## Question #:3

**CLINICAL SCENERIO**: A 54-year-old male business executive with no known past medical history or family history is found to have a heart murmur. Examination reveals a pansystolic murmur in the apex. ECG is normal. Transthoracic echocardiogram shows severe mitral regurgitation, preserved left ventricular function (EF 85%) and pulmonary arterial systolic pressure of 15 mmHg.

**QUESTION LINE**: Which of the following is appropriate management?

**OPTIONS**: - 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

**CORRECT-CHOICE LINE**: Correc answer is e.

**REASONING**: The patient has severe chronic non-ischaemic mitral regurgitation that has not decompensated and 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.

**>>DESCRIPTION**: A 54-year-old male with a heart murmur has severe mitral regurgitation but preserved LV function (EF 85%) and normal pulmonary arterial pressure. He is asymptomatic with no prior history.

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

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: 6 monthly echocardiogram

**>>REASONING**: The patient has severe mitral regurgitation but does not meet the criteria for intervention, such as symptoms, LV dysfunction, or pulmonary hypertension. Therefore, serial monitoring with 6-monthly echocardiograms is the appropriate management. Infective endocarditis prophylaxis is not indicated. Percutaneous repair is for high-risk surgical patients with NYHA III/IV heart failure.

## Question #:15

**CLINICAL SCENERIO**: A 78-year-old lady presents with syncope and lightheadedness over the past couple of months. She has a past medical history of angina, COPD, and hypertension. Her regular medicines include GTN, aspirin, ramipril, and furosemide. ECG shows a rate of 68 bpm, sinus rhythm, PR interval of 220 ms, QRS duration of 130 ms, RBBB, and left axis deviation.

**QUESTION LINE**: What investigation will you order?

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

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: 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.

**>>DESCRIPTION**: A 78-year-old with syncope, angina, COPD, and hypertension presents with lightheadedness. Medications include GTN, aspirin, ramipril, and furosemide. ECG: 68 bpm, sinus rhythm, PR 220 ms, QRS 130 ms, RBBB, left axis deviation.

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

**>>CORRECT-CHOICE LINE**: Correct answer is a.

**>>CORRECT-CHOICE\_TEXT**: 7-day ECG Holter monitor

**>>REASONING**: The ECG findings (RBBB, LAD, prolonged PR interval) suggest incomplete trifascicular block, necessitating a 7-day Holter monitor to rule out complete heart block. While a transthoracic echo could assess structural heart disease, arrhythmia is the immediate concern. Tilt-table test is for vasovagal syncope.

## Question #:92

**CLINICAL SCENERIO**: A 71-year-old gentleman is reviewed in hospital post-COPD exacerbation treatment. His systolic blood pressure ranges from 138mmHg to 156mmHg. He has ischaemic heart disease, gout, gallstones, and a repaired fractured neck of femur. He has an ACR of 31mg/mmol and takes allopurinol, aspirin, tiotropium, Symbicort, salbutamol.

**QUESTION LINE**: What is the most appropriate pharmacological management for his hypertension?

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

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: 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.

**>>DESCRIPTION**: 71-year-old male, post-COPD exacerbation, BP 138-156mmHg, with IHD, gout, gallstones, and a repaired hip fracture. ACR 31mg/mmol. Medications: allopurinol, aspirin, tiotropium, Symbicort, salbutamol.

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

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: ACE-inhibitor

**>>REASONING**: ACE-inhibitor is the most appropriate choice due to the presence of hypertension and proteinuria. Calcium channel blockers are suitable without proteinuria. Thiazide diuretics and alpha-blockers are later-line options. Beta-blockers may cause bronchospasm in COPD patients.

## Question #:48

**CLINICAL SCENERIO**: A 76-year-old man presents to the emergency department with shortness of breath on exertion, difficulty walking up stairs, and weight gain over 2 weeks. He needs to lay upright to sleep. His history includes 2 previous non-ST-segment elevation myocardial infarctions treated with coronary stenting. Examination shows a heart rate of 111 bpm, BP of 113/76 mmHg, shortness of breath at rest, JVP is 7cm, laterally displaced apical impulse, holosystolic murmur at the apex, bi-basal crepitations, and 2+ peripheral oedema. ECG shows Q-waves in inferior leads, and bloods show elevated troponins.

**QUESTION LINE**: Which of the following is the most appropriate next step in the management of this patient?

**OPTIONS**: - 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 rat - e) Administer a bolus of intravenous furosemide and then start him on an infusion

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: The most appropriate medication in patients with acute heart failure and a preserved ejection fraction who have signs of volume overload is addition/uptitration 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 a 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

**>>DESCRIPTION**: A 76-year-old man with a history of 2 prior MIs presents with shortness of breath, orthopnea, and weight gain. Exam: HR 111, BP 113/76, elevated JVP, displaced PMI, holosystolic murmur, bibasilar crackles, 2+ edema. ECG: Q-waves. Elevated troponins.

**>>OPTIONS**: a) Administer a bolus of intravenous furosemide and then start him on an infusion b) Commence bisoprolol to control his heart rat c) Load him with aspirin and clopidogrel d) Obtain an urgent echocardiogram e) Prescribe oral furosemide and discharge from the emergency department with a view to follow up with his GP

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Administer a bolus of intravenous furosemide and then start him on an infusion

**>>REASONING**: The patient has acute decompensated heart failure with volume overload. Intravenous loop diuretics are needed to improve symptoms and volume status. An echocardiogram is not the most immediate next step. The ECG changes reflect an old infarct, and a beta-blocker is contraindicated in acute decompensated heart failure.

## Question #:26

**CLINICAL SCENERIO**: A 70-year-old with a history of myocardial infarction presents with on-off palpitations and occasional fainting episodes. He is on secondary preventive medication, and his examination and observation are normal. An ECG reveals a trifascicular block.

**QUESTION LINE**: What is the most appropriate management plan for the patient?

**OPTIONS**: 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-CHOICE LINE**: Correct answer is b.

**REASONING**: The patient’s ECG shows trifascicular block with RBBB, left anterior hemiblock, and 1st-degree heart block. This, along with a history of presyncope and syncope, is concerning and indicates admission and pacemaker workup due to the risk of further arrhythmias or cardiac blocks. Symptomatic trifascicular block is an indication for a pacemaker. Reducing the beta-blocker dose won’t remove the risk, and a Holter monitor is unnecessary given the ECG findings.

