drug which would worsen the bradycardia and the patients symptoms.

Question #167

A 76-year-old woman presents to the acute medical assessment unit with a two-month history of palpitations. She reports regularly feeling her heart racing, occasionally associated with pre-syncope. She denies dyspnoea, syncope and chest pain.

She has a medical history significant for asthma, for which she takes regular inhaled beclometasone dipropionate and formoterol, and montelukast, with as required salbutamol. There is no other medical history.

Her observations are:

- Respiratory rate: 20/min
- Blood oxygen saturation: 98% on room air
- Heart rate: 116bpm
- Blood pressure: 135/68mmHg
- GCS: 15/15
- Temperature: 37.4°C

An ECG is performed which shows atrial fibrillation. Blood tests are unremarkable. A transthoracic echocardiogram shows no evidence of valvular heart disease and no evidence of congestive cardiac failure.

CHA ₂ DS ₂ -VASc score	
Risk factor	Points
Congestive heart failure	1
Hypertension (or treated hypertension)	1
Age ≥ 75 years	2
Age 65-74 years	1
Diabetes	1
Prior Stroke/TIA/thromboembolism	2
Vascular disease (including ischaemic heart disease and peripheral arterial disease)	1
Female sex	1



What is the most appropriate management?

- a) Apixaban + bisoprolol3
- b) Apixaban + digoxin
- c) Apixaban + diltiazem
- d) Warfarin + bisoprolol
- e) Warfarin + diltiazem

Apixaban + diltiazem is the correct answer. This patient has atrial fibrillation with no valvular abnormalities. Her CHA₂DS₂-VASc score is 3 and therefore qualifies for anticoagulation with a direct-acting oral anticoagulant (DOAC) such as apixaban. If this patient had mitral stenosis, weighed over 120kg, or had a contraindication to a DOAC, warfarin would be considered. This patient would be suitable for rate control rather than rhythm control - the two-month history suggests this is not acute, there is no clear reversible cause and there is no evidence of heart failure. This patient's asthma is a contra-indication for a beta-blocker, as beta-blockers can increase airway reactivity. Calcium channel blockers are second-line for rate control in atrial fibrillation. Therefore apixaban + diltiazem is the correct combination of medications.

Apixaban + bisoprolol is the incorrect answer. Beta-blockers are the first-line treatment for rate control in atrial fibrillation, but are relatively contraindicated in

asthma - given this patient is on fourth-line treatment for her asthma, beta-blockers are best avoided in this scenario.

Apixaban + digoxin is the incorrect answer. Digoxin is the third-line treatment for rate control in atrial fibrillation. Therefore a calcium channel blocker such as diltiazem is more appropriate. Digoxin would be more appropriate if this patient had evidence of heart failure due to the negative inotropic effects of calcium channel blockers.

Warfarin + bisoprolol is the incorrect answer. DOACs, such as apixaban, are used first line when anticoagulation is indicated for atrial fibrillation. Warfarin is recommended if there is evidence of mitral stenosis - however, this patient's echocardiogram has no evidence of valvular disease. As above, diltiazem is more appropriate than bisoprolol due to this patient's asthma.

Warfarin + diltiazem is the incorrect answer. As above, a DOAC is preferable to warfarin in this situation. Diltiazem is the correct drug for rate control in this scenario.

-

Question #167

A 76-year-old woman presents to the acute medical assessment unit with a two-month history of palpitations. She reports regularly feeling her heart racing, occasionally associated with pre-syncope. She denies dyspnoea, syncope and chest pain.

She has a medical history significant for asthma, for which she takes regular inhaled beclometasone dipropionate and formoterol, and montelukast, with as required salbutamol. There is no other medical history.

Her observations are:

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An ECG is performed which shows atrial fibrillation. Blood tests are unremarkable. A transthoracic echocardiogram shows no evidence of valvular heart disease and no evidence of congestive cardiac failure.

CHA ₂ DS ₂ -VASc score		Probability
Risk factor	Points	
Congestive heart failure	1	
Hypertension (or treated hypertension)	1	
Age ≥ 75 years	2	
Age 65-74 years	1	
Diabetes	1	
Prior Stroke/TIA/thromboembolism	2	
Vascular disease (including ischaemic heart disease and peripheral arterial disease)	1	
Female sex	1	

What is the most appropriate management?

- a) Apixaban + bisoprolol
- b) Apixaban + digoxin
- c) Apixaban + diltiazem
- d) Warfarin + bisoprolol
- e) Warfarin + diltiazem

Apixaban + diltiazem is the correct answer. This patient has atrial fibrillation with no valvular abnormalities. Her CHA₂DS₂-VASc score is 3 and therefore qualifies for anticoagulation with a direct-acting oral anticoagulant (DOAC) such as apixaban. If this patient had mitral stenosis, weighed over 120kg, or had a contraindication to a DOAC, warfarin would be considered. This patient would be suitable for rate control rather than rhythm control - the two-month history suggests this is not acute, there is no clear reversible cause and there is no evidence of heart failure. This patient's asthma is a contra-indication for a beta-blocker, as beta-blockers can increase airway reactivity. Calcium channel blockers are second-line for rate control in atrial fibrillation. Therefore apixaban + diltiazem is the correct combination of medications.

Apixaban + bisoprolol is the incorrect answer. Beta-blockers are the first-line treatment for rate control in atrial fibrillation, but are relatively contraindicated in asthma - given this patient is on fourth-line treatment for her asthma, beta-blockers are best avoided in this scenario.

Apixaban + digoxin is the incorrect answer. Digoxin is the third-line treatment for rate control in atrial fibrillation. Therefore a calcium channel blocker such as diltiazem is more appropriate. Digoxin would be more appropriate if this patient had evidence of heart failure due to the negative inotropic effects of calcium channel blockers.

Warfarin + bisoprolol is the incorrect answer. DOACs, such as apixaban, are used first line when anticoagulation is indicated for atrial fibrillation. Warfarin is recommended if there is evidence of mitral stenosis - however, this patient's echocardiogram has no evidence of valvular disease. As above, diltiazem is more appropriate than bisoprolol due to this patient's asthma.

Warfarin + diltiazem is the incorrect answer. As above, a DOAC is preferable to warfarin in this situation. Diltiazem is the correct drug for rate control in this scenario.

Discuss (2)Improve

Question #168

A 43-year-old male presents with central chest pain that spread into his jaw and arm. He is nauseous and sweaty and arrives to the emergency department. He is immediately given aspirin and glyceryl trinitrate spray. On examination, he has normal heart sounds and a clear chest. However, he is pale and clammy

Hb	145 g/l
Platelets	$400 * 10^9/l$
WBC	$8.6 * 10^9/l$

Na ⁺	135 mmol/l
K ⁺	4.3 mmol/l
Urea	4.5 mmol/l
Creatinine	67 µmol/l

Troponin	957 ng/ml (normal range <20)
ECG	Biphasic T waves in V2-V3

What is the likely explanation of the ECG?

- a) Normal variant
- b) Brugada Syndrome
- c) Wellens Syndrome
- d) Hypertrophic cardiomyopathy
- e) Takotsubo cardiomyopathy

Deeply inverted or biphasic T waves in V2-V3 suggests Wellen's syndrome and high risk of critical LAD stenosis

This gentleman has clear acute coronary syndrome. The ECG findings are unique to Wellens Syndrome in which there is a critical stenosis of the left anterior descending artery. These patients are at risk of imminent occlusion of this artery and should be admitted for coronary angiography and further intervention.

Takotsubo cardiomyopathy has ST elevation in the apical leads (V5, V6).

Hypertrophic cardiomyopathy typically has T wave inversion in the anteroseptal leads. Brugada has ST elevation and T wave inversion in V1-V2. Biphasic T-waves are not a normal variant.

Question #169

A 64-year-old man presents to cardiology outpatients with intermittent exertional chest pain. He has suffered from angina for several years and finds his glyceryl trinitrate spray effective, but he is keen on further treatment to help his symptoms.

His past medical history includes angina, asthma, and type 2 diabetes. His current medication includes salbutamol, montelukast, Fostair, metformin, amlodipine, glyceryl trinitrate, aspirin and simvastatin.

Observations are temperature 37°C, heart rate 52 beats/min, blood pressure 98/59 mmHg, respiratory rate 14 breaths/min, and oxygen saturation 96%.

What is the most appropriate additional therapy?

- a) Bisoprolol

- b) Isosorbide mononitrate
- c) Ivabradine
- d) Ranolazine
- e) Verapamil

For a patient with symptomatic stable angina on a calcium channel blocker but with a contraindication to a beta-blocker, the next line treatment should be long-acting nitrate, ivabradine, nicorandil or ranolazine

The correct answer is **ranolazine**. This patient has stable angina despite the use of first-line anti-anginal therapy with amlodipine. NICE recommends using either a beta-blocker or a dihydropyridine calcium channel blocker as first-line anti-anginal.

There are a number of options for next-line treatment for patients on a calcium channel blocker, including the addition of a beta-blocker, long-acting nitrate, ivabradine, nicorandil or ranolazine. In this case, **ranolazine** would be the most appropriate, as the other options have either cautions or contra-indications.

Given that this patient has asthma, it would be best practice to avoid a beta-blocker such as **bisoprolol**, especially if other equally appropriate therapy options are available.

Given that this patient already has a slightly low blood pressure on the candesartan alone, it would be best to avoid adding in **isosorbide mononitrate** as this risks dropping the blood pressure further.

Ivabradine is contra-indicated in bradycardia. The BNF states 'do not initiate for angina if heart rate below 70 beats per minute'.

The patient is already on a dihydropyridine calcium channel blocker, therefore adding a non-dihydropyridine calcium channel blocker such as **verapamil** should be avoided.

Discuss (4)Improve

Question #170

A 72-year-old man attends the emergency department with mild epistaxis. He denies any other bleeding. He has a past medical history of atrial fibrillation and severe mitral stenosis for which he takes warfarin. Observations are as follows: heart rate 80 beats per minute, blood pressure 145/85 mmHg, respiratory rate 14 breaths per minute, SpO₂ 96% (on air), and temperature 37.5°C.

Blood results are as follows:

Hb	144 g/L	Male: (135-180) Female: (115 - 160)
Platelets	$72 * 10^9/\text{L}$	(150 - 400)
WBC	$8.2 * 10^9/\text{L}$	(4.0 - 11.0)

Prothrombin time (PT)	114 secs	(10-14 secs)
INR	10.2	(<1.1)
Activated partial thromboplastin time (APTT)	54 secs	(25-35 secs)
Fibrinogen	3.4 g/L	(2 - 4)

You withhold the patient's warfarin.

What further measures are required?

- a) Fresh frozen plasma
- b) Intravenous 10mg vitamin K
- c) Intravenous 3mg vitamin K
- d) Platelet transfusion
- e) Prothrombin complex concentrate

INR > 8.0 (minor bleeding) - stop warfarin, give intravenous vitamin K 1-3mg, repeat dose of vitamin K if INR high after 24 hours, restart when INR < 5.0

The results of the coagulation screen suggest warfarin toxicity. The INR (derived from the PT) is used to monitor warfarin since it is the most representative, however, it is important to remember that warfarin can also prolong the APTT.

Intravenous 3mg vitamin K is correct. The patient has a minor bleed with an INR >8.0. Thus the warfarin should be withheld and the patient should receive intravenous vitamin K 1-3mg. The warfarin should be restarted when the INR < 5.0.

Fresh frozen plasma is incorrect. Fresh frozen plasma (FFP) is not recommended for use as a warfarin reversal agent as it can take hours to work due to the volume required. In warfarin-associated major haemorrhages, FFP should only be given if prothrombin complex concentrate is unavailable.

Intravenous 10mg vitamin K is incorrect. For minor haemorrhage, the dose of intravenous vitamin K recommended is 1-3mg.

Platelet transfusion is incorrect. In the context of haemorrhage, a platelet level of <50 is the standard transfusion trigger, with the exclusion of CNS or posterior eye bleeds in which the transfusion trigger is <100. The patient in this clinical case has a platelet count of 72 and thus does not require a platelet transfusion.

Prothrombin complex concentrate is incorrect. Prothrombin complex concentrate (PCC) is recommended only for major bleeding. PCC will reverse anticoagulation

within minutes of administration however administration of vitamin K is also required to counteract the long half-life of warfarin.

Question #171

A 66-year-old lady attended a cardiology clinic for follow up after a recent hospital admission with pulmonary oedema. She continued to experience significant shortness of breath at rest with exercise tolerance that was limited to a few steps around the house. She had a previous medical history of hypertension, diabetes and myocardial infarction 5 years previously treated with percutaneous coronary intervention. Her medications comprised of bisoprolol 10mg, ramipril 10mg, furosemide 80mg twice daily, spironolactone 25mg, simvastatin 40mg, metformin 1g twice daily and aspirin 75mg. On examination she appeared breathless, the jugular venous pressure was elevated at 7cm, there were bibasal fine crepitations and moderate pitting oedema to her knees. Her heart rate was 68 beats per minute and blood pressure was 95/65mmHg.

Investigations:

Haemoglobin	115 g/L
White cell count	5.6 $\times 10^9$ /L
Platelet Count	268 $\times 10^9$ /L
Serum sodium	132mmol/L
Serum potassium	4.3mmol/L
Serum urea	6.7mmol/L
Serum creatinine	68micromol/L

Electrocardiogram: normal sinus rhythm. Rate 65 beats per minute. QRS duration

155ms. No acute ST changes.

Echocardiography: left ventricular ejection fraction of 30%. No significant valvular abnormalities.

Coronary angiography: Patent left anterior descending coronary artery stent.
Minor diffuse coronary artery disease.

What is the most appropriate next management step?

- a) Increase dose of furosemide
- b) Cardiac resynchronisation therapy
- c) Coronary artery bypass graft
- d) Start amlodipine
- e) Home oxygen

This lady has ischaemic heart disease with New York Heart Association (NYHA) class 4 heart failure and is on maximal medical therapy. Her ECG shows a wide QRS complex which suggests ventricular dyssynchrony (i.e. The left and right ventricle are not beating at the same time). Resynchronisation of the ventricles with cardiac resynchronisation therapy with a paced device (CRT-P) involves simultaneous stimulation of both ventricles to reduce dyssynchrony and therefore increase cardiac output.

Nice supports the use of CRT-P in patients with:

- NYHA class three or four symptoms
- They are in normal sinus rhythm with either
 - → QRS duration of 150 ms or longer estimated by standard electrocardiogram (ECG)
 - → QRS duration of 120-149 ms estimated by ECG and mechanical dyssynchrony that is confirmed by echocardiography.

- They have a left ventricular ejection fraction of 35% or less.
- They are receiving optimal pharmacological therapy.

In this selected group of patients, it has been shown to improve symptoms by a reduction of about one NYHA classification, reduce hospitalisations and reduce mortality. These devices can be fitted with an implantable cardiac defibrillator (i.e. A CRT-D where the D stands for defibrillator as opposed to a CRT-P where P stands for paced and thus this device lacks the defibrillator component) for primary or secondary prophylaxis against ventricular dysrhythmias.

Increasing her dose of furosemide is unlikely to significantly improve her symptoms and is, therefore, an incorrect answer. Coronary artery bypass grafting has greater benefit than percutaneous coronary intervention at treating multivessel disease, however, this lady has disease of the left anterior descending artery which has been previously treated and only minor disease in her other coronary vessels; therefore this is an incorrect answer. She is hypotensive and therefore starting amlodipine is inappropriate. Home oxygen can be used for the palliation of shortness of breath in heart failure and is not appropriate in this case.

Question #172

An 82-year-old man sees his GP to discuss his medication. He complains of shortness of breath on minimal exertion and says that he now has to sleep upright in his armchair. His medication includes lisinopril, metoprolol, spironolactone, furosemide, aspirin, simvastatin, nifedipine and metformin.

Physical examination shows a heart rate of 69 beats per minute with a regular rhythm and blood pressure of 118/100 mmHg. His peripheries are cool, and his carotid pulse is difficult to palpate. He has a sustained apical impulse palpable in the 5th intercostal space in the mid-clavicular line. He has a palpable thrill in the second intercostal space at the right sternal border where his GP hears a loud crescendo-decrescendo murmur which radiates into his neck. He has a quiet S1, an inaudible S2, and a loud S4. His chest is clear to auscultation and he is noted to have 1+ peripheral oedema.

Which of the following physical examination findings are most suggestive of severe aortic stenosis?

- a) Loud crescendo-decrescendo murmur
- b) Quiet S1
- c) Inaudible S2
- d) Palpable thrill
- e) Narrow pulse pressure

Aortic stenosis - a soft S2 is a feature of severe disease

The feature identified on physical examination that is most suggestive of severe aortic stenosis is an inaudible second heart sound.

The loudness of a murmur does not correlate with its severity. In the case of regurgitant murmurs, for example, quiet murmurs can represent severe regurgitation as there is early equalisation between the two cardiac chambers involved.

A quiet S1 may reflect significant aortic valve calcification but does not necessarily reflect the severity of the stenosis

A palpable thrill and narrow pulse pressure can occur in severe aortic stenosis but are not as indicative of severe aortic stenosis as an absent second heart sound

Question #173

A 45-year-old male patient presents with a worsening shortness of breath to the medical assessment unit. He has an accompanying letter from his GP stating that his past medical history is significant only for a deteriorating renal function that is secondary to polycystic kidney disease.

On examination there is no evidence of fluid overload, with the lung fields remaining clear and the JVP not elevated. You do however discern a late systolic murmur heard best at the cardiac apex which is immediately preceded by a 'click'.

Observations

- heart rate 82bpm
- blood pressure 106/86mmHg
- respiratory rate 16 per minute
- temperature 36.5
- oxygen saturation 97% on room air

What is the most likely diagnosis?

- a) Mitral valve prolapse
- b) Mitral stenosis
- c) Pulmonary stenosis
- d) Mitral regurgitation
- e) Aortic stenosis

Adult polycystic kidney disease can be associated with extra-renal complications. Up to 85% of patients with autosomal dominant polycystic kidney disease (ADPKD) will exhibit liver cysts by the age of 30. Other complications include intracranial aneurysm and heart valve disease. Affected heart valves can occur in up to 25% of ADPKD patients and of them the majority have mitral valve prolapse. The majority of patients with mitral valve prolapse will be asymptomatic but when symptoms are present mitral valve prolapse has to be considered and investigated with an echocardiogram.

The 'click' coupled with the knowledge of the underlying ADPKD makes the murmur heard in this patient that of a mitral valve prolapse. The prolapse can worsen and cause a secondary mitral regurgitation leading to symptoms such as shortness of breath. The narrow pulse pressure present also suggests an element of left ventricular dysfunction.

Question #174

A 34-year-old gentleman with Marfan's syndrome and known mitral regurgitation presents to the emergency department with increasing shortness of breath for three days. He can currently walk 100 metres at a time before needing to stop. He feels restless at times and has noticed his heart is racing in his chest. There is no chest pain. He is under annual surveillance with echocardiography and is currently managed on furosemide and ramipril.

On examination, his pulse is irregularly irregular at 96 beats per minute. He is alert with a clear chest and no visible JVP. He has a soft S1 with a loud grade 4 pansystolic murmur with radiation into the axilla. He is skinny but alert.

Na ⁺	134 mmol/l
K ⁺	4.2 mmol/l
Urea	4.3 mmol/l
Creatinine	89 µmol/l

ECG	no visible P-waves, irregular narrow complex rhythm at 84 per minute
CXR	cardiomegaly with no effusions or lung shadowing
Bedside ECHO	No pericardial effusion, normal sized cardiac chambers, no regional wall motion abnormality

What is the most appropriate next action?

- a) Increase furosemide
- b) DC cardioversion
- c) Bisoprolol
- d) Refer to cardiothoracic surgery
- e) Repeat Echocardiography

New AF in mitral regurgitation -> refer for mitral valve replacement

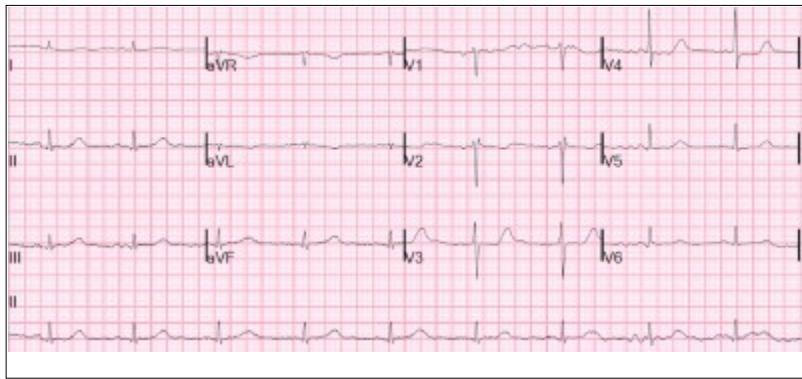
This gentleman has mitral regurgitation and has developed atrial fibrillation which is a common complication following dilation of the mitral annulus. He is not in pulmonary oedema and there is no haemodynamic compromise so DC cardioversion would be inappropriate. He shows no signs of cardiac failure so diuresis with furosemide will not help. Although, he is in atrial fibrillation, his rate is slow and does not need reducing at present with a beta blocker. A repeat ECHO will be useful to confirm current heart structure. The development of atrial fibrillation is an indication to refer for mitral valve surgery as is an ejection fraction less than 60%, pulmonary hypertension or a dilated left ventricle.

Question #175

A 26-year-old woman presents to the emergency department with generalised weakness. She has a past medical history of type 1 diabetes mellitus and reports having been vomiting repeatedly over the past 4 days and cannot keep any food or liquids down. There is no history of fever or diarrhoea.

On examination, she appears lethargic with dry mucous membranes. Chest sounds are clear and heart sounds are normal. Her abdomen is soft with generalised tenderness mostly within the epigastric region.

Her blood glucose is recorded as 7.4 mmol/L. An ECG is taken on arrival at the emergency department, as shown below:



What is the recommended initial management for this patient?

- a) Intravenous 0.9% sodium chloride with 40 mmol potassium
- b) Intravenous 1.26% sodium bicarbonate
- c) Intravenous calcium gluconate
- d) Intravenous magnesium sulphate
- e) Warmed intravenous 0.9% sodium chloride

This patient's ECG shows U waves that are most noticeable in leads V2 and V3. U waves are small positive deflections that appear shortly after a T wave and frequently represent hypokalaemia. There is also a prolonged QTc interval ($> 440\text{ms}$). Other features of severe hypokalaemia include increased P wave amplitude, PR interval prolongation and T wave flattening.

Intravenous 0.9% sodium chloride with 40 mmol potassium is correct. There are several causes of U waves including hypothermia, bradycardia, hypocalcaemia, hypomagnesaemia and severe hypokalaemia. There are also multiple causes of a prolonged QTc interval including hypokalaemia, hypomagnesaemia, hypocalcaemia and hypothermia. Given the presentation of prolonged vomiting in a patient with type 1 diabetes (likely representing gastroparesis), this patient is at high risk for having hypokalaemia. The clinical history and ECG are suggestive of hypokalaemia and therefore intravenous potassium replacement is necessary.

Intravenous 1.26% sodium bicarbonate is incorrect. Sodium bicarbonate 1.26% can be administered in instances of metabolic acidosis and certain arrhythmias (eg

broad complex tachycardia associated with salicylate overdose). It is not indicated for this patient.

Intravenous calcium gluconate is indicated for severe hypocalcaemia and hyperkalaemia with ECG changes such as tall tented T waves, QRS widening and flattened P waves. Although hypocalcaemia can cause a prolonged QTc interval which is seen here, it is a less common cause of U waves making calcium gluconate a less appropriate drug to administer.

Intravenous magnesium sulphate is given in the management of acute arrhythmias including atrial fibrillation and Torsades de Pointes (a monomorphic ventricular tachycardia associated with QTc prolongation). Although hypomagnesaemia can cause QTc prolongation, it is a less common cause of U waves in comparison to hypokalaemia, making potassium replacement the most appropriate initial management. However, it is important to remember that a normal magnesium level is required for the maintenance of a potassium level within the normal range and, therefore, replacement may also be necessary.

Warmed intravenous 0.9% sodium chloride is incorrect. Hypothermia is a less common cause of U waves and QTc prolongation and more commonly produces J waves which are not seen here. Secondly, there is no indication from the clinical presentation that this patient is hypothermic.

Question #176

A 68-year-old lady, who is a retired teacher, is admitted with lightheadedness. She has a history relevant for depression, osteoarthritis and sciatica. Her medication history includes codeine 30mg QDS, ibuprofen 400mg TDS, pregabalin 50mg TDS and amitriptyline 100mg ON. She is also on nitrofurantoin 50mg OD for recurrent urinary tract infections. Electrocardiogram (ECG) confirms normal sinus rhythm with a heart rate of 80bpm. There are no dynamic ST/T changes. PR interval is 140ms and QTc is prolonged at 526ms.

Which medication is most likely to be implicated?

- a) Nitrofurantoin
- b) Pregabalin
- c) Amitriptyline
- d) Ibuprofen
- e) Codeine

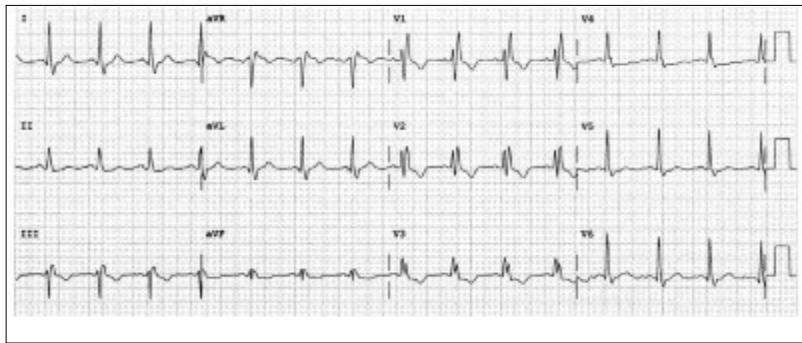
The correct answer is amitriptyline. Tricyclic anti-depressants are associated with prolongation of QTc interval. Other medications that can cause this include quinidine, erythromycin, digoxin, amiodarone and lithium. Prolongation of QTc interval, if marked, can result in R on T phenomenon, whereby a ventricular stimulus causes premature depolarisation of cells that have not completely repolarised. This may result in ventricular tachycardia or ventricular fibrillation. This lady is presenting with dizziness, and one should be concerned that this is a result of dysrhythmia secondary to pharmacotherapy. Any implicated medications should be temporarily discontinued, with alternatives sought.

Question #177

A 50-year-old man is brought into the emergency department following a collapse whilst gardening. He denies any presyncopal symptoms but now describes shortness of breath. His past medical history includes prostate cancer and hypertension.

On examination, there is good air entry bilaterally. His peripheral pulses are present bilaterally with a capillary refill time of < 2 seconds. There is no postural drop in blood pressure and his neurological examination is unremarkable.

An ECG is taken, as shown below:



What is the most likely cause of this patient's presentation?

- a) Aortic stenosis
- b) Hypercalcaemia
- c) Hypokalaemia
- d) Left ventricular hypertrophy
- e) Pulmonary embolism

This patient's ECG demonstrates right bundle branch block (RBBB) with an RSR pattern in leads V1-V3 (creating an 'M-shaped' QRS complex), with wide slurred S waves in the leads I, aVL, V5-V6 (creating a 'W-shaped' QRS complex). A widened QRS interval (i.e. > 120ms) is also a feature of RBBB. There are multiple causes of RBBB and in some cases, it may be found incidentally as a normal variant.

However, pathological causes commonly include increased right heart strain (e.g. right ventricular hypertrophy, cor pulmonale or pulmonary embolism). Given the history of malignancy, collapse and shortness of breath, a pulmonary embolism is the most likely cause of this patient's presentation to the hospital and ECG findings of RBBB.

Whilst aortic stenosis can cause the symptoms of collapse and breathlessness, it is not common for aortic stenosis to cause RBBB. Rather, aortic stenosis may be responsible for the appearance of left bundle branch block (LBBB) or left ventricular hypertrophy on an ECG.

The most common ECG finding in hypercalcaemia is QT interval shortening. In severe hypercalcaemia (> 3.4mmol/L) J waves may be seen. Although

hypercalcaemia is more likely to be present in a patient with a diagnosis of malignancy, it would not be responsible for this patient's RBBB and is unlikely to be the underlying diagnosis.

Hypokalaemia causes the following ECG changes: prolonged PR interval, widespread ST depression and T wave flattening or inversion and prominent U waves (most clearly in leads V2-V3). It does not cause RBBB and is, therefore, incorrect.

Left ventricular hypertrophy (LVH) has numerous causes, most commonly hypertension. There are lots of specific voltage criteria to be met to accurately diagnose LVH on an ECG including:

- R wave in V4, V5 or V6 > 26mm
- R wave in V5 or V6 plus S wave in V1 > 35 mm
- Largest R wave plus largest S wave in precordial leads > 45 mm

Additional features include left axis deviation and left atrial enlargement. Although this patient has hypertension, there are no features of LVH on his ECG and it is unlikely to cause his episode of syncope, making it the incorrect answer.

Question #178

An 82-year-old presents with suprapubic discomfort. He reports urgency but has been unable to pass any urine for over 24 hours. Three months ago, he underwent a third failed transurethral resection of the prostate for benign prostatic hypertrophy (PSA was normal). His past medical history includes hypertension, type 2 diabetes mellitus, chronic kidney disease (baseline creatinine 150 µmol/l) previous MI in 2007 and 2010. He remains relatively active, with an exercise tolerance of 500 yards. On examination, his mucous membranes are moist, his peripheries are warm and his JVP at 3 cm above the angle of Louis. His heart sounds and chest are unremarkable. His abdomen is tender and distended in the suprapubic region. On insertion of a urethral catheter, 900mls of residual urine is

noted. His blood tests are as follows:

Hb	115 g/l
Platelets	282 * 10 ⁹ /l
WBC	6.8 * 10 ⁹ /l

Na ⁺	144 mmol/l
K ⁺	5.8 mmol/l
Urea	12.1 mmol/l
Creatinine	201 µmol/l
Troponin T	0.08 (normal <0.03)

His ECG demonstrates left bundle branch block (old) and first-degree heart block at a rate of 49 beats/ minute and regular. What is the appropriate management?

- a) Treat as acute coronary syndrome only
- b) Intravenous fluids only
- c) Monitor renal function and consider long term catheter only
- d) Treat as acute coronary syndrome, monitor renal function and consider long term catheter
- e) Treat as acute coronary syndrome, intravenous fluids, monitor renal function and consider long term catheter

This patient is a typical elderly admission with multiple co-morbidities involving multiple organ systems. The key to this question is recognising which aspects are important and which are not. The patient is clearly euvoalaemic and intravenous fluids at this stage are not required. However, it would be prudent to monitor urine output and if significant amounts are produced, to replace output with oral intake or intravenous input. Troponin is raised but this is in the context of an acute on chronic kidney injury. In the context of no reported chest pain and no new dynamic ECG changes, treatment as an acute coronary syndrome is unnecessary. The patient is at greater risk of post-renal injury and his renal function tests should instead be monitored. A long-term catheter is a reasonable suggestion considering the number of failed TURPs.

Question #179

A 64-year-old hypertensive male presents to the emergency department with a history of sudden onset tearing chest pain. He is a chronic smoker with a 25 pack year history. He takes valsartan 160mg daily and amlodipine 5mg daily.

On examination, he is distressed and in severe pain. Blood pressure is 200/120 mmHg. The peripheral pulses are weak and his heart rate is 125 bpm.

Sublingual nitrates and oral aspirin are given to him in the emergency department and his twelve-lead ECG is performed which reveals tachycardia, left ventricular strain and deep S waves in lead V1-V3 and tall R waves in V4-V6.

Chest x-ray shows widening of the mediastinum with an irregular aortic contour.

Contrast-enhanced CT scan of the chest reveals an aortic intimal flap distal to left subclavian artery.

Which of the following is the most appropriate treatment option for this patient?

- a) Blood pressure control with IV beta-blockers
- b) Percutaneous stent insertion

- c) Surgical repair
- d) Blood pressure control with IV hydralazine
- e) Continuous infusion of isosorbide dinitrate

Correct answer is a.

The diagnosis in this question is aortic dissection type B (distal to the left subclavian artery), the management of which is blood pressure control. The most appropriate drugs used are IV beta-blockers.

Aortic dissections can be classified according to the anatomic site of the dissection. Type A involves the aortic arch and the aortic valve while type B involves the descending thoracic aorta distal to the left subclavian artery. (Stanford Classification)

Stanford types A and B are further sub-classified according to the De Bakey classification.

Stanford Type A constitutes De Bakey I (extending to the abdominal aorta) and De Bakey II (localised to the ascending aorta). Stanford type B is synonymous with De Bakey type III.

The importance of these classifications is the difference in treatment. Universally, blood pressure control is mandatory with a target reduction in systolic BP to 120mmHg. Type A dissection should undergo surgery (aortic arch replacement) if the patient is fit enough since medical management alone carries a high risk of mortality (50% within 2 weeks). Type B dissections carry a better prognosis with 89% survival at one month and should thus initially be managed medically, with surgical interventions only if complications develop.

Indications for endovascular stenting include:

- Rapidly expanding dissections (>1cm per year)
- Critical diameter (>5.5cm)
- Refractory pain

- Malperfusion syndrome
- Blunt chest trauma
- Penetrating aortic ulcers

Question #180

A 45-year-old man presents to the Emergency Department with shortness of breath, heavy chest pain and syncope. The chest pain and shortness of breath have been worsening over the last week. He is currently undergoing adjunct chemotherapy for a non-resectable soft tissue sarcoma. He has known metastasis in his thorax and mediastinum.

Hb	90 g/L	Male: (135-180) Female: (115 - 160)
Platelets	$100 * 10^9/\text{L}$	(150 - 400)
WBC	$12.3 * 10^9/\text{L}$	(4.0 - 11.0)

Na ⁺	131 mmol/L	(135 - 145)
K ⁺	3.1 mmol/L	(3.5 - 5.0)
Bicarbonat e	19 mmol/L	(22 - 29)
Urea	8.0 mmol/L	(2.0 - 7.0)
Creatinine	145 µmol/L	(55 - 120)

On physical examination his JVP is raised at 6cm, there are no precordial thrills, and he has quiet S1 and S2.

Observations show:

- Heart rate of 115/min and regular
- Blood pressure 95/55mmHg
- Respiratory rate 28/min
- Temperature 36.5°C
- AVPU - A

What is the more specific ECG finding associated with this diagnosis?

- a) Electrical alternans
- b) RBBB
- c) S1Q3T3
- d) ST elevation in leads V1-4
- e) Sinus tachycardia

Electrical alternans is suggestive of cardiac tamponade

Electrical alternans is the correct answer. The patient has metastatic disease within the thorax and mediastinum increasing his risk of a pericardial effusion. Electrical alternans is seen in massive pericardial effusions as an alternating height of QRS complex across the rhythm strip. Other ECG findings to look for are tachycardia and low voltage QRS. The website Life in the Fast Lane has a library of ECGs with examples of this.

RBBB is the incorrect answer because this is not a specific finding for massive pericardial effusion. There are a variety of causes of RBBB. Examples include

congenital heart disease, cardiomyopathy, pulmonary embolus, right ventricular hypertrophy, ischaemic heart disease and others. In an acute PE setting, which is a differential, in this case, you would also look for right heart strain due to the sudden increase in right ventricular pressure caused by the obstruction to the pulmonary vasculature.

S1Q3T3 is the incorrect answer as this is associated with a pulmonary embolism (PE). However, this is only seen in approximately 15% of PEs. The more common ECG finding is sinus tachycardia.

ST elevation in leads V1-4 is the incorrect answer because this is seen in an anterior occlusive, or type 1, myocardial infarction. This typically affects the left anterior descending artery and confers a high risk of a ventricular arrhythmia. The patient requires urgent PCI or, if that is not available within 120 minutes, then thrombolysis.

Sinus tachycardia is the incorrect answer as it is a non-specific finding on ECG. You would expect a patient with a massive pericardial effusion to mount a tachycardic response. This is because a decrease in diastolic relaxation and the subsequent volume of ventricular filling decreases your stroke volume. To maintain an adequate cardiac output, the patient will increase HR. As $CO = SV \times HR$. However, this is a non-specific finding and is seen in all types of shock; septic, cardiogenic, distributive and hypovolaemic. Answer 1 is a much more specific ECG finding.

Discuss (5)Improve

Question #181

A 78-year-old gentleman is seen in clinic with long-standing heart failure with reduced ejection fraction (28%). He has had numerous admissions this year with heart failure decompensation and is wondering if there is anything else that you can do for him. You review his ECG, which is in sinus rhythm with a heart rate of 58/min with a QRS of 98 msec and no signs of intraventricular delay. His blood pressure is: 98/55 mmHg. You also review his bloods:

Hb	129 g/L	Male: (135-180) Female: (115 - 160)
Platelets	$335 * 10^9/L$	(150 - 400)
WBC	$6.8 * 10^9/L$	(4.0 - 11.0)
Na^+	137 mmol/L	(135 - 145)
K^+	4.7 mmol/L	(3.5 - 5.0)
Urea	5.4 mmol/L	(2.0 - 7.0)
Creatinine	125 $\mu\text{mol}/\text{L}$	(55 - 120)

He is currently on carvedilol 25 mg BD, enalapril 10 mg BD, bumetanide 2 mg BD, aspirin 75 mg, ivabradine 2.5 mg BD, spironolactone 25 mg OD.

What alteration to this gentleman's medications could potentially decrease this gentleman's probability of being re-admitted?

- a) Increase ivabradine
- b) Add sacubitril/valsartan
- c) Increase spironolactone
- d) Stop enalapril - initiate sacubitril/valsartan
- e) Increase bumetanide

Sacubitril-valsartan should be initiated following ACEi or ARB wash-out period

This gentleman poses the issue of long-standing management of heart-failure. The ESC guidelines advice strictly on the uptitration of medication up to their maximal desired effect taking in consideration the patient's status - the frontline medications include ACE inhibitors, b-blockers and mineralocorticoid receptor

antagonists. The patient is on all three. If the patient remains symptomatic then a good choice would be dependable on their QRS morphology with a CRT-P device being appropriate in certain cases (e.g. QRS >130 msec) or ivabradine in someone with a heart rate of more than 70/min. The option of digoxin is also discussed in the ESC guidelines.

However the patient above is already bradycardic and hypotensive with a slightly raised potassium therefore making the possibility of increasing any of his current medications less appealing. In these patients the possibility of introducing sacubitril/valsartan is appropriate, however it is important for the clinician to be aware of the need for a wash-out period between the ACEi and the initiation of sacubitril/valsartan. The wash-out period advised is that of 36 hours.

The increase in diuretic might appear appealing, there is no evidence that diuretics improve mortality rates in such patients. The PARADIGM-HF trial that was comparing sacubitril/valsartan and enalapril indicated that sacubitril/valsartan was superior to enalapril in reducing cardiovascular mortality, heart failure hospitalisation and 30-day hospital readmission in heart failure patients with reduced ejection fraction.

Discuss (6) Improve

Question #182

A 53-year-old gentleman is reviewed in clinic due to abnormal blood tests. He has a past medical history of hypertension, type two diabetes, obesity, and depression. He started taking atorvastatin two months ago due to a routine assessment of QRISK and elevated cholesterol levels. He had a blood test as requested at three months following the start of treatment.

These shows that his alanine aminotransferase have increased from 28iU/L to 94iU/L. Other blood tests have remained within normal ranges apart from cholesterol which as improved from 5.4mmol/L to 4.9mmol/L.

How should his atorvastatin treatment be managed?

- a) Stop atorvastatin and repeat LFT within 4-6 weeks
- b) Continue atorvastatin and repeat LFT within 4-6 weeks
- c) Change atorvastatin to simvastatin
- d) Investigate for elevated creatinine kinase
- e) Stop atorvastatin and arrange for an urgent abdominal ultrasound

The correct answer is to continue atorvastatin and repeat LFT within 4-6 weeks. This patient has developed elevated transaminases following starting a statin, but these are within three times the upper range of normal. Please note that this is three times the maximum normal range, not three times above the patient's baseline result. Therefore it is acceptable to continue atorvastatin but monitor liver function with a repeat test in 4-6 weeks. If the level had been greater than three times the normal upper range then stopping the atorvastatin and repeating LFT would have been appropriate. There is no need to arrange for an ultrasound at the moment as there is a clear explanation, and there is no evidence of new muscle pain to justify testing for creatinine kinase. Changing statin can play a role if the statin is not tolerated, but that is not the case so far.

Question #183

A 72-year-old man attends the cardiology clinic. He has a past medical history of heart failure. He advises you that he has had worsening shortness of breath over the last few weeks. He is now symptomatic at rest. His regular medications include ramipril, bisoprolol, furosemide and eplerenone. His observations are as follows: heart rate 70 beats per minute, blood pressure 125/75 mmHg, respiratory rate 22 breaths per minute, and SpO₂ 94% on air. On examination, you note pitting oedema to the mid calves and bilateral coarse crepitations.

An echocardiogram and ECG is performed:

Ech	Left ventricular failure with an ejection fraction of 25%
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O

ECG

Sinus rhythm; QRS 140 msec; Dominant S wave in V1 and a broad monophasic R wave in lateral leads (I, aVL, V5-6)

What treatment is most likely to be beneficial to long-term survival?

- a) Bendroflumethiazide
- b) Bumetanide
- c) Cardiac resynchronisation therapy with defibrillator (CRT-D)
- d) Cardiac resynchronisation therapy with pacing (CRT-P)
- e) Ivabradine

Correct answer is e.

Cardiac resynchronisation therapy can be used in patients with a QRS duration of >130 msec and LBBB morphology to improve symptomatology

The patient has symptomatic (NYHA class IV) cardiac failure requiring escalation of treatment. He is already on an ACE-I, beta blocker, mineralocorticoid receptor antagonist, and loop diuretic. He has severe left ventricular systolic dysfunction (LVSD) with an ejection fraction of 25%. It is important to note the presence of LBBB with a significantly prolonged QRS. The dominant S wave in V1 and a broad monophasic R wave in the lateral leads are classic of LBBB.

Cardiac resynchronisation therapy with pacing (CRT-P) is correct. Cardiac resynchronisation therapy (CRT) is recommended as a treatment option for people with heart failure who have left ventricular dysfunction with a left ventricular ejection fraction (LVEF) of 35% or less, and LBBB with a QRS duration > 130 msec. The patient in this clinical case, therefore, fulfils these criteria. Many randomised controlled trials have shown that CRT significantly prolongs survival in patients with heart failure. The NICE guidelines have criteria on whether to choose CRT-D or CRT-P. In patients with NYHA class IV (as seen in this case), CRT-P is the

preferred modality. In this case, it would also be important to consider the use of sacubitril/valsartan. The PROVE-HF trial (as well as case reports and observational studies) have shown that sacubitril/valsartan is able to induce reverse cardiac remodeling, which has the potential to avoid ICD or CRT implantation.

Bendroflumethiazide is incorrect. Bendroflumethiazide can be used for mild or moderate heart failure. Although diuretics can have major benefits for patient symptomatology, they have no benefit to long-term survival.

Bumetanide is incorrect. Bumetanide is a type of loop diuretic which has been shown to have greater bioavailability compared to furosemide due to increased absorption in patients who have oedematous bowels. Although diuretics can have major benefits for patient symptomatology, they have no benefit to long-term survival.

Cardiac resynchronisation therapy with defibrillator (CRT-D) is incorrect. As the patient has NYHA class IV symptomatology, CRT-P is the correct option as per NICE guidelines.

Ivabradine is incorrect. Ivabradine is recommended as an option for treating chronic heart failure for patients who are in sinus rhythm with a heart rate of 75 beats per minute or more and who are already on standard therapy with ACE inhibitors, beta-blockers and aldosterone antagonists. The patient in this clinical case has a heart rate of 70 beats per minute and ivabradine is therefore not indicated.

Discuss (4)Improve

Question #184

A 28 year old woman is admitted to the medical assessment unit complaining of pleuritic chest pain. She is 32 weeks pregnant into her first pregnancy. An ECG reveals a sinus tachycardia. Her blood tests are unremarkable, although the referring accident and emergency doctor added on a D-dimer test for completion and this has come back positive. According to the Royal College of Obstetricians

and Gynaecologists guidelines, what is the next suitable investigation for this patient?

- a) Chest x-ray
- b) Ventilation/perfusion scan
- c) Compression duplex doppler of the legs
- d) CT pulmonary angiogram (CTPA)
- e) Cardiac catheterisation to assess right sided pressures

Correct answer is a.

In suspected pulmonary embolism in pregnant patients the Royal College of Obstetricians and Gynaecologists recommend the following:

- 1. Chest xray to look for an alternative diagnosis.
- 2. If the chest xray is normal consider a compression duplex doppler of both legs to exclude a DVT. If this is positive, the patient is treated with full dose low molecular weight heparin (LMWH) (warfarin is of course teratogenic).
- 3. If both the above investigations are normal, and there remains a strong suspicion of a pulmonary embolism, then clinicians should consider a CTPA or ventilation perfusion/scan weighing up the risks vs benefits of each and also the local hospital protocol. Current guidance however favours a perfusion scan as it has lower lung radiation doses than a CTPA.

Question #185

A 63-year-old man presents for review at his GP surgery. He is known to have hypertension and was started on amlodipine five years ago. Due to persistent poor hypertensive control, he began taking enalapril two years ago but has continued to struggle to keep his blood pressure within the required range. He reports no symptoms of postural hypotension, and his latest ambulatory blood pressure readings show an average blood pressure of 161/93 mmHg.

His latest blood test results are shown below:

Na ⁺	138 mmol/L	(135 - 145)
K ⁺	4.3 mmol/L	(3.5 - 5.0)
Urea	4.5 mmol/L	(2.0 - 7.0)
Creatinine	84 µmol/L	(55 - 120)

What medication should be offered to this patient as the next step in managing his hypertension?

- a) Bendroflumethiazide
- b) Bisoprolol
- c) Doxazosin
- d) Indapamide
- e) Spironolactone

Poorly controlled hypertension, already taking an ACE inhibitor and a calcium channel blocker - add a thiazide-like diuretic

The correct answer is **indapamide**. Current guidelines advise that in the absence of any clear contraindications, patients with poorly controlled hypertension already taking an ACE inhibitor and a calcium channel blocker should be offered a thiazide-like diuretic. Thiazide-like diuretics are sulfonamide-based diuretics that have similar pharmacological properties to thiazides but have been shown in recent trials to reduce systolic blood pressure more effectively than conventional thiazides, without a significant difference in side-effect profile. Indapamide is the only thiazide-like diuretic provided in the answer choices.

Bendroflumethiazide is incorrect as thiazide-like diuretics, rather than thiazide diuretics such as bendroflumethiazide now tend to be preferred as third-line antihypertensive therapy in most patients for the reasons outlined above.

Bisoprolol is incorrect. This would be an appropriate option for a patient with persistent hypertension already taking a calcium channel blocker, an ACE inhibitor and a thiazide-like diuretic with serum potassium > 4.5mmol/L.

Doxazosin is incorrect. Alpha blockade with doxazosin would be another option for a patient with persistent hypertension already taking a calcium channel blocker, an ACE inhibitor and a thiazide-like diuretic with serum potassium > 4.5mmol/L.

Spironolactone is incorrect. Aldosterone receptor antagonism with spironolactone may be offered to patients with persistent hypertension despite taking a calcium channel blocker, an ACE inhibitor and a thiazide-like diuretic with serum potassium < 4.5mmol/L, due to the propensity of spironolactone to cause hyperkalaemia.

Discuss (2)Improve

Question #186

You are working in the general medical clinic where a 42 year old woman comes for review following a recent, short admission to hospital where she was treated for a paracetamol overdose. She has a past history of depression but denies any other previous problems.

During the review, she is found to have a manual blood pressure reading of 165/85 mmHg. Clinical examination of cardiovascular and respiratory systems are normal, as is urine dip and fundoscopy. Given this information what should be your next course of management in relation to her blood pressure.

- a) Start ramipril

- b) Offer ambulatory blood pressure monitorin
- c) Arrange to check blood pressure again following a two week interval
- d) Start amlodipine
- e) Screen for causes of secondary hypertension

In 2011 the National Institute for Clinical Excellence updated its 2006 guideline for the management of hypertension (see the link below for the quick reference guide). Within this guideline, the first line use of ambulatory blood pressure monitoring (ABPM) to confirm hypertension in those found to have an elevated clinic reading ($> 140/90$ mmHg) is emphasised. When using ABPM to confirm a diagnosis of hypertension, two measurements per hour are taken during the persons waking hours. The average value of at least 14 measurements are then used to confirm a diagnosis of hypertension.

Generally speaking, secondary causes of hypertension should be sought in; patients under 40 who lack traditional risk factors for essential hypertension, patients with other sings and/or symptoms of secondary causes, and patients with resistant hypertension. Although in reality the most common cause of secondary hypertension is hyperaldosteronism, and as such a trial of an aldosterone antagonist such as spironolactone is often employed as both a therapeutic and diagnostic measure.

Drug treatment of essential hypertension can be summarised as follows, but for a more detailed explanation see the link below;

- Step 1; Age < 55 - ACE inhibitor. Age > 55 or of black African or Caribbean origin - calcium channel blocker
- Step 2; ACE inhibitor + calcium channel blocker
- Step 3; ACE inhibitor + calcium channel blocker + thiazide-like diuretic
- Step 4; consider further diuretic or beta-blockade or alpha blocker and seeking expert advice

Question #187

A 72-year-old gentleman with previous myocardial infarctions and coronary stents presents with increased leg swelling. This has arisen over two weeks. He is still well and denies any shortness of breath but has an occasional cough at night time. He is managed on aspirin, clopidogrel, ramipril and atorvastatin.

On further questioning, he admits feeling cold at times and feels low in energy for which his GP started citalopram 2 months ago.

He denies any further chest pains. His previous records indicate disease in the circumflex and right coronary arteries previously. He admits to drinking 20-30 units of alcohol a week.

On examination, he appears well. He has bilateral pitting leg oedema with extension into the sacral area. There is a pansystolic murmur over the sternum. His abdomen is soft with a tender hepatomegaly. There is no splenomegaly, ascites, jaundice or asterixis. His JVP is raised with a double flicker pattern which peaks up to the ear lobe on the second flicker. Current blood pressure is 145/86 mmHg.

Hb	95 g/l	Na ⁺	137 mmol/l
Platelets	476 * 10 ⁹ /l	K ⁺	4.2 mmol/l
WBC	10.2 * 10 ⁹ /l	Urea	4.3 mmol/l
Neuts	9.6 * 10 ⁹ /l	Creatinine	112 µmol/l
Bilirubin	4 µmol/l	ALP	165 u/l
ALT	103 u/l	Albumin	34 g/l

PT 12.2 (14-18 normal range)	APTT 34 (34-42 normal range)	
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Chest x-ray	enlarged cardiac shadow, no effusions or lung field shadowing
ECG	sinus rhythm with Q waves in V2-V3
Urine dip	2+ protein, no blood

What is the likely diagnosis?

- a) Tricuspid regurgitation
- b) Cirrhosis
- c) Hypertensive cardiomyopathy
- d) Nephrotic syndrome
- e) Hypothyroidism

Prominent V waves on JVP → tricuspid regurgitation

This gentleman presents with leg swelling and a raised JVP which are both signs of right-sided heart failure. Nephrotic syndrome and hypothyroidism can present with swollen legs but would not have a raised JVP. Hypertensive cardiomyopathy does occur but there is no mention of previous hypertension and we do not know his blood pressure. He does not have cirrhosis given there are no signs of liver disease and his clotting is normal though his LFTs are raised. This leaves tricuspid regurgitation as the answer. He most likely developed this after suffering a posterior myocardial infarction. The faint V waves on JVP are a classic sign.

Question #188

A 68-year-old male is admitted with confusion and an ataxic gait. His past medical history includes heart failure and atrial fibrillation. His drug history includes

dabigatran, bisoprolol and ramipril.

Investigation results are as follows:

Hb	110 g/l	Na ⁺	139 mmol/l
Platelets	$140 * 10^9/l$	K ⁺	3.8 mmol/l
WBC	$10.2 * 10^9/l$	Urea	6.5 mmol/l
Neuts	$8.2 * 10^9/l$	Creatinine	42 µmol/l
Lymphs	$1.4 * 10^9/l$	CRP	12 mg/l
PT ratio	$1.6 * 10^9/l$	aPTT	50 seconds

CT head	Large acute right-sided subdural haematoma with ventricular effacement and midline shift
---------	--

How will you manage this patient?

- a) Platelet transfusion
- b) Fresh frozen plasma
- c) Red cell transfusion
- d) Tranexamic acid
- e) Idarucizumab

Correct answer is e.

Bleeding on dabigatran? Can use idarucizumab to reverse

Dabigatran is an oral anticoagulant acting by direct inhibition of thrombin. In active bleeding, or life-threatening haemorrhage, idarucizumab should be used. This is a specific antidote which reverses the effect of dabigatran.

The PT ratio and INR is not a reliable marker following dabigatran ingestion, and should not be used to monitor the anticoagulant effects. The aPTT and, if available, the thrombin time (TT) should be used to measure the anticoagulant effect of dabigatran,

If idarucizumab is unavailable, and particularly if there is evidence of acquired coagulopathy, consider administration of red cell concentrate, fresh frozen plasma, and/or platelet transfusion.

Discuss (4)Improve

Question #189

An 80-year-old man with a history of atrial fibrillation is admitted following an acute coronary syndrome and is treated with percutaneous coronary intervention. His medication on admission was warfarin, co-codamol and allopurinol.

With regards to antithrombotic therapy, what should he be prescribed in the immediate aftermath of the event?

- a) Continue warfarin with the addition of 2 antiplatelets
- b) Continue warfarin with the addition of 1 antiplatelet
- c) Continue warfarin monotherapy
- d) Stop warfarin, start 2 antiplatelets
- e) Stop warfarin, start 3 antiplatelets

Correct answer is a,

In the initial phase following an ACS/PCI patients who also have AF

Question #190

clinic for her regular follow-up. She is asymptomatic, can perform daily tasks, and has a New York heart association (NYHA) functional classification of I. On examination, there is a loud rumbling pan-systolic murmur heard at the apex. She is noted to be in atrial fibrillation, which was not previously known. A recent echocardiogram shows an ejection fraction of 62% with mild left atrial dilatation.

Which feature from the history would be the strongest indication for referral for valve replacement?

- a) Age
- b) Atrial fibrillation
- c) Left atrial dilatation
- d) Loud murmur
- e) Ejection fraction <65%

Correct answer is b.

New AF in mitral regurgitation -> refer for mitral valve replacement

Surgery is indicated in patients with signs of left ventricular (LV) dysfunction. If LV function is preserved, surgery should be considered in asymptomatic patients with new-onset atrial fibrillation (AF) or pulmonary hypertension. Severe symptomatic mitral regurgitation is also an indication for replacement.

AF develops as a consequence of increased left atrial pressures (due to backflow), resulting in left atrial dilatation. This is, therefore, the strongest indication for replacement.

In the absence of AF, left atrial dilatation only becomes an indication for replacement if it is significant ($\geq 60 \text{ mL/m}^2$ body surface area [BSA]).

Age itself is not an indication for mitral valve replacement. Age will be considered alongside other co-morbidities and functional baseline when deciding if she will be suitable for a general anaesthetic for the procedure.

The loudness of a murmur typically doesn't impact the decision to proceed with

surgery.

Ejection fraction becomes relevant in asymptomatic patients. 2017 European society of cardiology (ESC) and European association for cardiothoracic surgery (EACTS) guidelines for the management of valvular heart disease suggest that replacement should be considered in asymptomatic patients with an ejection fraction of less than 60%.

Discuss (3) Improve

Question #191

A 64-year-old male presents sudden onset back pain while painting a wall at home, radiating to his left anterior chest and jaw, associated with nausea and vomiting. It appears to be constant for the past 3 hours since onset and is his first ever episode. His past medical history includes hypertension, hypercholesterolaemia and one previous transient ischaemic attack.

His ECG demonstrates left ventricular hypertrophy by voltage criteria and T-wave inversion in I, aVF and V5 and V6. On examination, his heart sounds are both present with a soft systolic murmur. The chest is clear and abdomen is soft and non-tender without a pulsatile mass. Both radial pulses are intact with no delay. There is a mild radio-femoral delay. His blood pressure is stable at 134/80 mmHg, heart rate 124/min and sinus rhythm. His bloods are as follows:

Hb	123 g/l
MCV	82 fl
Platelets	204 * 10 ⁹ /l
WBC	9.2 * 10 ⁹ /l
Troponin T	240 (normal range < 32)



His pain settles transiently with 2.5mg of subcutaneous morphine. What is the most appropriate immediate action?

- a) Aspirin 300mg and clopidogrel 300mg and treatment dose low molecular weight heparin
- b) Intravenous fluids
- c) Blood transfusion
- d) Coronary angiogram +/- stent as appropriate
- e) CT aorta

Correct answer is e.

The combination of an atypical history of back and chest pain, persistent pain, non-specific ECG changes, mildly raised troponin and an unexplained sinus tachycardia should raise suspicion that this is not a simple diagnosis of acute coronary syndrome. The clue lies in the distal pulses: the lack of radial-radial delay suggests any aortic pathology occurs below the subclavian artery origin. The mild radial-femoral delay suggests a descending artery lesion. The patient is unwell but not actively exsanguinating as evidenced by the stable Hb and blood pressure. An underlying diagnosis of acute aortic syndrome, a new term classifying all acute aortic pathology including dissections, ulcers and aneurysms, must be ruled out by high definition CT aorta with contrast.

Discuss (4)Improve

Question #192

A 50-year-old man is recovering 12 hours after a stent placement for a ST elevation myocardial infarction in which there was a small stenosis of the circumflex artery. He has returned to the coronary care unit and you are called to see him due to changes on the cardiac monitor.

There are runs of broad complexes lasting 3-10 seconds at a rate of 80 per minute. He is asymptomatic and on waking is able to tell you that he has no chest pain or palpitations.

Na ⁺	135 mmol/l
K ⁺	3.2 mmol/l
Urea	5.3 mmol/l
Creatinine	89 µmol/l
Magnesium	0.61mmol/l (normal range 0.6-1.0)
Troponin	3000pg/ml (normal range <20)
ECG	Broad complexes at 73 beats per minute running in sets of 3-15 with normal sinus rhythm between. PR 150ms, QTc 450ms. No ST elevation at present

What action should be taken?

Reassure
60% IV magnesium sulfate
30% Transcutaneous pacing
3% DC cardioversion
1% Amiodarone
6%

Accelerated idioventricular rhythm is common and un concerning following recent MI

Important for me
Less important

This gentleman has an MI that has been successfully treated and is recovering well. He is at risk of malignant arrhythmias like ventricular tachycardia during this time. However these broad complexes are not tachycardic. This phenomenon is

accelerated idioventricular rhythm which commonly follows an MI in which ventricular ectopic tissue take over the pacemaker role. This usually resolves with time but can be helped by atropine if the patient is symptomatic. In this case, there are no symptoms and so the best action is to reassure the patient and continue monitoring. All other options would not be appropriate given the benign nature of this rhythm.

Discuss (8)Improve

Question #193

A 73-year-old man was admitted as an emergency suffering from severe, central and crushing chest pain. ECG revealed evidence of anterior ST elevation MI and primary percutaneous coronary intervention was performed with the deployment of two drug-eluting stents to the left anterior descending artery. The patient had no previous coronary artery disease or significant family history. He is an ex-smoker and drinks approximately 10 units of alcohol per week.

Post-procedure the patient was well and mobilised on the ward without further chest pain or shortness of breath. Clinical examination was unremarkable.

The patient was commenced on aspirin, clopidogrel, ramipril, bisoprolol and atorvastatin therapy. Further investigations performed following intervention are summarised as below.

Haemoglobin	13.9 d / dL
White cell count	$8.6 * 10^9/l$
Platelets	$199 * 10^9/l$
Urea	7.2 mmol / L
Creatinine	110 micromol / L

Sodium	137 mmol / L
Potassium	4.3 mmol / L
Total cholesterol	5.4 mmol / L
LDL cholesterol	3.1 mmol / L
HDL cholesterol	1.2 mmol / L
Triglycerides	2.5 mmol / L

Transthoracic echocardiogram: no valvular abnormality; mild anterior dyskinesia with overall normal left ventricular systolic function; ejection fraction 55-60 %

Primary PCI report: total occlusion of mid-LAD, two DES deployed with good angiographic result; 50 % mid-vessel occlusion of the right coronary artery; circumflex unobstructed.

Prior to discharge, the patient asks to speak to you about any other possible strategies to reduce his risk of suffering a further heart attack.

Which of the following interventions is recommended for this patient by NICE guidance on secondary prevention of myocardial infarction?

- a) Percutaneous coronary intervention to right coronary artery
- b) Mediterranean-style diet
- c) Abstinence from alcohol
- d) Omega-3 fatty acid supplements
- e) Aldosterone antagonist (for example, eplerenone)

NICE recommends several lifestyle changes after a myocardial infarction. These include smoking cessation, a Mediterranean-style diet, achievement of a healthy weight, limitation of alcohol consumption to recommended healthy limits and 20-30 minutes of physical activity daily. NICE does not recommend routinely eating oily fish or the consumption of omega-3 supplements, although it is noted that there is no evidence of harm from these interventions.

Aldosterone antagonists are recommended for use in patients with symptoms and signs of heart failure or with evidence of systolic impairment on echocardiography. There is no evidence that percutaneous treatment of mild asymptomatic coronary artery disease (such as in this patient) reduces the future risk of myocardial infarction.

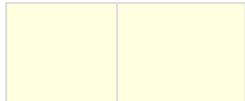
Jones K, Saxon L, Cunningham W, Adams P. Secondary prevention for patients after a myocardial infarction: summary of updated NICE guidance. BMJ 2013;347:f6544.

Question #194

You review a 65 year-old male who was admitted yesterday. He is a diabetic gentleman who was admitted with a non-ST elevation myocardial infarction and subsequent flash pulmonary oedema. He has been treated with aspirin, clopidogrel, fondaparinux and intravenous furosemide. He is achieving a good diuresis - producing 100mls of urine per hour.

You have been asked to see him as he continues to have significant breathlessness but no ongoing chest pain. His blood pressure is 92/87mmHg and oxygen saturations are 83% on 65% humidified oxygen. He has bibasal crepitations and JVP is raised. His ABG results are as follows:

pH	7.32
pCO ₂	4.6kPa
pO ₂	7.9kPa



What would be your next clinical intervention?

- a) Start non-invasive ventilation
- b) Start continuous positive airway pressure ventilation (CPAP)
- c) Refer for angiography and primary coronary intervention
- d) Start a tirofiban infusion
- e) Start a GTN infusion

Correct answer is b.

Acute heart failure not responding to treatment - consider CPAP

This gentleman has had a non-ST elevation myocardial infarction (NSTEMI) and is now struggling with hypoxia secondary to pulmonary oedema. He has been treated optimally for his NSTEMI and is now pain free. He is diuresing well but continues to be hypoxic with signs of ongoing pulmonary oedema. The next logical intervention would be to try CPAP to improve his oxygenation. GTN, tirofiban and angiography would all look to improve coronary perfusion, however the gentleman is pain free suggesting this is not a priority.

Question #195

A 31-year-old woman comes to the emergency department complaining of palpitations and dizziness. She has had palpitations before, but this is the first time she is feeling dizzy.

On examination, she is afebrile. Her heart rate is 140 bpm, and her blood pressure is 110/90 mmHg. She has a respiratory rate of 12 breaths per minute, and oxygen saturation is 96% on air. The rest of her examination is normal.

A 12-lead ECG shows polymorphic ventricular tachycardia, QTc 510ms.

Her laboratory studies show:

		Reference range
Haemoglobin	125 g/L	Men: 135-180 g/L Women: 115-160 g/L
White blood cells	$8.7 * 10^9/l$	$4.0-11.0 * 10^9/l$
Platelets	$280 * 10^9/l$	$150-400 * 10^9/l$
Sodium	137 mmol/L	135-145 mmol/L
Potassium	3.9 mmol/L	3.5-5.0 mmol/L
Magnesium	1.01 mmol/L	0.70-1.05 mmol/L
Urea	2.5 mmol/L	2.0-7 mmol/L
Creatinine	99 umol/L	55-120 umol/L

Which of the following is the best next step in the management of this patient?

- a) Intravenous amiodarone
- b) Intravenous lidocaine
- c) Intravenous magnesium sulphate
- d) Immediate defibrillation
- e) Synchronised DC cardioversion

Correct answer is c.

IV magnesium sulfate is used to treat torsades de pointes

This patient is presenting with an acute episode of torsades de pointes. Her ECG shows polymorphic ventricular tachycardia with a prolonged QT interval, which is consistent with torsades de pointes. It is characterised by rapid, irregular QRS complexes, which appear to be twisting around the ECG baseline. Torsades de

pointes is treated with IV magnesium sulphate, even when the serum magnesium levels are normal.

Immediate defibrillation is indicated in patients with pulseless torsades de pointes. This patient is conscious and has a pulse.

Synchronised DC cardioversion is indicated in patients with haemodynamically unstable torsades de pointes. This patient is haemodynamically stable.

IV amiodarone and IV lidocaine can be used to treat monomorphic ventricular tachycardia. Torsades de pointes is a polymorphic ventricular tachycardia.

Question #196

An 18-year-old male presents with shortness of breath. He states that it has gradually worsened over the past year. He denies cough, wheeze or chest pain. He has no past medical history of note. On examination you note a loud second heart sound. An ECG shows right bundle branch block (RBBB) with left axis deviation (LAD).

What is the most likely diagnosis?

- a) Coronary sinus atrial septal defect
- b) Ventricular septal defect
- c) Idiopathic pulmonary hypertension
- d) Chronic pulmonary emboli
- e) Ostium primum atrial septal defect

Correct answer is e.

Both primum and secundum atrial septal defects result in RBBB. The axis can be of use in distinguishing between ostium primum ASDs (usually have a LAD) and ostium secundums (usually have RAD).

The patient has presented with features of pulmonary hypertension, namely the progressive shortness of breath and loud second heart sound. The ECG is very suggestive of an atrial septal defect (ASD). Atrial septal defects result in RBBB. The

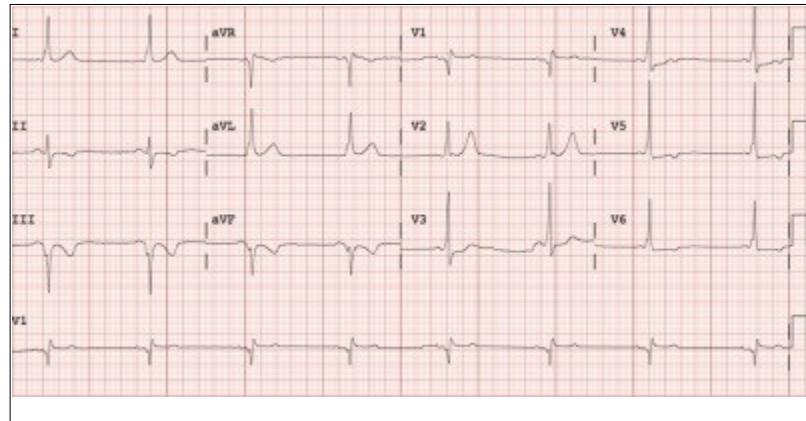
axis can be of use in distinguishing between ostium primum ASDs (usually have a LAD) and ostium secundums (usually have RAD). The ECG features in this case are therefore more suggestive of an ostium primum.

Furthermore, although ostium secundums are more common overall, they do not usually present as early as this. In contrast, ostium primum defects are usually much lower down in the septum and often involve the atrioventricular valves which accelerates the pathogenesis resulting in presentation at a younger age.

Discuss (11)Improve

Question #197

A 35-year-old man is investigated for recurrent palpitations associated with pre-syncopal symptoms and dyspnoea. Blood tests are unremarkable and a resting 12 lead ECG is shown below:



Given the likely diagnosis it is decided that radiofrequency ablation is the most appropriate treatment. What part of the heart should be ablated?

- a) Sinoatrial node
- b) Atrioventricular node
- c) Junction of right atrium and right ventricle

- d) Left atrial appendage
- e) Junction of left atrium and left ventricle

The ECG shows a short PR interval associated with a slurred upstroke (delta wave). Note the non-specific ST-T changes which are common in WPW and may be mistaken for ischaemia. The left axis deviation means that this is type B WPW, implying a right-sided pathway.

Discuss (5) Improve

Question #198

A 74-year-old man with a history of depression, chronic obstructive pulmonary disease and hypertension is admitted to hospital with severe central chest pain. He has no history of ischaemic heart disease or similar chest pains in the past. On admission blood pressure is 160/98 mmHg, pulse 110/min, respiratory rate 18/min and oxygen saturations are 93% on room air.

A CT chest (with contrast) is shown below:



What is the most appropriate management?

- a) Chest drain
- b) Cardiothoracic surgery
- c) Intravenous labetalol
- d) Thrombolysis with Tenecteplase
- e) Low-molecular weight heparin

Correct answer is c.

Dissection of the descending aorta can be seen. As this is a Stanford type B dissection it is treated conservatively with intravenous beta-blockers to lower blood pressure and help prevent any extension.

Discuss (7)Improve

Question #199

A 43-year-old lady with a history of schizophrenia presents to the emergency department with palpitations, headaches and dizziness for 3 days. She says that she can feel her heart pounding after which she becomes dizzy and feels faint. She

has noted these bouts about three to four times per day. She does not suffer from diabetes and is not hypertensive.

Regarding her headaches, she has had them for a long period of time and is quite convinced that there is some sort of sinister problem with her brain, although her doctors do not believe so. She has a younger sister who has had epilepsy for the last 15 years.

She was diagnosed as having a UTI 4 days ago which is currently being treated with ciprofloxacin. Her medication history includes olanzapine and occasional paracetamol for her headaches.

On examination, her pulse was 135 bpm with an irregular rhythm. Blood pressure was 90/60mmHg. Her systemic examination was unremarkable.

Attachment to a cardiac monitor revealed runs of ill sustained polymorphic tachycardia. She was immediately given IV lidocaine to which there was no response.

Which is the most appropriate next step in management?

- f) Immediate DC cardioversion
- g) IV amiodarone
- h) IV flecainide
- i) IV magnesium sulphate
- j) IV labetalol

Correct answer is d.

The scenario above is an acquired long QT syndrome secondary to both ciprofloxacin and olanzapine. Patients with long QT syndromes develop syncope and palpitations as a result of polymorphic ventricular tachycardia (torsades de pointes). Episodes may terminate spontaneously which is usual, but may also evolve into fatal ventricular fibrillation. The corrected QT interval in between the arrhythmia is usually >0.5s.

Question #200

A 71-year-old patient presents to the Emergency Department with a 30 minute history of crushing central chest pain. ECG shows tall R waves in V1-2. Which coronary territory is likely to be affected?

- f) Lateral
- g) Posterior
- h) Anteroseptal
- i) Anterolateral
- j) Inferior

Correct answer is b.

Question #201

A 54-year-old male business executive is referred to you after a heart murmur is detected at a medical examination he received after he transferred to a new company. He has no known past medical history or family history. He is well and leads an active lifestyle. On examination, you note a pansystolic murmur in the apex. Chest auscultation is unremarkable. An ECG demonstrates sinus rhythm at 64 beats/minute with no changes suggestive of ventricular hypertrophy. A transthoracic echocardiogram demonstrated good views with severe mitral regurgitation, preserved left ventricular function (EF 85%) and pulmonary arterial systolic pressure of 15 mmHg. Which of the following is appropriate management?

- f) Mitral valve replacement
- g) Mitral valve repair
- h) Percutaneous mitral valve repair (Mitraclip)
- i) Infective endocarditis prophylaxis and 6 monthly echocardiogram
- j) 6 monthly echocardiogram

Correc answer is e.

The patient has severe chronic non-ischaemic mitral regurgitation that has not decompensated. The aetiology is unclear: primary causes include mitral valve prolapse, flail leaflet or rheumatic heart disease. Secondary causes include cardiomyopathy or coronary artery disease. The patient does not meet indications for intervention on the mitral valve, which include symptoms, left ventricular dysfunction, pulmonary hypertension, new atrial fibrillation and dilated left ventricle. It is uncommon for intervention to take place without any of the indications mentioned above. Instead, serial monitoring with 6 monthly echocardiograms is appropriate. Infective endocarditis prophylaxis is no longer indicated for patients in the absence of a prosthetic valve repair or replacement.

Percutaneous mitral valve repair is an emerging technique that is available to patients considered too high risk for mitral valve surgery, which uses a device to individually approximate the regurgitant leaflet. Comparing Mitraclip with mitral valve replacement or repair in severe mitral regurgitation patients with NYHA III/IV heart failure symptoms, one-year and four-year survival were similar with similar improvements in symptoms, LV size and function. However, Mitraclip patients demonstrated significantly higher incidence of requiring further surgery but significantly lower risks of major adverse events post-procedure.

1. Feldman T, Foster E, Glower DD et al. Percutaneous repair or surgery for mitral regurgitation. *N Engl J Med*. 2011;364(15):1395

Question #202

A 60 year old man with a known history of congestive cardiac failure and asthma is reviewed in a cardiology clinic. He is noted to have a blood pressure of 95/63mmHg and a heart rate of 98bpm. An ECG confirms sinus rhythm. He has previously developed symptoms of wheeze with beta blockade. He is commenced on ivabradine 5mg twice daily by his cardiologist.

Which of the following should the patient be warned of as a recognised side effect of ivabradine?

- f) Neutrophilia
- g) Hypotension
- h) Phosphenes
- i) Diaphoresis
- j) Renal failure

Correct answer is c.

Ivabradine use may be associated with visual disturbances including phosphenes and green luminescence

Ivabradine is a second line agent for rate control in the treatment of chronic heart failure in those in whom beta-blockade is contraindicated, not tolerated or therapy fails. Other rate limiting drugs such as the calcium channel blockers diltiazem or verapamil may sometimes be used but hypotension often limits their use. Digoxin too can sometimes be used, even in sinus rhythm although the rate limiting effect it has is relatively modest; often digoxin and ivabradine are ultimately used in conjunction.

NICE guidelines on the use of ivabradine give clear criteria on its use; the patient must have at least a moderate degree of heart failure with a left ventricular ejection fraction of <35%, they must be stable on optimal doses of other heart failure medications including angiotensin system modulators, an aldosterone receptor antagonist and a beta blocker if not contraindicated. It is important the patient is regularly monitored by a community heart failure team to titrate the dose. Ivabradine can also only be used in patients in sinus rhythm. It works by selectively inhibiting the If [ionic funny] channel which is a sodium/potassium symporter channel largely expressed within the sinoatrial node. Inhibition of this channel slows the intrinsic rhythmicity of the hearts pacemaker function. Pharmaceutical bradycardia aids in the management of cardiac failure as it allows improved diastolic filling and reduced myocardial oxygen usage.

Side effects of ivabradine include bradycardia, ventricular escape rhythms, dizziness, headache, muscle cramps and eosinophilia. Neutrophilia is rarely seen with ivabradine. Hypotension is also not seen with ivabradine use which makes it an important drug in the arsenal when treating patients with cardiac failure in

whom other rate limiting drugs may be relatively contraindicated.

An important side effect with ivabradine however is that of visual disturbance including the phenomenon of phosphenes, or 'flashing lights'. Other visual symptoms described include green discolouration of visual field, blurring of vision and scintillating scotomata. These visual symptoms are usually transient and mild. They arise due to inhibition of similar ionic channels in the retina to the sinoatrial If channels. Approximately 20% of patients taking the drug develop some form of visual disturbance but only 1% need to discontinue therapy because of it.

The use of ivabradine in renal dysfunction is not well established however manufacturers recommend it may be used with estimated glomerular filtration rates above 15ml/min. There is no evidence its use causes renal dysfunction although mild rises in plasma creatinine levels may be seen.

Question #203

A 54-year-old woman presents to her general practitioner complaining of a rash on her chest, face and upper arms. The rash is transient and seems to mostly occur in the summertime. She has a past medical history of hypertension, palindromic rheumatism and heart failure with preserved ejection fraction. She takes amlodipine, furosemide, indapamide and hydroxychloroquine.

On examination, there is a maculopapular rash on her chest and distal forearms. It is not itchy.

Bloods tests:

Antinuclear antibody	negative
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What is the likely explanation for her symptoms?

- f) Furosemide
- g) Hydroxychloroquine
- h) Indapamide
- i) Solar urticaria
- j) Systemic lupus erythematosus

Correct answer is c.

Thiazides may cause photosensitivity

Indapamide is correct. Thiazide diuretics are a well-recognised cause of a photosensitive rash. The patient's rash occurs mostly in the summertime and on sun-exposed areas and is therefore consistent with this reaction.

Furosemide is incorrect. This is mainly associated with bullous skin reactions.

Solar urticaria is incorrect. The rash, in this case, is maculopapular and non-pruritic and therefore is not consistent with a diagnosis of urticaria.

Hydroxychloroquine is incorrect. This medication is associated with rashes but is actually used to treat conditions associated with photosensitive rashes and is therefore not the likely cause.

SLE is incorrect. Photosensitive rashes are common in lupus. However, the ANA is negative, which makes a diagnosis of the systemic form of lupus, overwhelmingly unlikely.

Question #204

A 52-year-old is brought from the renal outpatient clinic to the emergency department (ED) after appearing very short of breathing having walked up the stair. His breathlessness was associated with some mild, generalised chest discomfort, but the patient puts this down to a recent chest infection he has just recovered from.

When assessed in the ED the patient's breathlessness has improved and he denies any further chest discomfort. He reports he has been experiencing more frequent

similar episodes, gradually increasing in severity over several months. He reports he recently had a productive cough with coryzal symptoms for the last 3-4 days but he feels it is improving. On questioning he confirms his symptoms of breathlessness are often worse during and post these chest infections.

He has a past medical history of chronic renal artery stenosis and resultant poorly controlled hypertension for which he is on multiple medications including an ACE inhibitor, beta-blocker and a diuretic.

An ECG is performed as seen below.

What is the patient's most likely diagnosis?

- f) Brugada syndrome
- g) Hyperkalaemia
- h) Left ventricular hypertrophy
- i) Pericarditis
- j) Wellen's syndrome

Correct answer is c.

This patient has presented with symptoms and typical ECG changes in keeping with left ventricular (LV) hypertrophy. The thickening of the cardiac muscle is most likely a result of chronic uncontrolled hypertension, hence the prolonged, progressive history. LV hypertrophy results in signs and symptoms of LV failure including breathlessness, angina and reduced exercise tolerance. The patient's ECG demonstrates left ventricular hypertrophy with ST elevation in V2-3, without reciprocal lead changes and the combination of the S wave in V1 with the R wave in V5 or V6 clearly exceeding 40 mm.

Brugada syndrome is a genetic cardiac disorder resulting in electrical activity disorders and not structural issues. Features include syncope and sudden cardiac death. There are several types of Brugada syndrome with variation seen on ECG

however the only potentially diagnostic ECG abnormality is of coved ST-segment elevation in 2 or more of V1-3 followed by a negative T wave. This is classical of Brugada type 1 syndrome and is commonly known as Brugada sign.

There is no clear reason why this patient would be hyperkalaemia. Although he has renal impairment it is not indicated that he has end-stage renal failure or that he is on renal replacement therapy therefore hyperkalaemia would not be expected. Common ECG changes noted in hyperkalaemia included peaked T waves, P wave widening/flattening, PR prolongation progressing to the bradyarrhythmias and conduction blocks. No clear features of hyperkalaemia can be seen on this patient's ECG.

Pericarditis is inflammation of the tissue surrounding the heart and can be due to infections. Patients normally present with pleuritic, constant chest pain and fevers. Although this patient has a very recent chest infection, pericarditis normally develops a few weeks post the resolution of infection as it is an immune response and not a direct result of the infection itself. ECG findings associated with pericarditis include widespread concave ST elevation and PR depression which are not present on this patient's ECG.

Wellen's syndrome is the ECG pattern of biphasic or deeply inverted T waves in the chest leads V2-3. It is highly specific for critical stenosis of the left anterior descending (LAD) artery and therefore is normally seen in patients presenting with ischaemic like symptoms. You would expect the patient's chest pain to be continuous and to be more severe if he was experiencing stenosis of the LAD.

Question #204

A 45-year-old woman develops severe central chest pain. An ECG in the ambulance shows ST segment elevation in leads: I, aVL, V2-6. Shortly after she arrives in hospital the pain resolves and a second ECG is entirely normal. She has had three similar episodes of chest pain in the past. All episodes of chest pain have come on at rest. Blood pressure is 140/80 mmHg, heart rate is 90 beats per minute and heart sounds are normal. She underwent coronary angiography following a previous episode of chest pain three weeks ago, which showed no significant coronary artery disease. An echocardiogram is normal.

What is the likely diagnosis?

- f) Acute anterolateral myocardial infarction
- g) Crescendo angina
- h) Da Costas syndrome
- i) Prinzmetal's variant angina
- j) Takotsubo cardiomyopathy

Correct answer is d.

This woman is likely to be suffering from Prinzmetal's variant angina. Classically her pain occurs at rest and the ECG demonstrates ST segment elevation that disappears as the pain abates. Normal coronary angiography supports this diagnosis, however in many Prinzmetal's angina patients there is co-existing coronary artery disease. Symptoms and ECG changes are unlikely to be reproduced with exercise testing in Prinzmetal's angina.