**>>DESCRIPTION**: 70-year-old with prior MI presents with palpitations, occasional fainting. ECG shows trifascicular block.

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

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Admit for urgent pacemaker workup

**>>REASONING**: The ECG showing trifascicular block along with the patient’s history of syncope indicates the need for a pacemaker workup. Beta-blocker dose reduction is unlikely to resolve the risk, and a Holter monitor is insufficient.

## Question #:114

**CLINICAL SCENERIO**: A 59-year-old man with ischaemic heart disease and type 2 diabetes is 8 hours post right hemicolectomy for bowel malignancy. He is tachycardic with a pulse rate of 200bpm, blood pressure of 148/79mmHg, and oxygen saturations of 98% on 2L/min nasal oxygen. ECG shows a regular broad complex tachycardia with a monomorphic waveform at 200bpm. Labs are mostly unremarkable.

**QUESTION LINE**: What is the most appropriate initial management?

**OPTIONS**: - a) Magnesium sulphate 2g IV - b) Amiodarone 300mg IV6 - c) Synchronised DC shock - d) Metoprolol 5mg IV - e) Adenosine 6mg IV5

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: 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.

**>>DESCRIPTION**: 59M, ischemic heart disease, DM, 8hr post hemicolectomy, tachycardic (200bpm), BP 148/79, SpO2 98% on 2L O2. ECG: regular broad complex tachycardia (200bpm). Labs unremarkable.

**>>OPTIONS**: a) Adenosine 6mg IV5 b) Amiodarone 300mg IV6 c) Magnesium sulphate 2g IV d) Metoprolol 5mg IV e) Synchronised DC shock

**>>CORRECT-CHOICE LINE**: Correct answer is b.

**>>CORRECT-CHOICE\_TEXT**: Amiodarone 300mg IV6

**>>REASONING**: The patient presents with ventricular tachycardia. As per resuscitation guidelines, in the absence of shock, syncope, ischemia, or heart failure, amiodarone 300mg IV is the most appropriate initial management. DC cardioversion is reserved for unstable patients.

## Question #:10

**CLINICAL SCENERIO**: A 64-year-old man with a history of high cholesterol, managed with atorvastatin, presents with exertional chest pain. The pain resolves with rest. He is currently taking bisoprolol, but his symptoms are not well-controlled.

**QUESTION LINE**: What should be added next?

**OPTIONS**: a) Amlodipine b) Isosorbide mononitrate c) Ivabradine d) Nicorandil e) Verapamil

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: 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 betablocker.

**>>DESCRIPTION**: A 64-year-old man on atorvastatin and bisoprolol for high cholesterol and exertional angina, respectively, reports uncontrolled symptoms. What is the next appropriate addition to his treatment?

**>>OPTIONS**: a) Amlodipine b) Isosorbide mononitrate c) Ivabradine d) Nicorandil e) Verapamil

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Amlodipine

**>>REASONING**: Amlodipine, a dihydropyridine calcium channel blocker, should be added next as first-line treatment for stable angina that is not controlled with a beta-blocker like bisoprolol. Isosorbide mononitrate, ivabradine and nicorandil are third-line agents. Verapamil is contraindicated with a beta-blocker.

## Question #:80

**CLINICAL SCENERIO**: A 28-year-old pregnant lady at 37 weeks presented to the ED with sudden chest pain, worsened by inspiration, and progressive shortness of breath. She had a recent external cephalic version and resolved placenta praevia. Examination revealed a heart rate of 122bpm, respiratory rate 24/min, and oxygen saturations of 98% on air. Her condition rapidly deteriorated, with oxygen saturation dropping to 88%, blood pressure at 88/66mmHg, and oozing from venepuncture sites. Investigations revealed thrombocytopenia, prolonged INR/APTT, elevated D-dimer, and arterial blood gases showing pH 7.48, Pa02 5.9 kPa, and PaC02 2.2 kPa.

**QUESTION LINE**: What is the most likely diagnosis?

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

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: 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

**>>DESCRIPTION**: A 28-year-old pregnant woman at 37 weeks presents with sudden chest pain, dyspnea, and subsequent rapid deterioration. Examination reveals hypoxia, hypotension, and oozing from venepuncture sites. Labs show thrombocytopenia, coagulopathy, elevated D-dimer, and abnormal arterial blood gases.

**>>OPTIONS**: a) Amniotic fluid embolus b) Aortic dissection c) Peripartum cardiomyopathy d) Pulmonary embolus e) Septic shock

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Amniotic fluid embolus

**>>REASONING**: Pulmonary embolism is unlikely due to the presence of DIC and absence of DVT signs. Septic shock is less likely due to the lack of a clear infection source. Amniotic fluid embolus is the most likely diagnosis given the sudden onset of DIC and respiratory compromise in a pregnant patient.

## Question #:82

**CLINICAL SCENERIO**: A 28-year-old pregnant lady at 37 weeks presents with sudden chest pain, shortness of breath, and no cough or calf pain. Five days prior, she had an external cephalic version. Her history includes resolved placenta previa, smoking, and a maternal DVT history. Initial findings: HR 122, RR 24, SpO2 98%, T 37.6ºC, BP 112/72. She rapidly deteriorates: SpO2 88%, BP 88/66, RR 32, with blood oozing from venepuncture sites. Labs show low platelets, elevated INR/APTT, and high D-dimer. Chest X-ray and urinalysis are unremarkable; ECG shows sinus tachycardia and T wave inversion. ABG: pH 7.48, PaO2 5.9 kPa, PaCO2 2.2 kPa.

**QUESTION LINE**: What is the most likely diagnosis?

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

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: 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.

**>>DESCRIPTION**: A 37-week pregnant woman presents with acute chest pain, dyspnea, and rapid deterioration with hypoxia, hypotension, oozing from venipuncture sites, thrombocytopenia, coagulopathy, and elevated D-dimer. No signs of DVT or infection.

**>>OPTIONS**: a) Amniotic fluid embolus b) Aortic dissection c) Peripartum cardiomyopathy d) Pulmonary embolus e) Septic shock

**>>CORRECT-CHOICE LINE**: Correct answer is a.

**>>CORRECT-CHOICE\_TEXT**: Amniotic fluid embolus

**>>REASONING**: Amniotic fluid embolism is most likely due to DIC and the lack of evidence for DVT or sepsis.

## Question #:23

**CLINICAL SCENERIO**: A 46-year-old man presents with fever (38.9 o C), malaise, reduced appetite, intermittent sweating, and rigors for three weeks. He has no medical history or drug allergies. Observations: RR 24/min, SpO2 95% on room air, HR 105bpm, BP 101/62mmHg, GCS 15/15. Examination reveals a loud early diastolic murmur over the right second intercostal space and splinter hemorrhages in fingernails.

**QUESTION LINE**: What is the most appropriate antibiotic to start?

**OPTIONS**: a) Amoxicillin b) Benzylpenicillin c) Flucloxacillin d) Vancomycin + low-dose gentamicin 5. e) 6. Vancomycin + low-dose gentamicin + rifampicin

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: 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 + lowdose 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.

**>>DESCRIPTION**: A 46-year-old man with fever, malaise, sweating, and rigors presents with a new diastolic murmur and splinter hemorrhages. He has no significant history or allergies.

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

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Amoxicillin

**>>REASONING**: The patient’s presentation suggests native valve endocarditis, and IV amoxicillin is the appropriate empirical treatment. Benzylpenicillin is for Streptococcus, flucloxacillin for Staphylococcus, and vancomycin + gentamicin +/- rifampicin are for penicillin-allergic patients, MRSA, or prosthetic valve endocarditis, respectively.

## Question #:98

**CLINICAL SCENERIO**: 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 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 groundglass changes of the left ventricle. He has no chest pain or shortness of breath.

**QUESTION LINE**: What is the most likely explanation of these echocardiogram findings?

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

**CORRECT-CHOICE LINE**: The correct answer is amyloidosis.

**REASONING**: 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.

**>>DESCRIPTION**: A 54-year-old male with frothy urine, proteinuria, and mild kidney injury has a systolic murmur. Echocardiogram shows LVEF 55%, normal valves, LV thickness of 16mm, 2mm pericardial effusion, and ground-glass changes in the LV. No chest pain or shortness of breath.

**>>OPTIONS**: a) Amyloidosis b) Cor pulmonale c) Mitochondrial disease d) Myocarditis e) Type 2 diabetes mellitus

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Amyloidosis

**>>REASONING**: Amyloidosis is the most likely diagnosis given the proteinuria, cardiac findings (increased LV thickness, pericardial effusion, ground-glass appearance), and global speckled pattern on echocardiogram. Cor pulmonale is unlikely without respiratory disease, myocarditis doesn’t explain proteinuria, diabetes doesn’t explain echo changes and mitochondrial disease would not show ground-glass pattern.

## Question #:51

**CLINICAL SCENERIO**: A 55-year-old with previous rheumatic heart disease aged 32 presents with an 18month 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 |  |

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: - 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-CHOICE LINE**: Correct answer is e.

**REASONING**: 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.

**>>DESCRIPTION**: A 55-year-old with prior rheumatic heart disease presents with exertional dyspnea. Echo shows elevated pulmonary arterial pressures (77 mmHg). Cardiac catheterization reveals the following: Right atrium: 8 mmHg, 71% O2 sat; Right ventricle: 39/8 mmHg, 71% O2 sat; Pulmonary artery: 45/12 mmHg, 71% O2 sat; Capillary wedge: 20 mmHg, 93% O2 sat; Left ventricle: 165/11 mmHg, 93% O2 sat; Aorta: 90/58 mmHg.

**>>OPTIONS**: a) Aortic stenosis b) Aortic stenosis and mitral stenosis c) Aortic stenosis and pulmonary hypertension d) Aortic stenosis, mitral stenosis, pulmonary hypertension e) Mitral stenosis

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Aortic stenosis, mitral stenosis, pulmonary hypertension

**>>REASONING**: No oxygen saturation step-ups indicate no shunts. Elevated pulmonary arterial pressure suggests pulmonary hypertension. Aortic valve gradient >25mmHg indicates aortic stenosis. Mitral valve gradient of 9mmHg (wedge pressure - LVEDP) suggests mitral stenosis.

## Question #:134

**CLINICAL SCENERIO**: 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 0.7cm2 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.

**QUESTION LINE**: What is the most appropriate management plan?

**OPTIONS**: 1. Review in six months 2. Cardiac MRI 3. Exercise test 4. Aortic valve replacement 5. Transcatheter aortic valve implantation (TAVI)

**CORRECT-CHOICE LINE**: The correct answer is aortic valve replacement.

**REASONING**: 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.

**>>DESCRIPTION**: A 67-year-old woman with severe aortic stenosis (aortic valve 0.7cm2, mean pressure gradient 62mmHg, LVEF 43%) and normal pulmonary arterial pressure is seen in cardiology clinic. She is asymptomatic and has a normal chest X-ray (2 months prior). Past medical history includes hypothyroidism, osteoarthritis, and migraines. She is a non-smoker.

**>>OPTIONS**: 1. Aortic valve replacement 2. Cardiac MRI 3. Exercise test 4. Review in six months 5. Transcatheter aortic valve implantation (TAVI)

**>>CORRECT-CHOICE LINE**: 1

**>>CORRECT-CHOICE\_TEXT**: Aortic valve replacement

**>>REASONING**: The patient has asymptomatic severe aortic stenosis and an ejection fraction <50%, indicating a need for aortic valve replacement or TAVI. Exercise testing is indicated for EF >50%. Cardiac MRI is not helpful in this case. Review in six months is only appropriate with EF >50% and successful exercise testing.

## Question #:62

**CLINICAL SCENERIO**: A 78-year-old male with heart failure and reduced ejection fraction (32%) has frequent admissions. ECG shows sinus rhythm, HR 64, QRS 136 msec with LBBB. BP is 98/55 mmHg. Labs: Na 136, K 4.7, Urea 6.8, Creatinine 126. Medications: carvedilol 25 mg BD, enalapril 10 mg BD, bumetanide 2 mg BD, aspirin 75 mg, ivabradine 2.5 mg BD.

**QUESTION LINE**: What alteration to this gentleman’s management could potentially decrease this gentleman’s probability of being re-admitted?

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

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: 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.

**>>DESCRIPTION**: 78M, HFrEF (EF 32%), frequent admissions. ECG: sinus rhythm, HR 64, QRS 136ms, LBBB. BP 98/55. Na 136, K 4.7, Cr 126. Meds: carvedilol, enalapril, bumetanide, aspirin, ivabradine. What management change reduces readmission risk?