Prinzmetal's angina is caused by coronary artery spasm, however the underlying pathophysiology causing spasm is not currently well understood. In some patients it is associated with other vasospastic disorders such as Raynaud's phenomenon.

The main stay of treatment is:

- Avoiding precipitants of spasm such as smoking.
- Calcium channel blockers, nitrates and/or nicorandil.

Question #205

A 16-year-old female presents with a swollen knee. She states that she has had several similar episodes previously. She also complains of excessive bleeding after dental work. She states that her father has a bleeding disorder. On examination her right knee is hot and swollen. A knee aspirate reveals a bloody effusion.

Blood results are as follows:

Hb	110 g/l
Platelets	$682 * 10^9/l$
Prothrombin time (PT)	12 s (normal 10-13)
activated partial thromboplastin time (aPTT)	54 s (normal 25-36)
Factor VIII level	2% of normal (very low)
Factor IX level	Normal
von Willebrand Factor level	Normal

What disorder does she most likely have?

- f) Down syndrome
- g) Edwards syndrome
- h) Marfan syndrome
- i) Ehlers-Danlos syndrome
- j) Turner's syndrome

Correct answer is e.

Females with Turner's syndrome have just one X chromosome. Therefore they have the same probability of being affected by an X linked recessive disease as males

The patient clearly has haemophilia A as indicated by the prolonged aPTT and severely reduced factor VIII levels. Rare cases of females with severe haemophilia can occur due to extreme lyonization, homozygosity, mosaicism, or Turner

syndrome.

Females with Turner's syndrome have just one X chromosome. Thus they have the same probability of being affected by an X linked recessive disease as males. For the aforementioned reason, Turner's syndrome is the most likely underlying diagnosis out of the given options.

Question #206

A 75-year-old man is an inpatient on the orthopaedic ward recovering from an elective knee replacement performed three days previously. The patient's immediate post-operative recovery had been unremarkable and he had begun to mobilise with the ward physiotherapist. Routine observations recorded on the ward had been unremarkable during the previous 24 hours.

During her drug round, the patient's nurse saw the patient suddenly become unable to breathe and clutch at his chest. After calling for help the nurse went to her patient and found him to be in cardiac arrest. Resuscitations attempts were initiated following advanced life-support protocol. Please see the below table for a summary of the patient's electrical rhythm and treatments administered during the initial phases of the resuscitation attempt.

The patient was noted to be in good physical health with his only comorbidity being hypertension, well controlled with medication. The patient's admission clerking recorded that he was a retired schoolteacher who lived independently at home with his wife. A review of the patient's drug chart indicated that he had been receiving subcutaneous enoxaparin as prophylaxis against venous thromboembolism, but that the patient had refused to wear compression stockings during his admission as he found them uncomfortable.

Number of rhythm check	Result of rhythm check	Treatment administered
1	Pulseless electrical activity	IV adrenaline 1 mg

Number of rhythm check	Result of rhythm check	Treatment administered
2	Ventricular fibrillation (coarse)	DC shock 150 J
3	Ventricular fibrillation	???

In addition to a further DC shock, what is the appropriate choice of IV drug treatment following the third rhythm check?

- f) IV adrenaline 1 mg
- g) IV atropine 400 micrograms & IV amiodarone 300 mg
- h) No IV drug treatment indicated
- i) IV adrenaline 1 mg & IV amiodarone 300 mg
- j) IV atropine 400 micrograms

Correct answer is a.

The advanced life support algorithm for pulseless electrical activity (PEA) and asystole requires adrenaline to be given immediately and then continued every 3 to 5 minutes (in practice, after every second rhythm check). By contrast, the algorithm for ventricular fibrillation and ventricular tachycardia (VF / VT) requires IV adrenaline to not be given until after the third shock and then continued every 3 to 5 minutes.

However, once adrenaline has been given during a resuscitation attempt it should be given every 3-5 minutes even if the type of rhythm changes from PEA/asystole to VF / VT, even if this is only the second cycle of the rhythm being VF / VT (as in the example in the question).

As part of the VF / VT algorithm, IV amiodarone 300 mg should be given after three defibrillation attempts. A further dose of IV amiodarone 150 mg should be considered after a total of five defibrillation attempts. Atropine is not used as part of the advanced life-support algorithm.

Question #207

A 64-year-old man presents to his general practitioner (GP) with several months of chest pain that occurs only on exertion. When he pauses to rest, the pain dissipates within a few minutes. He has a past medical history of high cholesterol, for which he takes atorvastatin. The GP commences him on bisoprolol.

Several weeks later, the patient re-attends, stating that the symptoms are still not under control.

What should be added next?

- f) Amlodipine
- g) Isosorbide mononitrate
- h) Ivabradine
- i) Nicorandil
- j) Verapamil

Correct answer is a.

If angina is not controlled with a beta-blocker, a longer-acting dihydropyridine calcium channel blocker should be added

This patient is experiencing stable angina - the chest pain is occurring on exertion, but not at rest. NICE recommend the use of a beta-blocker or calcium channel blocker (CCB) first-line. As this patient has been commenced on bisoprolol already, the next step is to add a CCB. A dihydropyridine CCB should be used, to avoid reducing the heart rate further, and so **amlodipine** is the most appropriate option here.

Isosorbide mononitrate would be appropriate to use as a third agent, if the combination of a beta-blocker and CCB was not adequate. However, at this stage, a CCB should be added.

Similarly, **ivabradine**, which modulates the 'funny current', would be appropriate as a third agent instead of isosorbide mononitrate. A CCB should be added first, as

this patient is only on bisoprolol.

Nicorandil is another alternative agent to be added third, instead of isosorbide mononitrate or ivabradine. It would not be appropriate to add currently, as the patient is only on bisoprolol.

Verapamil is a non-dihydropyridine CCB. As such, it is negatively chronotropic and so should not be used alongside a beta-blocker; this may precipitate complete heart block. It would have been suitable as a first-line alternative to the beta-blocker.

Question #208

An 89-year-old gentleman presented to the general medical clinic with complaints of palpitations. He was diagnosed with atrial fibrillation (AF) three months ago and started on bisoprolol. This was part of a plan to rate control, rather than rhythm control, his AF. The bisoprolol has been up-titrated as his symptoms did not come under control with his dose. Palpitations occur almost every day but are not associated with any other symptoms. He also has a history of ischaemic heart disease with a myocardial infarction five years ago and type 2 diabetes mellitus. He currently takes bisoprolol, metformin, aspirin and simvastatin. He has declined anticoagulation with warfarin or NOAC despite the risk as his wife died from an intracranial bleed whilst taking warfarin.

On examination, his heart rate is 94/min and irregular. His chest is clear on auscultation and there is no peripheral oedema. How should he be further managed?

- f) Amiodarone
- g) Dronedarone
- h) Amlodipine
- i) Diltiazem
- j) Left atrial ablation

Correct answer is d.

The correct answer is diltiazem. This is a gentleman with symptomatic, but not

decompensating, permanent AF which is not responding to first-line treatment with a beta-blocker (bisoprolol). NICE advises that first line treatment for a rate-control strategy in AF should be either a beta-blocker or a rate-limiting calcium channel blocker, but digoxin can be considered for non-paroxysmal AF in patients who are not very active. If the first-line treatment fails, either with continuing symptoms or poor response of ventricular rate then a combination therapy with any of the following two can be used: beta-blocker, diltiazem, digoxin. Amiodarone, dronedarone and left atrial ablation are all strategies for cardioversion. Amlodipine is a calcium channel blocker but is used in hypertension rather than in AF as it is not rate-limiting, and would therefore not help.

Question #209

A 29-year-old man is admitted to the cardiology ward following a collapse whilst playing football. He has no recollection of events other than running on the pitch and waking up in the ambulance. In the last 2-3 months, he has been complaining of intermittent palpitations but put this down to work-related stress. There is no history of chest pain and he has no past medical history of note.

His observations are recorded within normal limits. On auscultation, chest sounds are clear and heart sounds are normal. He has a soft and non-tender abdomen and his neurological examination is unremarkable.

A bedside echocardiogram demonstrates a hypokinetic right ventricle.

Given the likely diagnosis, what is the most characteristic finding on this patient's ECG?

- f) Left ventricular hypertrophy
- g) Positive deflection at the J point
- h) Right bundle branch block
- i) Slurred upstroke in QR
- j) Small positive deflection at the end of QRS complex

The most characteristic ECG finding in arrhythmogenic right ventricular dysplasia (ARVD) is the epsilon wave (a small positive deflection at the end of the QRS complex)

This patient has arrhythmogenic right ventricular dysplasia (ARVD), a type of inherited cardiovascular disease. ARVD can present as palpitations, syncope, or sudden cardiac death similar to hypertrophic obstructive cardiomyopathy (HOCM). Over time, the right ventricular myocardium is replaced by fibrofatty tissue and patients develop signs of right ventricular failure. This patient's echocardiogram finding of a hypokinetic right ventricular wall is suggestive of the diagnosis. The most characteristic ECG finding in ARVD is an **epsilon wave**. An epsilon wave is a positive deflection following the end of the QRS complex caused by post-excitation of right ventricular myocytes.

Left ventricular hypertrophy is a common ECG finding of HOCM. HOCM is the most common cause of sudden cardiac death and can also present with palpitations and syncope, as seen in this patient. However, this patient's echocardiogram findings are in keeping with ARVD, of which the epsilon wave is the most characteristic ECG finding.

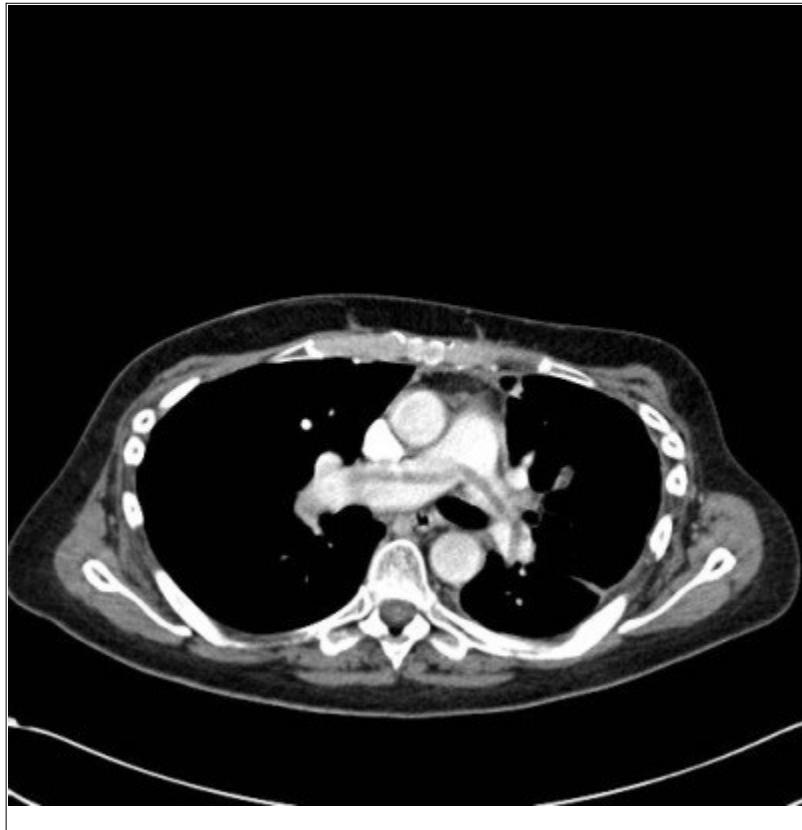
An **Osborn wave (or J wave)** is a positive deflection at the J point. There are multiple causes, of which the most common is hypothermia. Other causes of J waves include left ventricular hypertrophy and Brugada syndrome. However, an Osborn wave is not associated with ARVD.

There are multiple causes of **right bundle branch block**, including right ventricular hypertrophy. In ARVD, rather than hypertrophy of the right ventricle, the myocardium is replaced by fatty tissue. Right bundle branch block is not a sign of ARVD. Instead, the left bundle branch block is more likely to feature on an ECG of a patient with ARVD.

A **delta wave** is a slurred upstroke in the QRS complex that is commonly associated with pre-excitatory activity, such as that seen in Wolff-Parkinson White syndrome. It is not associated with ARVD.

Question #210

A 72-year-old man is admitted to hospital with shortness-of-breath. On examination his pulse is 96/min, BP 100/64 mmHg, respiratory rate 20/min, temperature 37.5°C, oxygen saturations 96% on room air. A 12-lead ECG shows sinus rhythm, at a rate of 94/min with no diagnostic ST-T changes. The troponin I level is < 0.05 µg/L. A CT chest (with contrast) is ordered:



What is the most likely diagnosis?

- f) Superior vena cava obstruction
- g) Aortic dissection (Stanford type A)
- h) Infective endocarditis
- i) Aortic dissection (Stanford type B)
- j) Pulmonary embolism

Correct answer is e.

The CT shows a large saddle embolus where the pulmonary trunk splits to form

the right and left pulmonary arteries.

Most image-based questions can be answered without looking at the image. If we therefore concentrate on the history a few clues point towards a pulmonary embolism (PE). The first is tachypnoea. Around 95% of patients with a PE have a respiratory rate > 16/min. Tachypnoea is not as common in any of the other diagnoses. A low-grade temperature is also an under appreciated sign of pulmonary embolism. This could of course be consistent with infective endocarditis but there are no other features to support this diagnosis.

Question #211

You are the on-call medical doctor called to review a patient in the Emergency Department. A 54 year old male patient with a past history asthma, ischaemic heart disease and transient ischaemic attack has presented with palpitations. His admission ECG shows a regular, narrow complex tachycardia. Vagal manoeuvres have been tried in the department with no success. You decide to give intravenous adenosine in an attempt to chemically cardiovert. An initial dose of 6mg is given into a proximal vein with a large flush. The patient soon loses consciousness and an 11 second ventricular standstill is noted on the rhythm strip before slow return of sinus rhythm.

Which of the patient's medications is most likely to be responsible for this?

- f) Phyllocontin
- g) Dipyridamole
- h) Bisoprolol
- i) Simvastatin
- j) Montelukast

Correct answer is b.

The effects of adenosine are enhanced by dipyridamole

Adenosine is a useful medication in the investigation and treatment of regular narrow complex tachycardias. With reference to the Resuscitation UK guidelines, it has a place as a second line measure to vagal manoeuvres when no 'adverse

'features' are present. Adenosine works by transiently blocking the AV node by effect on the A1 receptor, causing potassium efflux and hyperpolarisation.

Dont be side-tracked by the fact that this patient is asthmatic, yes this is a relative contraindication to the administration of adenosine, but that is not the question. Theophyllines have been known to antagonise adenosine necessitating higher dosages. Dipyridamole blocks the cellular uptake of adenosine, increasing concentration at receptors and potentiating its effect. Adenosine should therefore be used with caution, if at all, in patients taking dipyridamole, and certainly low starting doses should be considered.

Question #212

A 78-year-old lady presents with syncope. She states that she has had several episodes of light headedness over the past couple of months. There are no obvious precipitants of the dizzy spells. She has a past medical history of angina, COPD and hypertension. Her regular medicines include GTN, aspirin, ramipril and furosemide.

ECG results are as follows:

Rate	68 beats per minute
Rhythm	Sinus rhythm
PR interval	220 ms
QRS duration	130 ms
QRS morphology	Right bundle branch block
Cardiac axis	Left axis deviation

What investigation will you order?

- f) 7-day ECG Holter monitor
- g) Transthoracic echocardiogram
- h) Transesophageal echocardiogram
- i) Brain natriuretic peptide (BNP)
- j) Tilt-table test

Correct answer is a.

Patients who present with syncope and have an ECG showing incomplete trifascicular block need a Holter test to assess for episodes of complete heart block

The right bundle branch block (RBBB), left axis deviation (LAD) and prolonged PR interval are suggestive of incomplete trifascicular block. These patients require a 7-day ECG Holter monitor to assess for episodes of complete (third degree) heart block. It is likely that a permanent cardiac pacemaker will be required to prevent further syncopal episodes.

A tilt-table test is useful for investigating vasovagal syncope.

The patient may also have underlying structural heart disease so a transthoracic echo would also be prudent to perform. However, the most immediate concern is of arrhythmia, making a 7 day holter the better answer.

Question #213

A 78-year-old man who is usually fit and well and takes levothyroxine for hypothyroidism presents to the acute medical take after two episodes of syncope. He recalls sitting at the dinner table and passing out with no dizziness prior. He can usually walk 100 metres at a time.

On examination, he has a loud systolic murmur heard over the 2nd intercostal space on the left side radiating to the carotid. He has a slow rising pulse and his

chest is clear. No JVP is seen and there is no ankle oedema.

Chest x-ray	enlarged heart with calcification of the aortic knuckle
ECG	sinus, 75 beats per minute
ECHO	Aortic valve cross sectional area of 0.8mm^2 with a pressure gradient of 42mmHg, cusps appear calcified and poorly mobile

What other investigation should be performed?

- f) Coronary angiography
- g) Thyroid function test
- h) CT chest
- i) Treadmill ECG testing
- j) HbA1c

Correct answer is a.

Aortic stenosis co-occurs with atherosclerotic disease -> perform angiogram prior to surgical intervention

This gentleman meets the ECHO criteria for severe aortic stenosis and should be listed for aortic valve replacement (AVR) immediately. He shows no signs of failure or haemodynamic compromise and is therefore fit. Given that AVR involves opening the chest, it is a perfect time to perform coronary artery bypass grafting and since there is a correlation of aortic stenosis with atherosclerosis, it is beneficial to perform an angiogram prior to the AVR procedure. The patient effectively gets two procedures for one. CT chest would be important if he had respiratory disease. HbA1c and thyroid function tests are not essential unless there was a suspicion that these were abnormal. Treadmill testing is contraindicated in symptomatic and severe aortic stenosis where it can cause syncope.

Question #214

A 72-year-old female presents with a 6-month history of gradual onset, progressive exertional dyspnoea associated bilateral increased lower limb pitting oedema. She has no previous cardiac history and prior to 6 months ago, had no limitations to exercise tolerance. Her past medical history includes B cell lymphoma diagnosed four years ago and in remission one course of chemotherapy. On examination, the jugular venous pulse is raised at 6cm above the angle of Louis. A soft systolic murmur and bibasal inspiratory crackles can be heard on auscultation. Abdominal examination demonstrates a pulsatile 3cm liver edge. Her blood pressure is 125/77 mmHg, heart rate 68/min and sinus, saturations 92% on 2l, respiratory rate 22/min. ECG reveals sinus rhythm of low voltage. Chest radiography demonstrates bibasal alveolar shadowing with prominent upper lobe vasculature.

Urine dip is as follows: blood -ve, leucocytes -ve, nitrates -ve protein 3+ pH 5.5.

Blood tests are as follows:

Hb	111 g/l
Platelets	$275 * 10^9/l$
WBC	$8.3 * 10^9/l$

Na^+	139 mmol/l
K^+	4.9 mmol/l
Urea	7.4 mmol/l

Creatinine	140 µmol/l
CRP	3 mg/l
Trop	<0.03 (normal range < 0.03)

Bilirubin	6 µmol/l
ALP	32 u/l
ALT	260 u/l

A transthoracic echocardiogram is performed, reported as significant increase in echogenicity, symmetrical left ventricular wall thickening, impaired bilateral diastolic ventricular dysfunction, bilaterally enlarged atria, pulmonary artery systolic pressure 45 mmHg, a 5mm pericardial effusion

What is the most likely underlying diagnosis for this patient?

- f) Amyloid cardiomyopathy
- g) Non-ST elevation myocardial infarction (NSTEMI)
- h) Idiopathic dilated cardiomyopathy
- i) Chemotherapy induced cardiomyopathy
- j) Viral pericarditis leading to tamponade

Correct answer is a.

A clinical picture of decompensated congestive cardiac failure is described with subtle details of hepatomegaly (possible congested, possibly related to the underlying diagnosis), associated heavy proteinuria, echogenic TTE and low voltage criteria ECG, on a background of previous B-cell lymphoma. The unifying

diagnosis is amyloid cardiomyopathy, likely secondary to AL amyloid from B-cell dyscrasia, with cardiac, renal and possibly liver involvement. There is no evidence of a sudden onset cardiac event suggestive of an ischaemic cause. Echocardiogram does not demonstrate ventricular dilation, only impaired function. A 5mm pericardial effusion is not sufficiently significant to cause tamponade. Although chemotherapy-induced cardiomyopathy is possible, it is difficult to tie in the delayed onset from the end of treatment with renal involvement. It also does not explain the echogenic pictures on transthoracic echo.

Question #215

A 35-year-old woman presented to the emergency department with intermittent palpitations, breathlessness, and non-specific chest discomfort. The previous day she had found out that she was 14-weeks pregnant which had caused significant stress and anxiety. She has a long history of anxiety and depression, managed by cognitive behavioural therapy. She reported that she had intermittently had episodes of palpitations and shortness of breath for the past 10 years and had attributed this to her anxiety. These symptoms had worsened over the past couple of weeks and today her symptoms were intolerable. Her medications consisted of over-the-counter vitamin supplements.

She looked anxious, but otherwise, the examination was unremarkable.

An ECG was performed:

ECG #1: Normal sinus rhythm. T wave inversion in V1 - V3.

Whilst in the emergency department, she complained of severe palpitations and dizziness.

Her ECG was repeated:

ECG #2: Wide complex regular tachycardia with left bundle branch block (LBBB) pattern. Heart rate 150bpm. Fusion beats and capture beats present.

As she was being transferred into resus, she spontaneously cardioverted back to

sinus rhythm.

An echocardiogram was performed:

The right ventricle is dilated with depressed right ventricular systolic function and localised apical aneurysm. Left ventricular size and function are normal.

What is the most likely cause of her presentation?

- f) Aortic dissection
- g) Takutsubo cardiomyopathy
- h) Peripartum cardiomyopathy
- i) Arrhythmogenic right ventricular cardiomyopathy
- j) Brugada syndrome

Arrhythmogenic right ventricular cardiomyopathy - T wave inversion in V1-3

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is characterised by the replacement of the myocardium of the right ventricle with fibrofatty tissue. The abnormal fibrofatty tissue predisposes to ventricular tachycardia (VT), right ventricular systolic dysfunction, and can cause sudden death. It is often inherited but can be sporadic. Presentation is often with nonspecific palpitations, chest discomfort, and syncope. This patient has a typical presentation: the pregnancy-associated increase in stroke volume is likely the trigger for her symptoms recently worsening. Her two ECGs are characteristic: typical changes include inverted T waves in the precordial leads V1-V3 (looking at the right ventricle) with episodes of ventricular tachycardia originating in the right ventricle (giving the ECG a LBBB morphology as the arrhythmia takes an abnormal route of conduction from right to left through slowly-conducting myocardium rather than conducting tissue). Epsilon waves may also be seen in ARVC. Her echocardiogram is also characteristic - infiltration and thinning of the right ventricular wall can occur to the point that systolic function is impaired and an aneurysm forms.

Note that the differential for a wide-complex regular tachycardia on an ECG

includes SVT with aberrant conduction (as well as VT). However, the fusion beats and capture beats are more in keeping with VT in this case.

This patient's presentation is not typical for aortic dissection. An echo might see cardiac tamponade or new aortic regurgitation if this was the diagnosis but this is not the case here.

Takutsubo cardiomyopathy is caused by transient systolic dysfunction of the left ventricle (LV), presenting similarly to an acute coronary syndrome. Patients typically have chest pain, a raised troponin, and ECG findings consistent with ischaemia such as ST elevation/T wave inversion. This is typically triggered by recent physical or emotional stress. Patients are initially managed as an ACS but cardiac angiography shows no significant coronary artery disease. Instead, the LV wall shows hypokinesis or akinesis which creates the shape of an 'octopus pot' during systole. It may be argued that this patient has experienced a recent stressor (the news of pregnancy) however her presentation, long history of symptoms, and echo findings are more in keeping with ARVC.

Peripartum cardiomyopathy (PPCM) is a rare type of cardiomyopathy that is often dilated and occurs in late pregnancy or early postpartum (typically one month before or after birth). The aetiology is unclear but is thought to be multifactorial. Criteria for diagnosis of PPCM include impairment of LV systolic function (LVEF <45%) which was not the case in this patient. Furthermore, this patient presented in the early second trimester - too early for PPCM but late enough for pregnancy-related haemodynamic changes to trigger her underlying ARVC.

Lastly, Brugada syndrome is another important differential to consider in young patients with palpitations, ECG changes in V1-V3, and episodes of VT. Brugada is caused by an inherited mutation in the cardiac sodium channel. However, echocardiography is typically normal and an ECG may show unusual ST elevation in V1-V3 with T wave inversion in these leads.

Question #216

You are called to review a 65-year-old man who was admitted following a pre-syncopal episode. He has a history of previous NSTEMI 3 years ago. His nurse

explains he is feeling lightheaded and feels his heart racing in his chest.

On arrival, he appears sweaty and unwell. He opens his eyes to pain, localises to pain, and makes incomprehensible sounds. His pulse is palpable at 180 bpm with BP 88/54 mmHg.

ECG demonstrates a wide complex monomorphic tachycardia with prolonged QRS 150msec.

What is the most appropriate next step?

- f) Adrenaline
- g) Amiodarone
- h) Magnesium sulphate
- i) Synchronised DC cardioversion
- j) Unsynchronised DC cardioversion

Correct answer is d.

A synchronised cardioversion is the treatment for an unstable patient in VT

Synchronised DC cardioversion is the preferred treatment for tachyarrhythmia in unstable patients displaying life-threatening adverse signs. In this situation, unstable VT with a pulse should be managed with synchronised DC cardioversion. To convert atrial or ventricular tachyarrhythmias, the shock must be synchronised to occur with the R wave of the ECG. An unsynchronised shock could coincide with a T wave and cause ventricular fibrillation (VF). Please see adult Advanced Life Support guidelines.

Adrenaline 1mg 1:10000 is used in the ALS algorithm in the treatment of unconscious patients with shockable and non-shockable rhythms. In the ALS algorithm, shockable rhythms include pulseless VT and VF. In this case, the patient is unconscious with VT and a pulse can be felt.

While **amiodarone** can be used to treat ventricular tachycardia, it would not be the most appropriate next step here, as this patient has signs of unstable VT,

which must be managed with synchronised DC cardioversion in the first instance. Amiodarone is also used as part of the ALS algorithm for the treatment of patients with pulseless VT or VF. However, given the palpable pulse, synchronised DC cardioversion would be more appropriate here.

Magnesium sulphate is the treatment of choice in broad-complex polymorphic tachycardia. Given this patient is unstable with a monomorphic VT, synchronised DC cardioversion would be the most appropriate next step.

Unsynchronised DC cardioversion would be used in the treatment of patients with pulseless VT or VF. Given the palpable pulse, synchronised DC cardioversion would be more appropriate.

Question #217

A 26-year-old female presents with a third episode of palpitations associated with shortness of breath and chest discomfort. She has no other past medical history, thyroid function tests unremarkable. She denies taking any recreational drugs and has no significant family history. Her ECG is as follows:

Which of the following drugs would be safe to administer immediately if she becomes tachycardic?

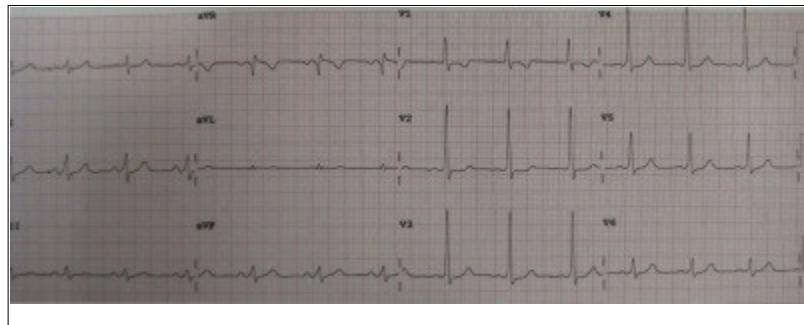
- a) Adenosine
- b) Digoxin
- c) Diltiazem
- d) Verapamil
- e) Procainamide

correct answer is e.

Question #218

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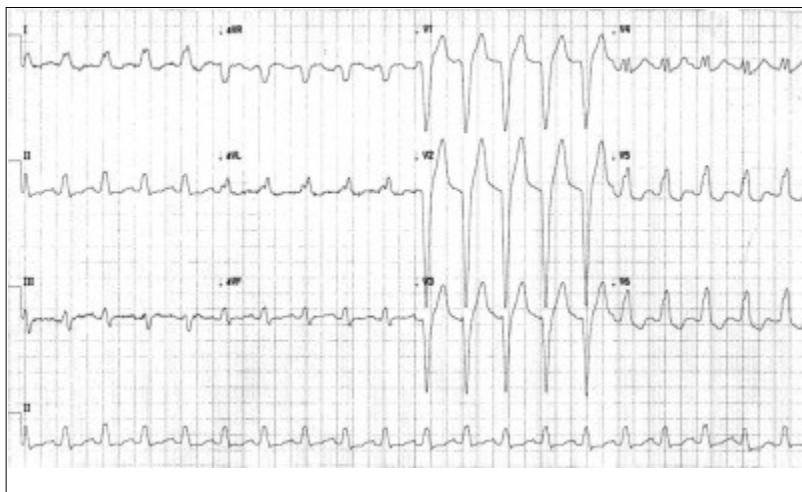
Correct answer is e.

The ECG demonstrates Wolff-Parkinson-White (WPW) syndrome, with a delta wave upstroke as a result of pre-excitation prior to the QRS complex. WPW is an atrioventricular re-entry tachycardia, with an accessory pathway allowing rapid conduction between the two channels, bypassing the slow-conducting AV node. Any drug that increases the delay of the normal conduction pathway via the AV node increases conduction via the accessory pathway, potentially inducing ventricular fibrillation or tachycardias. The only drug that does NOT act on the AV node in this setting is procainamide.

Question #219

A 72-year-old woman presents with palpitations. Her past medical history includes ischaemic heart disease and chronic obstructive pulmonary disease. Her pulse is 120/min, blood pressure 110/76 mmHg and the chest is clear on auscultation.

The ECG is shown below:



What is shown on the ECG?

- f) Left bundle branch block
- g) Anterior ST elevation myocardial infarction
- h) Right bundle branch block
- i) Narrow complex tachycardia
- j) Atrial flutter

Correct answer is a.

The morphology of the QRS complexes is diagnostic for left bundle branch block (LBBB). This finding can be associated with a wide variety of underlying problems but in an acutely unwell patient myocardial ischaemia needs to be excluded.

Question #220

A 46-year-old man is referred to the acute medical assessment unit with a fever of 38.9 °C. He reports a three-week history of general malaise, reduced appetite, intermittent sweating, and occasional rigors. The patient has no medical history and has had no previous operations or procedures. He denies any drug allergies.

His observations are:

- Respiratory rate: 24/min
- Oxygen saturation: 95% on room air
- Heart rate: 105bpm
- BP: 101/62mmHg
- Temperature: 38.9 °C
- GCS: 15/15

On examination, the patient has a loud early diastolic murmur over the right second intercostal space. Splinter haemorrhages are noted in three of his fingernails.

What is the most appropriate antibiotic to start?

- f) Amoxicillin
- g) Benzylpenicillin
- h) Flucloxacillin
- i) Vancomycin + low-dose gentamicin
- j) Vancomycin + low-dose gentamicin + rifampicin

Correct answer is a.

IV amoxicillin is the empirical treatment of choice in native valve endocarditis

Fever + a new cardiac murmur raises suspicion of infective endocarditis. A loud early diastolic murmur over the right second intercostal space is suspicious for aortic regurgitation secondary to endocarditis. This will cause the patient's shortness of breath.

Amoxicillin is the correct answer. Given the patient has no medical or surgical history, he should be treated for native valve endocarditis by starting high-dose intravenous amoxicillin. Ideally, six sets of blood cultures should be sent, with as

many as possible before the initiation of treatment (although this should not delay treatment if the patient is unstable).

Benzylpenicillin is the incorrect answer. Benzylpenicillin is the treatment for infective endocarditis caused by a fully sensitive *Streptococcus* organism.

Flucloxacillin is the incorrect answer. Flucloxacillin is used to treat infective endocarditis caused by a fully sensitive *Staphylococcus* (usually *Staphylococcus aureus*.)

Vancomycin + low-dose gentamicin is the incorrect answer. Vancomycin + low-dose gentamicin can be used for infective endocarditis in penicillin-allergic patients or patients with methicillin-resistant *Staphylococcus aureus* (MRSA) infective endocarditis.

Vancomycin + low-dose gentamicin + rifampicin is the incorrect answer. Vancomycin + low-dose gentamicin + rifampicin is the treatment for infective endocarditis on a prosthetic valve. This patient has no history of valve replacement.

Question #221

A 30-year-old man presents with a fever. He complains that he has had a fever, headache and lethargy for three weeks. On examination, his temperature is 38.5C. You notice a healed human bite mark on his right forearm, he tells you he was bitten in a pub brawl about a couple months ago. Diastolic and systolic murmurs are heard on auscultation. A trans-oesophageal echo demonstrates an oscillating mass on a bicuspid aortic valve, with aortic regurgitation. Three blood cultures are taken before administration of empirical antibiotics, two grow small colonies of tiny pleomorphic gram-negative bacilli.

What is the likely causative organism?

- f) *Eikenella corrodens*
- g) *Escherichia coli*

- h) *Phoenicoparrus andinus*
- i) *Staphylococcus aureus*
- j) *Streptococcus viridans*

Correct answer is a.

This patient fits the Duke criteria for infective endocarditis as there is: echocardiographic evidence of an intra-cardiac mass associated with a valve, a fever of >38 and two blood cultures of an endocarditis causing microorganism. Additionally, this patients bicuspid aortic valve is a risk factor for developing endocarditis.

Eikenella corrodens is a gram-negative bacilli, which is a commensal of the human mouth. It is a member of the HACEK group, an acronym for a group of organisms that can cause gram-negative endocarditis (*Haemophilus* species, *Actinobacillus actinomycetemcomitans*, *Cardiobacterium hominis*, *Eikenella corrodens*, and *Kingella* species.) The human bite injury and gram-negative culture make *Eikenella corrodens* the most likely causative organism.

Streptococcus viridans and *Staphylococcus aureus* are commoner causes of infective endocarditis, which are easily cultured and are gram-positive.

Escherichia coli could cause endocarditis and is a gram-negative bacilli, but is not the most likely in this case.

Phoenicoparrus andinus is the binomial nomenclature for the rare Andean Flamingo, which are not known to cause infective endocarditis.

Question #222

A 67-year-old male presents with increasing chest pain on exertion over the past 3 weeks, stopping him from going to work. His past medical history includes hypertension, type 2 diabetes mellitus, sick sinus syndrome, recent abstinence from alcohol after a history of excess (with an episode of acute liver decompensation 9 months ago) and stable angina. He previously tried isosorbide mononitrate but reported significant headaches and facial flushing. He has not previously suffered myocardial infarctions, with a recent echo demonstrating 70%

ejection fraction. An ECG demonstrates first-degree heart block with normal QRS complexes at 50 beats/ minute, blood pressure is 140/76 mmHg. He currently takes bisoprolol 5mg OD alone and has been using his GTN spray with increasing frequency without effect. What is the most appropriate next step in management?

- f) Reperfusion therapy with coronary artery bypass graft or percutaneous coronary intervention
- g) Ranolazine
- h) Ivabradine
- i) Diltiazem
- j) Nicorandil

Correct answer is e.

NICE recommends the routine prescription of a short-acting nitrate for stable angina, followed by either a beta blocker or calcium channel blocker as first-line therapy with insufficient symptomatic control. Next, NICE recommends combining both beta blockers and calcium channel blockers. Patients requiring more than 2 antianginals should be considered for reperfusion therapies¹, with the addition of a third drug only if the patient is not a candidate for PCI or CABG.

In this case, the patient has first-degree heart block with relative bradycardia and calcium channel blockers would be contraindicated. NICE recommends the addition of either ranolazine, nicorandil, ivabradine or a long-acting nitrate. Sick sinus syndrome would be a contraindication to ivabradine and calcium channel blocker. In addition, alcohol excess and recent decompensation, despite current abstinence, is likely to have resulted in liver dysfunction, an absolute contraindication to ranolazine. Nicorandil is only contraindicated in LV failure and cardiogenic shock, acting as a potassium channel opener, and is thus the only appropriate anti-anginal in this scenario.

1. NICE Guidelines 73. Management of stable angina. Dec 2012

Question #223

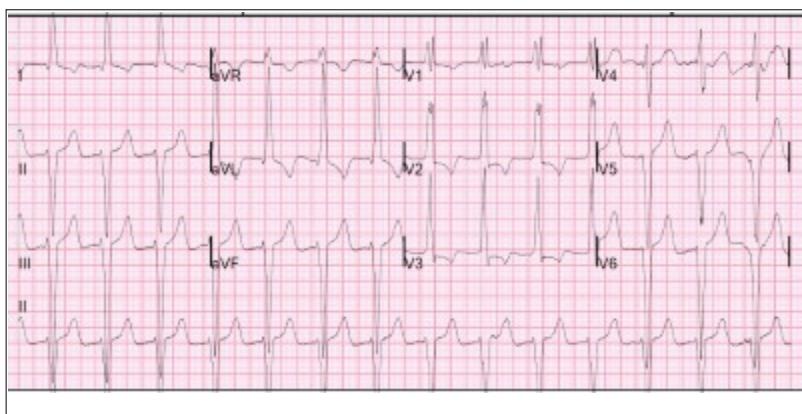
A 70-year-old presents to the outpatients' department having been referred by his GP with on-off palpitations for several months. The patient has a myocardial infarct over 20 years ago which was successfully managed via thrombolysis but otherwise, he has no significant past medical history.

The patient reports he has spontaneous episodes of palpitations approximately once every 2 weeks, and occasional feeling faint. He fainted once when walking quickly for the bus and so now takes it slowly when exerting himself and has no further similar episodes. He is otherwise very well and denies any episodes of chest pain etc.

He is on secondary preventive medication including:

- Aspirin low dose
- Bisoprolol 10mg twice daily
- Atorvastatin 40mgs once daily
- Ramipril 10mg once daily

His examination and observation are normal and an ECG performed is shown below.



What is the most appropriate management plan for the patient?

- f) Admit for urgent ablation workup
- g) Admit for urgent pacemaker workup
- h) No additional management is required
- i) Reduce the dose of his beta-blocker
- j) Arrange for urgent 24-hour Holter monitor

Correct answer is b.

The patient's ECG show trifascicular block with a right bundle branch block (RBBB), left anterior hemiblock and 1st-degree heart block. This with the history of pre-syncope and syncope is concerning and is an indication for admission and pacemaker workup as the patient is at risk of further significant arrhythmias or cardiac blocks.

Trifascicular block is a ventricular escape rhythm that usually arises from the left anterior or posterior fascicle regions following ischaemic heart disease or structural heart disease. As such ablation is not a viable treatment option.

Symptomatic trifascicular block is an indication for implantable cardiac pacemaker or permanent pacemaker work up.

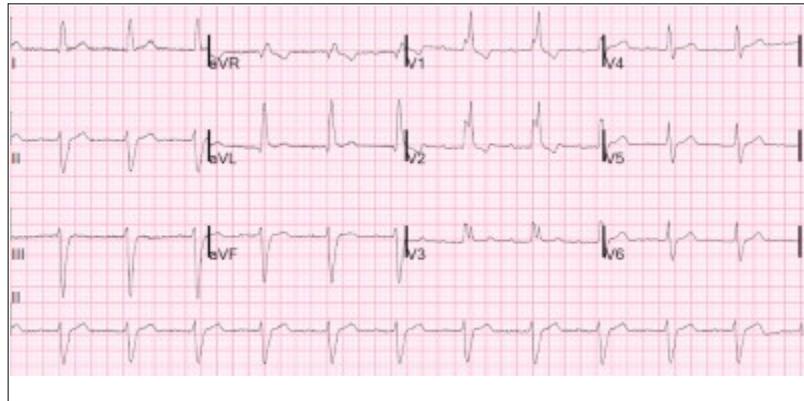
The patient has concerning features of symptomatic trifascicular block with pre-syncope and syncope on exertion which warrants urgent assessment for a pacemaker.

The patient's trifascicular block is very unlikely to be associated with his beta-blocker medication and therefore reducing the dose will not remove the risk of further significant arrhythmias or cardiac blocks.

Arranging for an urgent 24-hour Holter monitor is unnecessary as the patient's ECG is unlikely to change and the current risk of arrhythmias or heart block requires urgent intervention.

Question #224

A 77-year-old man is reviewed in the cardiology. He presented to his GP with intermittent dizziness and reduced exercise tolerance. An ECG accompanies the referral letter:



What does the ECG show?

- f) Left bundle branch block
- g) Long QT syndrome
- h) ECG changes consistent with hypokalaemia
- i) Bifascicular block
- j) Trifascicular block

Correct answer is e.

RBBB +left anterior or posterior hemiblock + 1st-degree heart block = trifascicular block

The ECG demonstrates RBBB + left anterior hemiblock + 1st-degree heart i.e. trifascicular block.

Question #225

A 58-year-old man attends cardiology clinic with retrosternal chest pain on exertion. He reports the onset of his symptoms when walking more than 50 metres on flat ground or when playing on the floor with his grandchildren. The symptoms have been present at the current level for at least the previous 9 months and the patient denied any episodes of pain at rest. The patients GP has

previously attempted treatment with bisoprolol and amlodipine but both medications were discontinued for unwanted effects (bradycardia and ankle swelling respectively). The patient has no other medical history except for hypercholesterolaemia. Regular medications are aspirin 75 mg daily and pravastatin 10 mg daily with nitrate spray and sildenafil used as required. He is formerly a heavy smoker but managed to quit the previous year.

Clinical examination is unremarkable with no evidence of cardiac failure. Blood pressure is 102 / 72 mmHg.

Please see below for the results of previous investigations.

Electrocardiogram: sinus rhythm at 58 beats per minute; borderline left axis deviation; normal QRS complex; non-specific lateral ST segment abnormalities; normal T waves.

Transthoracic echocardiogram: normal valvular function; no regional wall motion abnormality; ejection fraction 55-60 %.

Urea	6.7 mmol / L
Creatinine	80 micromol / L
Sodium	138 mmol / L
Potassium	4.1 mmol / L

Cardiac stress magnetic resonance imaging: significant evidence of ischaemia in the region of the lateral left ventricle; estimated 20 % of LV myocardium with significant evidence of ischaemia.

What is the most appropriate management of the patients chest pain?

- f) Ticagrelor
- g) Nicorandil
- h) Percutaneous coronary intervention
- i) Ivabradine
- j) Long-acting isosorbide mononitrate

Correct answer is e.

The role of percutaneous coronary intervention in stable angina is a controversial subject. Revascularisation has not been shown to reduce mortality or the rate of myocardial infarction in such patients. However, sub-group analysis of randomised controlled trials suggests some benefit for the above outcomes over medical therapy. Therefore, ESC guidelines recommend offering revascularisation to patients with stable coronary artery disease and ischaemia in > 10 % of the left ventricle. Also, the patient's age and lack of co-morbidities make this the most appropriate strategy in this case.

The other possible answers that are options for the medical management of stable angina are contraindicated in this patient. Both nicorandil and ISMN are contra-indicated for this patient due to his borderline hypotension and use of sildenafil. Ivabradine is contra-indicated due to the patient's history of bradycardia. It is also not used in patients with moderate to severe angina symptoms as has been shown to increase the rate of cardiovascular events in such patients.

Ticagrelor is a P2Y12 adenosine diphosphate receptor blocker with utility as an oral antiplatelet agent.

Al-Lamee R, Davies J, Malik I. What is the role of coronary angioplasty and stenting in stable angina? BMJ 2016;352:i205

Question #226

A 36 year old woman presents to the medical assessment unit with a 2 day history of pleuritic right sided chest pain, and shortness of breath. She is 38 weeks pregnant with her first child, her pregnancy so far had been uncomplicated.

On examination, she is slightly tachypnoeic, with no added sounds on chest auscultation.

There is no clinical evidence of a deep vein thrombosis. Her oxygen saturations are 95% on room air, heart rate 98/min.

What is the next appropriate investigation?

- f) CTPA
- g) D Dimer
- h) Chest x-ray
- i) V/Q scan
- j) Bilateral leg dopplers

Correct answer is c.

Venous thromboembolic event (VTE) is an important cause of maternal death in the UK. Pregnant women are 10x more likely to have a VTE than non-pregnant women of the same age.

Where there is suspicion of a pulmonary embolism (PE), a chest x-ray should be performed initially. If this does not explain the patient's symptoms, compression duplex dopplers should be performed. A diagnosis of deep vein thrombosis (DVT) can indirectly confirm a PE, reducing radiation doses for the mother.

If both tests are negative, and the clinical suspicion of PE remains high, further imaging should be organised; either CTPA or ventilation-perfusion scan (V/Q scan). British thoracic society guidelines (BTS) recommend a CTPA in non-pregnant women.

The decision as to which scan to perform should be ideally taken with the input from the patient. V/Q scanning carries a slightly increased risk of childhood cancer compared with CTPA but carries a lower risk of maternal breast cancer. The ventilation component of the V/Q scan may be able to be omitted, reducing the radiation dose.

A D Dimer would not assist in the diagnosis, as it may be raised anyway in

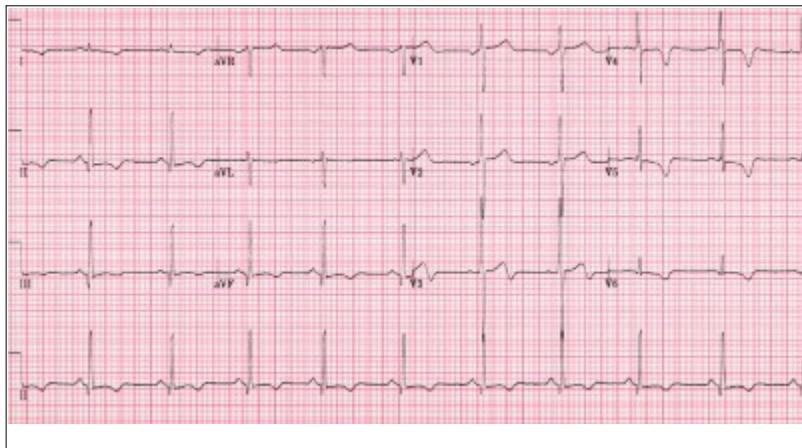
pregnancy due to disturbances in the coagulation system.

Guidelines: RCOG- The acute management of thrombosis and embolism during pregnancy and the puerperium

Question #227

A 34-year-old is brought into the emergency department following a syncope episode whilst playing tennis. The patient reports he is now feeling well with no symptoms. On further questioning, he only plays tennis occasionally and does not exercise regularly. He reports experienced shortness of breath and chest discomfort when playing and has had a few episodes of feeling light-headed but has never fainted before. He is otherwise well with no recent illness or significant past medical history.

Observations are all within normal range and an ECG performed is shown below.



What is the patient's most likely diagnosis?

- f) Brugada syndrome
- g) Dilated cardiomyopathy
- h) Hypertrophic obstructive cardiomyopathy
- i) Arrhythmogenic right ventricular dysplasia (ARVD)
- j) Wellen's syndrome

Correct answer is c.

This patient has presented with features in keeping with hypertrophic obstructive cardiomyopathy (HOCM). The condition is due to a genetic defect that causes a disorder of the cardiac muscle and, although patients can be asymptomatic, it is the commonest cause of sudden cardiac death in the young. Patients who do present with symptoms typically experience exertional dyspnoea, angina and syncopal episodes, commonly following or during exertion. ECG findings are of left ventricular hypertrophy, non-specific ST-segment and T-wave abnormalities, progressive T wave inversion and deep Q waves. On this patient's ECG widespread T wave inversion can be seen, especially in lead I & II as well as non-specific ST-segment changes in V2 & 3. The diagnosis can be confirmed via cardiac echo and management is dependant on the extent of obstruction and patient symptoms.

Brugada syndrome is another genetic cardiac disorder however unlike HOCM it results in electrical activity disorders and not structural issues. Features include syncope and sudden cardiac death however it is not always associated with exertion or exercise. There are several types of Brugada syndrome, each with variations seen on ECG. However, the only potentially diagnostic ECG abnormality is of coved ST-segment elevation in 2 or more of V1-3 followed by a negative T wave. This is classical of Brugada type 1 syndrome and is commonly known as the Brugada sign.

Dilated cardiomyopathy is a disease of the myocardial and characterised by progressive ventricular dilation and dysfunction. Presentation is normal with worsening biventricular failure including peripheral and pulmonary oedema. Syncope episodes are rare and symptoms are not fluctuant, as seen in this patient. ECG abnormalities are those associated with atrial and ventricular hypertrophy with conduction delays (e.g. LBBB), left axis deviation and poor R wave progression.

Arrhythmogenic right ventricular dysplasia (ARVD), also known as arrhythmogenic right ventricular cardiomyopathy (ARVC), is a result of a genetic defect affecting the desmosomes of the myocardium. ARVD results in non-ischemic cardiomyopathy, mainly affecting the right ventricle. The condition leads to hypokinetic areas of the ventricle wall and myocardium fibrofatty replacement

with which results in associated arrhythmias. Patients typically present with palpitations, syncope, and potentially sudden cardiac death however ECG findings are of T wave inversion in leads V1 to V3 and of right bundle branch block which are not present in this case.

Wellen's syndrome is the ECG pattern of biphasic or deeply inverted T waves in the chest leads V2-3. It is highly specific for critical stenosis of the left anterior descending artery and therefore is normally seen in patients presenting with ischaemic-like symptoms.

Question #228

A 72-year-old man presents to the emergency department with palpitations. An ECG confirms atrial fibrillation. His CHA₂DS₂-VASc score is 2 and he is commenced on bisoprolol and edoxaban.

He is referred and attends for catheter ablation, which he undergoes successfully. Post-procedure ECG confirms sinus rhythm.

He is followed up 4 weeks later in the clinic and a repeat ECG confirms the maintenance of sinus rhythm.

What is the best course of action at this stage?

- f) Continue edoxaban for a further 4 weeks then stop
- g) Continue edoxaban for a further 8 weeks then stop
- h) Continue edoxaban long-term
- i) Discontinue edoxaban
- j) Switch to aspirin

Patients who've had a catheter ablation for atrial fibrillation still require long-term anticoagulation as per their CHA₂DS₂-VASc score

The correct answer is **continue edoxaban long-term**. Although the ablation has been successful and rhythm control has been achieved, patients in this setting

have the same stroke risk as those remaining in atrial fibrillation. Anticoagulation should, therefore, be continued long-term.

Continue edoxaban for a further 4 weeks then stop is incorrect.

Continue edoxaban for a further 8 weeks then stop is incorrect.

Discontinue edoxaban is incorrect.

Switch to aspirin is incorrect.

Question #229

A 22-year-old woman presents to the emergency department with a 1-day history of abdominal pain and vomiting. She denies fever or diarrhoea and disclosed that she went out to a party the night before and consumed 7 glasses of wine. She reports that there is a heart condition that runs in her family, but she cannot remember the name of the condition.

Her observations are within normal limits. On examination, there is central and epigastric tenderness but no guarding or peritonism. Her respiratory and cardiovascular examinations were unremarkable.

Venous blood gas:

pH	7.35	(7.35 - 7.45)
Na ⁺	138 mmol/L	(135 - 145)
K ⁺	4.1 mmol/L	(3.5 - 5.0)
Bicarbonate	22 mmol/L	(22 - 29)
Lactate	3.5 mmol/L	(< 1.6)

Glucose	6.2 mmol/L	(4 - 8)
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ECG: Sinus rhythm, normal axis, QRS duration 110ms, QTc 550ms.

What drug is it most important to avoid?

- f) Codeine
- g) Cyclizine
- h) Hyoscine butylbromide
- i) Omeprazole
- j) Ondansetron

5HT-3 receptor antagonists shouldn't be used as antiemetics in patients with long-QT syndrome

This patient has a prolonged QTc interval of 550ms. A normal QTc interval for women should lie between 350-460ms. Patients with a prolonged QTc interval are at particular risk of developing critical arrhythmias such as Torsades de pointes (a polymorphic ventricular tachycardia). It is therefore extremely important to avoid drugs that may prolong the QTc interval further. **Ondansetron** is a type of 5HT-3 receptor antagonist that is associated with QTc interval prolongation and should therefore be avoided.

Codeine is a weak opioid analgesic with common side effects of nausea, drowsiness and constipation. It does not prolong the QTc interval.

Cyclizine is a H1-receptor antagonist. Side effects of cyclizine include urinary retention, dry mouth, and blurred vision. Prolongation of the QTc interval is not a common feature of this medication.

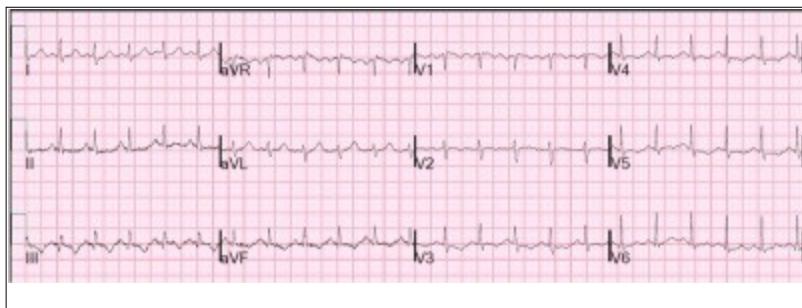
Hyoscine butylbromide is used in the treatment of gastric spasm. It is an anticholinergic agent and is, therefore, contraindicated in myasthenia gravis, urinary retention and gastrointestinal obstruction. It does not prolong the QTc

interval.

Omeprazole is a proton-pump inhibitor used in the treatment of gastritis and gastro-oesophageal bleeding. Complications include electrolyte disturbances (e.g. hypomagnesaemia) and increased risk of *Clostridium difficile* infections but not prolongation of the QTc interval.

Question #230

A 19-year-old student is brought to the Emergency Department by her friends. Around one hour ago she 'collapsed' whilst playing hockey. Her friends describe her complaining that she felt light-headed and then 'fainting' to the ground. She lost consciousness for a few seconds before returning to normal quite quickly. There is no past medical history of note other than the use of Microgynon 30 (a combined oral contraceptive pill). For the past 4-5 days the patient has experienced shortness-of-breath and a central chest pain which is worse when she coughs. On examination her pulse is 120/min, blood pressure 96/60 mmHg and chest auscultation reveals scattered wheezes. An ECG done on admission is shown below:



What is the most likely diagnosis?

- f) Hypertrophic obstructive cardiomyopathy
- g) Vasovagal attack
- h) Acute coronary syndrome
- i) Pulmonary embolism
- j) Asthma attack

Correct answer is d.

It is usually taught that pulmonary embolism (PE) presents with pleuritic chest pain, dyspnoea and haemoptysis. This combination of symptoms is however only found in less than 20% of cases. As PE is a potentially life-threatening condition it is important to be aware of the wide variety of symptoms and signs that may accompany cases.

A lot of patients who develop a PE have risk factors. There is one present in this case - combined oral contraceptive pill use. Tachycardia is also a common sign.

It would be unusual to develop an asthma attack with no previous history of asthma. Occasional wheezes are a relatively common finding in patients following a pulmonary embolism.

The ECG shows a sinus tachycardia and a partial S1Q3T3 - the S wave is not particularly convincing.

Discuss (5) Improve

Question #231

A 22-year-old Afro-Caribbean man presents with chest pain. He states the pain is sharp in nature, worse on inspiration, and localised to the right lateral chest wall. He has no past medical history and takes no regular medicines.

An ECG is performed:

P waves	Normal morphology
PR interval	140ms
QRS	110ms
QTc	420ms

T waves	Inverted in V1-V6
ST segments	No elevation or depression

What is the most likely explanation of these ECG results?

- f) NSTEMI
- g) STEMI
- h) Normal variant
- i) Prinzmetal's angina (variant angina)
- j) Stable angina pectoris

Widespread T wave inversion in the chest leads can be a normal variant in patients with Afro-Caribbean ethnicity

Coronary artery disease would be very unlikely in a man of this age. The absence of ST elevation makes an acute STEMI even more unlikely. T-wave inversion can of course be a feature of an NSTEMI or stable angina, however due to the absence of risk factors, and the patients age, it would be a very unlikely diagnosis.

Prinzmetal's angina due to vasospasm would be a possibility. However this usually presents with ST elevation.

Widespread T-wave inversion in the chest leads can be a normal variant in patients with Afro-Caribbean ethnicity and this would be the most likely diagnosis in a young man with no cardiovascular risk factors.

The patients pain is pleuritic in nature and an alternative diagnosis should therefore be considered (e.g. pneumothorax).

Discuss (7) Improve

Question #232

A 52-year-old female who you see regularly represents to your clinic after routine blood tests having recently been started on ramipril for her hypertension. Her blood pressure (BP) prior to initiation of ramipril in the clinic was 145/98mmHg and her baseline creatinine prior to treatment was 100umol/L. On review today her clinic BP appears to be well controlled at 132/84mmHg and her bloods show her creatinine has risen to 125umol/L. What is the most appropriate step in the management of her hypertension?

- f) Reduce dose of ramipril
- g) Continue current dose of ramipril
- h) Stop ramipril and consider angiotensin receptor blocker
- i) Increase dose of ramipril
- j) Stop ramipril and consider calcium channel blocker

Correct answer is b.

The main consideration here is the renal function, which shows an increase in creatinine by 25%. Her BP appears to be well controlled. The BNF recommends the angiotensin-converting enzyme inhibitors should only be stopped if the creatinine increases by 30% or eGFR falls by 25% or greater. This lady's results are within these limits and have shown good effect. It would therefore be pertinent to continue the ramipril at the current dose and monitor the renal function as per normal protocol

Question #233

A patient attends a cardiology clinic following a recent myocardial infarct (MI) which was successfully managed via percutaneous coronary intervention (PCI). The patient has been taking their secondary prevention medications regularly and reports no issues or symptoms.

Their examination is unremarkable and the patient's observations are as follows:

- Heart rate 55 beats per minute
- Blood pressure 134/80 mmHg
- Respiratory rate 16 breaths per minute
- Oxygen saturations 98% on air
- Temperature 36.2 °C

An ECG performed has widened QRS complexes and atrioventricular dissociation in keeping with accelerated idioventricular rhythm (AIVR) with a rate of 55 beats per minute.

What management should be advised?

- f) Arrange for urgent repeat PCI
- g) Arrange for urgent pacemaker insertion
- h) No additional management is required
- i) Stop the patient's beta blocker and continue all other medications
- j) Urgent atropine

Correct answer is c.

Accelerated idioventricular rhythm is common and un concerning following recent MI

AIVR is a benign ectopic rhythm of ventricular origin. It is common post-MI following the reperfusion of an ischaemic myocardium. AIVR is usually self-limiting and therefore treatment is not necessary, especially as the patient is asymptomatic and haemodynamically stable with a normal heart rate and blood pressure.

Arranging for urgent repeat PCI is unnecessary as there is no indication of new/further ischaemic damage or an ischaemic event having occurred. AIVR is

due to an ectopic rhythm and therefore PCI is of no value.

Pacemaker insertion is not required as AIVR is due to a benign ectopic rhythm and therefore pacemaker insertion would not remove the cause. As the patient is asymptomatic and haemodynamically stable no treatment is required.

Stopping the patient's beta-blocker is unnecessary as this will not eliminate the AIVR. Beta-blockers are an important part of secondary prevention post-MI and therefore should be continued.

Atropine could be considered to overcome AIVR via increase the sinus rate however this is rarely required. As the patient is symptom-free and haemodynamically stable the use of atropine is unnecessary.

Question #234

An 84-year-old man comes for review. Four weeks ago an opportunistic blood pressure reading was taken and recorded as 150/92 mmHg. You therefore arranged ambulatory blood pressure monitoring (ABPM) along with a standard hypertension work-up. You did not calculate his 10-year cardiovascular risk on account of his age. The following results were obtained:

Na ⁺	141 mmol/l
K ⁺	4.2 mmol/l
Urea	6.5 mmol/l
Creatinine	101 µmol/l

Total cholesterol	4.9 mmol/l
HDL cholesterol	1.2 mmol/l
Fasting glucose	5.5 mmol/l

Urine dipstick was normal. The ECG showed sinus rhythm, 72 bpm and first degree heart block.

The daytime average blood pressure reading was 145/80 mmHg. What is the most appropriate course of action?

- f) Diagnose stage 1 hypertension and advise about lifestyle changes
- g) Start treatment with an ACE inhibitor
- h) Start treatment with a calcium channel blocker
- i) Start treatment with a thiazide-like diuretic
- j) Repeat the ABPM

Stage 1 hypertension is defined by an ABPM reading of $\geq 135/85$ mmHg, with stage 2 hypertension having a cut-off of $\geq 150/95$ mmHg.

This patient therefore has stage 1 hypertension. As they are > 80 years they do not need treatment.

Question #235

A 56-year-old woman presents to her GP with discolouration around her eyes which has been present for some months. She feels very conscious about its appearance but denies any pain, itch or discomfort.

On examination, she appears well. The area in question appears as follows:

Given the likely diagnosis, which of the following would be an appropriate management option?

- f) Topical emollient
- g) Topical hydrocortisone
- h) Topical ketoconazole
- i) Topical trichloroacetic acid
- j) Urgent referral to secondary care

Correct answer is d.

The photo above demonstrates xanthelasma - high lipid levels leading to soft yellow/orange plaques, periorbitally. They are not of clinical concern - except for the underlying lipid profile which should be investigated and treated accordingly. They can be left alone, but if patients are keen for treatment, a commonly used option is topical trichloroacetic acid.

Topical emollient is unnecessary. This will not help to alleviate the appearance of the xanthelasma.

Topical hydrocortisone is inappropriate here. As a steroid, this would not help to reduce the appearance of xanthelasma.

Topical ketoconazole is also inappropriate. This is an antifungal and may be used for seborrhoeic dermatitis, which would instead present with dry, flaky skin and erythema - not yellow/orange plaques.

An urgent referral to a hospital is also unwarranted. Xanthelasma is of no clinical concern and requires no further investigation.

Question #236

A 68-year-old man presents with occasional lightheadedness over the past few weeks. During his morning walk today, he experienced a brief spell of dizziness. He

denies experiencing chest pain, breathlessness, or any other significant complaints.

His medical history includes diabetes, hypertension, and myocardial infarction for which he underwent coronary artery bypass grafting (CABG) seven years ago. He is currently on medications including aspirin, bisoprolol, lisinopril, atorvastatin, and vildagliptin.

Observations are as below:

- BP 140/85 mmHg
- Heart rate 74 bpm
- Respiratory rate 16/min
- Oxygen saturation 98% on room air

An ECG was subsequently carried out.

What is the most appropriate management option?

- f) Discharge him and recommend a follow up ECG
- g) Give atropine
- h) Intravenous infusion of isoproterenol
- i) Refer for cardiac resynchronisation therapy
- j) Review his medications

The correct answer is **review his medications**.

The ECG findings demonstrate a progressive prolongation of the PR interval until a beat is dropped. This is characteristic of a 2nd-degree Mobitz 1 or Wenckebach-

type block. This is usually a relatively benign rhythm with a low risk of progression to higher-degree blocks. This patient has symptoms of lightheadedness and dizziness, coupled with stable vital signs. As long as there is no hemodynamic compromise, there is no need for urgent intervention. Searching for a reversible cause should be the first step. This patient is on bisoprolol, a known cause of AVN block, and should be reviewed.

Discharge him and recommend a follow up ECG is incorrect. It is indicated if the patient is asymptomatic with a first or 2nd-degree Mobitz 1 heart block. This patient has symptoms of light-headedness and dizziness, which require intervention.

Give atropine is incorrect. Atropine is generally indicated for acute symptomatic AVN block with bradycardia and adverse features (heart failure, shock, syncope, myocardial ischaemia). There are no adverse features in this case.

Intravenous infusion of isoproterenol is also an incorrect answer. Reviewing any potential causes should be the initial approach for symptomatic hemodynamically stable patients. IV isoproterenol is indicated for hemodynamically unstable patients who fail to respond to transcutaneous pacing. Given the patient's history of coronary artery disease, it is important to avoid it if possible, due to the potential development of ischemic chest pain.

Refer for cardiac resynchronisation therapy is incorrect. Cardiac resynchronisation can be beneficial if there is heart failure with an ejection fraction of less than or equal to 35% and a widened QRS (e.g. left bundle branch block) complex on the ECG. It has no role in the management of the AVN block.

Question #237

A 34-year-old male is brought in from a nearby bar having collapsed. His friends who accompany him tell you that the patient had been moderately intoxicated but had not reported any symptoms prior to his collapse.

On arrival, the patient is conscious but reports feeling generally weak and light-headed. He has never had an episode like this before but has had episodes of

palpitation with associated shortness of breath previously.

The patient states he smokes 20 cigarettes/day, often binge drinks consuming 10 plus pints of beer most weekends and occasionally uses cocaine.

On examination, the patient is conscious and reports some lightheadedness and palpitation but feels otherwise ok when lying down. He has a fast, thready pulse between 130-150bpm and a blood pressure of 89/65mmHg. He has a mildly raised respiratory rate but his chest is clear and peripheral saturations are 94% on room air. Heart sounds are normal and examination is otherwise unremarkable.

What is the most appropriate management?

- f) IV adenosine
- g) IV metoprolol
- h) Cardioversion
- i) Pacemaker insertion
- j) Percutaneous coronary intervention

Correct answer is c.

This patient has presented with a tachyarrhythmia and is haemodynamically unstable with an acute collapse, most likely syncope induced, and shock, demonstrated by the hypotension and cool extremities, etc. As seen on the ECG the patient's rhythm is fast and irregular with a varied rate of between 100 and 150 bpm. The QRS complexes are narrow and clear 'p' waves cannot be seen before each complex. This is in keeping with a tachyarrhythmia, most likely secondary to atrial fibrillation however the specific cause is not relevant as urgent synchronised cardioversion is required in any unstable patient presenting with a tachyarrhythmia. Cardioversion should be carried out in a resuscitation setting, with procedural sedation if possible. Other indications of instability in arrhythmias include evidence of myocardial ischaemia and heart failure. Following cardioversion, further management is based on the QRS complexes (narrow vs broad) and the rhythm (regular vs irregular).

Adenosine is the treatment of choice in patients presenting with supraventricular tachycardias (SVT), which may be the underlying diagnosis in this case, however, it should be used in hemodynamically stable patients only. As the patient is unstable cardioversion is required.

Metoprolol is an IV beta-blocker and, although it can be used in some tachyarrhythmias, this patient is unstable and requires urgent electrical cardioversion. IV beta-blockers can be used in stable patients with tachyarrhythmias (e.g. atrial fibrillation) for rate control as so long as they have an adequate systolic blood pressure and there are no other contraindications (e.g. asthma).

Pacemaker insertion is used in the management of bradycardia arrhythmias and does not have a role in acute arrhythmias causing compromise.

Percutaneous coronary intervention is used in the management of myocardial infarction (MI). Although the patient has some risk factors, and cocaine use can induce an MI, it is unlikely to be the underlying issue in this case with the patient not reporting chest pain symptoms and no clear evidence of ST-elevation seen on the ECG.

Question #237

A 43-year-old gentleman who is admitted and found to have a STEMI has chest pain on the ward. He is currently recovering from his percutaneous coronary intervention the day before and has felt well until now. The pain is over the left side of the chest and severe if he breathes. He has no cough or fevers and his observations are within normal limits.

On examination, he has normal heart sounds and a clear chest. He is mildly tender over the chest wall. His femoral puncture sites are clean with no haematoma. He is in sinus rhythm on the cardiac monitor and has soft non-oedematous calves. No JVP is seen.

Na ⁺	156 mmol/l
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K ⁺	3.4 mmol/l
Urea	5.6 mmol/l
Creatinine	78 µmol/l
CRP	45 mg/l

Hb	134 g/l
Platelets	398 * 10 ⁹ /l
WBC	13.0 * 10 ⁹ /l

ECG	sinus rhythm, T wave inversion in V1-V3, PR 180ms and flat in most leads
Chest x-ray	clear lung fields, heart is at the upper limit of normal for size

What is the likely diagnosis?

- f) Hospital acquired pneumonia
- g) Pericarditis
- h) Pulmonary embolism
- i) Coronary artery dissection
- j) Pleurisy

Pleuritic chest pain at <48hrs after MI -> pericarditis

This gentleman develops chest pain in the first 24-48hrs after an MI. A hospital-acquired pneumonia would usually take longer to develop. A coronary artery dissection can be a complication of PCI but there would most likely be ischaemic changes seen on the ECG. Pleurisy is usually a post inflammatory condition after a lower respiratory tract infection. Pulmonary embolism is possible but patients with MI are anticoagulated and this is early for a PE to develop. It is recognised that a number of MI patients develop acute pericarditis in the first 48hrs. Treatment is supportive and it is key to rule out other complications first.

Question #238

A patient attends a cardiology clinic following a recent myocardial infarct (MI) which was successfully managed via percutaneous coronary intervention (PCI). The patient has been taking their secondary prevention medications regularly and reports no issues or symptoms.

Their examination is unremarkable and the patient's observations are as follows:

- Heart rate 55 beats per minute
- Blood pressure 134/80 mmHg
- Respiratory rate 16 breaths per minute
- Oxygen saturations 98% on air
- Temperature 36.2 °C

An ECG performed has widened QRS complexes and atrioventricular dissociation in keeping with accelerated idioventricular rhythm (AIVR) with a rate of 55 beats per minute.

What management should be advised?

- f) Arrange for urgent repeat PCI
- g) Arrange for urgent pacemaker insertion
- h) No additional management is required
- i) Stop the patient's beta blocker and continue all other medications
- j) Urgent atropine

Accelerated idioventricular rhythm is common and un concerning following recent MI

AIVR is a benign ectopic rhythm of ventricular origin. It is common post-MI following the reperfusion of an ischaemic myocardium. AIVR is usually self-limiting and therefore treatment is not necessary, especially as the patient is asymptomatic and haemodynamically stable with a normal heart rate and blood pressure.

Arranging for urgent repeat PCI is unnecessary as there is no indication of new/further ischaemic damage or an ischaemic event having occurred. AIVR is due to an ectopic rhythm and therefore PCI is of no value.

Pacemaker insertion is not required as AIVR is due to a benign ectopic rhythm and therefore pacemaker insertion would not remove the cause. As the patient is asymptomatic and haemodynamically stable no treatment is required.

Stopping the patient's beta-blocker is unnecessary as this will not eliminate the AIVR. Beta-blockers are an important part of secondary prevention post-MI and therefore should be continued.

Atropine could be considered to overcome AIVR via increase the sinus rate however this is rarely required. As the patient is symptom-free and haemodynamically stable the use of atropine is unnecessary.

Question #239

An 84-year-old man comes for review. Four weeks ago an opportunistic blood pressure reading was taken and recorded as 150/92 mmHg. You therefore arranged ambulatory blood pressure monitoring (ABPM) along with a standard hypertension work-up. You did not calculate his 10-year cardiovascular risk on account of his age. The following results were obtained:

Na ⁺	141 mmol/l
K ⁺	4.2 mmol/l
Urea	6.5 mmol/l
Creatinine	101 µmol/l
Total cholesterol	4.9 mmol/l
HDL cholesterol	1.2 mmol/l
Fasting glucose	5.5 mmol/l

Urine dipstick was normal. The ECG showed sinus rhythm, 72 bpm and first degree heart block.

The daytime average blood pressure reading was 145/80 mmHg. What is the most appropriate course of action?

- a) Diagnose stage 1 hypertension and advise about lifestyle changes
- b) Start treatment with an ACE inhibitor
- c) Start treatment with a calcium channel blocker
- d) Start treatment with a thiazide-like diuretic
- e) Repeat the ABPM

Stage 1 hypertension is defined by an ABPM reading of $\geq 135/85$ mmHg, with stage 2 hypertension having a cut-off of $\geq 150/95$ mmHg.

This patient therefore has stage 1 hypertension. As they are > 80 years they do not need treatment.

Question #240

A 56-year-old woman presents to her GP with discolouration around her eyes which has been present for some months. She feels very conscious about its appearance but denies any pain, itch or discomfort.

On examination, she appears well. The area in question appears as follows:

Given the likely diagnosis, which of the following would be an appropriate management option?

- a) Topical emollient
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- c) Topical ketoconazole
- d) Topical trichloroacetic acid
- e) Urgent referral to secondary care

Correct answer is d.

The photo above demonstrates xanthelasma - high lipid levels leading to soft yellow/orange plaques, periorbitally. They are not of clinical concern - except for the underlying lipid profile which should be investigated and treated accordingly. They can be left alone, but if patients are keen for treatment, a commonly used option is topical trichloroacetic acid.

Topical emollient is unnecessary. This will not help to alleviate the appearance of the xanthelasma.

Topical hydrocortisone is inappropriate here. As a steroid, this would not help to reduce the appearance of xanthelasma.

Topical ketoconazole is also inappropriate. This is an antifungal and may be used for seborrhoeic dermatitis, which would instead present with dry, flaky skin and erythema - not yellow/orange plaques.

An urgent referral to a hospital is also unwarranted. Xanthelasma is of no clinical concern and requires no further investigation.

Question #241

A 68-year-old man presents with occasional lightheadedness over the past few weeks. During his morning walk today, he experienced a brief spell of dizziness. He denies experiencing chest pain, breathlessness, or any other significant complaints.

His medical history includes diabetes, hypertension, and myocardial infarction for which he underwent coronary artery bypass grafting (CABG) seven years ago. He is currently on medications including aspirin, bisoprolol, lisinopril, atorvastatin, and vildagliptin.

Observations are as below:

- BP 140/85 mmHg
- Heart rate 74 bpm
- Respiratory rate 16/min
- Oxygen saturation 98% on room air

An ECG was subsequently carried out.

What is the most appropriate management option?

- a) Discharge him and recommend a follow up ECG
- b) Give atropine
- c) Intravenous infusion of isoproterenol
- d) Refer for cardiac resynchronisation therapy
- e) Review his medications

The correct answer is **review his medications**.

The ECG findings demonstrate a progressive prolongation of the PR interval until a beat is dropped. This is characteristic of a 2nd-degree Mobitz 1 or Wenckebach-type block. This is usually a relatively benign rhythm with a low risk of progression to higher-degree blocks. This patient has symptoms of lightheadedness and dizziness, coupled with stable vital signs. As long as there is no hemodynamic compromise, there is no need for urgent intervention. Searching for a reversible cause should be the first step. This patient is on bisoprolol, a known cause of AVN block, and should be reviewed.

Discharge him and recommend a follow up ECG is incorrect. It is indicated if the patient is asymptomatic with a first or 2nd-degree Mobitz 1 heart block. This patient has symptoms of light-headedness and dizziness, which require intervention.

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Question #242

A 34-year-old male is brought in from a nearby bar having collapsed. His friends who accompany him tell you that the patient had been moderately intoxicated but had not reported any symptoms prior to his collapse.

On arrival, the patient is conscious but reports feeling generally weak and light-headed. He has never had an episode like this before but has had episodes of palpitation with associated shortness of breath previously.

The patient states he smokes 20 cigarettes/day, often binge drinks consuming 10 plus pints of beer most weekends and occasionally uses cocaine.

On examination, the patient is conscious and reports some lightheadedness and palpitation but feels otherwise ok when lying down. He has a fast, thready pulse between 130-150bpm and a blood pressure of 89/65mmHg. He has a mildly raised respiratory rate but his chest is clear and peripheral saturations are 94% on room air. Heart sounds are normal and examination is otherwise unremarkable.

An ECG is performed as below.

What is the most appropriate management?

- a) IV adenosine
- b) IV metoprolol
- c) Cardioversion

- d) Pacemaker insertion
- e) Percutaneous coronary intervention

Correct answer is c.

This patient has presented with a tachyarrhythmia and is haemodynamically unstable with an acute collapse, most likely syncope induced, and shock, demonstrated by the hypotension and cool extremities, etc. As seen on the ECG the patient's rhythm is fast and irregular with a varied rate of between 100 and 150 bpm. The QRS complexes are narrow and clear 'p' waves cannot be seen before each complex. This is in keeping with a tachyarrhythmia, most likely secondary to atrial fibrillation however the specific cause is not relevant as urgent synchronised cardioversion is required in any unstable patient presenting with a tachyarrhythmia. Cardioversion should be carried out in a resuscitation setting, with procedural sedation if possible. Other indications of instability in arrhythmias include evidence of myocardial ischaemia and heart failure. Following cardioversion, further management is based on the QRS complexes (narrow vs broad) and the rhythm (regular vs irregular).

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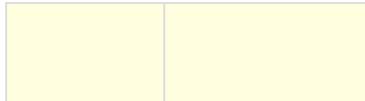
Question #243

A 43-year-old gentleman who is admitted and found to have a STEMI has chest pain on the ward. He is currently recovering from his percutaneous coronary intervention the day before and has felt well until now. The pain is over the left side of the chest and severe if he breathes. He has no cough or fevers and his observations are within normal limits.

On examination, he has normal heart sounds and a clear chest. He is mildly tender over the chest wall. His femoral puncture sites are clean with no haematoma. He is in sinus rhythm on the cardiac monitor and has soft non-oedematous calves. No JVP is seen.

Na ⁺	156 mmol/l
K ⁺	3.4 mmol/l
Urea	5.6 mmol/l
Creatinine	78 µmol/l
CRP	45 mg/l

Hb	134 g/l
Platelets	398 * 10 ⁹ /l
WBC	13.0 * 10 ⁹ /l



ECG	sinus rhythm, T wave inversion in V1-V3, PR 180ms and flat in most leads
Chest x-ray	clear lung fields, heart is at the upper limit of normal for size

What is the likely diagnosis?

- a) Hospital acquired pneumonia
- b) Pericarditis
- c) Pulmonary embolism
- d) Coronary artery dissection
- e) Pleurisy

Correct answer is b.

Pleuritic chest pain at <48hrs after MI -> pericarditis

This gentleman develops chest pain in the first 24-48hrs after an MI. A hospital-acquired pneumonia would usually take longer to develop. A coronary artery dissection can be a complication of PCI but there would most likely be ischaemic changes seen on the ECG. Pleurisy is usually a post inflammatory condition after a lower respiratory tract infection. Pulmonary embolism is possible but patients with MI are anticoagulated and this is early for a PE to develop. It is recognised that a number of MI patients develop acute pericarditis in the first 48hrs. Treatment is supportive and it is key to rule out other complications first.

Question #244

A 76-year-old man presents to the emergency department with complaints of shortness of breath on exertion, difficulty walking up stairs, and significant weight gain over the last 2 weeks. His daughter who is with him expresses concerns about

him and says that he is needing to lay upright in his armchair in order to get any sleep.

His medical history includes 2 previous non-ST-segment elevation myocardial infarctions for which he was treated with coronary stenting and hypertension. Physical examination shows a heart rate of 111 beats per minute and blood pressure of 113/76 mmHg. He is visibly short of breath at rest and is using accessory muscles. His JVP is 7cm above the angle of Louis, his apical impulse is laterally displaced, and heart sounds are audible with a holosystolic murmur heard loudest at the apex. Auscultation of his chest reveals audible crepitations bi-basally and he has 2+ peripheral oedema extending to his mid-thighs. An ECG shows Q-waves in his inferior leads, and his bloods show elevated troponins.

Which of the following is the most appropriate next step in the management of this patient?

- f) Obtain an urgent echocardiogram
- g) Prescribe oral furosemide and discharge from the emergency department with a view to follow up with his GP
- h) Load him with aspirin and clopidogrel
- i) Commence bisoprolol to control his heart rate
- j) Administer a bolus of intravenous furosemide and then start him on an infusion

Correct answer is e.

The most appropriate medication in patients with acute heart failure and a preserved ejection fraction who have signs of volume overload is addition/up-titration of a loop diuretic

This patient presents with acute decompensated congestive heart failure. He has evidence of significant volume overload on exam, with an elevated JVP and signs of pulmonary and peripheral oedema. The aetiology of his heart failure is likely to be ischaemic cardiomyopathy related to his history of myocardial infarctions and manifests with a displaced apical impulse and a holosystolic murmur heard loudest at the apical region, suggestive of significant ventricular dilatation. His

symptoms are severe and he requires inpatient hospitalisation and treatment with intravenous loop diuretics to improve his symptoms and to optimise his volume status.

An echocardiogram would be useful in the evaluation of this patient but is not the most appropriate next step as he is currently significantly symptomatic and needs intravenous loop diuretics to improve his volume status.

Although this patient does need to be treated with loop diuretics, he is in significant distress and likely needs to be admitted as an inpatient and should be treated more aggressively with intravenous loop diuretics.

This patient does have a history of ischaemic heart disease and is at risk of having further ischemic events. However, the findings on his ECG represent an old inferior infarct, and there are no features to suggest an acute event. In addition, he may have elevated troponins simply as a result of having ischaemic cardiomyopathy. He does not necessarily need to be loaded on dual antiplatelet therapy at this moment, but he should have his troponins trended to ensure there is no significant rise in their values.

Starting a beta-blocker in acute decompensated heart failure is contraindicated

Question #245

A 58-year-old woman was admitted with a stroke following a month's history of recurrent fevers, anorexia and weight loss. On examination, she had a left-sided hemiparesis and facial droop. Cardiovascular examination revealed splinter haemorrhages in 5 of her fingers across both hands, and a soft diastolic murmur heard loudest in expiration over the aortic area.

A trans-thoracic echocardiogram showed an oscillating vegetation on an aortic leaflet, in the path of regurgitant jets. Two blood cultures were positive for Streptococci spp. She was diagnosed with infective endocarditis and started on intravenous benzylpenicillin 1.2g every 4 hours and gentamicin 1mg/kg twice daily therapy.