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

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Arrange cardiac resynchronisation therapy-pacemaker (CRT-P) implantation

**>>REASONING**: CRT-P is indicated for patients with QRS >130ms and LBBB to improve symptoms and outcomes. Ivabradine is contraindicated due to slow HR and spironolactone due to borderline hypotension and raised K.

## Question #:18

**CLINICAL SCENERIO**: A 35-year-old woman with a history of anxiety and depression presented with intermittent palpitations, breathlessness, and chest discomfort. She is 14 weeks pregnant and experiencing significant stress. ECG showed normal sinus rhythm, T wave inversion in V1 - V3. Later, she developed wide complex regular tachycardia with LBBB pattern, HR 150 bpm, with fusion and capture beats. She spontaneously cardioverted. Echocardiogram revealed a dilated right ventricle with depressed systolic function and apical aneurysm. Left ventricle was normal.

**QUESTION LINE**: What is the most likely cause of her presentation?

**OPTIONS**: - a) Aortic dissection - b) Takutsubo cardiomyopathy - c) Peripartum cardiomyopathy - d) Arrhythmogenic right ventricular cardiomyopathy - e) Brugada syndrome

**CORRECT-CHOICE LINE**: Correct Answer: d

**REASONING**: 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 pregnancyassociated 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 AVRC.

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 pregnancyrelated 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.

**>>DESCRIPTION**: A 35-year-old pregnant woman (14 weeks) with anxiety/depression presented with palpitations, breathlessness, and chest discomfort. ECG: T wave inversion in V1-V3. Later, wide complex tachycardia (LBBB, 150 bpm) with fusion/capture beats occurred, followed by spontaneous cardioversion. Echo: dilated RV with depressed function and apical aneurysm. LV normal.

**>>OPTIONS**: a) Aortic dissection b) Arrhythmogenic right ventricular cardiomyopathy c) Brugada syndrome d) Peripartum cardiomyopathy e) Takutsubo cardiomyopathy

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Arrhythmogenic right ventricular cardiomyopathy

**>>REASONING**: The most likely diagnosis is arrhythmogenic right ventricular cardiomyopathy (ARVC) due to T wave inversion in V1-V3, wide complex tachycardia originating from the right ventricle (LBBB morphology), and echocardiogram findings (dilated RV, apical aneurysm). Aortic dissection is less likely given the absence of typical presentation/echo findings. Takutsubo is less likely because of long history of symptoms, and echo findings. Peripartum cardiomyopathy is less likely due to normal LV systolic function and early second trimester presentation. Brugada syndrome is less likely because echocardiography is typically normal in Brugada.

## Question #:113

**CLINICAL SCENERIO**: A 68-year-old man presents with left-sided upper and lower limb weakness upon waking. Symptoms persist in hospital. Examination reveals homonymous hemianopia. No significant medical history or regular medications. CT head is unremarkable. ECG shows atrial fibrillation.

**QUESTION LINE**: How would you manage this patient’s anti-thrombotic medication?

**OPTIONS**: - 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-CHOICE LINE**: Correct answer is b.

**REASONING**: 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 CHA2 DS2 -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.

**>>DESCRIPTION**: 68-year-old male with left-sided weakness and homonymous hemianopia. CT is unremarkable but ECG shows atrial fibrillation.

**>>OPTIONS**: 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-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Aspirin 300mg once daily for 14 days, then apixaban for life

**>>REASONING**: The patient likely had a stroke secondary to atrial fibrillation (CHA2DS2-VASc score of 3). Initial treatment includes aspirin 300mg daily for 14 days, followed by lifelong apixaban to prevent further strokes. Apixaban alone is incorrect due to the risk of hemorrhagic transformation if started immediately. Clopidogrel alone is insufficient for stroke prevention in atrial fibrillation. Thrombolysis is not indicated due to symptom onset on waking.

## Question #:117

**CLINICAL SCENERIO**: A 72-year-old male with atrial fibrillation on flecainide and warfarin presents with syncope and palpitations. He is tachycardic (HR 215/min) with a regular narrow complex tachycardia on ECG.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: - 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-CHOICE LINE**: Correct answer is b.

**REASONING**: Flecainide can transform AF into flutter, and slows cardiac conduction, resulting in an atrial rate around 200/min which can conduct via the AV node at a 1:1 ratio. The regular rhythm on ECG makes atrial fibrillation and atrial flutter with variable conduction less likely. No specific risk factors for AVRT or AVNRT.

**>>DESCRIPTION**: 72M, AF on flecainide/warfarin, presents with syncope, palpitations, HR 215, regular narrow complex tachycardia on ECG.

**>>OPTIONS**: a) Atrial fibrillation b) Atrial flutter with 1:1 conduction c) Atrial flutter with variable conduction d) Atrio-ventricular nodal re-entry tachycardia (AVNRT) e) Atrio-ventricular re-entry tachycardia (AVRT)

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Atrial flutter with 1:1 conduction

**>>REASONING**: Flecainide increases the risk of atrial flutter with 1:1 conduction because it slows conduction, allowing a rate around 200/min to conduct through the AV node. The rhythm is regular, making AF and atrial flutter with variable conduction less likely. No specific risk factors for AVRT/AVNRT.

## Question #:100

**CLINICAL SCENERIO**: A 40-year-old male presents with dyspnoea on exertion, progressing over a year to limiting him to 0.5 miles. He denies cough or chest pain, has a 25-year smoking history (15 cigarettes/day), and a family history of MI. BMI is 24 kg/m², HR 80/min, BP 130/77mmHg, RR 18/min, SpO2 97%. Examination reveals occasional expiratory wheeze, no pedal oedema, a fixed split S2, and normal JVP. Chest X-ray shows dilated pulmonary vessels, and ECG shows sinus rhythm with RBBB.

**QUESTION LINE**: What is the likely cause of his exertional dyspnoea?

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

**CORRECT-CHOICE LINE**: Correcv answer is a.

**REASONING**: 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.

**>>DESCRIPTION**: A 40-year-old male presents with exertional dyspnoea, a smoking history, and a family history of MI. Examination reveals occasional expiratory wheeze, a fixed split S2, normal JVP, dilated pulmonary vessels on X-ray, and RBBB on ECG.