She was reviewed after 5 days on antibiotic therapy with the following results.

Na+	139 mmol/l
K+	4.7 mmol/l
Urea	14.2 mmol/l
Creatinine	178 µmol/l
Serum bilirubin	16 µmol/l
Serum alkaline phosphatase	115 IU/l
Serum aspartate aminotransferase	18 IU/l
C Reactive protein	89 mg/l
Haemoglobin	138 g/l
White cell count	13.6 x 10 ⁹ /L
INR	1.1
Blood cultures	<i>Streptococcus Bovis</i>
Penicillin Minimum Inhibitory Concentration (MIC)	0.6 mg/L (high)
ECG	Prolonged PR interval (not present on admission ECG)

What is the next most important management?

- f) Increase antibiotics to intravenous benzylpenicillin 2.4g every 4 hours and gentamicin 1mg/kg twice daily
- g) Organise an urgent colonoscopy
- h) Switch antibiotics to ceftriaxone 2g once daily
- i) Refer to cardiothoracic surgeons
- j) Organise urgent trans-oesophageal echocardiogram

Correct answer is d.

This patient has infective endocarditis, as diagnosed by two positive major criteria from the Duke's criteria for infective endocarditis. In addition, she has a number of minor criteria, including fever, vascular events (stroke), immunological events (splinter haemorrhages, renal impairment secondary to glomerulonephritis).

Despite starting on appropriate empirical treatment she appears to have an ongoing infection from her blood tests, and her ECG demonstrates a prolonged PR interval which may be reflective of an enlarging aortic abscess disrupting the atrioventricular node, which is an indication for referral for cardiothoracic surgery in infective endocarditis.

Other surgical indications include

- Heart failure: valve obstruction resulting in pulmonary oedema or shock, severe acute regurgitation
- Uncontrolled infection: abscess, false aneurysm, persisting fever and positive blood cultures for greater than 10 days despite appropriate antibiotics, multiresistant microorganisms
- Prevention of embolism: large vegetations resulting in one or more embolic episodes despite appropriate antibiotic therapy, or other predictors of complications eg. heart failure.

She has a penicillin-resistant streptococci, according to the British Society of

Antimicrobial Chemotherapy guidelines, should be treated with vancomycin and gentamicin, so options (a) and (c) are incorrect. *Streptococcus Bovis* bacteraemia is associated with bowel malignancy, and a colonoscopy should be arranged to rule out malignancy, however, the worsening PR prolongation takes priority and should be managed first.

An urgent trans-oesophageal echocardiogram may be useful to evaluate the size of the aortic abscess, however, it would not change management as this patient needs to be referred to the cardiothoracic surgeons as there is already evidence of enlarging vegetation.

Question #246

A 54-year-old male previously treated for lymphoma is seen in clinic with a 6-month history of exertional dyspnoea which is progressive. He was treated four years ago and was told that there was no evidence of disease on the final CT scan. His GP treated him for a presumed lower respiratory tract infection two weeks ago. He returned from a business trip to Thailand six months ago, during which he consumed more alcohol than he usually does. He takes no medications and is otherwise fit and well, and is a non-smoker. On examination there is mild pedal oedema, his chest is clear and jugular venous pressure(JVP) is raised on inspiration. Auscultation of his heart reveals an extra heart sound very soon after S2.

What is the likely cause of his symptoms?

- f) Superior vena cava obstruction
- g) Cardiac tamponade
- h) Left ventricular failure
- i) Chronic obstructive pulmonary disease
- j) Constrictive pericarditis

Correct answer is e.

Examination of this patient demonstrates Kussmaul's sign - paradoxical elevation of the JVP on inspiration. This sign is seen in constrictive pericarditis, cardiac tamponade and restrictive cardiomyopathy. This presentation gives no other

features to suggest tamponade. His constrictive pericarditis here is likely cause by the previous radiotherapy for lymphoma. Other causes of constrictive pericarditis include TB and chronic pericarditis. Definitive management involves surgical pericardial stripping.

Question #247

A 55-year-old with previous rheumatic heart disease aged 32 presents with an 18-month history of exertional dyspnoea. An initial echo demonstrated significant raised pulmonary arterial pressures of 77 mmHg, she undergoes a left and right heart catheter with results as follows:

		Oxygen saturations
Right atrium	8 mmHg	71%
Right ventricle	39/8 mmHg	71%
Pulmonary artery	45/12 mmHg	71%
Capillary wedge	20 mmHg	93%
Left ventricle	165/11 mmHg	93%
Aorta	90/58 mmHg	

What is the most likely diagnosis?

- f) Aortic stenosis
- g) Mitral stenosis
- h) Aortic stenosis and mitral stenosis
- i) Aortic stenosis and pulmonary hypertension
- j) Aortic stenosis, mitral stenosis, pulmonary hypertension

Correct answer is e.

The key to questions regarding saturations and cardiac catheters is to spot the 'step-up' in oxygen saturation and abnormalities in gradients across valves. There are no 'step-ups' in oxygen saturations, demonstrating no shunts. However, you will note that the pulmonary arterial pressure is greater than the normal one-fifth of systolic measurements; hence pulmonary hypertension is present. In addition, there is a greater than 25mmHg gradient across the aorta valve, demonstrating moderate aortic stenosis. Lastly, the capillary wedge pressure is equivalent to the left atrial pressure, which should also be the same as the left ventricular diastolic pressure. A normal mitral valve expects less than 5mmHg pressure difference. Using these inferences, the mitral valve gradient is calculated by the capillary wedge pressure of 20mmHg (same as the left atrial pressure) minus the diastolic left ventricular pressure of 11mmHg: the 9mmHg difference thus also demonstrates mitral stenosis.

Question #248

You are called to urgently review a 45-year-old woman in the emergency department. She presented earlier in the day with fever, a productive cough and confusion. She had been diagnosed by the emergency department doctors as having community acquired pneumonia after seeing consolidation on her chest X-ray and sepsis secondary to this with associated delirium. Collateral history from her partner establishes that she had been feeling unwell for two days with a cough and become increasingly unwell, confused and agitated. The partner also explained that she had recently been diagnosed with an overactive thyroid and been told to start urgently on anti-thyroid tablets, but had not done so when she had read the label of these tablets and found that they should not be taken during pregnancy and she is trying to become pregnant. She had been taking paracetamol only for fever.

Despite treating with IV co-amoxiclav and oral clarithromycin and fluid resuscitation with three liters of fluid as well as IV paracetamol the patient has not improved.

On examination she is very agitated and aggressive and is difficult to examine. She feels very hot and clammy to touch, but peripheries are well perfused.

Auscultation shows left sided crepitations. Her heart rate is 170 bpm, blood pressure 81/40 mmHg, temperature 39.2°C, oxygen saturations 100% on 2L via nasal cannulae.

Blood tests from this admission are still pending. Out-patient blood tests show a undetectable low TSH two weeks ago, as well as a free T4 of 53mmol/ml.

What is the most appropriate immediate treatment?

- a. Change to IV meropenem and monitor
- b. Start carbimazole
- c. Urgent referral for thyroidectomy
- d. Start IV propranolol
- e. IV adenosine

Answer is D.

In thyroid storm with IV beta-blockers are a important first-line treatment

This is a patient with severe confusion, fever and evidence of chest infection and untreated hyperthyroidism. The clinical history suggests that she has developed a thyroid storm as she has fever, severe tachycardia, confusion and agitation with hyperthyroidism. This is a medical emergency, and the most urgent matter is to control her heart rate which is likely the cause for her low blood pressure. IV beta-blocker should be started for this purpose, and propranolol would further help reduce the conversion of T3 into T4. In addition, fluids and dexamethasone should be given, as well as anti-thyroid medications.

Changing to meropenem is unlikely to add any further benefit as the effects of the co-amoxiclav probably have not been seen yet. As the patient is not stable it is not appropriate to refer for surgery as management, but may be a viable long-term strategy to treat her hyperthyroidism. Starting carbimazole now is not the most appropriate action as the patient is unstable and this needs to be corrected first. Adenosine is would be appropriate for a patient with SVT without infection and thyrotoxicosis

Question #249

A 28-year-old man presents to the clinic for review of his familial hypercholesterolaemia. He is currently on 80mg once daily atorvastatin.

His blood results are as follows:

Hb	135 g/l	Na+	138 mmol/l
Platelets	322 * 10 ⁹ /l	K+	4.6 mmol/l
WBC	10.5 * 10 ⁹ /l	Urea	6.6 mmol/l
Neuts	6.2 * 10 ⁹ /l	Creatinine	72 µmol/l

Total cholesterol	7.5 mmol/L (normal range < 5)
LDL cholesterol	5.5 mmol/L (normal range < 3)

What treatment would you begin?

- a) Fenofibrate
- b) Nicotinic acid
- c) Ezetimibe
- d) Evolocumab
- e) Cholestyramine

The answer is C.

First line management for familial hypercholesterolaemia is high dose statins. Ezetimibe is used as a second line agent

High dose statin therapy is first line for familial hypercholesterolaemia.

If serum total or low-density lipoprotein (LDL) cholesterol concentration is not appropriately controlled on statin therapy, then the second line agent ezetimibe can be co-administered with the statin. Ezetimibe acts by decreasing cholesterol absorption in the small intestine

Question #250

A 45-year-old man presents to the endocrine clinic for review. He has had 3 stones in weight gain over the past 6 months and his GP is concerned about a possible diagnosis of Cushing's syndrome and has checked an initial 24hr urinary free cortisol which is elevated. He is hypertensive with a blood pressure of 155/90 mmHg, his pulse is 75 beats per minute and regular. His body mass index is 35 kg/m² and there are obvious abdominal striae. Which of the following would be most suggestive of an adrenal adenoma producing cortisol?

- a. Normal 9am serum cortisol
- b. Raised urinary free cortisol on repeat testing
- c. Serum cortisol of 220 nmol/l at 9am after an overnight dexamethasone suppression test
- d. Serum potassium of 2.4 mmol/l
- e. Undetectable levels of ACTH6

The answer is E.

The key fact is that an adrenal adenoma producing cortisol would suppress the release of ACTH. For this reason, if ACTH is undetectable then an adrenal tumour is the most likely cause. In this case, a CT abdomen would be the obvious next step.

Normal 9 am serum cortisol would make a diagnosis of Cushing's less likely, and a raised urinary free cortisol on repeat testing is not specific for the underlying cause. Failure to suppress after overnight dexamethasone suppression test merely confirms a diagnosis of Cushing's. Serum potassium levels as low as 2.4 mmol/l would be very unusual in a case of Cushing's.

Question #251

A 38-year-old woman is referred to the outpatient department by her GP with pain in her calves when walking 50 meters. She reports no other symptoms and has no other past medical history other than migraine. She is on no regular medication and her family history includes her mother having diabetes and her father dying of a heart attack aged 46. She currently smokes 35 cigarettes per day and drinks a glass of wine every evening. Her occupation is as a financial advisor.

Examination reveals tendon xanthomas affecting the extensor tendons of his fingers. On examining her face, it is noticed she has xanthelasma around both eyes and corneal arcs.

Which of the following is the most likely diagnosis?

- a) Tangier disease
- b) Homozygous familial hypercholesterolaemia
- c) Heterozygous familial hypercholesterolaemia
- d) Familial hypertriglyceridaemia
- e) Apo CII deficiency

Answer is C.

This patient's symptoms suggest intermittent claudication and combined with the signs of hypercholesterolaemia especially the tendon xanthomas would suggest one of the familial hypercholesterolaemias. Patients with homozygous familial hypercholesterolaemia, present with early cardiovascular disease, sometimes as early as the second decade of life, whereas, patients with heterozygous familial hypercholesterolaemia rarely present before the age 30. Thus, this case is more suggestive of heterozygous familial hypercholesterolaemia.

In heterozygous familial hypercholesterolaemia, total cholesterol would typically be above 7.9 mmol/l, with normal triglyceride levels. In homozygotes it is typically above 15 mmol/l

Question #252

A 67-year-old man with a history of ischaemic heart disease and type 2 diabetes mellitus is noted to have non-visible haematuria during an annual review. He is currently feeling well and is asymptomatic. The urine dipstick showed blood ++, with no protein and no leucocytes. This result is repeated one week later.

His current medications include aspirin, bisoprolol, atorvastatin, ramipril, metformin and pioglitazone.

Which one of the following drugs should be stopped whilst awaiting further investigations?

- a. Aspirin
- b. Ramipril
- c. Atorvastatin
- d. Metformin
- e. Pioglitazone

Thiazolidinediones are associated with an increased risk of bladder cancer

The correct answer is **Pioglitazone**. Pioglitazone, an oral antidiabetic agent used in the management of type 2 diabetes mellitus, has been associated with an increased risk of bladder cancer. In the presence of non-visible haematuria (blood in urine), this medication should be discontinued until further investigations are completed.

Aspirin is not typically associated with haematuria and its use as antiplatelet therapy in a patient with ischemic heart disease should not be interrupted without a compelling reason.

Ramipril, an angiotensin-converting enzyme (ACE) inhibitor, is used for hypertension and cardiac conditions. While it can occasionally cause renal impairment leading to proteinuria, it does not typically cause haematuria.

Atorvastatin, a statin used for hyperlipidaemia and cardiovascular risk reduction, may have some side effects such as myopathy or liver dysfunction but it is not commonly associated with haematuria.

Finally, **Metformin**, another oral antidiabetic agent, can lead to lactic acidosis in cases of renal insufficiency but it does not usually cause haematuria. Therefore, there would be no need to stop metformin based on the given clinical scenario

Question #253

A 65-year-old man who is known to have type 2 diabetes mellitus presents for advice. He is a Muslim and is considering fasting for Ramadan. His diabetes is currently controlled with a combination of diet and metformin 500mg tds. Looking at his records the last HbA1c was 6.4% (46 mmol/mol). If he decides to fast during Ramadan, what is the most appropriate advice to give regarding his metformin?

- a) Metformin should be stopped
- b) Metformin 1.5g after sunset
- c) Metformin 500mg before sunrise, 1g after sunset
- d) Metformin 500mg after sunset
- e) Metformin 1g before sunrise, 500mg after sunset

During Ramadan, one-third of the normal metformin dose should be taken before sunrise and two-thirds should be taken after sunset

The correct answer is **Metformin 500mg before sunrise, 1g after sunset**. This is because during Ramadan, individuals who fast are required to refrain from consuming food and drink from sunrise to sunset. In order to maintain adequate glycaemic control while minimizing the risk of hypoglycaemia, it is recommended that metformin be taken in a divided dose - a smaller dose before sunrise (pre-dawn meal or Suhoor) and a larger dose after sunset (evening meal or Iftar). This allows for better distribution of the medication throughout the day while still adhering to the fasting requirements.

Metformin should be stopped is incorrect because stopping metformin abruptly may result in poor glycaemic control and increased risk of hyperglycaemia. It is important for patients with diabetes to continue their medications during Ramadan, with appropriate adjustments made under medical supervision.

Metformin 1.5g after sunset is not the best option because taking the entire daily dose at once can increase the risk of gastrointestinal side effects and may not provide optimal glycaemic control throughout the day. Dividing the dose as mentioned earlier helps maintain better blood glucose levels during fasting hours.

Metformin 500mg after sunset is also incorrect because this dosing regimen significantly reduces the total daily dose of metformin compared to his current regimen, which may lead to inadequate glycaemic control. Additionally, it does not account for any pre-dawn medication intake.

Finally, **Metformin 1g before sunrise, 500mg after sunset** is not ideal as it provides a higher dose before sunrise than necessary. The recommended approach is to take a smaller dose before sunrise and a larger one after sunset in order to minimize potential hypoglycaemia during fasting hours while maintaining adequate glycaemic control

Question # 254

A 29-year-old female is admitted to the Emergency Department following an episode of collapse. She denies prodromal symptoms and woke up after an undetermined period to find herself lying face down on the ground. She has recently consulted her GP regarding feelings of generalised weakness, intermittent palpitations and dizziness.

Her past medical history is remarkable for hypothyroidism and rheumatoid arthritis, previously managed with infliximab, and complicated 3 months ago by a diagnosis of TB. Her regular medications include methotrexate, folic acid, levothyroxine, artificial tears, rifampicin, and isoniazid.

During the examination, the patient complains of recurrent palpitations. The cardiac monitor shows broad-complex tachycardia. Her blood pressure is stable at 117/68mmHg and she is given a bolus of amiodarone.

Her venous blood gas is as follows:

pH	7.31
pCO ₂	4.3 kPa
pO ₂	6.3 kPa
Na ⁺	137 mmol/l
K ⁺	2.1 mmol/l
Cl ⁻	114 mmol/l
iCa ²⁺	1.05 mmol/l
Glucose	5.4 mmol/l
HCO ₃	15.6 mmol/l
BE	-9.5 mmol/l

Some additional investigations are requested:

Urine dipstick	pH 5.0 glucose ++ protein +
XR chest	unremarkable

XR abdomen

normal bowel gas pattern, no evidence of abnormal renal calcification

What is the most likely cause of the metabolic abnormalities described?

- a. Addison's disease
- b. Fanconi syndrome
- c. Rheumatoid arthritis
- d. Sjogren's syndrome
- e. Liver cirrhosis

Answer is B.

The patient has a normal anion gap hyperchloraemic metabolic acidosis associated with hypokalaemia. This, in combination with the absence of GI symptoms, should alert you to the possibility of underlying renal tubular acidosis.

Fanconi syndrome is a disorder of proximal renal tubular function that leads to abnormal loss of bicarbonate, glucose, potassium, phosphate, uric acid and amino acids in the urine. Patients have features of type 2 renal tubular acidosis, accompanied by hypophosphataemia, glycosuria, and aminoaciduria. Presenting features include polyuria, polydipsia, osteomalacia (rickets and growth failure in children) and symptoms secondary to the electrolyte abnormalities associated with the disorder. Rifampicin has recently been implicated in the development of Fanconi syndrome and is the most likely cause in this case.

Rheumatoid arthritis and Sjogren's syndrome are causes of type 1 (distal) renal tubular acidosis; a failure of hydrogen ion secretion in the distal tubule that causes hypokalaemia and nephrocalcinosis. The absence of abnormal renal calcification on the patient's x-ray points away from this as the diagnosis.

Addison's disease typically causes hyperkalaemia, often associated with other electrolyte abnormalities including hyponatraemia and hypoglycaemia. It would, therefore, be inconsistent with the biochemistry provided.

Liver cirrhosis does not typically cause renal tubular acidosis unless it is itself a manifestation of Wilson's disease. There is nothing in the question to suggest Wilson's disease as the underlying diagnosis, however, and the occurrence of this pattern of metabolic abnormality would still be predicated on the development of a complicating Fanconi syndrome

Question # 255

A 29-year-old man with known Addison's disease contacts his endocrinologist. He has signed up for a marathon and is wondering what to do with hydrocortisone and fludrocortisone for the event. He is normally fit and well without any other medical problems. His diagnosis was established five years ago and since then he has had two admissions due to an inability of taking hydrocortisone and now has emergency doses of IM hydrocortisone. What is the most appropriate advice to give?

- a) Take doses as normal for the marathon
- b) Double the hydrocortisone dose but not the fludrocortisone dose for the marathon
- c) Double the fludrocortisone dose but not hydrocortisone dose for the marathon
- d) Double both the fludrocortisone dose and the hydrocortisone dose for the marathon
- e) Omit both medications for the marathon

Answer is B.

A patient with Addisons' disease who undertakes significantly strenuous activity should double the dose of glucocorticoid and mineralocorticoids

This is a patient with Addison's disease who is planning significantly strenuous activity. In such a situation, both fludrocortisone dose and the hydrocortisone dose should be doubled, with advice to increase fluid intake as well. If the patient was just on hydrocortisone then no additional fludrocortisone would be needed.

Glucocorticoid therapy should ideally mimic endogenous cortisol rhythm with the lowest level at time of falling asleep and highest at waking. Treatment should aim to control symptoms at the smallest dose possible. Hydrocortisone has a short half-life but excellent bioavailability.

Mineralocorticoid therapy will be eventually required in adrenal insufficiency to counter intravascular volume depletion. It is important in the presence of increased fluid loss that the mineralocorticoid dose is adjusted. This is why doubling of the dose is advised.

If less strenuous activity, such as a long hike, was planned than increasing the dose of hydrocortisone by 5-10mg would be reasonable, without any change in fludrocortisone. This change would also apply for any day that increased activity is planned for. They should also be advised to increase fluid intake in the proportion to the increase in hydrocortisone dose.

If a patient is taking an alternative steroid regime, such as dexamethasone or prednisolone, then endocrinology advise would be needed.

Omitting any medications could be dangerous and precipitate an Addisonian crisis.

Question #256

You review a 28-year-old woman who is 26 weeks pregnant. She has just had a routine oral glucose tolerance test as her BMI is 34 kg/m^2 . The following results were obtained:

Time (hours)	Blood glucose (mmol/l)
0	7.4
2	11.2

There have been no other antenatal problems and her anomaly scan was normal. What is the most appropriate action?

- a. Repeat oral glucose tolerance test in 4 weeks
- b. Start metformin + advice about diet / exercise + self-monitor glucose levels
- c. Advice about diet / exercise + self-monitor glucose levels
- d. Start insulin + advice about diet / exercise + self-monitor glucose levels
- e. Reassure results within normal limits

Answer is D.

NICE have recently changed their gestational diabetes guidelines. Insulin should be started in the fasting glucose is ≥ 7 mmol/l. Aspirin should also be considered given the increased risk of pre-eclampsia

Question #257

An 18-year-old male with no prior medical history is admitted to the resuscitation room of Emergency Department where you are asked to assess him. He is noted to be hypotensive, tachycardic and febrile. Further assessment highlights a widespread, non-blanching, purple rash. He is diagnosed with meningococcal septicaemia and is treated appropriately. Nevertheless, his condition deteriorates further when he also develops Waterhouse-Friderichsen syndrome. Which of the following sets of blood results would be most consistent with his condition at this point?

- a) Na⁺ 147mmol/L, K⁺ 5.8mmol/L, Glucose 7.5mmol/L
- b)
Na⁺ 147mmol/L, K⁺ 3.0mmol/L, Glucose 2.0mmol/L
- c) Na⁺ 129mmol/L, K⁺ 5.8mmol/L, Glucose 2.0mmol/L
- d) Na⁺ 147mmol/L, K⁺ 5.8mmol/L, Glucose 11.5mmol/L
- e) Na⁺ 129mmol/L, K⁺ 5.8mmol/L, Glucose 11.5mmol/L

Answer is C.

Waterhouse-Friderichsen syndrome is defined as adrenal failure due to bleeding into the adrenal glands (otherwise referred to as haemorrhagic adrenalitis) and is most commonly caused by meningococcal septicaemia.

The biochemical pattern which results is analogous to that seen in an Addisonian crisis, and so you would expect to see hyponatraemia, hyperkalaemia and hypoglycaemia

Question #258

A 35-year-old woman presents with weight gain for the last couple of years. She feels that most weight has been gained on her face and abdomen. On further questioning, her periods have been irregular for some time. She was diagnosed with type 2 diabetes mellitus last year. The patient drinks approximately 15 units of alcohol per day.

On examination, she has violaceous striae across the abdomen. Proximal muscle weakness is also evident when asking the patient to stand. Blood tests are taken:

Na ⁺	143 mmol/L	(135 - 145)
K ⁺	2.8 mmol/L	(3.5 - 5.0)

The team suspects that the patient's alcohol use may be the underlying cause of her presentation, rather than an endogenous cause, and want to differentiate the two.

What is the most appropriate investigation?

- a. High-dose dexamethasone suppression test
- b. Insulin stress test
- c. Low-dose dexamethasone suppression test

- d. Petrosal sinus sampling
- e. Short synacthen test

The insulin tolerance test can be used to distinguish Cushing's syndrome from pseudo-Cushing's

Important for meLess important

This patient may have pseudo-Cushing's syndrome as a result of alcohol use, as opposed to true Cushing's syndrome. Patients with pseudo-Cushing's syndrome display clinical and biochemical features similar to those seen in Cushing's syndrome, such as the scenario here, but the presentation is caused by alcohol abuse, rather than an endogenous source or steroid use. Other causes of pseudo-Cushing's include severe depression, eating disorders, and chronic illness. Of the options listed above, the **insulin stress test** is used to differentiate between true Cushing's and pseudo-Cushing's.

The **high-dose dexamethasone suppression test** is used to differentiate pituitary from ectopic sources of Cushing's syndrome. It is not useful in pseudo-Cushing's.

The **low-dose dexamethasone suppression test** is used as a first-line test for patients with suspected Cushing's syndrome. It will not help to differentiate between Cushing's and pseudo-Cushing's.

Petrosal sinus sampling is performed for patients with confirmed ACTH-dependent Cushing's syndrome without an obvious pituitary lesion on MRI. Blood is sampled peripherally and from the inferior petrosal sinuses and the ratio of ACTH is measured. It is not useful to distinguish Cushing's from pseudo-Cushing's.

The **short synacthen test** is not relevant here. It is involved in the diagnosis of Addison's disease, rather than Cushing's syndrome.

Question #259

A 22 year old lady presents over a year with mild-moderate, intermittent abdominal pain. She has felt low in mood over this period and also her periods have stopped. Her history includes two previous attacks of renal calculi formation managed conservatively. She had a car crash recently, where she says that the car just 'came out of nowhere'. She is intermittently getting global headaches that can be very severe in nature but are otherwise featureless. On prompting, she tells you that she has sometimes noticed a white nipple discharge on her clothing. She has had low blood pressure and several faints over this last year and after her GP discovered a low serum cortisol level he has started her on oral hydrocortisone and referred her to your clinic. On examination today her blood pressure 130/80. She has a blistering, red rash across her lower abdomen and back. Her abdomen is largely non-tender with no palpable organomegally or peritonism. Visual fields are reduced bitemporally. Her urine dipstick shows glycosuria. The remainder of the examination is unremarkable. Which of the following is likely to treat the underlying condition most effectively?

- a) Bisphosphonates
- b) Surgery
- c) Cabergoline
- d) Octreotide
- e) Insulin

The unifying diagnosis here is is multiple endocrine neoplasia (MEN) type 1. MEN1 consists of tumours of the parathyroid, pituitary, and pancreas.

This patient has symptoms of hypercalcaemia (abdominal groans, renal stones, and psychic moans of the 'bones, stones, groans, psychic moans'). This can be caused by a parathyroid tumour. She has symptoms suggestive of a non-functioning pituitary macro-adenoma causing hyperprolactinemia (amenorrhea and galactorrhoea) and hypopituitarism presenting as Addison's from suppressed ACTH. Dopamine is the inhibitor of prolactin in the pituitary, and comes from the hypothalamus. A nonfunctioning pituitary macro-adenoma compresses the pituitary stalk, interrupting dopamine flow to the pituitary, and therefore abolishing dopaminergic inhibition of prolactin, resulting in hyperprolactinaemia.

At the same time, the macro-adenoma causes hypopituitarism through local pressure effects on the pituitary itself. 76% of pituitary tumours in MEN1 are prolactinomas, with the remainder being nonfunctioning adenomas.

Prolactinomas are extremely sensitive to medical management with e.g. cabergoline or bromocriptine and even shrink in size subsequently. First line treatment for nonfunctioning adenomas however is surgical removal. The glycosuria is suggestive of hyperglycaemia, which in the context of this MEN picture suggests a glucagonoma.

The answers try and trick you into looking to manage just one of the abnormalities here in an isolated fashion, e.g. targeting the pituitary with cabergoline, the pancreas with insulin, or the parathyroid with bisphosphonates. This may happen if you do not recognise that this is MEN. However, as the question asks what treatment is likely to treat the underlying condition, clearly insulin alone, cabergoline alone, or bisphosphonate alone will not suffice. Of the options available, only surgery can tackle all of the problems, and indeed is what most patients with MEN end up needing.

MEN is an autosomal dominant condition. MEN1 consists of tumours of the parathyroid, anterior pituitary, and pancreas:

- The parathyroid tumours cause hypercalcaemia and its symptoms (which don't forget include polyuria and polydypsia). The management of hypercalcaemia is fluid resuscitation and bisphosphonates.
- Pituitary tumours can be a prolactinoma, somatotroph adenoma (causing acromegaly by secreting growth hormone), or an ACTH-secreting tumour causing Cushing's disease. There can be any combination of these, although the question will usually point you toward one particular pituitary abnormality. Remember that you are unlikely to elicit a galactorrhoea history unless you specifically ask and are sensitive about it.

- The pancreas tumours can be an insulinoma (persistent hypoglycaemia -check C-peptide which will be high in endogenous insulin secretion as opposed to exogenously given insulin e.g. in self harm/non-accidental injury, where c-peptide levels remain low.), gastrinoma (Zollinger-Ellison, presenting as refractory gastric ulcers), glucagonoma causing persistent hyperglycaemia and also a necrolytic migratory erythema (as in this case), or a VIPoma causing profuse watery diarrhoea (VIP is the antagonist to gastrin and therefore tries to get you to flush the gut out rather than hold and digest contents).

MEN II has two forms. MEN2a presents with medullary thyroid carcinoma (neck lump), parathyroid tumour (hypercalcemia) and pheochromocytoma (hypertension, flushing, tachycardia intermittently). MEN2b presents with medullary thyroid carcinoma and pheochromocytoma.

The key message for the exam is that if you are presented with a case where you suspect there is an endocrine abnormality (even just a high calcium), look again at the stem to make sure you are not missing other endocrine abnormalities being present (e.g. amenorrhoea) that might alert you to there being an underlying diagnosis of MEN. If you know a few key features of each of the possible abnormalities within each type of MEN, you should be able to pinpoint which MEN type is present.

Question #260

A 43-year-old man is referred by his GP with a 4 week history of a lump appearing on the right side of his neck. The lump is roughly 7 mm and is located on the right side of the thyroid gland, in the anterior triangle. It does not move when the patient sticks out his tongue, but it does move on swallowing. There is no history of weight loss or night sweats.

Blood tests are performed and reveal:

Hb	12.9 g/l
Platelets	210 * 10 ⁹ /l
WBC	6.0 * 10 ⁹ /l
Na+	141 mmol/l
K+	3.9 mmol/l
Urea	4.1 mmol/l
Creatinine	33 µmol/l

What is the most appropriate first-line investigation?

- a) Radioisotope scan of thyroid
- b) Magnetic resonance scan of head and neck
- c) Excision biopsy
- d) Fine needle aspiration biopsy
- e) Ultrasound scan of thyroid

Answer is E.

High-resolution ultrasound scanning is an ideal first-line initial imaging investigation for most neck lumps. Because most lesions in the neck are site-specific, once a lesion has been located, specific ultrasound features can be used to establish the diagnosis

Question #261

A 34 year-old woman is seen by her GP for the annual review of her type 1 diabetes. Her most recent HbA1c is 58 mmol/mol and the only problem she has noticed are severe hypoglycaemic episodes during the night around 2-4am, that she noticed she was getting while working night shifts for a local supermarket. However, by breakfast time, her blood glucose levels often rise to around 15

mmol/mol. Her insulin regimen is currently a twice daily mixed insulin.

What is the most appropriate change to her current insulin treatment?

- a. Move to a basal bolus of insulin
- b. Take 1 dextrose tablet at 9pm
- c. Reduce nocturnal insulin dose
- d. Add gliclazide

Answer is A .

This patient is experiencing severe nocturnal hypoglycaemic episodes followed by rises in blood glucose by breakfast time. The most appropriate change to the insulin treatment regimen is changing to a basal bolus regime, with one daily injection of background long-acting insulin and three short acting injections of insulin

Question #262

A 53-year-old woman is referred to an endocrinology clinic. She has a history of Grave's disease which had previously been controlled on carbimazole however recently presented to her general practitioner due to a two-month history of diarrhoea, palpitations, and heat intolerance. She is post-menopausal and has not had any recent illness or relevant past medical history. The general practitioner performed some thyroid function tests, results of which are as follows:

TSH	0.2	(0.5-5.5 mu/L)
Free T4	25. 2	(9-18 pmol/L)

On examination in the clinic, she is warm, tachycardic but otherwise well. A smooth, non-tender goitre is noted however there are no eye signs.

What is the most appropriate treatment?

- a) Advice on medication compliance
- b) Prednisolone
- c) Propylthiouracil
- d) Radioiodine therapy
- e) Thyroidectomy

Answer is D.

Radioiodine therapy is the treatment of choice for patients with a relapse of Graves disease in the absence of contraindications, such as pregnancy and active severe Graves ophthalmopathy

Given this woman's medical history, findings of a smooth non-tender goitre, and laboratory results, the most likely diagnosis here is a relapse of her Grave's disease. In these cases and the absence of any contraindications, radioiodine therapy is the preferred treatment.

Prednisolone can be used in subacute thyroiditis however this is not the diagnosis here as evidenced by the lack of recent illness and non-tender goitre.

Propylthiouracil is an alternative antithyroid drug to carbimazole and is preferred in early pregnancy. As this woman is presenting with a relapse of Grave's disease, radioiodine therapy is preferred. Also, note she is post-menopausal, and therefore pregnancy is not a contraindication here to either radioiodine therapy or carbimazole.

Thyroidectomy is not the first-line treatment in a relapse of previously well-controlled Grave's disease.

Question #263

A 77-year-old female is admitted to the acute medical unit with a chest infection. Her past medical history includes COPD and heart failure. Her current medications include salbutamol, tiotropium, bisoprolol and ramipril. On examination you note left basal crepitations. The JVP is at 2cm above the sternal angle. There is no peripheral oedema. Her blood pressure is 145/85 mmHg.

You note that her blood results are as follows:

Na+	120 mmol/l
Urine osmolarity	400 mosmol/l
Urine sodium	50 mosmol/l
Urea	6.2 mmol/l
Creatinine	54 µmol/l
fT3	6.2 pmol/l (normal range 3.5 - 7.8)
fT4	6 pmol/l (normal range 9.0 - 25.0)
TSH	0.2 mU/l (normal range 0.4 - 4.0)
morning cortisol	normal

You water restrict the patient to 1.5 litres per day. On day 4 her bloods are reported as follows:

Na +	118 mmol/l
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How would you manage this patient?

- a. Furosemide
- b. Levothyroxine
- c. Demeclocycline
- d. Hypertonic saline (3%)

e. Hypotonic saline (0.45%)

If SIADH is resistant to fluid restriction then the drug demeclocycline can be used

The differential for hyponatraemia is wide. The fluid status of the patient is of paramount importance for identifying the cause. In this example, the patient is clearly euvoalaemic. Causes of euvoalaemic hyponatraemia include SIADH, hypothyroidism, and adrenal insufficiency. The serum and urine osmolarities, and urinary sodium are consistent with either of these diagnoses.

SIADH is effectively a diagnosis of exclusion - primary hypothyroidism and hypoadrenalinism must be excluded to make the diagnosis.

In this case, the thyroid function tests are suggestive of euthyroid sick syndrome. This would not be an indication for initiating levothyroxine. Although hypothyroidism is a well-known cause of hyponatraemia euthyroid sick syndrome would not effect sodium balance. It is often stated that to make a diagnosis of SIADH relies upon 'normal thyroid function tests'. It is important to note that this is referring to primary hypothyroidism. Euthyroid sick syndrome does not preclude the diagnosis of SIADH.

The morning cortisol is normal which suggests that hypoadrenalinism is not present.

Therefore the most likely diagnosis is SIADH secondary to a chest infection. The initial treatment for non-severe SIADH is fluid restriction.

This patient has been trialled with fluid restriction however the sodium has not improved and has in fact got worse. The next step in the management would be demeclocycline. Demeclocycline inhibits the effects of ADH on the renal tubules. It effectively causes a nephrogenic diabetes insipidus.

The patient's symptoms are not severe enough to warrant use of hypertonic saline. Hypertonic saline has risks associated with it, as rapid correction of serum Na can result in central pontine myelinolysis.

If the patient had been hypervolaemic then furosemide would be indicated

Question #264

A middle age woman is being treated for symptomatic hypercalcaemia associated with a squamous cell lung cancer (serum calcium 3.60 mmol/L). She is slow to respond to initial measures of saline hydration and intravenous pamidronate. Whilst awaiting surgical resection for her underlying cancer what may be the next best step in her management?

Na+	142 mmol/l
K+	4.3 mmol/l
Urea	7.0 mmol/l
Creatinine	89 µmol/l
Glucose	4.8 mmol/l

What is the most appropriate management?

- a) High dose loop diuretics
- b) Calcitonin 4 units/kg
- c) Insulin actrapid 50 units in 50% dextrose
- d) IV colloid administration instead of crystalloid
- e) Plasma exchange

Answer is B.

General symptoms of hypercalcaemia may include malaise, lethargy, depression, dehydration and can lead to depressed consciousness. Bone pain and abdominal pain may feature and can be summarised by the classic 'bones, stones, moans and abdominal groans'.

Alongside searching for the underlying cause, management initially involves

aggressive rehydration, typically 4-6 L saline on the first day. Bisphosphonates act by interfering with osteoclastic bone resorption and typically IV pamidronate is used at a dose of 60-90mg over 2-4 hours. Calcitonin (extracted from salmon) also interferes with osteoclast activity as well as increasing renal calcium excretion.

Diuretics may lead to further dehydration. Dialysis may be a last line treatment for life threatening hypercalacemia, but not plasma exchange

Question #265

A 23-year-old woman attends a fertility clinic with her partner. She complains of oligomenorrhoea and galactorrhoea and has failed to get pregnant after 18 months of regular unprotected intercourse. Blood tests reveal a serum prolactin level of 6000 mIU/l (normal <500 mIU/l). A pituitary MRI is arranged which shows a microprolactinoma.

Which of the following is the best initial treatment?

- a. Octreotide
- b. Bromocriptine
- c. Trans-sphenoidal hypophysectomy
- d. Pituitary radiotherapy
- e. Transfrontal hypophysectomy

Answer is B.

Dopamine agonists (e.g. cabergoline, bromocriptine) are first-line treatment for prolactinomas, even if there are significant neurological complications

This patient has a prolactinoma. In the majority of cases, symptomatic patients are treated medically with dopamine agonists (e.g. bromocriptine) which inhibit the release of prolactin from the pituitary gland. Surgery is performed for patients who cannot tolerate or fail to respond to medical therapy. A trans-sphenoidal approach is generally preferred unless there is a significant extra-pituitary extension. Radiotherapy is rarely performed and octreotide is a somatostatin analogue used in the treatment of acromegaly.

Question #266

A 45-year-old man with headaches returns to the endocrinology clinic following investigations. He originally complained of headaches waking him up at night, low energy, weight loss, low libido and postural dizziness. Investigations have revealed a normal full blood count, low free T4, low testosterone, low morning cortisol, low LH and FSH and low TSH. He also had an MRI which is suspicious for pituitary adenoma. He is keen to start whichever is necessary to feel better. He has no other medical problems and takes no regular tablets. Which hormonal replacement would be contra-indicated in the immediate setting?

- a) Hydrocortisone
- b) Prednisolone
- c) Dexamethasone
- d) Testosterone
- e) Levothyroxine

The Answer is E.

In hypothyroidism with adrenal failure do not start thyroid hormone replacement before glucocorticoid replacement as otherwise an adrenal crisis can be precipitated

This patient has panhypopituitarism secondary to a pituitary adenoma. It is critical for patients with adrenal and thyroid insufficiency to replace steroid function prior to replacing thyroid function as otherwise an adrenal crisis can be precipitated. Therefore levothyroxine is contra-indicated in the immediate setting, even though the patient will need replacement after he has started steroid replacement. Typically he would need hydrocortisone split into three doses to replace his steroid needs, and this is preferred over dexamethasone and prednisolone.

Ultimately the treatment may be trans-sphenoidal surgery

Question #267

A 45-year-old lady is admitted to hospital with abdominal pain and malaise. She has no past medical history and takes no regular medications or supplements. Bloods tests show:

Ca 2++ 2.70 mmol/l

PO4 + 1.2 mmol/l

Creatinine 60 µmol/l

Chest X-ray - normal appearances

She denies taking any medications or supplements. Her chest X-ray is normal in appearance, and renal function normal. You ring the GP and find out her calcium was also slightly raised 8 years ago. What is the most likely diagnosis?

- a. Secondary hyperparathyroidism
- b. Malignancy with bony metastasis
- c. Primary hyperparathyroidism
- d. Familial hypocalciuric hypercalcaemia
- e. Sarcoidosis

Answer is D.

PO4 would normally be low in primary hyperparathyroidism. Her renal function is normal excluding secondary hyperparathyroidism. Sarcoidosis is unlikely with a normal CXR. This leaves malignancy or familial hypocalciuric hypercalcaemia. Although malignancy is possible her raised Ca2+ 8 years makes familial hypocalciuric hypercalcaemia more likely

Question #268

A 35-year-old woman is known to have a family history of multiple endocrine neoplasia type 2a. She has tested positive for the RET oncogene and is concerned as her mother had suffered from thyroid cancer. However, she is reluctant to undergo a prophylactic thyroidectomy and asked if there is another option

available.