**>>OPTIONS**: a) Atrial septal defect b) Bronchial malignancy c) Chronic obstructive pulmonary disease d) Chronic pulmonary embolism e) Ischaemic heart disease

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Atrial septal defect

**>>REASONING**: Atrial septal defect is the likely cause, presenting later in life with RBBB and a fixed split S2. Symptoms appear as the right atrium dilates, decreasing cardiac efficiency. Other options are less likely given the absence of typical findings.

## Question #:108

**CLINICAL SCENERIO**: A 65-year-old gentleman with known cardiomyopathy presents with progressive exertional shortness of breath. An echocardiogram reveals a provoked left ventricular outflow gradient of 64mmHg. He has no peripheral oedema or chest signs.

**QUESTION LINE**: What would be the most appropriate medical therapy?

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

## e) Ace-inhibitor

**CORRECT-CHOICE LINE**: The correct answer is beta-blocker.

**REASONING**: 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.

**>>DESCRIPTION**: A 65-year-old with cardiomyopathy has progressive exertional dyspnea. Echo shows a left ventricular outflow gradient of 64mmHg. No peripheral edema or chest signs.

**>>OPTIONS**: a) Ace-inhibitor b) Beta-blocker c) Digoxin d) Nitrate e) Phosphodiesterase type 5 inhibitor

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Beta-blocker

**>>REASONING**: Beta-blockers are appropriate as they control heart rate, preventing ventricular outflow obstruction. Nitrates and ACE inhibitors are contraindicated due to the risk of hypotension. Phosphodiesterase type 5 inhibitors and digoxin offer no benefit.

## Question #:95

**CLINICAL SCENERIO**: A 72-year-old man with a history of NSTEMI and a drug-eluting stent presents to the cardiology clinic for routine follow-up. He reports stable angina symptoms (retro-sternal chest pain on exertion) despite being on aspirin, clopidogrel, atorvastatin, and ramipril. Examination is unremarkable, and blood pressure is 125/75 mmHg. Angiography from the previous year showed 90% stenosis in the LAD treated with a stent and 60% stenosis in the mid-RCA. Echocardiogram showed mild hypokinesis and an EF of 40-45%. ECG showed sinus rhythm with T-wave inversions in V3-V5. He uses sildenafil and nitrate spray as needed.

**QUESTION LINE**: What is most appropriate next line therapy for the patients chest pain?

**OPTIONS**: - a) Amlodipine - b) Bisoprolol - c) Percutaneous coronary intervention - d) Long-acting isosorbide mononitrate - e) Nicorandi

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: 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 show 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 stunting in stable angina? BMJ 2016;352:i205.

**>>DESCRIPTION**: 72-year-old man with prior NSTEMI and stent presents with stable angina. He is on aspirin, clopidogrel, atorvastatin, and ramipril. Exam is normal, BP 125/75. Angiography showed LAD stent and 60% RCA stenosis. EF is 40-45%. He uses sildenafil and nitrate spray PRN.

**>>OPTIONS**: a) Amlodipine b) Bisoprolol c) Long-acting isosorbide mononitrate d) Nicorandi e) Percutaneous coronary intervention

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Bisoprolol

**>>REASONING**: Bisoprolol (a beta-blocker) is the preferred next-line therapy for stable angina in this patient with controlled blood pressure. Amlodipine is also reasonable but beta-blocker preferred. ISMN and nicorandil are contraindicated with sildenafil. PCI is not first-line for stable angina.

## Question #:103

**CLINICAL SCENERIO**: A 83 year old lady with a history of NSTEMI and gallstones presents to heart failure follow up clinic. She is able to walk around her bungalow, but struggles to walk to her local shops 100m away due to breathlessness. She is on senna, ramipril 10mg, aspirin 75m, frusemide 40mg bd, simvastatin 40mg, and spironolactone 50mg. Her latest echo reveals an ejection fraction of 25%. Her ECG is sinus rhythm Her last BNP was 1000 pg/ml. Her observations at clinic are: oxygen saturations: 94% on room air, blood pressure: 126/66 mmHg, heart rate: 84/min

**QUESTION LINE**: Which additional medication would be beneficial?

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

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: This patient is already taking some prognostically beneficial medications; ACE inhibitor and aldosterone antagonist. The additional 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.

**>>DESCRIPTION**: 83-year-old female with NSTEMI and gallstones presents with poorly controlled heart failure (EF 25%, BNP 1000 pg/ml, HR 84/min). Medications include ramipril, aspirin, frusemide, simvastatin, and spironolactone. O2 sat is 94% on room air, BP is 126/66 mmHg. She experiences breathlessness with minimal exertion.

**>>OPTIONS**: a) Atenolol b) Bisoprolol c) Diltiazem d) Digoxin e) Ivabradine

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Bisoprolol

**>>REASONING**: Bisoprolol (beta blocker) is beneficial for heart failure due to its role in preventing remodeling and reducing heart rate. Ivabradine is inappropriate as the patient is not on maximal beta-blocker therapy. Diltiazem is for angina, which the patient doesn’t have. Digoxin is a later-line therapy.

## Question #:110

**CLINICAL SCENERIO**: A 22-year-old man, recently granted asylum in the UK after fleeing Syria, presents with 3 months of fevers, night sweats, and fatigue. He reports reduced exercise tolerance and breathlessness. He has a history of inadequate food/shelter and consumption of unpasteurized sheep’s milk. Examination reveals diaphoresis with malodorous sweat, splinter hemorrhages, elevated JVP, pitting edema, a harsh systolic murmur over the aortic area, basal crackles, and palpable spleen. Investigations show lymphocytosis, thrombocytopenia, elevated ESR and CRP, hematuria, and CXR findings of blood diversion, airspace shadowing and pleural effusions. Echocardiogram shows aortic valve vegetation, moderate-severe aortic stenosis, and ejection fraction of 30%.

**QUESTION LINE**: Which of the following organisms is the most likely cause of the patient’s presentation?

**OPTIONS**: - a) Clostridium burnetti - b) Bartonella quintana - c) Haemophilus parainfluenzae - d) Kingella kingae - e) Brucella melitensis

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: The patient’s presentation, including aortic valve endocarditis, history of consuming unpasteurized milk products, contact with livestock from an endemic region, and malodorous sweat, strongly suggests Brucella melitensis as the causative organism. Lymphocytosis and thrombocytopenia are also typical findings in Brucellosis. While the other options are rare causes of endocarditis, there is nothing in the presentation to specifically suggest them.

**>>DESCRIPTION**: A 22-year-old Syrian refugee presents with fever, night sweats, fatigue, dyspnea, and signs of endocarditis (splinter hemorrhages, aortic murmur). History includes unpasteurized sheep’s milk consumption. Examination shows malodorous sweat. Labs show lymphocytosis and thrombocytopenia. Echo reveals aortic valve vegetation. What is the most likely organism?

**>>OPTIONS**: a) Bartonella quintana b) Brucella melitensis c) Clostridium burnetti d) Haemophilus parainfluenzae e) Kingella kingae

**>>CORRECT-CHOICE LINE**: Correct answer is b.

**>>CORRECT-CHOICE\_TEXT**: Brucella melitensis

**>>REASONING**: Brucella melitensis is the most likely cause given the patient’s history of unpasteurized milk consumption, signs of endocarditis, malodorous sweat, lymphocytosis and thrombocytopenia. The other options are less likely without specific supporting evidence.

## Question #:112

**CLINICAL SCENERIO**: 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:

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: - a) Hypertrophic obstructive cardiomyopathy - b) Anterior myocardial infarction - c) Long QT syndrome type - d) Arrhythmogenic right ventricular dysplasia - e) Brugada syndrome

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: The ECG shows a Brugada pattern, most marked in V1, which has an incomplete RBBB, a downsloping ST segment and an inverted T wave.

**>>DESCRIPTION**: A 30-year-old man collapses at work with no prior history. Examination shows BP 102/68 mmHg, pulse 88/min, SpO2 99%, RR 16/min. ECG is taken.

**>>OPTIONS**: a) Anterior myocardial infarction b) Arrhythmogenic right ventricular dysplasia c) Brugada syndrome d) Hypertrophic obstructive cardiomyopathy e) Long QT syndrome type

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Brugada syndrome

**>>REASONING**: The ECG shows a Brugada pattern with incomplete RBBB, downsloping ST segment, and inverted T wave in V1.

## Question #:54

**CLINICAL SCENERIO**: A 45-year-old male with typical angina symptoms, no smoking history, and no past medical history experiences chest pain in the center of his chest, exacerbated by exertion. According to NICE guidelines, what is the most appropriate initial investigation?

**QUESTION LINE**: According to the NICE guidelines, which is the most appropriate investigation?

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

**CORRECT-CHOICE LINE**: The correct answer is CT-coronary angiography .

**REASONING**: 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 firstline 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.

**>>DESCRIPTION**: A 45-year-old male with typical angina, no smoking history, and no prior medical issues presents with exertion-related chest pain. What is the most appropriate initial investigation according to NICE guidelines?

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

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: CT-coronary angiography

**>>REASONING**: NICE guidelines recommend CT coronary angiography as the first-line investigation for patients with typical or atypical angina and intermediate risk. Treadmill ECG is less accurate, echocardiograms don’t directly visualize coronary arteries, nuclear perfusion stress tests expose patients to radiation, and diagnostic coronary angiography is invasive and reserved for inconclusive cases.

## Question #:89

**CLINICAL SCENERIO**: An 81-year-old man with a history of multiple myeloma, treated with melphalan, presents to the cardiology clinic with worsening heart failure symptoms including shortness of breath, ankle swelling, and elevated JVP. He is already taking bisoprolol, ramipril, and atorvastatin. Examination reveals crackles in both lung bases and pitting oedema to his knees. ECG shows low voltage QRS complexes.

**QUESTION LINE**: An ECG accompanies the referral:

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

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: 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 asssociated with poor R wave progression in the chest leads (a pseudoinfarction pattern).

**>>DESCRIPTION**: 81-year-old man with multiple myeloma on melphalan presents with worsening heart failure. Examination shows elevated JVP, crackles, and pitting edema. ECG shows low voltage QRS complexes.

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

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Cardiac amyloidosis

**>>REASONING**: The patient’s history of myeloma, coupled with heart failure symptoms and low voltage QRS complexes, points towards cardiac amyloidosis secondary to AL amyloidosis. Other options are less likely given the absence of specific findings related to those conditions.

## Question #:40

**CLINICAL SCENERIO**: A 34-year-old male collapsed after moderate alcohol intoxication. He reports weakness and lightheadedness with a history of palpitations and shortness of breath. Examination reveals a fast, thready pulse (130-150bpm) and hypotension (89/65mmHg). Heart sounds are normal.

**QUESTION LINE**: What is the most appropriate management?

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

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: This patient presents with a tachyarrhythmia and hemodynamic instability (hypotension, possible syncope/shock). The fast, irregular rhythm with narrow QRS complexes suggests atrial fibrillation or another tachyarrhythmia. Urgent synchronized cardioversion is required for any unstable patient with a tachyarrhythmia.

Adenosine is for stable SVT patients. Metoprolol is for rate control in stable tachyarrhythmias. Pacemaker insertion is for bradycardia. Percutaneous coronary intervention is for MI, which is unlikely in this case given the lack of chest pain or ST-elevation.

**>>DESCRIPTION**: A 34-year-old male collapsed after moderate alcohol intoxication, presenting with weakness, lightheadedness, palpitations, a pulse of 130-150bpm, and BP of 89/65mmHg.

**>>OPTIONS**: a) Cardioversion b) IV adenosine c) IV metoprolol d) Pacemaker insertion e) Percutaneous coronary intervention

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Cardioversion

**>>REASONING**: The patient is hemodynamically unstable with a tachyarrhythmia, necessitating urgent cardioversion. Adenosine and metoprolol are for stable patients. A pacemaker is for bradycardia, and PCI is for MI, which is less likely given the presentation.

## Question #:29

**CLINICAL SCENERIO**: A 36-year-old woman, 38 weeks pregnant with her first child, presents with a 2-day history of pleuritic right-sided chest pain and shortness of breath. Examination reveals slight tachypnoea, no added chest sounds, and oxygen saturation of 95% on room air. Heart rate is 98/min. No clinical evidence of DVT.

**QUESTION LINE**: What is the next appropriate investigation?

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

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: 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 .

**>>DESCRIPTION**: A 36-year-old woman, 38 weeks pregnant, presents with 2-day history of pleuritic right-sided chest pain and shortness of breath. Exam: slight tachypnoea, clear chest, SpO2 95% RA, HR 98. No DVT signs.