Which of the following management options would be appropriate for her case?

- a) Annual monitoring of thyroid peroxidase (TPO)
- b) Annual monitoring of thyroglobulin
- c) Annual 5 hydroxyindoleacetic acid levels (5-HIAA)
- d) Annual monitoring of calcitonin
- e) Annual monitoring of chromogranin A

Answer is D.

Calcitonin can be used as a marker to monitor for Medullary thyroid carcinoma in MEN type II in those unwilling to undergo prophylactic thyroidectomy

Calcitonin is a marker for medullary thyroid carcinoma which is associated with MEN type II.

Chromogranin A and 5-HIAA can be used as markers for neuroendocrine tumours and carcinoid syndrome respectively.

Thyroid peroxidase antibody titre can be used as a marker in autoimmune thyroid conditions such as Hashimoto's thyroiditis and Graves disease.

Thyroglobulin can be used as a marker for papillary or follicular thyroid carcinoma. For example, a rise in this marker following a thyroidectomy can indicate the recurrence of these particular cancers

Question #269

A 72-year-old male was admitted drowsy and confused. His family describe a 4-day history of shortness of breath and a productive cough. His past medical history includes type 2 diabetes mellitus, hypertension and hypercholesterolaemia. He usually takes metformin 500 mg three times daily, gliclazide 80 mg twice daily, amlodipine 5 mg daily and simvastatin 40 mg nightly.

On examination he is confused with dry mucous membranes, blood pressure of 100/50 mmHg, a pulse of 110/min, a temperature of 37.6 °C and a respiratory rate of 20/min. Coarse crepitations were found at the right base and his pulse was thready with a capillary refill of 3 seconds; jugular venous pressure was not visible. Capillary blood glucose was found to be H1.

A venous blood sample is taken:

Hb	129 g/l	Na+	161 mmol/l
Platelets	204 * 10 ⁹ /l	K+	4.9 mmol/l
WBC	13.1 * 10 ⁹ /l	Urea	15.2 mmol/l
Neuts	11.9 * 10 ⁹ /l	Creatinine	97 µmol/l
Glucose	56 mmol/l	eGFR	62 mg/l
Ketones	1.9 mmol/l	HbA1c	75 mmol/mol
pH	7.35	HCO ₃	20 mmol/mol

What treatment would you initiate first?

- a. 0.9% normal saline
- b. 0.45 % normal saline
- c. Hartmann's solution
- d. Intravenous insulin
- e. 5% dextrose

Answer is A.

The underlying diagnosis is hyperosmolar hyperglycaemic state (HHS) precipitated by a lower respiratory tract infection. The diagnosis is made when a patient shows marked hyperglycaemia and hypovolaemia with a serum osmolality > 320 mosm/kg in the absence of marked ketonaemia or acidosis. This patient is clinically dry with a glucose of 56 and a calculated osmolality of $2(161+4.9) + 56 +$

$15 = 403 \text{ mosm/kg}$ with mild ketonaemia.

Despite this gentlemen's hypernatraemia, the first line fluid therapy is 0.9% normal saline as patients in HHS are sodium deplete. One litre should be administered rapidly and electrolytes then repeated to gauge potassium requirements in further fluid bags. A small initial rise in sodium is expected and this should not discourage further use of normal saline. Insulin is not routinely started immediately in the absence of significant ketonaemia as glucose will fall with fluid therapy alone. When glucose stops falling insulin may be started if glucose still remains high. Too rapid correction of osmolality i.e. with aggressive insulin and fluids in combination can precipitate cerebral oedema.

Question #270

An 18-year-old man presents to the clinic with poor development of secondary sexual characteristics. On examination, you note a lack of testicle development and sparse axillary and pubic hair.

Blood results are as follows:

Testosterone	2 nmol/L	(6 -27)
FSH	4.6 IU/L	(1.8 - 22.5)
LH	2.8 IU/L	(1.2 - 103)

What is the most likely diagnosis?

- a) Androgen insensitivity syndrome
- b) Kallmann's syndrome
- c) Klinefelter syndrome
- d) Mullerian agenesis
- e) Turner's syndrome

Kallman's syndrome - LH & FSH low-normal and testosterone is low

Kallmann's syndrome is correct. Kallmann's syndrome is a recognised cause of delayed puberty secondary to hypogonadotropic hypogonadism. The biochemical profile is in keeping with hypogonadotropic hypogonadism due to the presence of low testosterone and inappropriately normal LH and FSH. Kallmann's syndrome is the only listed condition which results in hypogonadotropic hypogonadism.

Androgen insensitivity syndrome is correct. Androgen insensitivity syndrome in its complete form is a disorder of hormone resistance characterised by a female phenotype in a genetic male due to the resistance to the effector functions of testosterone. The hormonal profile findings include very high testosterone, elevated LH, and a relatively normal FSH. Thus the patient's phenotypical sex and hormonal profile exclude this diagnosis.

Klinefelter syndrome is incorrect. Klinefelter syndrome is an aneuploid genetic condition where a male has an additional copy of the X chromosome. The primary features are infertility and small, poorly functioning testicles. This condition results in primary gonadal failure, and thus a compensatory elevation in LH and FSH.

Mullerian agenesis is incorrect. Mullerian agenesis is a congenital malformation characterised by a failure of the Mullerian ducts to develop, resulting in a missing uterus and variable degrees of vaginal hypoplasia of its upper portion. Given that the patient is phenotypically male, this diagnosis is unlikely.

Turner's syndrome is incorrect. Turner's syndrome can cause primary ovarian failure due to gonadal dysgenesis. This would present with low sex hormone levels, and markedly raised LH and FSH levels. It only affects females. Thus the patient's phenotypic sex, and biochemical results are not in keeping with this diagnosis.

Question #271

A 68-year-old man is referred to the cardiology outpatient department with shortness of breath on exertion. He becomes breathless when ascending stairs.

There is no chest pain. He has a past medical history of ischaemic heart disease and heart failure with reduced ejection fraction (30%). He is on aspirin, bisoprolol, ramipril, spironolactone, atorvastatin and lansoprazole. He does not smoke or drink alcohol.

His observations are heart rate 83 beats per minute, blood pressure 110/85 mmHg, respiratory rate 18/minute, oxygen saturations 97% on room air and temperature 37.1°C.

On examination, he is euvolemic. His heart sounds are normal and there is no peripheral oedema. Chest auscultation is normal. The pulse is regular.

From the options listed, what is the best choice of medication to improve his symptoms?

- a) Flecainide
- b) Ivabradine
- c) Nicorandil
- d) Ranolazine
- e) Verapamil

Ivabradine should be considered in heart failure if the patient has sinus rhythm > 75/min and a LVEF < 35% and have not responded to ACE-inhibitor, beta-blocker and aldosterone antagonist therapy

Ivabradine is the correct answer. This man has NYHA class II/III heart failure despite treatment with an ace inhibitor, beta-blocker and mineralocorticoid receptor antagonist. Ivabradine is an option in heart failure for those patients who have not responded to the previously described combination of medications and have a heart rate > 75 beats per minute and an LVEF < 35%.

Nicorandil and ranolazine are incorrect. These medications are options for the treatment of angina, not heart failure.

Flecainide is incorrect. This is a medication used to chemically cardiovert atrial fibrillation. This patient is in sinus rhythm.

Verapamil is incorrect. Negatively inotropic calcium channel blockers should be avoided in heart failure, especially in the context of current beta blocker treatment.

Question #272

An 84-year-old gentleman is admitted to hospital with a short history of episodes of loss of consciousness. The patient has no clear recollection of the events, but his family informs you that they have not identified any specific pattern to them. Most of them have been unwitnessed, with the exception of the last one, where according to his wife he became pale, his eyes rolled backwards and fell on the chair. Some minor flickering was noted. Once he regained consciousness he was briefly confused and anxious, but soon realised where he was. Cardiovascular examination was unremarkable, with no postural hypotension but a blood pressure of 103/55 mmHg, and no focal neurology was isolated. His bloods were unremarkable.

His ECG was that of sinus rhythm: 67/min with Right Bundle Branch Block (RBBB). You see from a previous echocardiogram in his notes, that he has a degree of heart failure with an ejection fraction of 45%. A 72 hour holter monitor revealed no significant pauses.

Which action would you take next?

- a) Arrange MRI head
- b) Arrange EEG
- c) CRT-D placement
- d) Arrange insertion of Reveal Device
- e) Arrange performance of Carotid sinus and Tilt-test

In patient with BBB and unexplained syncope but an ejection fraction >35% proceed with further testing (e.g. carotid sinus massage, electrophysiological studies) prior to

As per the ESC Guidelines on Cardiac Pacing and Cardiac Resynchronization therapy (2013) it is important to exclude any other probable causes of collapse/syncope prior to installing any device. It is important to note that 'Less than half of the patients with BBB and syncope have a final diagnosis of cardiac syncope, albeit the probability is different among different type of BBB'. Given his symptomatology and the examination findings alongside the 72-hour holter monitor this is unlikely to be purely cardiogenic therefore further investigation is needed. With his age and presentation, autonomic dysfunction could be the cause, therefore carotid sinus and tilt-test may provide the answer. An MRI head and an EEG remain plausible options, but given the lack of solid seizure-like activity, can be arranged later on, provided that no answer is given by other tests.

Discuss (9)Improve

Question #273

An 82-year-old lady presented with shortness of breath and palpitations. These came on suddenly whilst she was watching television. She had a sedentary lifestyle and recently had been confined to bed with an upper respiratory tract infection. After this she had noticed that her ankles and legs were more swollen than usual, in particular her left leg. On further questioning she said that she had always needed three pillows to sleep but had recently required an extra pillow.

Her past medical history included hypertension, hypothyroidism, asthma and known coronary artery disease.

On examination her pulse was irregularly irregular. The jugular venous pressure was 6cm above the manubriosternal angle. Heart sounds were normal with no added sounds. On auscultation of the lung fields there were coarse crepitations at both bases. There was bilateral pitting oedema to the knees more marked on the left. The circumference of the left leg was 2cm greater than the right.

Observations:

- Heart rate: 128 beats per minute
- SaO₂: 92% on room air
- Respiratory rate: 22 breaths per minute
- Temperature: 36.8 degrees Celsius
- Blood pressure: 110/68 mmHg

Investigations:

- 12-lead electrocardiogram: atrial fibrillation with a fast ventricular response, left ventricular hypertrophy
- Chest X-ray: cardiomegaly, Kerley B lines, small bilateral pleural effusions, pulmonary oedema
- Echocardiogram: poor biventricular function with an ejection fraction of 35%
- Computed Tomography Pulmonary Angiogram: small segmental pulmonary embolus

This lady was anticoagulated appropriately. Following this acute episode she improved clinically but remained in atrial fibrillation.

Given the clinical information available which of the following is the most appropriate drug to control her heart rate in the long term?

- a) Metoprolol
- b) Digoxin

- c) Diltiazem
- d) Flecainid
- e) Warfarin

Digoxin is a good choice as this patient also has evidence of congestive cardiac failure which may also benefit from digoxin therapy. It is important to note that NICE recommends the use of digoxin only in sedentary patients and not those with an active lifestyle.

Metoprolol, a beta blocker, is not an appropriate choice here given the past medical history of asthma.

Warfarin is not a drug that is used for rate control; it is an anticoagulant.

Diltiazem is a rate-limiting calcium channel blocker and unlike digoxin does not have the added benefits in congestive cardiac failure.

Flecainide, a class 1c anti-arrhythmic drug, is used for chemical cardioversion in patients with no underlying structural heart disease as part of a pill-in-the-pocket regimen.

Question #274

A 24-year-old man is brought in by ambulance to the emergency department after a witnessed collapse. He was unconscious for 10-15 seconds before quickly recovering. There were no preceding symptoms and no witnessed seizure activity. He has no past medical history and is not on any regular medications.

Observations:

- Heart rate 82 beats per minute
- Blood pressure 120/80 mmHg
- Respiratory rate 16/minute

- Oxygen saturations 96% on room air
- Temperature 37C

The examination is unremarkable and his Glasgow coma scale is 15/15.

An ECG demonstrates possible coved ST elevation in leads V1-V3 without reciprocal changes.

Troponin	6 ng/L	(< 14)
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Given the likely diagnosis, the administration of which anti-arrhythmic will make the ECG changes more apparent?

- a) Dobutamine
- b) Flecainide
- c) Isoprenaline
- d) Metoprolol
- e) Quinidine

ECG changes in Brugada syndrome are more apparent following the administration of flecainide or ajmaline - this is the investigation of choice in suspected cases

Flecainide is the correct answer. The patient presents with syncope and ECG changes that raise the possibility of Brugada syndrome (coved ST elevation in leads V1-V3). Administration of flecainide can make these ECG changes more apparent and aid diagnosis.

Dobutamine is incorrect. This medication can be used in stress echocardiography to determine if there is any inducible ischaemia but is not used to make the ECG changes of Brugada syndrome more apparent.

Metoprolol is incorrect. Typically, a short-acting beta-blocker is administered prior to CT coronary angiography with the aim of reducing the heart rate to a level of approximately 60 to facilitate interpretation of this form of imaging. It is not used to unmask the ECG changes of Brugada syndrome.

Quinidine is incorrect. This has been suggested in some reports to be useful as an anti-arrhythmic in Brugada syndrome. It does not make the ECG changes more prominent.

Isoprenaline is incorrect. This has been shown to normalise the electrocardiographic pattern in Brugada syndrome.

Discuss (2)Improve

Question #275

You are called to review a 68-year-old who is complaining of chest pain on the ward. She was admitted 6 days ago after having a stroke which has left her with a mild degree of right-sided hemiparesis. She has some difficulty articulating the nature of the pain but describes it as central.

Her past medical history includes hypertension and hypothyroidism. She has not had a stroke or transient ischaemic attack previously.

On examination, the heart rate is 102/min, blood pressure 122/78 mmHg, respiratory rate 22/min and oxygen saturations 94% on room air.

The nurse recorded an ECG:



What is the most likely diagnosis?

- a) Subarachnoid haemorrhage
- b) Inferior myocardial infarction
- c) Hypokalaemia
- d) Posterior myocardial infarction
- e) Pulmonary embolism

S1Q3T3 is a classic but uncommon ECG finding in PE

This woman is at risk of a pulmonary embolism given her relative immobility following the stroke.

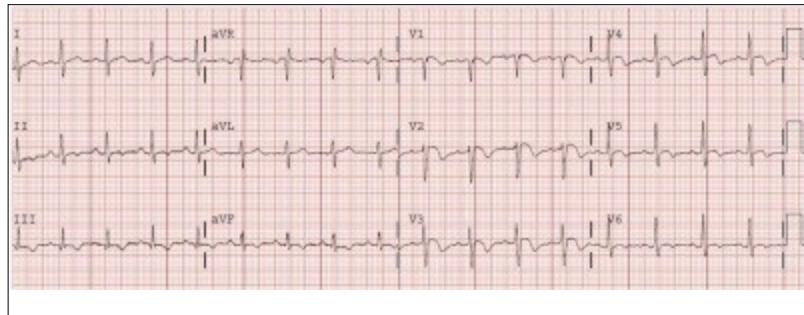
The tachycardia and tachypnoea are also signs associated with pulmonary embolism.

The ECG shows some of the features that may be associated with pulmonary embolism (sinus tachycardia, RBBB, right ventricular strain). Other features such as the left axis deviation are atypical.

Discuss (8)Improve

Question #276

A 35-year-old man presents to his GP with a five day history of dyspnoea, cough, chest pain and palpitations. He describes the chest pain as being worse when he takes a deep breath in and the only relieving factor is ibuprofen. There has been no sputum production or haemoptysis. He has no significant past medical history of note. On examination his pulse is 102/min, blood pressure is 124/78 mmHg and oxygen saturations are 95% on room air. Auscultation of his chest is normal. An ECG is taken:



What is the most likely diagnosis?

- a) Pulmonary embolism
- b) Acute coronary syndrome
- c) Pericarditis
- d) Musculoskeletal chest pain
- e) Viral pleurisy

S1Q3T3 is a classic but uncommon ECG finding in PE

The unifying diagnosis here is pulmonary embolism. He has dyspnoea and pleuritic chest pain. The tachycardia and low oxygen saturations are also suggestive. Low oxygen saturations in someone who is otherwise fit and well are unusual and need explaining. Acute coronary syndrome, pericarditis, viral pleurisy and musculoskeletal chest pain would not normally drop your saturations unless complications develop.

The ECG however is the clincher - he has the classic S1Q3T3 changes.

Discuss (15)Improve

Question #277

A 43-year-old with no past medical history presents with 6 months history of exertional dyspnoea. Her echocardiogram demonstrated impaired right ventricular function with raised pulmonary arterial pressure of 78 mmHg, good systolic function and ejection fraction 80%. Right and left heart catheterisation demonstrated pulmonary arterial hypertension, with a pulmonary arterial pressure of 49/24 mmHg, V/Q scan demonstrated no chronic pulmonary emboli, CTPA no acute emboli, ultrasound abdomen no portal hypertension. She is at present comfortable at rest but is short of breath on minimal activity. What is the appropriate management?

- a) No treatment
- b) IV prostaglandin
- c) Oral sildenafil
- d) Atrial septostomy
- e) List for lung transplantation

Treatment for pulmonary arterial hypertension is guided by the patients functional status. Patients with class I require no symptomatic treatment while those with class IV require prostaglandins, most likely intravenously. This patient is short of breath on minimal activity and hence functional class III. Endothelin receptor antagonists (e.g. bosentan) or phosphodiesterase V inhibitor (e.g. sildenafil) are appropriate. Atrial septostomy and lung transplantation are appropriate for patients who remain symptomatic at functional class IV despite intravenous prostaglandins and subsequent combination therapy with bosentan or sildenafil.

Question #278

A 79-year-old woman is reviewed in cardiology clinic as part of the ongoing assessment for possible intervention for severe aortic stenosis. The patient had been diagnosed with severe aortic stenosis 4 months previously, after experiencing progressive exertional breathlessness and reduced exercise tolerance. At her initial review at cardiology clinic, the patient had expressed interest in undergoing intervention for aortic stenosis: either surgical aortic valve replacement (SAVR) or transcatheter aortic valve insertion (TAVI). Subsequently, the patient had undergone a variety of investigations to assess her suitability for the above procedures.

The patient had a good functional status, leaving independently with her daughter and still participating in a wide range of community activities. The patient had a long-standing diagnosis of hypertension and had suffered a left cortical stroke 3 years previously. In addition, the patient had chronic obstructive pulmonary disease, although she had stopped smoking 30 years previously. The patient's regular medications were amlodipine, ramipril, clopidogrel, simvastatin and an ipratropium inhaler. The patient used a salbutamol inhaler as required. The patient had no known drug allergies.

A summary of the investigations undergone by the patient is given below.

Haemoglobin	128 g / dL
Mean cell volume	87.1 fl
White cell count	6.3 x 10 ³ / microlitre
Platelets	324 x 10 ³ / microlitre
Urea	5.3 mmol / L
Creatinine	124 micromol / L

Sodium	136 mmol / L
Potassium	4.6 mmol / L

Investigation	Result
Transthoracic echocardiogram	Severe aortic stenosis (valve area 0.85 cm ²); no other valve disease; normal systolic function; no anatomic contraindications to TAVI
Coronary angiography	No evidence of coronary artery disease
Iliofemoral angiography	Severe calcification and tortuosity of iliac arteries; unsuitable for transfemoral TAVI
Pulmonary function tests	Moderate obstructive lung disease

Following the above assessment, surgical aortic valve replacement was estimated to carry a 4.1 % risk of mortality and 3.7 % risk of permanent stroke (intermediate risk). No contraindications to transapical transcatheter aortic valve insertion were identified.

What is the recommended choice of intervention for the patient's aortic stenosis?

- a) Surgical aortic valve replacement and transcatheter aortic valve insertion inappropriate
- b) Surgical aortic valve replacement with mechanical valve
- c) Transapical transcatheter aortic valve insertion
- d) Surgical aortic valve replacement with bioprosthetic valve

e) Transfemoral transcatheter aortic valve insertion

The patient has severe, symptomatic aortic stenosis and without intervention is likely to have a poor prognosis. Given the patient's age, if she was to undergo surgical aortic valve replacement (SAVR) then a bioprosthetic valve would be preferred over a mechanical valve, freeing the patient from the need for lifelong anticoagulation. Typical risk stratification for SAVR defines the estimated risk of peri-operative death as low (< 4 %), intermediate (4-8 %) and high (> 8 %). Due to her comorbidities, the patient has an intermediate risk associated with SAVR, calculated using the STS risk calculator (<http://riskcalc.sts.org/stswebriskcalc/#/>).

Transcatheter aortic valve insertion (TAVI) was initially developed for patients considered unfit for SAVR due to high predicted mortality. However, a recent randomised controlled trial comparing outcomes for TAVI versus SAVR as an intervention for severe, symptomatic aortic stenosis has demonstrated that transfemoral TAVI can be a suitable intervention for some patients with a low or intermediate risk associated with SAVR. Transfemoral TAVI is increasingly favoured over SAVR as patient age increases.

In contrast, outcomes for transapical TAVI are inferior to SAVR, so this intervention is only appropriate for individuals with an unacceptably high surgical risk. For patients where both SAVR and transapical TAVI are possible, SAVR is strongly favoured at all patient ages.

Therefore, for this patient, bioprosthetic SAVR is the intervention of choice, favoured over transapical TAVI. Transfemoral TAVI is technically impossible in this patient due to the anatomy of her iliac arteries.

Question #279

A 74-year-old man attends the emergency department with epistaxis. He denies any other bleeding. He has a past medical history of atrial fibrillation and severe mitral stenosis for which he takes warfarin. Observations are as follows: heart rate 85 beats per minute, blood pressure 135/75 mmHg, respiratory rate 16 breaths per minute, SpO₂ 95% (on air), and temperature 37.5°C.

Blood results are as follows:

Hb	140 g/L	Male: (135-180) Female: (115 - 160)
Platelets	$88 * 10^9/L$	(150 - 400)
WBC	$6.2 * 10^9/L$	(4.0 - 11.0)

Prothrombin time (PT)	96 secs	(10-14 secs)
INR	6.8	(<1.1)
Activated partial thromboplastin time (APTT)	44 secs	(25-35 secs)
Fibrinogen	3.2 g/L	(2 - 4)

You withhold the patient's warfarin.

What further measures are required?

- a) Fresh frozen plasma
- b) Intravenous 10mg vitamin K
- c) Intravenous 3mg vitamin K
- d) Platelet transfusion
- e) Prothrombin complex concentrate

INR 5.0-8.0 (minor bleeding) - stop warfarin, give intravenous vitamin K 1-3mg, restart when INR < 5.0

The results of the coagulation screen suggest warfarin toxicity. The INR (derived from the PT) is used to monitor warfarin since it is the most representative, however, it is important to remember that warfarin can also prolong the APTT.

Intravenous 3mg vitamin K is correct. The patient has a minor bleed with an INR between 5.0 - 8.0. Thus the warfarin should be withheld and the patient should receive intravenous vitamin K 1-3mg. The warfarin should be restarted when the INR < 5.0

Fresh frozen plasma is incorrect. Fresh frozen plasma (FFP) is not recommended for use as a warfarin reversal agent as it can take hours to work due to the volume required. In major haemorrhages, FFP should only be given if prothrombin complex concentrate is not available.

Intravenous 10mg vitamin K is incorrect. For minor haemorrhage, the dose of intravenous vitamin K recommended is 1-3mg.

Platelet transfusion is incorrect. In the context of haemorrhage, a platelet level of <50 is the standard trigger, with the exclusion of CNS or posterior eye bleeds in which the transfusion trigger is <100.

Prothrombin complex concentrate is incorrect. Prothrombin complex concentrate (PCC) is recommended only for major bleeding. PCC will reverse anticoagulation within minutes of administration however administration of vitamin K is also required to counteract the long half-life of warfarin.

Discuss (2)Improve

Question #280

An 89-year-old lady presents acutely short of breath and distressed. She has a background of previous myocardial infarction (MI) and hypertension. She is now

coughing up white sputum. Examination reveals bilateral coarse crepitations throughout the lung fields. She has a raised jugular venous pressure and peripheral oedema. Observations are a respiratory rate of 35/min, oxygen saturations of 92% on 15 litres of oxygen per minute, blood pressure 135/90 mmHg, heart rate 100/min. Chest X-ray reveals widespread interstitial shadowing. Intravenous furosemide has been given but the patient fails to improve. Which of the following would be useful in treating this patient?

- a) Intravenous antibiotics
- b) Oral furosemide
- c) Bilevel positive airway pressure
- d) Continuous positive airway pressure
- e) Chest physiotherapy

Acute heart failure not responding to treatment - consider CPAP

The patient is suffering from severe pulmonary oedema with bilateral coarse crackles and cough productive of white sputum. The patient has signs of right sided heart failure with raised JVP and peripheral oedema. They also have a history of MI and hypertension that are two risk factors for heart failure.

NICE guidance on Acute Heart Failure 2014 states that a patient who has failed medical management of pulmonary oedema with severe dyspnoea should be considered for CPAP. BIPAP is not used in acute pulmonary oedema.

Question #281

A 80-year-old man with a past medical history of gout, reflux and ischaemic heart disease is admitted to the emergency department with a atrial fibrillation with fast ventricular response. He is managed according to ALS protocol and is stabilised.

A full set of bloods are sent and are displayed below:

Hb	135 g/l
----	---------

Platelets	$260 * 10^9/l$
WBC	$6 * 10^9/l$

Mg	0.34 $\mu\text{mol/l}$
Ca (adj)	2.1 u/l
PO4	0.8 u/l

This is discussed with the cardiology registrar, who advises correction of the magnesium.

What medication is the most likely cause of hypomagnesaemia in this case?

- f) Aspirin
- g) Omeprazole
- h) Ranitidine
- i) Colchicine
- j) Ramipril

Correct answer is b.

Careful electrolyte balance is important in the management of arrhythmias.

During the generation of the action potential in cardiac pacemaker cells, phase 4 (inflow of potassium) is dependant on magnesium channels, and, although the exact effect *in vivo* of magnesium administration is unclear, restoring normomagnesaemia is important in patient presenting with dysrhythmias.

PPI use is associated with hypomagnesaemia - the exact mechanism of this is not

known, but may be related to poorer intestinal absorption from dietary sources in patients on PPIs.

Question #282

A 38-year-old female of Asian descent, with no significant past medical history, presents after a syncopal event while pruning hedges. She has had a 6-month history of fever, arthralgia and for the past few weeks has had multiple episodes of vertigo and one syncopal event. She denies headaches or visual complaints. Her examination reveals a diminished radial pulse in the left arm and a systolic blood pressure difference in the upper extremities of 14 mmHg. A bruit is auscultated along the left upper extremity. Dopplers of the upper extremities indicate a stenotic area along the subclavian that is later confirmed by Magnetic Resonance Angiography (MRA). She is diagnosed with subclavian steal syndrome. Laboratory tests reveal a normocytic normochromic anaemia, elevated CRP and ESR, negative ANA and ANCA, and all other laboratory tests are within normal range. Of the following, What is the most likely diagnosis?

- f) Fibromuscular dysplasia
- g) Ehlers-Danlos syndrome
- h) Takayasu arteritis
- i) Giant cell arteritis
- j) Wegener's granulomatosis

Correct answer is c.

This patient likely has Takayasu arteritis which is essentially a chronic vasculitis primarily of the aorta and its branches. These patients can present in a variety of different ways depending on the vessels affected, however, they all typically have a prodrome of systemic symptoms including fatigue, weight loss and low-grade fevers prior to developing any vascular complaints. From a pathology standpoint biopsies of vessels are very similar to giant cell and are typically not performed.

There are 6 criteria for the diagnosis of Takayasu. Presence of 3 of the 6 has 90% sensitivity and specificity for diagnosis:

- 1. Age onset <=40 years
- 2. Claudication of the extremities
- 3. Decreased pulsation of one or both brachial arteries
- 4. Difference of at least 10 mm Hg in systolic blood pressure between the arms
- 5. Bruit over one or both subclavian arteries or the abdominal aorta
- 6. Arteriographic narrowing or occlusion of the entire aorta, its primary branches, or large arteries in the proximal upper or lower extremities, not due to arteriosclerosis, fibromuscular dysplasia, or other causes

Fibromuscular dysplasia typically affects the renal arteries leading to renal artery stenosis and hypertension, and it is not accompanied by other systemic manifestations like fever and malaise. Ehlers-Danlos syndrome is a genetic defect in type III collagen and can lead to aneurysms along with hyperelasticity of the skin and hypermobile joints, but other systemic manifestations are typically not present. Giant cell arteritis is most similar to Takayasu in pathology, however typically affects older patients and usually presents with headaches and tenderness over the temporal artery. Lastly, Wegener's is actually a small vessel vasculitis and its most common presenting symptoms include persistent rhinorrhoea, purulent/bloody nasal discharge, oral and/or nasal ulcers, polyarthralgias, myalgias, or sinus pain. Most with Wegener's are ANCA positive.

Question #283

A 78-year-old gentleman is seen in clinic with long-standing heart failure with reduced ejection fraction (32%). He has had numerous admissions this year with heart failure decompensation and is wondering if there is anything else that you can do for him. You review his ECG, which is in sinus rhythm with a heart rate of 64/min with a QRS of 136 msec and left bundle branch block. His blood pressure is: 98/55 mmHg. You also review his bloods:

Hb	128 g/L	Male: (135-180)
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		Female: (115 - 160)
Platelets	$335 * 10^9/L$	(150 - 400)
WBC	$6.8 * 10^9/L$	(4.0 - 11.0)
Na^+	136 mmol/L	(135 - 145)
K^+	4.7 mmol/L	(3.5 - 5.0)
Urea	6.8 mmol/L	(2.0 - 7.0)
Creatinine	126 $\mu\text{mol}/L$	(55 - 120)

He is currently on carvedilol 25 mg BD, enalapril 10 mg BD, bumetanide 2 mg BD, aspirin 75 mg, ivabradine 2.5 mg BD.

What alteration to this gentleman's management could potentially decrease this gentleman's probability of being re-admitted?

- f) Arrange cardiac resynchronisation therapy-pacemaker (CRT-P) implantation
- g) Increase ivabradine
- h) Increase bumetanide
- i) Increase spironolactone
- j) Initiate sacubitril/valsartan

Correct answer is a.

Cardiac resynchronisation therapy can be used in patients with a QRS duration of >130 msec and LBBB morphology to improve symptomatology

The European society of cardiology (ESC) guidelines have highlighted the importance of cardiac resynchronisation in patients with heart failure. Evidence exists indicating the importance of implantation of such devices in patients with intraventricular delay with most studies focusing on the impact of left bundle

branch block. The stronger evidence related CRT-P in such patients include left bundle branch block (LBBB) and QRS >150 msec; however the presence of LBBB on its own provided that the QRS is longer than 120 msec is associated with improved mortality/morbidity. There is no evidence of benefit from CRT for patients with QRS <120 msec, while the presence of right bundle branch block (RBBB) implies a worse disease state where CRT may not necessarily benefit such patients.

The possibility of altering medications in this case remains attractive however one should be aware of the potential of introducing detriment to the patient in doing so.

This patient is already tolerating a relatively slow heart rate therefore ivabradine could potentially cause further slowing.

He is borderline hypotensive with raised potassium (making the possibility of increasing spironolactone less favourable).

Question #284

A medical opinion was sought from the obstetrics team regarding a 38-year-old 28 weeks pregnant lady. A routine blood pressure check revealed a blood pressure of 158/98 mmHg. Other than suffering from hyperemesis gravidarum, her pregnancy had proceeded without complication. She specifically denied the presence of headaches, vomiting, change in vision, abdominal pain, seizures or bleeding per vagina. She had noticed no change in the frequency of foetal movements, and her 20-week antenatal scan revealed the presence of a healthy foetus with a rate of growth within the expected range. Her past medical history was unremarkable; she was a non-smoker and did not consume alcohol. Her blood pressure at the booking antenatal appointment was 148/88 mmHg. Her sister suffered from pre-eclampsia during her pregnancy necessitating delivery by caesarean section.

Examination revealed the presence of a well 28-week old pregnant lady. Her blood pressure was indeed 158/98 mmHg, her heart rate was 86 bpm and temperature 36.5°C. Examination of the cardiovascular system revealed normal heart sounds, a JVP of 3cm and the absence of pedal oedema. Examination of the respiratory

system was unremarkable. Examination of the gastrointestinal system revealed the presence of a symphysiofundal height appropriate for the stage of gestation with easily obtainable foetal heart sounds on hand held Doppler examination. Examination of the neurological system was unremarkable with normal reflexes, cranial nerve function and peripheral motor and sensory function. Urinalysis revealed no abnormality.

What is the next best management step?

- f) Commence ramipril
- g) Commence labetalol
- h) Commence indapamide
- i) Commence magnesium sulphate
- j) Transfer to high dependency unit to observe for signs of pre eclampsia

Correct answer is b.

This lady has pre-existing hypertension; her blood pressure at the antenatal booking clinic was elevated and continues to be elevated throughout (as opposed to gestational hypertension in which hypertension develops after 20 weeks). If untreated hypertension is associated with adverse maternal and foetal outcomes including intrauterine growth restriction, placental abruption, cerebrovascular accidents and prematurity. There are no clinical features of preeclampsia, including notably the presence of proteinuria and peripheral oedema, and there is, therefore, no indication for admission to a high dependency unit or to commence magnesium sulphate. Of the remaining options, labetalol is the safest antihypertensive to use in pregnancy; methyldopa is an alternative. The usual first line ACE inhibitors are absolutely contraindicated in pregnancy.

Question #285

A 45-year-old gentleman is advised by his GP to attend hospital following a routine blood test demonstrating an increase in creatinine two weeks following a dose increase in his ramipril. His ramipril was increased from 5mg daily to 5mg twice a day.

He has a letter which demonstrates investigation results, shown below. He reports

no new symptoms. He reports that home monitoring of blood pressure has shown his control to be better.

	21/11/2016	6/11/2016
Na ⁺	140 mmol/l	138 mmol/l
K ⁺	4.5 mmol/l	4.1 mmol/l
Urea	5.5 mmol/l	5.4 mmol/l
Creatinine	110 µmol/l	92 µmol/l

How should his ramipril be managed and monitored?

- f) Continue ramipril and repeat U&Es in 1-2 weeks
- g) Reduced the dose of ramipril and repeat U&Es in 1-2 weeks
- h) Stop ramipril and repeat U&Es in 1-2 weeks
- i) Stop ramipril and arrange for an urgent out-patient imaging to exclude renal artery stenosis
- j) Continue ramipril and repeat U&Es within 48 hours

Correct answer is a.

The correct answer is to continue ramipril and repeat U&Es in 1-2 weeks. Up to an increase in creatinine of 30% can be tolerated following the start or increase in an ACE-inhibitor such as ramipril, but U&Es should be repeated in 1-2 weeks. If the increase is greater than 30% then the ACE-inhibitor should be stopped.

Question #286

A 34-year-old male presents to the emergency department with chest tightness worse on inspiration. He informs you that over the past 3 days he has had a low-grade temperature and a degree of myalgia. Prior to all this he had no limitations

to his activity and never experienced any chest tightness. He was given glyceryl trinitrate spray and aspirin in the ambulance, neither of which have made any difference.

Examination is unremarkable, however you note that his temperature is 37.4°C. He is a smoker and he informs you that his father, mother and paternal uncle all have had heart attacks around the age of 60. His bloods are the following:

Hb	165 g/L	Male: (135-180) Female: (115 - 160)
Platelets	537 * 10 ⁹ /L	(150 - 400)
WBC	12.3 * 10 ⁹ /L	(4.0 - 11.0)
Na ⁺	138 mmol/L	(135 - 145)
K ⁺	4.3 mmol/L	(3.5 - 5.0)
Urea	4.6 mmol/L	(2.0 - 7.0)
Creatinine	93 µmol/L	(55 - 120)
CRP	34 mg/L	(< 5)
Troponin T (0hrs)	586 ng/L	(<14)
Troponin T (3hrs)	615 ng/L	(<14)
D-dimer	Negative	

An ECG is performed indicating sinus rhythm, no intraventricular delay and widespread ST elevations; you also notice was subtle PR depression.

What treatment should be initiated in the case of this gentleman?

- f) Arrange urgent angioplasty and potential revascularization
- g) Load patient with aspirin and clopidogrel
- h) Load patient with aspirin and ticagrelor
- i) Prescribe ibuprofen TDS
- j) Prescribe ibuprofen TDS and colchicine BD

Correct answer is e.

First line management of acute pericarditis involves combination of NSAID and colchicine

The history, examination and blood investigations are consistent with acute pericarditis/myopericarditis. The high troponin results indicate myocardial injury which in this case can be attributed to myopericarditis. Troponin is a marker of myocardial injury which can result due to primary ischaemia (plaque rupture or intraluminal coronary artery thrombus), ischaemia secondary to misbalance of supply and demand (tachy/bradyarrhythmias, aortic dissection or severe aortic valve disease, sepsis, severe anaemia or respiratory failure etc.) and myocardial injury not necessarily associated to ischaemia or multifactorial (such as myopericarditis, severe pulmonary embolism with right heart strain, cardiac stunning, cardiac surgery or intervention etc.) There is no strong, dynamic rise in the already high troponin level.

The fact that he is a smoker undeniably puts this gentleman at a higher risk of coronary event, however his blood tests, ECG and history point further to the direction of pericarditis. Given this, options 1, 2 and 3 which are associated with the management of acute coronary syndrome (ACS) are wrong.

The current guidelines as per the European Society of cardiology state that the treatment of choice in such patients is a combination of non-steroidal anti-inflammatory drugs (e.g. ibuprofen) and colchicine (option 5) and do not advocate the use of a single agent, such as ibuprofen (option 4).

Question #287

A 25-year-old female presents with palpitations and dizziness. She states that she has had a few episodes similar to this in the past and each time they have spontaneously resolved. On examination she has a blood pressure of 85/65 mmHg and a heart rate of 180bpm. An ECG shows an irregularly irregular rhythm, no identifiable P waves, and a QRS of 135ms.

Your senior decides to electrically cardiovert her. While the cardioversion is being prepared you instruct the patient to perform a vagal manoeuvre. Shortly after this she loses consciousness and goes into cardiac arrest.

What is the most likely underlying disorder?

- f) Torsades de Pointes
- g) Atrial fibrillation
- h) Atrioventricular nodal re-entry tachycardia (AVNRT)
- i) Monomorphic ventricular tachycardia
- j) Wolff-Parkinson-White syndrome (WPW)

Correct answer is e.

AV blocking drugs and vagal manoeuvres are absolutely contraindicated in patients with AF and pre-excitation

There are few arrhythmias, which can cause an irregularly irregular broad complex tachycardia. These include irregular atrial arrhythmias with bundle branch block (e.g. atrial fibrillation), and polymorphic ventricular tachycardia (including Torsades de Pointes). Another important group of conditions to be aware of include the pre-excitation syndromes.

Pre-excitation refers to early activation of the ventricles due to impulses bypassing the AV node via an accessory pathway. This results in a broad QRS complex e.g. the slurred upstroke of QRS (delta wave) as is seen in WPW. When present with atrial fibrillation, the accessory pathway allows for rapid conduction directly to the ventricles bypassing the AV node. This results in an irregularly irregular broad

complex tachycardia.

The Valsalva manoeuvre increases the vagal tone resulting in AV node block. This promotes conduction of the atrial signals through the accessory pathway with a resultant increase in ventricular rate and possible degeneration into VT or VF, as is seen in this case.

AV blocking drugs and vagal manoeuvres are absolutely contraindicated in patients with AF and pre-excitation. In a haemodynamically unstable patient, urgent synchronised DC cardioversion is required. Medical treatment options in a stable patient include procainamide, although DC cardioversion may be preferred. Procainamide is a class 1 anti-arrhythmic which preferentially blocks the accessory pathway.

Question #288

A 59-year-old woman presents to the emergency department with shortness of breath. She has been progressively getting worse over two weeks and has now started to feel short of breath on rest. She is known to have mitral valve prolapse and is awaiting surgery but has so far not been given a date. She also has polycystic ovarian syndrome, type 2 diabetes and depression. She takes metformin, sertraline and furosemide.

On examination, she looks unwell. She has bilateral crepitations with no wheeze on auscultation of her chest, a raised JVP, and a systolic murmur. A chest X-ray shows pulmonary oedema. She is treated with IV diuretics but remains breathless and hypoxic. What kind of ventilatory support would be most appropriate?

- f) No ventilator support is appropriate
- g) Negative pressure ventilation
- h) Intubation
- i) Bilevel positive airway pressure (BIPAP)
- j) Continuous positive airway pressure (CPAP)

Correct answer is e.