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

**>>CORRECT-CHOICE LINE**: Correct answer is c.

**>>CORRECT-CHOICE\_TEXT**: Chest x-ray

**>>REASONING**: Initial investigation for suspected PE in pregnant women is a chest x-ray. If negative, proceed to leg Dopplers. CTPA or V/Q are considered if suspicion remains high after these initial tests. D-dimer is unreliable in pregnancy.

## Question #:83

**CLINICAL SCENERIO**: A 77-year-old man presents to the ED with a purple lacy rash on his arms and legs for a few days. He has a history of hypertension, type 2 diabetes, diverticulitis, and atrial fibrillation. Medications include amlodipine, ramipril, metformin, and warfarin (started one week ago). Examination reveals a widespread purple mottled rash on all four limbs and the abdomen. Chest, heart, and abdomen are normal. Blood results are provided.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: - a) Amyloidosi - b) Antiphospholipid syndrome - c) Cholesterol atheroemboli - d) Churg-Strauss syndrome - e) Focal segmental glomerulosclerosis

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: 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 a recognised precipitant of cholesterol atheroemboli which makes it the most likely diagnosis here.

**>>DESCRIPTION**: 77M presents with a purple, lacy rash on limbs and abdomen, appearing over days. PMH: HTN, T2DM, diverticulitis, AF. Meds: amlodipine, ramipril, metformin, warfarin (started 1 week prior). Exam: widespread mottled rash. Labs provided.

**>>OPTIONS**: a) Amyloidosi b) Antiphospholipid syndrome c) Churg-Strauss syndrome d) Cholesterol atheroemboli e) Focal segmental glomerulosclerosis

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Cholesterol atheroemboli

**>>REASONING**: Livedo reticularis can be caused by antiphospholipid syndrome, Churg-Strauss syndrome, and cholesterol atheroemboli. While Churg-Strauss and cholesterol atheroemboli can cause elevated eosinophils, recent warfarin initiation points towards cholesterol atheroemboli as the most likely diagnosis.

## Question #:119

**CLINICAL SCENERIO**: A 65-year-old man with hypertension and diet-controlled diabetes is admitted with central chest pain, radiating to the left arm, associated with nausea and vomiting. ECG shows T wave inversion in leads I, V4, V5, and V6. He is diagnosed with NSTEMI and treated. Post-angiogram, he develops a rash on his legs, reduced urine output, and bluish lacey discolouration. Repeat bloods show deranged renal function.

**QUESTION LINE**: What is the most likely cause of his symptoms and deranged blood tests?

**OPTIONS**: - a) Cholesterol embolism - b) Contrast nephropathy - c) Endocarditis - d) Hypovolaemia during NSTEMI - e) Newly started ACE inhibitor

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: This gentleman as developed an acute kidney injury following angiography. The combination of a lacey 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.

**>>DESCRIPTION**: A 65-year-old man post-NSTEMI and angiogram presents with a rash, reduced urine output, bluish leg discoloration, and AKI.

**>>OPTIONS**: a) Cholesterol embolism b) Contrast nephropathy c) Endocarditis d) Hypovolaemia during NSTEMI e) Newly started ACE inhibitor

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Cholesterol embolism

**>>REASONING**: The lacey rash (livedo reticularis) and raised eosinophils following angiography point to cholesterol embolism. Contrast nephropathy lacks the rash and eosinophilia. ACE inhibitors and hypovolemia are less likely given the rapid onset and uncomplicated angiogram, respectively. Endocarditis is unlikely without fever or systemic signs.

## Question #:106

**CLINICAL SCENERIO**: A 73-year-old man presents with a 3-month history of thoracic back pain and recent night sweats/shivers. Blood tests show anemia (Hb 87 g/L), elevated white cells (11.6x10^9/L), low MCV (70 fl), and low MCH (20 pg). Blood cultures grow Streptococcus gallolyticus. MRI shows discitis at T8/9. ECHO shows no vegetation.

**QUESTION LINE**: What is the next investigation for this patient?

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

**CORRECT-CHOICE LINE**: Correct anmswer is a.

**REASONING**: 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.

**>>DESCRIPTION**: 73-year-old man with thoracic back pain, night sweats/shivers. Labs: anemia, elevated WBCs, low MCV/MCH. Blood cultures: Streptococcus gallolyticus. MRI: discitis T8/9. ECHO: no vegetation.

**>>OPTIONS**: a) Colonoscopy b) CT chest/abdomen/pelvis c) PET scan d) Repeat blood cultures e) Ultrasound abdomen/pelvi

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Colonoscopy

**>>REASONING**: Streptococcus gallolyticus bacteremia is associated with a significant risk of underlying colonic malignancy. Colonoscopy is the gold standard for investigating potential colorectal cancer. CT chest/abdomen/pelvis, ultrasound, and PET scan are less sensitive or not first-line. Repeat blood cultures do not aid in diagnosing the underlying malignancy.

## Question #:63

**CLINICAL SCENERIO**: 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 preeclampsia 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.

**QUESTION LINE**: What is the next best management step?

**OPTIONS**: - 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-CHOICE LINE**: Correct answer is b.

**REASONING**: 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.

**>>DESCRIPTION**: A 38-year-old, 28-week pregnant woman has a blood pressure of 158/98 mmHg on routine check. She has hyperemesis gravidarum but otherwise uncomplicated pregnancy. No headaches, vision changes, abdominal pain, seizures, or bleeding. Fetal movements normal. Antenatal booking BP was 148/88 mmHg. Sister had preeclampsia. Examination is unremarkable except for elevated BP.

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

**>>CORRECT-CHOICE LINE**: Correct answer is b.

**>>CORRECT-CHOICE\_TEXT**: Commence labetalol

**>>REASONING**: The patient has pre-existing hypertension. Labetalol is the safest antihypertensive in pregnancy. ACE inhibitors are contraindicated. No clinical features of preeclampsia, so no need for HDU or magnesium sulfate.

## Question #:50

**CLINICAL SCENERIO**: A 54-year-old male previously treated for lymphoma presents with progressive exertional dyspnoea for 6 months. 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. He takes no medications 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.

**QUESTION LINE**: What is the likely cause of his symptoms?

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

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: 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.

**>>DESCRIPTION**: A 54-year-old male, previously treated for lymphoma, presents with progressive exertional dyspnoea. Examination reveals raised JVP on inspiration (Kussmaul’s sign) and an extra heart sound soon after S2.