Acute heart failure not responding to treatment - consider CPAP

The correct answer is continuous positive airway pressure (CPAP). This is a patient with a known mitral valve prolapse presenting with decompensation. It can be assumed that the lesion is significant as she is due to have surgery to repair it. She has developed signs of pulmonary oedema and she is not responding to treatment with diuretics. Vasodilators and opiates could be a plan of further medical management, but CPAP could help reduce pulmonary oedema. BIPAP would be appropriate in ventilatory failure in COPD, whilst negative pressure ventilation is more of historical treatment for polio.

Mitral valve prolapse has a prevalence of 2-3% in the population and the majority of cases will be asymptomatic. Risk factors for progression the severe disease include:

- Comorbidities: atrial fibrillation, left sided cardiac failure, hypertension and obesity.
- Echocardiogram findings: mitral leaflet thickness >5mm, prolapse of the posterior leaflet, moderate or severe regurgitation.
- Stress echocardiogram findings: regurgitation during exercise but not at rest.

Question #289

A 75-year-old male presents to the emergency department with central crushing chest pain which has been ongoing for 4 hours. It is associated with shortness of breath.

Blood results are as follows:

Hb	125 g/L	Male: (135-180) Female: (115 - 160)
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Platelets	$424 * 10^9/L$	(150 - 400)
WBC	$14.6 * 10^9/L$	(4.0 - 11.0)
Na^+	135 mmol/L	(135 - 145)
K^+	4.4 mmol/L	(3.5 - 5.0)
Urea	7.8 mmol/L	(2.0 - 7.0)
Creatinine	134 $\mu\text{mol}/L$	(55 - 120)
Troponin	8422 ng/L	(< 15)

An ECG is performed:



What is the most likely diagnosis?

- f) Anterolateral STEMl
- g) Inferolateral STEMl
- h) Myocarditi
- i) Pericarditis
- j) Posterior STEMl

Correct answer is b.

The ECG demonstrates ST elevation in the inferior (II, III, and aVF) and lateral leads (V5-6) confirming a diagnosis of an **inferolateral STEMI**. ST depression in V1-2 is suggestive of an associated posterior infarction. This constellation of ECG abnormalities is typically produced by occlusion of the proximal circumflex artery.

Anterolateral STEMI would present with ST elevation in the anterior (V1-V4) and lateral leads.

Posterior STEMI presents with reciprocal changes in the anterior leads including horizontal ST depression and a dominant R wave in V2.

Both **myocarditis and pericarditis** present with global concave ST elevation. The troponin is usually normal in pericarditis and raised in myocarditis.

DiscussImprove

Question #290

A 67-year-old woman is complaining of a six-hour history of chest pain and dizziness in the high-dependency unit. She underwent a mitral valve replacement five days ago and had some temporary trans-venous pacing wires removed earlier today.

On examination, she has oxygen saturations of 93% on 2 litres via nasal cannula, has a heart rate of 110/min, a blood pressure of 76/43mmHg, has a temperature of 37.9°C, and is responsive to voice. She feels cool peripherally. An ECG is performed at the bedside which shows sinus rhythm with QRS complexes of alternating amplitude.

Blood tests from this morning show the following:

Hb	89 g/L	(115 - 160)
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Platelets	$121 * 10^9/L$	(150 - 400)
WBC	$13.1 * 10^9/L$	(4.0 - 11.0)
Na ⁺	143 mmol/L	(135 - 145)
K ⁺	3.1 mmol/L	(3.5 - 5.0)
Urea	18.2 mmol/L	(2.0 - 7.0)
Creatinine	151 µmol/L	(55 - 120)
CRP	282 mg/L	(< 5)
Magnesium	0.83 mmol/L	(0.85 - 1.10)

What is the most appropriate management given the likely diagnosis?

- f) Electrolyte replacement
- g) Emergency percutaneous coronary intervention
- h) Pericardiocentesis
- i) Prolonged course of antibiotics
- j) Urgent temporary pacing wire insertion

Correct answer is c.

Electrical alternans is suggestive of cardiac tamponade

This woman has clinical signs of cardiac tamponade which is confirmed by electrical alternans on ECG. Electrical alternans occurs due to the movement or 'swinging' of the heart within the pericardium as a result of the fluid present. This results in a variable amplitude of the QRS complex depending on the hearts proximity to the ECG leads. In this case, this was likely precipitated by a traumatic removal of trans-venous pacing wires causing bleeding into the pericardial space. As a result, she requires an urgent echocardiogram to confirm the diagnosis and

subsequent **pericardiocentesis** or re-sternotomy to drain the blood. It is worth noting that her acute kidney injury is the result of poor cardiac output as a result of her tamponade.

Although there are electrolyte abnormalities on this patient's blood tests that should be replaced, this is not likely to be the cause of her acute presentation. An arrhythmia post-operatively could cause a reduction in cardiac output however the ECG performed here demonstrates sinus rhythm and therefore **electrolyte replacement** would not reverse her current condition.

An acute coronary syndrome must be considered in cardiac post-operative patients complaining of chest pain however the electrical alternans on ECG with no remarkable ST changes to speak of is more consistent with a diagnosis of cardiac tamponade. Therefore, **emergency percutaneous coronary intervention** is not the most appropriate option here.

A **prolonged course of antibiotics** may be employed if a diagnosis of infective endocarditis is suspected however this is not the case here for a variety of reasons. Firstly, infective endocarditis post-operatively is unlikely to present within 5 days. Furthermore, the electrical alternans on ECG is indicative of cardiac tamponade rather than infective endocarditis. Finally, although the patient has a fever and a raised CRP, these are common findings post-operatively due to the systemic inflammatory response to surgery and are not specific for infective endocarditis.

Arrhythmias are common in the post-operative period for patients who have undergone cardiac surgery, particularly valve surgery, however, this patient is in sinus rhythm at an acceptable rate. Therefore, as this patient's pathology is mechanical rather than electrical, **urgent temporary pacing wire insertion** will not help with her current issue.

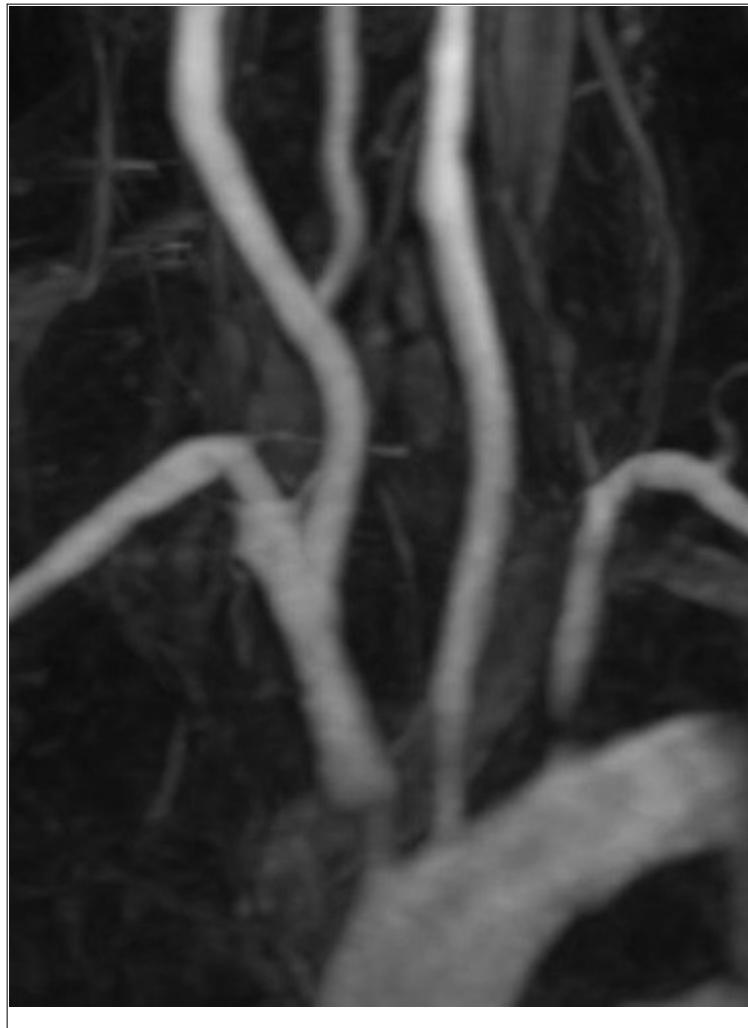
Discuss (3)Improve

Question #291

A 26-year-old woman is investigated for persistent malaise, fever, headaches and raised inflammatory markers. She was diagnosed with Raynaud's syndrome three years ago after complaining of cold hands. The most recent bloods in the referral letter show a CRP of 64 mg/l.

On examination blood pressure is 80/64 mmHg in the right arm and 72/54 mmHg in the left arm. The heart rate is 72/min and a diastolic murmur is noted on auscultation of the heart.

Magnetic resonance angiography is requested:



What is the most appropriate management?

- f) Plasma exchange
- g) Rituximab
- h) Prednisolone
- i) Cardiothoracic surgery referral
- j) Intravenous immunoglobulin

Correct answer is c.

The magnetic resonance angiography (MRA) demonstrates stenoses of the supra-aortic arteries, especially the brachiocephalic trunk and the left subclavian artery with near-occlusion of the left vertebral artery. These findings are consistent with a diagnosis of Takayasu's arteritis.

Discuss (4) Improve

Question #292

A 68 year old gentleman with a past medical history of type 2 diabetes mellitus and hypertension attends the Emergency Department complaining of general malaise and feeling unwell. He had undergone successful primary coronary intervention (PCI) post myocardial infarction 5 weeks previously. He has a temperature of 38.2 degrees Celsius, blood pressure 164/87 mmHg and the doctor notes a lacy reticular rash extending over his legs.

Hb	12.2g/dl
Eosinophils	$1.2 * 10^9/l$
WBC	$14.5 * 10^9/l$
Urea	8mmol/l

Creatinine	142 μ mol/l
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What is the most likely cause of his symptoms?

- f) Allergic rash to contrast
- g) Infected femoral site
- h) Cholesterol embolus
- i) Infective endocarditis
- j) Polyarteritis nodosa

Correct answer is c.

This is a classic story for a cholesterol embolus post an invasive arterial procedure, whereby a plaque has been ruptured and there is lodging of debris in small/medium arteries causing mechanical occlusion, inflammation and end-organ damage. Patients may have the lacy reticular rash of livedo reticularis and this, together with acute renal failure and an eosinophilia should lead the clinician to consider this diagnosis.

Patients tend to present with non-specific symptoms such as fever, weight loss and myalgia for weeks to months before developing end-organ damage or stroke.

The current mainstay of treatment is supportive.

Question #295

A 31-year-old woman is brought into the Emergency Department by her husband. He tells you that she is abnormally drowsy and he has difficulty rousing her. She has a background of depression which is medicated by her General Practitioner, but is otherwise well.

On examination she is afebrile but has dry, warm skin. Her heart rate is 140bpm with a blood pressure of 105/90mmHg. She has a respiratory rate of 8 breaths per minute and saturating at 92% on air. Her Glasgow coma scale (GCS) is 12 (E3V4M5) and she appears agitated. Her pupils are dilated and she has hyperreflexia. Whilst examining her abdomen it is noted that she has a palpable

bladder and bowel sounds are absent.

Investigations:

Arterial blood gas (ABG):

pH	7.29	(7.35-7.45)
PCO ₂	7.2 kPa	(4.9-6.1 kPa)
PO ₂	10.1 kPa	(10-13.1 kPa)
Bicarbonate (HCO ₃ -)	28 mmol/L	(22-28 mmol/L)
Base Excess (BE)	3.1 mmol/L	(-2 to 2 mmol/L)
Sodium	134 mmol/L	(135-145 mmol/L)
Potassium	4.4 mmol/L	(3.5-5.5 mmol/L)
Chloride	105 mmol/L	(95-110 mmol/L)
Lactate	2.1 mmol/L	(0.2-1.6 mmol/L)
Glucose	5.3 mmol/L	(4-7 mmol/L)

ECG - Polymorphic ventricular tachycardia, QTc 500ms.

What is the most important next step in the management of this patient?

- f) Synchronised DC cardioversion
- g) Intravenous amiodarone
- h) Intravenous magnesium sulphate

- i) Intravenous lidocaine
- j) Intravenous sodium bicarbonate

Correct answer is c.

IV magnesium sulfate is used to treat torsades de pointes

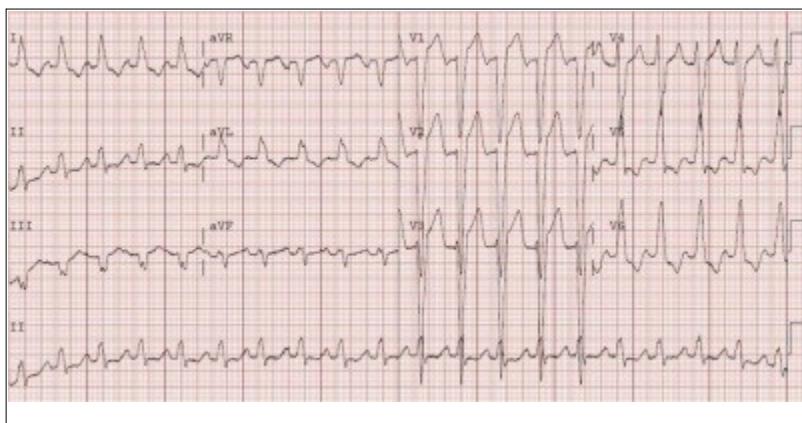
This woman has had a tricyclic antidepressant (TCA) overdose. In excess, TCAs can cause QT interval prolongation which can predispose to ventricular arrhythmias. A polymorphic ventricular arrhythmia with a prolonged QT interval is characteristic of torsades de pointes. The treatment for this is intravenous magnesium sulphate.

As she currently has a stable blood pressure (systolic > 90mmHg), DC cardioversion would not be appropriate at this stage. Intravenous amiodarone and lidocaine can be used to medically cardiovert a monomorphic ventricular tachycardia, but are not the treatment of choice in torsades de pointes.

Sodium bicarbonate is the first-line treatment of TCA overdose. However, in the presence of torsades de pointes, intravenous magnesium sulphate is the more appropriate choice to give initially.

Question #296

A 58-year-old man presents with intermittent chest pains for the past two days. Whilst you are taking the history he complains of worsening pain and you arrange an ECG immediately:



He is tachycardic and sweaty with a blood pressure of 128/88mmHg. What does the ECG show?

- f) Narrow complex tachycardia with ST elevation
- g) Right bundle branch block with ST elevation
- h) Left bundle branch block with ST elevation
- i) Ventricular tachycardia with ST elevation
- j) Posterior myocardial infarction

Correct answer is c.

The ECG shows left bundle branch block (LBBB). The relationship between new LBBB and acute coronary syndrome (ACS) is complicated with many cardiologists ignoring the standard guidelines (both UK and US) to initiate reperfusion therapy due to the poor correlation seen in clinical practice between coronary lesions and standard LBBB. The Sgarbossa criteria have therefore been developed to evaluate whether new LBBB represents an ACS. In this particular ECG there is more ST elevation than is normally expected with LBBB indicating a likely ACS.

Question #297

A 71-year-old patient presents to the Emergency Department with a two hour history of crushing central chest pain. He is known to have a history of ischaemic heart disease. The ECG shows the following:

- ST elevation greater in lead II than in lead III with abnormal Q waves in II, III, and aVF
- ST depression, tall, broad R waves and upright T waves in V1-3. Dominant R wave in V2
- ST elevation in V5-V6

Where is the lesion most likely to be?

- f) Left anterior descending
- g) Left circumflex

- h) Right coronary artery
- i) Left main stem
- j) Posterior interventricular

Ischaemic changes in leads I, aVL +/- V5-6 - left circumflex

These are classical findings of a circumflex occlusion. The table below shows how the changes correspond to the cardiac anatomy:

ECG changes	Component of infarction
ST elevation greater in lead II than in lead III with abnormal Q waves in II, III, and aVF	Inferior component of infarction
ST depression, tall, broad R waves and upright T waves in V1-3. Dominant R wave in V2	Posterior component of infarction
ST elevation in V5-V6	Lateral component of infarction

Please see the link for an example ECG with a description of the changes.

Question #298

A 24-year-old female is brought into the emergency department by ambulance. She has a family history of Wolff-Parkinson-White syndrome and was binge drinking last night. She is complaining of palpitations which she says started suddenly 1 hour ago.

Her observations are as follows

- Heart rate 180 bpm

- Blood pressure 100/60 mmHg
- Saturations 98% on air
- Respiratory rate 26/min
- Temperature 36.8°C

An ECG (electrocardiogram) is done which shows pre-excited atrial fibrillation (AF)

Which of the following is an appropriate treatment?

- f) Adenosine
- g) Atenolol
- h) Flecainide
- i) Diltiazem
- j) Digoxin

Correct answer is c.

In pre-excited AF don't give anything that blocks conduction at AV node (including calcium channel blockers, adenosine or digoxin) as this can cause ventricular tachycardia

Flecainide is the only appropriate treatment on this list

All of the other options are drugs which block transmission through the AV (atrioventricular) node. Giving these drugs in pre-excited AF (atrial fibrillation) can precipitate VT (ventricular tachycardia) or VF (ventricular fibrillation).

Note that flecainide is contraindicated in patients with structural heart abnormalities. In this situation then amiodarone can be given instead.

If any of the following are present: hypotension, signs of shock, altered mental status, chest pain or acute heart failure then synchronised cardioversion should be used rather than medication.

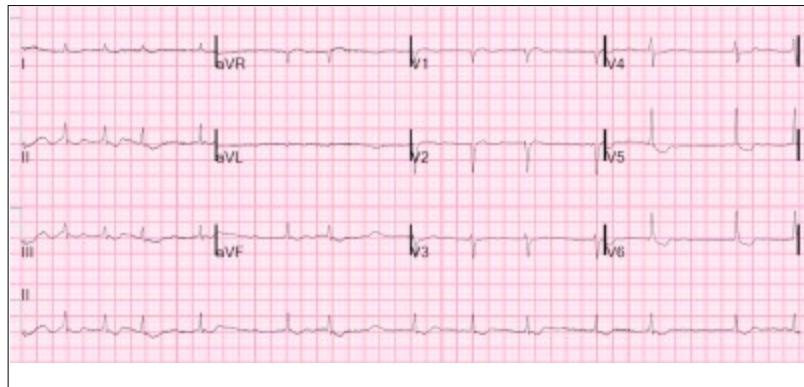
Discuss (6)Improve

Question #299

A 77-year-old woman is seen in a heart failure clinic. Her past medical history includes congestive cardiac failure, atrial fibrillation, hypertension, and depression. Her regular medications include bisoprolol, digoxin, amlodipine, furosemide, and citalopram.

Repeat digoxin levels are displayed below:

Digoxin level	1.1 ng/mL	(0.7-2.0)
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Which agent is responsible for the abnormalities displayed in her ECG?

- f) Amlodipine
- g) Bisoprolol
- h) Citalopram
- i) Digoxin
- j) Furosemide

Correct answer is d.

This woman's ECG displays 'scooped' ST depression in leads II, III, aVF, V5, and V6 in the context of atrial fibrillation. This is a classical sign seen in the digoxin effect,

the ECG changes that arise with digoxin within normal therapeutic levels. It is important to note that in digoxin toxicity, the most common dysrhythmias are supraventricular tachycardia with a slow ventricular response due to blocking of the AV node.

Whilst calcium channel blockers can significantly disrupt the ECG, this is associated primarily with non-dihydropyridines such as verapamil or diltiazem, rather than dihydropyridines such as amlodipine which are less cardioselective.

Bisoprolol can give rise to bradycardia and PR prolongation on ECG but the ST changes observed here are consistent with the digoxin effect. Therefore, bisoprolol is incorrect.

Citalopram is a selective serotonin reuptake inhibitor and therefore can result in QT prolongation. The ST changes as described here are not consistent with this syndrome and therefore this is an incorrect answer.

Furosemide is a loop diuretic that can result in hypokalemia and therefore flattened and inverted T waves. The ST segment in this ECG shows the classical 'scooped' appearance and therefore is more consistent with the digoxin effect rather than hypokalemia. Furosemide is therefore incorrect.

Question #300

A 62-year-old man sees his GP with complaints of leg pain. Over the last few months, he notices a crampy uncomfortable feeling in the back of both of his calves when he walks to the shops. The discomfort can be so bad that he has to stop and rest for a few minutes after which he notices that his symptoms are improved. On occasions, he also gets the symptoms when he is shopping in the supermarket. He attributes this to overexerting himself and tries to rest on his shopping trolley while walking but does not seem to help. He has a 40-pack year smoking history and takes amlodipine for his blood pressure, and paracetamol and ibuprofen for lower back pain that has troubled him for years.

Physical examination reveals mild atrophy of his thigh and calf muscles bilaterally, in addition to shiny pale skin with significant hair loss throughout his lower limbs.

His pedal pulses are bilaterally impalpable, and popliteal pulses are faint. Power in both lower limbs is normal throughout all movements, and he has normal patellar reflexes bilaterally and absent ankle reflexes. His Babinski reflex is downgoing on the left side and equivocal on the right side. A recently obtained ankle brachial pressure index test yielded a result of 0.70 on the right side and 0.95 on the left side. X-rays of his lumbar spine show evidence of joint space narrowing and osteophytes.

Which one of the following is the next best step in the management of this patient?

- f) MRI scan of lumbar spine
- g) Check his HbA1c to screen for diabetes
- h) Refer to vascular surgery for consideration of peripheral arterial stenting or bypass surgery
- i) Starting him on duloxetine to manage his pain
- j) Screen for coronary artery disease with ECG stress testing

Correct answer is c.

In patients with cardiovascular risk factors and symptoms suggestive of claudication with an equivocal/borderline ankle brachial pressure index study result, the next best study is an ankle brachial pressure index after exercise

This gentleman has symptoms of claudication, in particular, vascular claudication. Although he does have a history of chronic back pain for which he takes analgesia, he is less likely to have neurogenic claudication compared to vascular claudication. An important discriminating factor between neurogenic claudication and vascular claudication is that patients with neurogenic claudication have symptoms on exertion that improve with manoeuvres such as leaning forward, and sitting down, whereas vascular claudication does not change with these manoeuvres and only improves with rest. In addition, his physical exam shows evidence of peripheral arterial disease characterised with muscle atrophy, shiny skin with hair loss and impalpable pedal pulses. Absent ankle jerk reflexes and equivocal Babinski reflexes can be normal variants and do not always indicate neurological pathology. This patient has rightly already undergone an ankle brachial pressure index

assessment, and the results indicate a diagnosis of peripheral arterial disease. He is functionally impaired by his symptoms and so the next best step in his management would be to refer him to vascular surgery for consideration of treatment strategies which may include percutaneous interventions with stenting and/or surgical bypass.

Although this patient has evidence of degenerative joint disease on his lumbar spine x-ray, his clinical presentation is not consistent with neurogenic claudication and so an MRI scan of his lumbar spine is not indicated.

Although it is important to screen for and aggressively manage cardiovascular risk factors in patients with peripheral arterial disease, this in itself would not address the patient's symptoms or the disease course.

This patient's pain is most consistent with vascular claudication as opposed to neuropathic pain, for which an agent like duloxetine would be appropriate.

Although individuals with peripheral arterial disease have a significant likelihood of having concomitant coronary disease, this patient is not complaining of angina or symptoms suggestive of coronary disease and so screening for this is not indicated. The next best step in managing this patient is to address and treat his symptoms related to his peripheral arterial disease.

Question #301

A 77-year-old man with known atrial fibrillation is admitted following an upper gastrointestinal haemorrhage. His atrial fibrillation is managed using bisoprolol and warfarin. Since his admission, he has had four large episodes of haematemesis. You, the emergency department doctor, request the patient's INR to be checked as one of a series of investigations. The haematology laboratory phone through and inform you his INR is 8.5. He is currently hypotensive (90/45 mmHg) and tachycardic (120 beats per minute). You begin resuscitation using 0.9% saline, and send a cross match, group and save. What is the most appropriate treatment of this patient's INR?

- f) Fresh frozen plasma + stop warfarin
- g) Vitamin K + stop warfarin
- h) Prothrombin complex concentrates
- i) Prothrombin complex concentrates + vitamin K + stop warfarin
- j) Stop warfarin

Correct answer is d.

Major bleeding - stop warfarin, give intravenous vitamin K 5mg, prothrombin complex concentrate

The nub of this question is the emergency management of haemorrhage in patients on warfarin. This patient has an INR greater than 8 and is actively bleeding. Therefore the answer is 4.

Patients on warfarin have reduced levels of Factor X, IX, VII and II. Rapid correction is most effectively achieved through administration of prothrombin complex concentrates.

The British Journal of Haematology states that: 'Emergency anticoagulation reversal in patients with major bleeding should be with 2550 u/kg four-factor prothrombin complex concentrate and 5 mg intravenous vitamin K'

Question #302

An 89-year-old woman presents to the emergency room with increased shortness of breath and decreased mobility of 1-week duration. Her shortness of breath is worse at night and she sometimes wakes up gasping for breath. She has fallen over twice in the last week which isn't normal for her. She has a past medical history of diabetes mellitus type 2 and hypertension. She has had no previous surgeries. She lives independently with her husband and her daughter lives close by and helps with the shopping.

On examination, she is found to have coarse crackles bi-basally on auscultation of her chest. She has a regular heart rate with a pan-systolic murmur loudest over the apex.

Investigations show:

Haemoglobin	11g/dl
WCC	$6 \times 10^9/l$
Platelets	$178 \times 10^9/l$
Sodium	139 mmol/l
Potassium	4.2 mmol/l
Urea	8 mmol/l
Creatinine	92 μ mol/l
Blood cultures	<i>Methicillin-sensitive Staphylococcus aureus</i>

Echocardiogram severe mitral regurgitation with large mobile structure on valve leaflet

Chest X-ray: bilateral blunting of the costophrenic angles and upper lobe diversion

What is the best treatment for this lady?

- f) Flucloxacillin orally
- g) Flucloxacillin intravenously
- h) Amoxicillin orally and vancomycin intravenously
- i) Ceftriaxone intravenously
- j) Amoxicillin intravenously and vancomycin intravenously

Correct answer is b.

This lady has a native mitral valve endocarditis with a bacteraemia caused by a methicillin-sensitive *Staphylococcus aureus*. Once cultures have grown the causative bacteria and it is found to be sensitive to methicillin guidelines suggest flucloxacillin intravenously to be the treatment of choice. Flucloxacillin orally would not provide sufficient antimicrobial cover nor will ceftriaxone, amoxicillin and vancomycin.

There is some debate about the optimal length of treatment, but 6 weeks of intravenous therapy is generally accepted as the length of treatment needed. Shorter therapeutic regimens may be effective in selected patients with right-sided endocarditis and with endocarditis due to highly susceptible *Streptococcus viridans* treated with synergistic antimicrobials.

Question #303

A 28-year-old lady pregnant lady of 37 weeks gestation presented to the Emergency Department a few hours earlier with new onset chest pain. The pain was sharp and made worse with inspiration. She also complained of rapid onset progressively increasing shortness of breath, affecting her ability to complete sentences. She denied the presence of a cough or sputum production and did not suffer from haemoptysis or calf pain. Other than a successful external cephalic version for a breech presentation five days ago and a placenta praevia which spontaneously resolved, her pregnancy was unremarkable. She was in good health with an unremarkable past medical history and normal routine investigations throughout her pregnancy. She smoked 15 cigarettes per day and did not consume alcohol. Her mother suffered from an unexplained deep vein thrombosis when she was 42 years old but otherwise her family history was unremarkable.

Initial examination revealed a heart rate of 122bpm, respiratory rate 24/min, oxygen saturations of 98% on air, a temperature of 37.6°C and a blood pressure of 112/72 mmHg. She was struggling to complete full sentences. Examination of her cardiorespiratory system revealed good air entry in both bases, a JVP of 3cm, the absence of pedal oedema and soft and non-tender calves. Examination of her gastrointestinal system was unremarkable and ultrasound auscultation revealed the presence of a fetal heartbeat.

As she was being tended to for initial investigations, her condition rapidly deteriorated. Her oxygen saturation dropped to 88% on air, and her blood pressure was recorded as 88/66mmHg. She appeared cool and clammy, and her respiratory rate increased to 32/min. The doctor tending to venepuncture noted the presence of oozing of blood from the wound. She was promptly transferred to the intensive care unit and the following investigations were conducted:

Hb	101 g/l
Platelets	$75 * 10^9/l$
WBC	$12.2 * 10^9/l$

Na ⁺	136 mmol/l
K ⁺	4.8 mmol/l
Urea	14.1 mmol/l
Creatinin e	158 μ mol/l

INR	3.9
APTT	84 s
D-	2920 ng/ml

Dimer	
-------	--

Urinalysis: ketones ++, leucocytes/nit/prot/blood/glucose negative

Portable chest x-ray: normal appearance of heart and lung fields

ECG: sinus tachycardia 148bpm, T wave inversion leads V3-V6

Arterial blood gases on air:

pH	7.48
PaO ₂	5.9 kPa
PaCO ₂	2.2 kPa
BE	1
HCO ₃	24 mmol/l

She was commenced on 15 litres/min oxygen via non-rebreath mask, and an arterial line and central venous line were sited, as well as the presence of two large bore peripheral cannulae. She was immediately commenced on 2 litres of Hartmann's solution stat.

What is the most likely diagnosis?

- f) Pulmonary embolus
- g) Septic shock
- h) Peripartum cardiomyopathy
- i) Aortic dissection
- j) Amniotic fluid embolus

Correct answer is e.

In this instance, pulmonary embolism would not account for the presence of DIC and there is no evidence of deep vein thrombosis clinically. Septic shock can follow a similar path to amniotic fluid embolus, but in this instance, there is little evidence for a focus of sepsis

Question#304

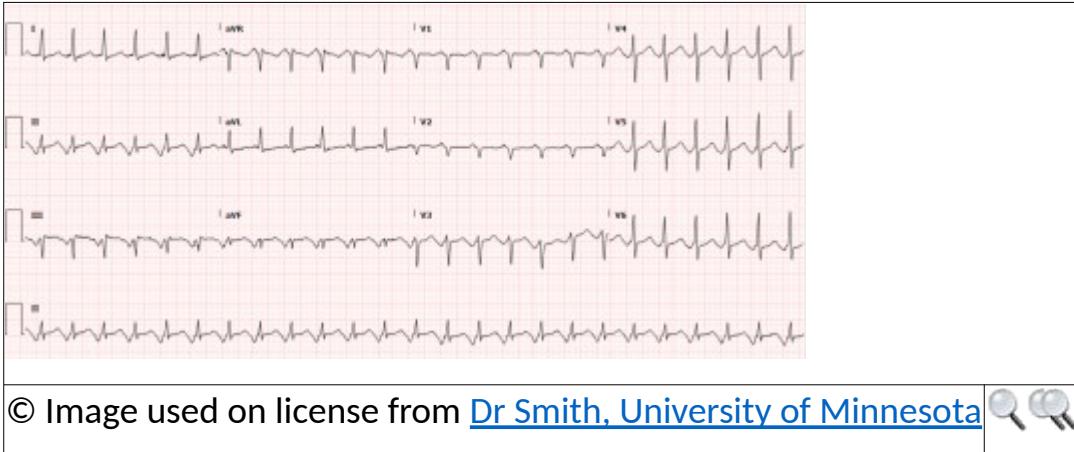
A 26-year-old woman has attended the emergency department with palpitations and shortness of breath. She has a background of asthma for which she has beclometasone and salbutamol inhalers.

On further questioning, she denies chest pain, dizziness, and loss of consciousness.

Observations are as follows:

Heart rate	200 bpm
Blood pressure	102/56 mmHg
Oxygen saturations	98%
Respiratory rate	28 /min
Temperature	36.8°C

Her ECG is shown below:



© Image used on license from [Dr Smith, University of Minnesota](#)



Of the following options, what is the most appropriate management?

- a) Direct current (DC) cardioversion
- b) Intravenous adenosine 12mg
- c) Intravenous adenosine 6mg
- d) Intravenous metoprolol
- e) Intravenous verapamil

The above ECG shows a narrow complex tachycardia of approximately 200bpm. This is also associated with an absence of P waves, except for P wave inversion in the inferior leads, otherwise known as retrograde conduction. These findings are in keeping with atrioventricular nodal re-entry tachycardia, or AVNRT, the most common form of supraventricular tachycardia.

The correct management option in the above scenario is **intravenous verapamil**. All five of the above options can be used in patients with supraventricular tachycardia, however, verapamil is the most appropriate given the patient's clinical state and past medical history of asthma.

Following advanced life support algorithms, **DC cardioversion** should be used in narrow complex tachyarrhythmias when the patient is unstable. The above scenario does not describe evidence of heart failure, syncope, chest pain, or haemodynamic instability (shock). This option is therefore incorrect.

Intravenous adenosine 12mg is used in chemical cardioversion of narrow complex tachyarrhythmias, however, should only be used when 6mg of adenosine has failed to cardiovert. Furthermore, adenosine is contraindicated in patients with asthma. For these reasons, this option is incorrect.

Intravenous adenosine 6mg is often the initial medical management in narrow complex tachyarrhythmias. Although this would normally be the next option in management, adenosine is contraindicated in patients with asthma, making this option incorrect.

Intravenous metoprolol is also an option in the management of supraventricular tachycardias. Cardioversion is caused by heart rate reduction leading to self-termination of the tachyarrhythmia. However, due to the patient's asthma history, beta-blockers should be avoided.

Discuss (11)Improve

Question#305

A 32-year-old primigravida at 37 weeks attends the antenatal unit complaining of abdominal pain which is worse on the right side. She has also been vomiting. Her blood pressure is 148/97 mmHg. She denies any abnormal discharge and reports that fetal movements are still present. Her blood results are shown below.

Hb	93 g/l
Platelets	89 * 10 ⁹ /l
WBC	9.0 * 10 ⁹ /l
Urate	0.49 mmol/l
Bilirubin	32 µmol/l

ALP	203 u/l
ALT	190 u/l
AST	233 u/l

You are phoned for advice on the results. What is the most likely diagnosis?

[HELLP syndrome](#) 92% [Obstetric cholestasis](#) 4% [Acute fatty liver](#) 4% [Hyperemesis gravidarum](#) 0% [Gout](#) 0%

Correct answer is a.

The most likely diagnosis here is HELLP syndrome (haemolysis, elevated liver enzymes and low platelets), a serious manifestation of pre-eclampsia. The clinical features of hypertension, vomiting and abdominal pain support the diagnosis but are not pre-requisites. The abdominal pain here may be a sign of liver inflammation and resulting stretch of the liver capsule.

Obstetric cholestasis is associated with intense pruritus and the most sensitive marker is a rise in serum bile acids. Acute fatty liver is another serious condition, which also has associations with pre-eclampsia. It would typically cause greater elevations in liver enzymes and a deep jaundice. Hyperuricaemia may be a useful marker of pre-eclampsia and does not necessarily indicate an attack of gout. Urate is thought to rise due to diminished kidney function and reduced clearance. Hyperemesis gravidarum should be a diagnosis of exclusion and would be unlikely to present for the first time this late into pregnancy.

Discuss (3) Improve

Question#306

A 32-year-old primigravida at 37 weeks attends the antenatal unit complaining of abdominal pain which is worse on the right side. She has also been vomiting. Her blood pressure is 148/97 mmHg. She denies any abnormal discharge and reports that fetal movements are still present. Her blood results are shown below.

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Discuss (3)Improve

Question#307

A 55-year-old obese man with a history of hypertension, diabetes and angina develops central crushing chest pain whilst gardening alone. He is short of breath and clammy. He takes two puffs of his glyceryl trinitrate spray but has only mild relief from this.

He calls an ambulance, but before the ambulance arrives he suffers a cardiac arrest. The ambulance crew commence cardiopulmonary resuscitation (CPR). Heart monitoring shows him to be in ventricular tachycardia and after a DC shock and a further round of CPR, he recovers cardiac output.

He is taken to the local hospital intensive care unit where he remains unconscious. The doctors explain to his family that because of the time between his cardiac arrest and CPR commencing, his brain function may not recover well.

What is the most reliable indicator of this patient's prognosis at 72 hours post-arrest?

Electroencephalogram 19%
Glasgow coma scale motor score 23%
Pupillary reflex 35%
Somatosensory evoked potential 15%
Gag reflex 8%

Ability to predict poor outcome (death or severe disability) in a comatose patient following cardiac arrest is important for decision making regarding withdrawal of treatments as well as communicating with families.

No clinical neurological signs have been shown to correlate reliably with poor outcomes when measured less than 24 hours after cardiac arrest.

Work by Rittenberger and colleagues (2010) looked at patients who suffered ventricular tachycardia or ventricular fibrillation cardiac arrests. They found that GCS motor score did not correlate with cardiac arrest outcome when measured at 24 or 72 hours. However, lack of pupillary light or corneal reflex at 72 hours was a reliable predictor of death.

Vestibulo-ocular reflexes are also used but are less reliable. No biochemical, imaging or neurophysiological studies are currently recommended for the assessment of prognosis.

References:

Question#308

A 65-year-old man is referred to cardiology outpatient clinic with exertional shortness of breath. He becomes breathless after ascending a single flight of stairs. He has a background of ischaemic heart disease and heart failure with reduced ejection fraction (30%). He is on aspirin, bisoprolol, ramipril, spironolactone, atorvastatin and lansoprazole. He does not smoke or drink alcohol.

His observations are heart rate 63 beats per minute, blood pressure 120/77 mmHg, respiratory rate 18/minute, oxygen saturations 96% on room air and temperature 37°C.

On examination, he is euvolemic. Chest auscultation is normal. There is no peripheral oedema. The pulse is regular.

From the listed options, what is the best medication choice to improve his symptoms?

Add ivabradine6%
Add nicorandil4%
Add sacubitril-valsartan13%
Replace ramipril with ivabradine1%
Replace ramipril with sacubitril-valsartan76%

Sacubitril-valsartan is considered in heart failure patients with a LVEF < 35% who are still symptomatic on ACE-inhibitors & beta-blockers

Important for me
Less important

Replace ramipril with sacubitril-valsartan is the correct answer. This man has NYHA class II/III heart failure not controlled with an ace inhibitor, beta blocked and mineralocorticoid receptor antagonist. Given his ejection fraction is < 35%, replacing ramipril with sacubitril-valsartan is a suitable option to improve the clinical situation.

Add ivabradine is incorrect. This would be an option if his heart rate was > 75 beats per minute.

Add sacubitril-valsartan is incorrect. This should replace an ace inhibitor rather than be added to this treatment.

Replace ramipril with ivabradine is incorrect. This is not a substitution recommended by guidance and the patient's heart rate is too low for the addition of ivabradine.

Add nicorandil is incorrect. This medication is used in the treatment of stable angina, rather than heart failure.

Discuss (4)
Improve

Question#309

A 24-year-old man presents to the emergency department with one hour of palpitations and dizziness. He has no significant past medical history and is on no regular medications. He works as an accountant. There is no history of illicit drug use.

His observations are heart rate 180 beats per minute, blood pressure 88/59 mmHg, respiratory rate 22/minute, oxygen saturations 96% on air and temperature 36.4°C.

Clinical examination is unremarkable except for a regular bounding tachycardia.

An ECG demonstrates ventricular tachycardia with positive concordance throughout the chest leads and similar morphology throughout.

As he is haemodynamically unstable, emergency electrical cardioversion is undertaken in the emergency department resuscitation area, which is successful.

Post cardioversion, his observations are heart rate 84 beats per minute, blood pressure 120/84 mmHg, respiratory rate 18/minute, oxygen saturations 96% on air and temperature 36.6°C.

A repeat ECG demonstrates sinus rhythm and T wave inversion in leads V1-V3 with no other significant abnormalities.

Blood tests:

Hb	137 g/L	Male: (135-180) Female: (115 - 160)
Platelets	$188 * 10^9/L$	(150 - 400)
WBC	$7.2 * 10^9/L$	(4.0 - 11.0)

Na ⁺	141 mmol/L	(135 - 145)
K ⁺	4.4 mmol/L	(3.5 - 5.0)
Urea	5.6 mmol/L	(2.0 - 7.0)
Creatinine	89 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Adjusted calcium	2.41 mmol/L	(2.2 - 2.6)
Magnesium	0.92 mmol/L	(0.85 - 1.10)
Troponin	17 ng/L	(<14)

A CT pulmonary angiogram excludes a pulmonary embolism.

What is the likely diagnosis?

Arrhythmogenic right ventricular cardiomyopathy

Brugada syndrome 23%
Catecholaminergic Polymorphic Ventricular Tachycardia 6%
Right ventricular outflow tract ventricular tachycardia 5%
Romano-Ward syndrome 2%

Correct answer is a.

Arrhythmogenic right ventricular cardiomyopathy - T wave inversion in V1-3

Important for me Less important

Arrhythmogenic right ventricular cardiomyopathy (AVRC) is the correct answer.
 AVRC is a condition inherited with an autosomal dominant pattern, which is

characterised by replacement of the normal right ventricular myocardium with fibrofatty tissue. It typically presents in the 2nd-4th decade of life with palpitations, syncope or sudden cardiac death and is a cause of monomorphic ventricular tachycardia. The resting ECG is typically abnormal and may demonstrate T wave inversion in V1-V3. The mildly elevated troponin in this case is simply secondary to ventricular tachycardia. Imaging will demonstrate structural and functional abnormalities of the right ventricle.

Brugada syndrome is incorrect. This is also an inherited cause of sudden cardiac death (autosomal dominant) but it traditionally causes a polymorphic ventricular tachycardia and the resting ECG is characterized by 'coved' ST segment elevation >2mm in >1 of V1-V3 followed by a negative T wave.

Catecholaminergic Polymorphic Ventricular Tachycardia is incorrect. This is an inherited cause of arrhythmia and sudden cardiac death characterized by palpitations, syncope of cardiac arrest precipitated by exercise or emotions. It is typically associated with polymorphic ventricular tachycardia as suggested by the name. Usually, the resting ECG is normal.

Right ventricular outflow tract ventricular tachycardia (RVOT-VT) is incorrect. Idiopathic RVOT-VT is not an inherited condition and the resting ECG is normal.

Romano-Ward syndrome is incorrect. This is a cause of congenital long QT syndrome, which can cause a polymorphic ventricular tachycardia. We are told there are no abnormalities on the resting ECG apart from T wave inversion. Therefore, we can assume the QT interval is normal.

Discuss (2) Improve

Question#310

A 72-year-old man presents to the emergency department with dizziness. There is no chest pain, palpitations or shortness of breath. He has a past medical history of hypertension. He takes amlodipine. He does not smoke or drink alcohol.

His observations are as follows:

- Heart rate 48 beats per minute
- Blood pressure 125/84 mmHg
- Respiratory rate 18/minute
- Oxygen saturations 96% on room air
- Temperature 37.1C

On examination, he is now asymptomatic. His heart rate is irregular. There are no murmurs. Chest auscultation is normal. His jugular venous pulse is not elevated. His Glasgow coma scale is 15/15.

An ECG demonstrates intermittently non-conducted P waves associated with a prolonged PR interval that is constant and not progressive. The QRS is broad. There are no ischaemic changes.

Bedside echocardiography reveals an approximately normal ejection fraction.

Given the likely diagnosis, what is the most appropriate management?

Atropine5%
Biventricular implantable cardioverter defibrillator (ICD)
insertion9%
Dual chamber pacemaker insertion62%
Monitoring12%
Single chamber pacemaker insertion12%

Correct answer is c.

In patients with Mobitz type II AV block, or complete heart block, a DDD or DDDR pacemaker is indicated

Important for meLess important

Dual chamber pacemaker insertion is the correct answer. The patient has high grade atrioventricular (AV) block (2nd degree, Mobitz type II). This is characterised

by intermittent non-conduction of P waves associated with a prolonged PR interval. In contrast to Mobitz type I, the PR interval is constantly prolonged and does not progressively lengthen. Mobitz type II is typically caused by significant structural damage to the conducting system. Mobitz II is much more likely than Mobitz I to degenerate to haemodynamic compromise and progress to complete heart block. Pacemaker insertion is warranted. It should be a dual chamber pacemaker on setting DDD or DDDR. DDD pacing means that the pacemaker can record both atrial and ventricular rates and can pace either chamber as necessary. DDDR is the same but can adjust the heart rate to account for the need for higher cardiac output.

Atropine is incorrect. 2nd degree AV block is typically not responsive to atropine. Additionally, while this patient certainly needs a pacemaker, he is now asymptomatic with no adverse features and therefore does not need atropine urgently.

Biventricular implantable cardioverter defibrillator (ICD) insertion is incorrect. This is typically indicated in patients with an ejection fraction of < 35% and symptomatic heart failure. It is not strictly inserted for high risk bradyarrhythmias.

Monitoring is incorrect. He is at risk of sudden deterioration and needs definitive management.

Single chamber pacemaker insertion is incorrect. Single chamber pacemakers are predominantly used for the management of atrial arrhythmias and are not indicated in those with AV block.

Discuss (8)Improve

Question#311

A 69-year-old gentleman is reviewed in cardiology clinic. He has been suffering from angina for five years and despite optimal medical management he finds that his symptoms are worsening. He has a past medical history of type 2 diabetes mellitus, gout and hypertension. He is currently taking felodipine, atenolol,

atorvastatin, aspirin, ramipril and metformin. He has recently undergone an angiogram which has delineated triple vessel disease. He is very keen to optimise his survival chances as he is concerned about dying of a heart attack.

What intervention would have the greatest survival advantage?

Aggresive control of blood pressure, cholesterol and diabetes10%
Percutaneous coronary intervention (PCI) with insertion of stents10%
Coronary arterial bypass graft (CABG)77%
No intervention will give a further survival advantage2%
Heart transplant1%

Correct answer is c.

The correct answer is coronary arterial bypass graft (CABG). This is a gentleman with severe ischaemic heart disease and angina. The greatest benefits are in either PCI or CABG as they address the underlying problem; reduced coronary artery blood flow. CABG is favoured over PCI in the case of people who are over 65 years old, have diabetes or have triple vessel disease. This patient has all three and therefore is a better option, but both options should be discussed in an MDT setting and with the patient.

Question#312

A 76-year-old female presents to the clinic for evaluation in the company of her daughter. She has a history of ischaemic cardiomyopathy with a recent echocardiogram showing an ejection fraction of 30%, increased filling pressures, moderate aortic stenosis, moderate mitral regurgitation, and pulmonary arterial systolic pressures of 32 mmHg. At present, she can walk approximately 100 yards before she gets significantly short of breath, and this has been the case since her last hospitalisation 4 months ago. Otherwise, she feels reasonably well and has no new complaints today. She monitors her weight regularly at home and says that these have been stable.

Her medications include aspirin, amlodipine, metoprolol, lisinopril, furosemide, gabapentin, as required paracetamol, and timolol and brinzolamide eye drops. Physical examination shows a heart rate of 63 beats per minute and blood pressure of 129/76 mmHg. Her JVP is about 4cm above the angle of Louis, her

apical impulse is laterally displaced, and heart sounds are audible with a holosystolic murmur heard loudest at the apex. Auscultation of her chest is clear and she has trace peripheral oedema.

What is the next best step in the management of this patient?

Discontinue amlodipine7%
Add spironolactone68%
Undertaken ECG to assess her QRS interval19%
Up-titrate the dose of furosemide4%
Go down on the dose of her metoprolol2%

Correct answer is b.

Patients with heart failure and an ejection fraction < 35% who are euvoalaemic and are already taking an ACE-inhibitor and a beta-blocker should have an aldosterone antagonist added to their regimen as a next step

Important for me
Less important

This patient has congestive heart failure related to her history of ischaemic cardiomyopathy. She seems to be doing relatively well, and her symptoms are stable. In addition, she has been monitoring her weight at home which has also been stable, and she does not have signs of volume overload on physical exam. Her heart rate and blood pressure are also in acceptable limits. Of note, her recent echocardiogram shows an ejection fraction of 30%. She, therefore, qualifies for the addition of an aldosterone antagonist to her drug regimen. Individuals with congestive heart failure who already on an ACE-inhibitor and beta-blocker, and to have an ejection fraction of less than 35% who are started on an aldosterone antagonist have been shown to have low mortality compared to those who do not have an aldosterone antagonist added to their medications.

Her blood pressure is acceptable at the moment and so she does not need to discontinue amlodipine.

Cardiac resynchronization therapy is indicated in heart failure patients with NYHA functional class III and IV patients who remain symptomatic despite stable, optimal heart failure medical therapy and who have an LVEF ≤ 35% and a prolonged QRS duration on an ECG. This patient is not yet on optimal medical

therapy and should be started on an aldosterone antagonist before she can be considered for this approach.

She does not have signs of volume overload and therefore does not require changes to the dose of her furosemide today.

Her heart rate is in acceptable limits today and she does not have symptoms to suggest that the dose of her beta-blocker needs to be decreased.

Question#313

A 59-year-old man presents with stridor which came on gradually over the course of the day. On examination you note swelling of the tongue and the mucous membranes of the oropharynx. He has a past medical history of rheumatoid arthritis, type 2 diabetes mellitus and heart failure.

He was recently started on a new medication. What medicine would be the most likely cause of this presentation?

Nimodipine11%Losartan4%Lisinopril82%Prednisolone1%Sitagliptin2%

Correct answer is c.

Drug-induced angioedema without urticaria is most frequently associated with angiotensin converting enzyme inhibitors or, less frequently, angiotensin II receptor blockers

Important for meLess important

Angioedema is a well recognised side effect of angiotensin-converting enzyme (ACE) inhibitor therapy. Angioedema can also be seen with angiotensin receptor blocker (ARB) therapy but much less frequently than is the case with ACE inhibitors. Therefore lisinopril is a better answer than losartan.

ACE inhibitors block the enzyme ACE resulting in increased levels of bradykinin. Bradykinin then accumulates potentially resulting in angioedema.

All the other options can cause peripheral oedema, however not specifically angioedema as described.

DiscussImprove

Question#314

A 52-year-old gentleman works as a lorry driver. His past medical history includes hypertension and raised BMI. He is admitted to the Emergency Department with chest pain. An ECG confirms ST depression in V1 to V4, with no pathological Q waves. Subsequent blood tests reveal a troponin of 10,000 consistent with a non-ST-elevation myocardial infarction (NSTEMI). He is commenced on appropriate secondary prevention, including aspirin 75mg OD, clopidogrel 75mg OD and fondaparinux 2.5mg SC. The patient is transferred to the cardiology unit and awaits in-patient angiography.

You are asked to review him urgently as he describes acute breathlessness. He denies chest pain. Observations are as follows: heart rate 130 beats per minute, blood pressure 95/62 mmHg, temperature 36.1°C, respiratory rate 28/min, saturations 94% on 10 litres oxygen. On examination, heart sounds are normal with no added sounds. JVP is mildly elevated. There is no evidence of ascites or peripheral oedema. A repeat ECG confirms sinus tachycardia (heart rate 130bpm) but no dynamic changes. A portable chest x-ray reveals cardiomegaly with upper lobe diversion and peri-hilar shadowing. An urgent bedside echocardiogram demonstrates significant LV systolic dysfunction with a 2cm of pericardial effusion but no features of tamponade.

What is the single next best management step?

Continuous positive airway pressure (CPAP)21%
Ultrasound-guided pericardiocentesis22%
Urgent angiography16%
IV fluids3%
Inotrope and IV diuretic39%

Correct answer is e.

This patient is demonstrating features of cardiogenic shock, secondary to anterior

wall myocardial infarction. With anterior infarcts, the mainstay of treatment includes diuretics to reduce pulmonary congestion and inotropic support to enhance cardiac output and perfusion.

Echocardiography demonstrates a reasonable-sized pericardial effusion but there is no indication for pericardiocentesis in the absence of features consistent with cardiac tamponade. Intravenous fluids would exacerbate pulmonary oedema in the context of anterior infarcts and are relatively contraindicated for this reason. They are of relevance, however, in inferior infarcts where preload needs to be maintained. This patient is preserving oxygen saturations via Venturi mask and therefore, CPAP is not currently indicated. Angiography is required to clarify the extent of coronary disease in due course, but the patient requires stabilisation before this can occur.

Discuss (13)Improve

Question#315

A 38-year-old man is referred to the pharmacology clinic for review. He is currently managed for hypertension with three anti-hypertensive agents, (ramipril, amlodipine and indapamide). His GP is concerned as he has still not achieved blood pressure target. On examination his blood pressure is 155/95 mmHg, pulse is 78 beats per minute and regular. There are no heart murmurs or bruits. Abdomen is soft and non tender with no masses and his body mass index is 24 kg/m².

Investigations:

Na ⁺	142 mmol/l
K ⁺	3.1 mmol/l
HCO ₃ ⁻	30 mmol/l

Urea	7.0 mmol/l
Creatinine	90 µmol/l

Which of the following is the most likely diagnosis?

- a) Bartter's syndrome
- b) Conn's syndrome
- c) Cushing's syndrome
- d) Licorice overdose
- e) Renal artery stenosis

This patient has resistant hypertension in the presence of hypokalaemia and high bicarbonate, despite initiation of three different anti-hypertensive medications. Although the lack of symptoms or signs of cardiovascular disease doesn't rule out renal artery stenosis completely, it makes it less likely versus Conn's or licorice overdose as a cause of hypertension with hypokalaemia. Licorice overdose is made less likely by the fact that most licorice sold in shops in the UK now has glycyrrhetic acid removed, and this is the active agent which leads to hypokalaemia and hypertension. Conn's is the single most common cause of secondary hypertension, making up between 5 and 10% of cases of hypertension overall.

Bartter's syndrome leads to hypokalaemia with hypotension and is therefore not an appropriate answer here.

Question#316

A 70-year-old man is admitted to hospital with chest pain associated with nausea and vomiting. There was no recent history of shortness of breath or wheeze. ECG demonstrated inferolateral ST depression and troponin was significantly elevated at 12 hours after symptom onset.

His past medical history included chronic obstructive pulmonary disease, hypertension and a previous MI 3 years previously. The patient reported two exacerbations of his COPD within the last 18 months, neither of which required hospital admission. He had never required intubation or non-invasive ventilation due to his COPD.

Regular medications included bendroflumethiazide 2.5 mg OD, inhaled tiotropium 18 microg OD and inhaled salbutamol 100 microg PRN. There were no known drug allergies. The patient was a retired engineer, an ex-smoker and lived independently with his wife.

The initial impression was of non-ST elevation myocardial infarction and treatment was initiated with aspirin, clopidogrel, fondaparinux, ramipril and atorvastatin. The patient subsequently mobilised pain-free on the ward and was discharged home with a plan for outpatient stress echocardiogram.

Recent pulmonary function tests are given in the table below.

Forced vital capacity	105 % predicted
Forced expiratory volume (1s)	67 % predicted
FVC / FEV1	64 % predicted

Transthoracic echocardiogram: no valvular abnormality; mild-moderate systolic impairment of lateral left ventricle; normal right ventricular function

What is the most appropriate plan for beta-blockade therapy for this patient?

- a) Low dose bisoprolol with slow up-titration of dose
- b) Low dose carvedilol with slow up-titration of dose

- c) Low dose bisoprolol
- d) Low dose carvedilol
- e) Beta-blockade therapy inappropriate

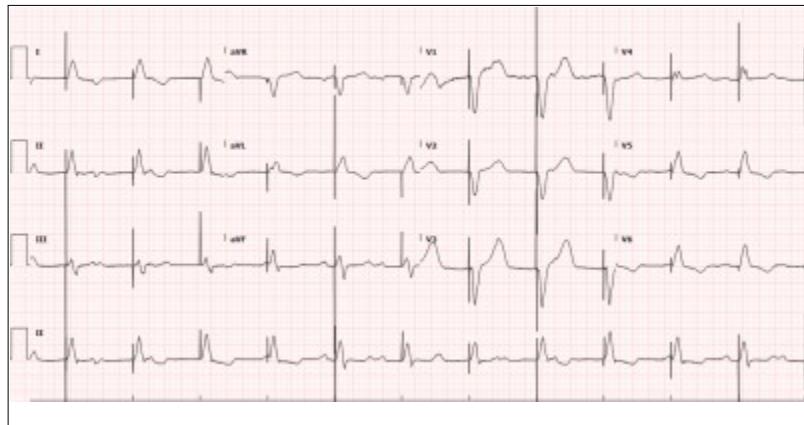
NICE guidance on secondary prevention following myocardial infarction includes a recommendation for beta-blocker therapy as it has been shown to reduce mortality. In patients with COPD or asthma, beta-blockers have sometimes been held due to concerns about precipitating bronchospasm. However, a large observational study has shown substantial survival benefits for patients with COPD who have suffered a myocardial infarction in starting beta-blockers during hospital admission (adjusted hazard ratio 0.50).

Therefore, beta-blocker therapy is indicated in this patient. The most appropriate choice is the cardio-selective agent (bisoprolol) started at a low dose and slowly up-titrated as per current guidance.

Qunit J, Herrett E, Bhaskaran K, Timmis A, Hemingway H, Wedzicha J et al. Effect of beta-blockers on mortality after myocardial infarction in adults with COPD: population-based cohort study of UK electronic healthcare records. *BMJ* 2013;347:f6650.

Question#317

A 62-year-old man is recovering from an aortic valve replacement via median sternotomy in the high-dependency unit. You are called to see him overnight as he is experiencing chest pain, worse on breathing. An ECG is performed and displayed below. He had temporary trans-venous pacing wires inserted during the procedure.



What does the ECG show?

- a) AAI pacing
- b) Complete heart block with loss of capture
- c) DDD pacing
- d) Myocardial infarction
- e) VVI pacing

Correct answer is e.

Although atrial pacing wires are commonly inserted following cardiac surgery, it is particularly important to insert ventricular wires following aortic valve surgery due to the significantly higher incidence of complete heart block postoperatively. The ECG shows pacing spikes immediately preceding QRS complexes, indicating ventricular (**VVI**) pacing with good capture. This patient has a degree of heart block underlying his paced rhythm, hence the discordance between the p waves and the QRS complexes, and the reason why the patient is paced.

AAI pacing would show pacing spikes prior to p waves, and in instances of heart block as shown here there would be abnormal or potentially absent conduction to the ventricles. As the pacing spikes are associated with the QRS complex here, AAI cannot be the mode in use.

Although this patient may have **complete heart block**, there is good capture as shown by a 1:1 ratio between pacing spikes and subsequent QRS complexes. Therefore loss of capture is an incorrect answer.

DDD pacing refers to the pacing of both the atria and ventricles. Although this can be useful post-operatively to increase cardiac output, it is not being used here. DDD pacing would either show pacing of the atria (pacing spikes before p waves) or tracking of the patient's intrinsic atrial rate (ventricular pacing spikes that follow p waves and immediately precede QRS complexes).

Pain is very common following a sternotomy and is classically sharp and pleuritic. It can be difficult to interpret ischaemia on a paced ECG however the Sgarbossa criteria is a useful tool for this. As this patient does not fit this criterion, and the pain is classical for sternotomy pain rather than cardiac, **myocardial infarction** is unlikely.

Discuss (2)Improve

Question#318

A 75-year-old female is admitted to hospital with community-acquired pneumonia. She is currently being treated with intravenous benzylpenicillin and oral clarithromycin. On the third day of admission, she complains of a sensation of fullness on the lateral aspect of her right leg.

Her past medical history includes osteoarthritis, temporal arteritis (now off steroids) and a uterine prolapse.

Bloods performed the day before show:

Hb	131 g/l
Platelets	520 * 10 ⁹ /l
WBC	14.2 * 10 ⁹ /l

Na ⁺	141 mmol/l
K ⁺	4.1 mmol/l
Urea	6.2 mmol/l
Creatinine	86 µmol/l
CRP	78 mg/L

A doppler ultrasound scan is performed that shows a superficial vein thrombosis in the right leg. What is the most appropriate management?

- a) Treatment dose low molecular weight heparin (LMWH) and warfarin until warfarin therapeutic then anticoagulate for three months
- b) Prophylactic dose low molecular weight heparin (LMWH) for 30 days.
- c) Treatment dose low molecular weight heparin (LMWH) for 30 days.
- d) Non-steroidal anti-inflammatory drugs for 8-12 days.
- e) Low molecular weight heparin (LMWH) and warfarin until warfarin therapeutic then anticoagulate for six months

Correct answer is b.

Superficial vein thrombosis (also referred to as thrombophlebitis) is a common condition and in the majority of cases is self-limiting. Patients presenting with a superficial vein thrombosis are at increased risk of a venous thromboembolic event and up to 20% of patients will already have a deep vein thrombosis (DVT) on presentation. Patients are more likely to have thrombus extension into the deep venous system if they have more than 5cm of thrombus within 10cm of the saphenofemoral junction (i.e. within the proximal long saphenous vein).

The evidence for how superficial vein thrombosis should be managed comes from a Cochrane review and is used to support the Scottish intercollegiate guidelines network (SIGN) guidelines on the prevention and management of venous

thromboembolism (guide 122). The guidelines recommend that all patients with clinical signs of a superficial vein thrombosis should have an ultrasound scan to exclude a DVT.

Patients with a confirmed diagnosis of a superficial vein thrombosis should be considered for treatment with anti-embolism stockings and prophylactic doses of low molecular weight heparin (LMWH) for 30 days or fondaparinux for 45 days. In cases where LMWH is contraindicated, 8-12 days of oral non-steroidal anti-inflammatory drugs should be offered as this has been shown to reduce the risk of thrombus extension.

Question#319

A 45-year-old male patient presents with a worsening shortness of breath to the medical assessment unit. He has an accompanying letter from his GP stating that his past medical history is significant only for a deteriorating renal function that is secondary to polycystic kidney disease.

On examination there is no evidence of fluid overload, with the lung fields remaining clear and the JVP not elevated. You do however discern a late systolic murmur heard best at the cardiac apex which is immediately preceded by a 'click'.

Observations

- heart rate 82bpm
- blood pressure 106/86mmHg
- respiratory rate 16 per minute
- temperature 36.5
- oxygen saturation 97% on room air

What is the most likely diagnosis?

- a) Mitral valve prolapse
- b) Mitral stenosis
- c) Pulmonary stenosis
- d) Mitral regurgitation
- e) Aortic stenosis

Correct ans is a.

Adult polycystic kidney disease can be associated with extra-renal complications. Up to 85% of patients with autosomal dominant polycystic kidney disease (ADPKD) will exhibit liver cysts by the age of 30. Other complications include intracranial aneurysm and heart valve disease. Affected heart valves can occur in up to 25% of ADPKD patients and of them the majority have mitral valve prolapse. The majority of patients with mitral valve prolapse will be asymptomatic but when symptoms are present mitral valve prolapse has to be considered and investigated with an echocardiogram.

The 'click' coupled with the knowledge of the underlying ADPKD makes the murmur heard in this patient that of a mitral valve prolapse. The prolapse can worsen and cause a secondary mitral regurgitation leading to symptoms such as shortness of breath. The narrow pulse pressure present also suggests an element of left ventricular dysfunction.

Discuss (4)Improve

Question#320

A 75-year-old gentleman with a background of type II diabetes mellitus, ischaemic heart disease (IHD) and New York Heart Association (NYHA) III heart failure presents to your clinic with continuing fatigue and shortness of breath on minimal exertion. He currently takes Aspirin 75mg, Bisoprolol 10mg, Ramipril 5mg, Furosemide 40mg twice daily and Spironolactone 25mg. His electrocardiograph

demonstrates left bundle branch block (old) and his ejection fraction is 32% measured by echocardiography at the clinic today. His chest x-ray shows an enlarged cardio-thoracic ratio but no obvious pulmonary oedema. Blood tests taken with his general practitioner this week are as follows:

Haemoglobin	110g/l
White cell count	11.0×10^9
Neutrophils	7.2×10^9
Platelets	240×10^9
Na+	137 mmol/l
K+	4.2 mmol/l
Urea	9 mmol/l
Creatinine	125 µmol/l

What evidence-based treatment, if any, is available to treat this gentleman's cardiac failure?

- a) Increase dose of Spironolactone to 50mg once daily
- b) Increase dose of Furosemide to 80mg twice daily
- c) Cardiac-resynchronisation therapy
- d) Angiography +/- coronary stenting
- e) He is already on maximal treatment

NICE guidance has recommended that cardiac resynchronisation therapy can be

offered to patients who fulfil all of the following criteria:

- NYHA III or IV heart failure
- Ejection fraction of <35%
- The heart is beating regularly with evidence of electrical conduction disease
- They are medication that is most effective for them

This patient is on appropriate doses of secondary-prevention and heart failure medications and there is no evidence of an acute coronary syndrome but the previous IHD is likely the cause of his cardiac failure. He satisfies all of the above criteria for cardiac resynchronisation and this has a good evidence-base.

Discuss (22)Improve

Question#321

A 58-year-old woman was referred to the acute medical unit with headaches and hypertension. She had no history of fever, neck stiffness, limb weakness, seizures or blurring of her vision. Her pulse rate was 70 beats per minute and blood pressure was 200/110 mmHg. Her heart sounds, chest examination, abdominal and neurological examination were normal. Fundoscopy was also normal.

Hb	138g/l
Platelets	238 * 10 ⁹ /l
WBC	6.2 * 10 ⁹ /l
Na ⁺	135 mmol/l
K ⁺	3.8 mmol/l

Urea	6.4 mmol/l
Creatinine	75 µmol/l
ECG	normal sinus rhythm
Chest x-ray	normal
CT Head	normal
Urinalysis	normal

What is the best initial management?

- a) Intravenous labetalol
- b) Intravenous glyceryl trinitrate
- c) Oral amlodipine
- d) Intravenous sodium nitroprusside
- e) Sublingual nifedipine

Correct answer is c.

In an hypertensive urgency, treatment aims to lower blood pressure with the use of oral anti-hypertensive medication like a calcium channel blocker

This woman has hypertensive urgency, which is a severe increase in blood pressure (>180 mmHg systolic or >110 mmHg diastolic) not associated with target organ damage. This may be associated with headache, shortness of breath, or nosebleeds. The treatment is to lower the blood pressure within 24-48 hours and oral antihypertensive medications are recommended. Hospitalisation is not required. Amlodipine is a dihydropyridine calcium channel antagonist and a potent antihypertensive that can be used in this situation.

Intravenous antihypertensives like labetalol, glyceryltrinitrate are used in hypertensive emergencies which require lowering of blood pressure within

minutes to hours e.g hypertensive encephalopathy, aortic dissection. Sublingual nifedipine is not recommended for treatment of hypertensive crises.

Question #322

A 24-year-old primigravida presents to the emergency department with a 72-hour history of nausea and vomiting with associated right upper quadrant pain. She has no past medical history of note and takes only pregnancy vitamins. During her 32-week midwife appointment 1 week ago, it was noted that she had gained weight and had borderline hypertension. The decision at that appointment was to monitor.

On examination, she is clinically dehydrated and appears unwell. Respiratory and cardiovascular examinations are unremarkable and abdominal examination demonstrates a gravid uterus and right upper quadrant tenderness. There is pitting oedema to the mid-shin. Observations are all within normal limits aside from a blood pressure of 145/90mmHg. She is alert and orientated, reports no headaches and has no rashes.

Blood tests are performed, results are as demonstrated:

Hb	100 g/L	Male: (135-180) Female: (115 - 160)
Platelets	$97 * 10^9/L$	(150 - 400)
WBC	$7.3 * 10^9/L$	(4.0 - 11.0)
PT	13.0 seconds	(9.5-13.5)
APTT	39.0 seconds	(30-40)

Na ⁺	132 mmol/L	(135 - 145)
K ⁺	3.4 mmol/L	(3.5 - 5.0)
Bicarbonate	22 mmol/L	(22 - 29)
Urea	7.5 mmol/L	(2.0 - 7.0)
Creatinine	100 µmol/L	(55 - 120)
Bilirubin	45 µmol/L	(3 - 17)
ALP	150 u/L	(30 - 100)
ALT	350 u/L	(3 - 40)
Albumin	34 g/L	(35 - 50)

An ultrasound abdomen shows patchy areas of hepatic enhanced echogenicity. Fetal monitoring is satisfactory, but the baby is in the breech position.

Given the likely diagnosis, what is the most appropriate management?

- f) Commence IV antibiotics
- g) Give steroids and organise an induction of labour
- h) Organise a plasma exchange with fresh frozen plasma
- i) Rehydrate with IV fluids and monitor
- j) Give steroids and organise a caesarean section within 48 hours

Delivery of the baby is the treatment of HELLP syndrome

This patient has HELLP syndrome as shown by her anaemia (secondary to haemolysis), thrombocytopenia and deranged LFTs. She is 33 weeks pregnant and

thus steroids would be recommended prior to delivery if possible to aid the maturation of her child's lungs. She does not have any evidence of a liver haematoma, disseminated intravascular coagulation or haemodynamic instability and thus she does not need an immediate delivery at this stage. Induction of labour would not be appropriate as her child is breech.

Differentials would include cholecystitis and thrombotic thrombocytopenic purpura (TTP).

IV antibiotics would be appropriate in the treatment of cholecystitis, but this patient has no features of infection, going against a diagnosis of cholecystitis.

Plasma exchange with fresh frozen plasma would be the recommended treatment for TTP, however, TTP would typically present with neurological symptoms such as confusion, headaches, and seizures in severe cases, which our patient does not have.

Question #323

A 24-year-old male presents to the emergency department with a collapse. The previous night he had been out drinking heavily with his friends in a nightclub. A friend attends with him and states that some of the lads had taken MCAT (Mephedrone) but he is unsure whether Michael also took MCAT. Michael felt well when he woke up but realised that he was late for his football match so rushed out of the house without any breakfast. He had just scored the first goal of the match when he collapsed. When he came around he complained of palpitations.

His past medical history includes asthma and hay fever for which he takes regular antihistamines. During the ambulance ride to hospital the paramedic performed some observations and an ECG. He was found to have a blood glucose of 3.6 and was given some GlucoGel and a biscuit. Other observations included a blood pressure of 90/60 mmHg, heart rate of 135/min which was irregular, irregular in character, respiratory rate of 16/min, afebrile.

The emergency department doctor reviewed the ECG and diagnosed atrial fibrillation with a fast ventricular response and prescribed digoxin in view of his

asthma history and blood pressure readings. The patient had a normal QTc. Five minutes later the cardiac monitor showed a broad complex tachycardia and Michael became unresponsive.

What was the cause of the patient's collapse?

- f) Hypertrophic cardiomyopathy (HCM)
- g) MCAT related arrhythmia
- h) Wolff-Parkinson-White syndrome
- i) Alcohol induced atrial fibrillation
- j) Torsades de pointes

Correct answer is c.

Wolff-Parkinson-White syndrome (WPW) is a disorder of the conduction system also known as a pre-excitation syndrome. The incidence of WPW is between 0.1% and 0.3% in the general population. Sudden cardiac death in people with WPW is rare (incidence of less than 0.6%).

In WPW there is an abnormal accessory pathway called the 'Bundle of Kent' found between the atria and ventricles. If this pathway is used signals may stimulate the ventricles to contract prematurely, resulting in atrioventricular re-entrant tachycardia.

Normally the AV node acts as a gatekeeper limiting the amount of electrical activity that can reach the ventricles. If a patient has an arrhythmia whereby the atria generate an excessively fast rhythm such as atrial flutter or fibrillation the AV node is able to reduce the amount of electrical activity that reaches the ventricles. However, the Bundle of Kent does not possess the ability to limit the electrical signals to the ventricles. If the person has an atrial arrhythmia the signals can pass down the bundle and cause the ventricles to contract at the same rate. In the extreme heart rates and when the accessory pathway is used the patient may develop dangerous cardiac arrhythmias which can trigger ventricular fibrillation and lead to death.

In WPW certain medications that can block the AV node or those that can

enhance conduction down the accessory pathway by increasing the refractory period in the AV node should be avoided. These include digoxin, adenosine, diltiazem, verapamil, other calcium channel blockers and beta blockers. In this case, the patient's WPW was worsened by administration of digoxin and her arrhythmia developed into ventricular fibrillation.

People with WPW are usually asymptomatic. Symptomatic patients may complain of shortness of breath, palpitations, dizziness and syncope. Diagnosis is usually made by ECG as patients may have the characteristic 'delta wave'. Patients may also be found to be in atrial fibrillation or have SVT. Treatment can include cardioversion in emergencies otherwise amiodarone. Definitive treatment would be ablation.

Question #324

A 65-year-old with exertional dyspnoea over the past 3 months is referred to the pulmonary hypertension team. An initial echocardiogram demonstrated 65% ejection fraction, preserved left ventricular function with a pulmonary arterial pressure of 72 mmHg. She undergoes a right and left heart catheter, revealing the following saturations:

Right atrium high	60%
Right atrium mid	89%
Right atrium low	70%
Right ventricle high	70%
Right ventricle mid	73%
Right ventricle low	72%

Pulmonary artery	71%
Capillary wedge	96%

What is the most likely diagnosis?

- f) Aortic stenosis
- g) Atrial septal primum defect
- h) Atrial septal secundum defect
- i) Pulmonary stenosis
- j) Mitral stenosis

The key to questions regarding saturations and cardiac catheters is to spot the 'step-up' in oxygen saturation. It is present between the high and mid right atrium, demonstrating the presence of a left-to-right shunt. Primum ASD defects typically occur lower in the septum than secundum defects, which typically can be found in the mid-atrial region.

Question #325

A 48-year-old man is brought in by ambulance to the Emergency Department after experiencing sudden onset crushing central chest pain radiating down his arm. His ECG showed an inferior STEMI and he undergoes successful PCI with 2 stents inserted into his right coronary artery. He is transferred to CCU and started on secondary prevention medication.

24 hours after his presentation he becomes bradycardic at 34bpm and his blood pressure falls to 80/47 mmHg. He denies chest pain but feels dizzy and light headed. His cardiac monitor shows 3rd-degree heart block. He is given atropine but his heart rate does not raise and his blood pressure remains low.

What is the most appropriate next step in his management?

- f) Isoprenaline infusion
- g) Temporary pacing wire
- h) Permanent pacemaker
- i) Repeat angiography
- j) Adrenaline

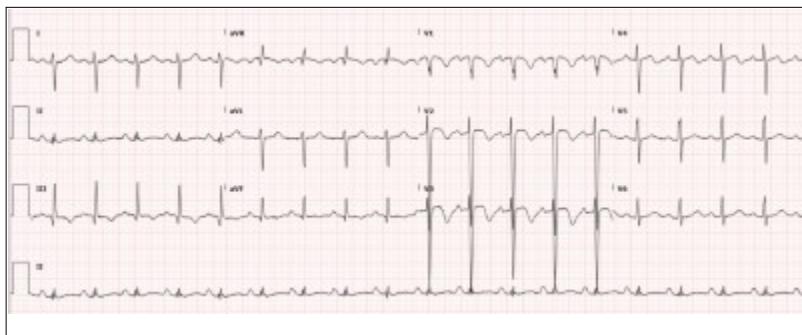
Correct answer is .b

This gentleman has suffered an inferior STEMI. Complete heart block is a recognised complication of both inferior and anterior STEMI. Inferior STEMI CHB is usually more transient and they only require treatment if they become symptomatic. Anterior STEMI CHB is a more serious complication and they are more likely to need a permanent pacing system. In this case, as he has not responded to atropine then isoprenaline infusion is unlikely to work. There is no suggestion of repeat infarction so angiography is not indicated. He will require a temporary pacing wire to be placed.

Question #326

A man in his 50's is brought to the emergency department by ambulance and was found collapsed in the street. His vital signs are as follows: Blood pressure 90/60 mmHg, pulse 120 bpm, respirations 25 bpm and temperature 37.9 °C.

The 12 lead ECG showed:



What is the most likely cause of the patient's presentation?

- f) Non-ST elevation myocardial infarction
- g) Pulmonary embolism

- h) ST elevation myocardial infarction
- i) Severe hypokalaemia
- j) Takotsubo cardiomyopathy

The ECG shows sinus tachycardia with right axis deviation, P pulmonale, Q waves in lead III and inverted T waves in the right precordial leads (V1-V3) and lead III.

Pulmonary embolism is the correct answer. Although the ECG is neither sensitive nor specific for pulmonary embolism, the classical findings of S1Q3T3 highly suggest right ventricular strain. Hence, the clinical picture is highly suspicious for pulmonary embolism, and the patient should promptly have an urgent CT pulmonary angiogram and thrombolysis if indicated. Other acute causes for right ventricular strain, like pneumothorax, upper airway obstruction and exacerbation of asthma/COPD, should also be considered in the differentials.

Non ST elevation myocardial infarction is incorrect. ECG changes like ST depression or T wave inversion in NSTEMI are often non-specific. If new, the T-wave inversions in this patient may suggest acute coronary syndrome, particularly right ventricular infarction, given the right ventricular strain pattern; however, NSTEMI is unlikely to cause collapse. The classical S1Q3T3 pattern in ECG, along with the clinical findings of tachycardia, tachypnoea and low-grade temperature, is more suggestive of pulmonary embolism in this patient.

ST elevation myocardial infarction is incorrect. Classically, STEMI is diagnosed if there is > 1-2 mm ST elevation in 2 contiguous leads or a new left bundle branch block (LBBB). Both of these are not found in this patient's ECG.

Severe hypokalaemia is incorrect. Although hypokalaemia may present with T-wave inversions in the precordial leads, there are no ST depression or U waves commonly seen in hypokalaemia. While hypokalaemia can cause muscle tetany, paralysis and respiratory failure, it is unlikely to cause collapse.

Takotsubo cardiomyopathy is incorrect. ECG changes like T wave inversion in Takotsubo cardiomyopathy are often non-specific. Moreover, the classical S1Q3T3

pattern in ECG, along with the clinical findings of tachycardia, tachypnoea and low-grade temperature, is more suggestive of pulmonary embolism in this patient.

Question #327

A 58 year-old man with a background of ischaemic heart disease and Crohn's disease has developed colonic enterocutaneous fistulae. He is admitted to hospital under the surgical team and a temporary ileostomy is formed to defunction the bowel and promote healing.

Two days post-operatively he develops palpitations and the surgical team request your assistance.

On examination the pulse rate is 220bpm and the blood pressure is 135/90mmHg. Oxygen saturations are 96% on 2L nasal oxygen.

The chest is clear to auscultation.

A 12-lead ECG reveals a wide-complex tachycardia with a polymorphic waveform.

Blood tests from the morning reveal:

Hb	129 g/l
Platelets	643 * 10 ⁹ /l
WBC	13.8 * 10 ⁹ /l
Na ⁺	129 mmol/l
K ⁺	3.3 mmol/l
Phosphate	0.63 mmol/l

Mg ⁺⁺	0.59 mmol/l
Urea	8.1 mmol/l
Creatinine	97 µmol/l
Bilirubin	15 µmol/l
ALP	143 u/l
ALT	53 u/l
Albumin	31 g/l

What is the most appropriate initial management?

- f) Adenosine 6mg Iv
- g) Magnesium sulphate 2g
- h) Metoprolol 5mg IV
- i) Synchronised DC shock
- j) Amiodarone 300mg IV

Correct answer is b.

In this scenario this patient has developed polymorphic ventricular tachycardia with no adverse features. This is likely to be secondary to electrolyte derangement in this case.

Initial management steps include stopping all drugs known to prolong the QT interval. Correct electrolyte abnormalities, especially hypokalaemia. Give magnesium sulphate 2 g IV over 10 min (= 8 mmol).

If adverse features are present (shock, syncope, myocardial ischaemia or heart failure) arrange immediate synchronised cardioversion.

Discuss (6) Improve

Question #328

A 57-year-old man attends his GP surgery for review. For the past year, he has experienced exertional chest pain, which initially only came on whilst walking uphill, but has now started to affect him when walking on the flat. The pain is alleviated by the use of glyceryl trinitrate spray. He denies ever having had chest pain at rest and does not report any symptoms of postural hypotension. His other medications include aspirin, atorvastatin and atenolol, which has been maximally titrated.

What is the most appropriate next step in management for this patient?

- f) Refer for assessment for percutaneous coronary intervention (PCI) or CABG
- g) Start amlodipine
- h) Start isosorbide mononitrate
- i) Start ranolazine
- j) Start verapamil

If angina is not controlled with a beta-blocker, a longer-acting dihydropyridine calcium channel blocker should be added

The correct answer is **start amlodipine**. This patient has a likely diagnosis of stable angina, as he suffers from exertional chest pain which is alleviated by the use of GTN spray. As he is currently taking a beta-blocker at the maximum permissible dose, the next most appropriate step in management is to add in a calcium channel blocker. In this scenario, dihydropyridine calcium channel blockers are preferred to non-dihydropyridine calcium channel blockers, due to the increased risk of the latter causing complete heart block in combination with beta-blockers.

Refer for assessment for percutaneous coronary intervention (PCI) or CABG is incorrect. As the patient has stable angina, with no symptoms at rest, he does not warrant urgent referral for PCI. Non-urgent referral for PCI or CABG would be

appropriate if his symptoms were not improved by the addition of amlodipine.

Start isosorbide mononitrate is incorrect. This would be an appropriate third-line management option for stable angina in this patient if amlodipine failed to alleviate his symptoms. However, it should only be started whilst awaiting assessment for revascularization with PCI or CABG.

Start ranolazine is incorrect. This would also be more appropriate as a third-line management option whilst awaiting specialist assessment for PCI or CABG.

Start verapamil is incorrect. Although it would be reasonable to start a calcium channel blocker in this patient, starting a non-dihydropyridine calcium channel blocker such as verapamil in a patient already on a beta-blocker would run the risk of precipitating complete heart block.

Discuss (2)Improve