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

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Constrictive pericarditis

**>>REASONING**: Kussmaul’s sign (paradoxical elevation of JVP on inspiration) points to constrictive pericarditis, cardiac tamponade, or restrictive cardiomyopathy. Given the history of lymphoma and radiotherapy, constrictive pericarditis is the most likely cause. The presentation lacks features suggestive of tamponade.

## Question #:58

**CLINICAL SCENERIO**: 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

**QUESTION LINE**: What is the most appropriate course of action?

**OPTIONS**: - 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-CHOICE LINE**: Correct answer is b.

**REASONING**: 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 postreperfusion 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.

**>>DESCRIPTION**: A 63-year-old female with inferior STEMI (ST elevation in leads II, III, aVF) underwent primary PCI. Six hours post-PCI, she develops complete heart block but remains asymptomatic and haemodynamically stable (Pulse 44bpm, BP 123/75mmHg).

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

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Continue close monitoring and observation of the patient

**>>REASONING**: Complete heart block post-inferior MI often resolves without intervention. Given the patient is asymptomatic and haemodynamically stable, close monitoring is appropriate. Temporary pacing would be considered if the patient were unstable.

## Question #:35

**CLINICAL SCENERIO**: A 52-year-old female on ramipril for hypertension presents with controlled BP (132/84mmHg) but elevated creatinine (125umol/L, baseline 100umol/L).

**QUESTION LINE**: What is the most appropriate step in the management of her hypertension?

**OPTIONS**: - 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-CHOICE LINE**: Correct answer is b.

**REASONING**: 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 greatre 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

**>>DESCRIPTION**: A 52-year-old female on ramipril presents with controlled BP but creatinine increased by 25%.

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

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Continue current dose of ramipril

**>>REASONING**: Creatinine increased by 25%, which is within acceptable limits as per BNF guidelines (30% increase or 25% eGFR fall required to stop ACEi). BP is well controlled, so continuing current dose is appropriate. Other options are not indicated based on the creatinine level and BP control.

## Question #:118

**CLINICAL SCENERIO**: A 28-year-old woman at 10 weeks gestation presented with hyperemesis gravidarum and a history of intravenous drug use, now on methadone treatment. An ECG showed a prolonged corrected QT interval of 490 ms. Her QT interval was 430ms three weeks earlier. She takes methadone and ondansetron.

**QUESTION LINE**: With regard to the patient’s prolonged corrected QT interval, what is the most appropriate strategy for managing her medications?

**OPTIONS**: - 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-CHOICE LINE**: Correct answer is d.

**REASONING**: The patient has a prolonged QT interval (490 ms) and is taking both methadone and ondansetron, which can prolong the QT interval. Discontinuing ondansetron is the most appropriate step, given the importance of methadone for opiate replacement therapy and the availability of alternative anti-emetics.

**>>DESCRIPTION**: A 28-year-old woman, 10 weeks pregnant, presents with hyperemesis gravidarum. ECG shows prolonged QT interval (490 ms) while on methadone and ondansetron.

**>>OPTIONS**: a) Continue both methadone and ondansetron and admit for cardiac monitoring b) Continue both methadone and ondansetron and arrange outpatient ambulatory ECG c) Continue methadone, stop ondansetron d) Continue ondansetron, stop methadone e) Stop both methadone and ondansetron

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Continue methadone, stop ondansetron

**>>REASONING**: The patient’s prolonged QT interval is likely due to the combination of methadone and ondansetron. Continuing methadone is important for her long-term health, therefore, stopping ondansetron is the best option. The other options involve continuing both drugs or stopping methadone, which are less appropriate.

## Question #:84

**CLINICAL SCENERIO**: A 66-year-old gentleman post-myocardial infarction is concerned about starting atorvastatin due to relatives’ negative experiences with simvastatin (muscle pain, disturbed sleep). He has no family history of early hypercholesterolaemia, but several relatives use statins after cardiac events.

**QUESTION LINE**: What options exist with regards to his secondary prevention?

**OPTIONS**: - 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-CHOICE LINE**: Correct answer is b.

**REASONING**: Statins are the primary lipid-regulating drugs for secondary prevention of cardiovascular disease (except ezetimibe in primary hypercholesterolaemia). NICE guidelines currently approve only statins for secondary prevention. Other listed drugs are not licensed for this purpose, except ezetimibe in primary hypercholesterolaemia. There is no indication of primary hypercholesterolaemia in this patient. Assuming the patient understands the lack of alternatives, continuing the current atorvastatin dose is the most appropriate action. Dose reduction or switching to pravastatin could be considered if a specific dose was provided.

**>>DESCRIPTION**: A 66-year-old man, post-MI, is concerned about atorvastatin due to family history of statin intolerance, but has no personal history of early hypercholesterolemia. What are his secondary prevention options?

**>>OPTIONS**: a) Continue with atorvastatin at the current dose b) Ezetimibe 10 mg OD c) Fenofibrate 160 mg OD d) Lomitapide 5 mg OD e) Niacin (as per product literature)

**>>CORRECT-CHOICE LINE**: Correct answer is a.

**>>CORRECT-CHOICE\_TEXT**: Continue with atorvastatin at the current dose

**>>REASONING**: Statins are the primary and NICE-approved treatment for secondary prevention of CVD. Other options like fenofibrate, lomitapide and niacin are not indicated, and ezetimibe is only used in primary hypercholesterolemia. Thus, continuing atorvastatin is the best option.

## Question #:16

**CLINICAL SCENERIO**: A 78-year-old man with hypothyroidism presents with syncope. Examination reveals a loud systolic murmur radiating to the carotid, a slow rising pulse, and an aortic valve area of 0.8mm² with a pressure gradient of 42mmHg. Chest X-ray shows an enlarged heart with calcification of the aortic knuckle.

**QUESTION LINE**: What other investigation should be performed?

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

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: 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 is 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 is can cause syncope.

**>>DESCRIPTION**: A 78-year-old man with hypothyroidism presents with syncope. He has a systolic murmur radiating to the carotid, slow rising pulse, and severe aortic stenosis (0.8mm², 42mmHg gradient). Chest X-ray shows an enlarged heart.

**>>OPTIONS**: a) CT chest b) Coronary angiography c) HbA1c d) Thyroid function test e) Treadmill ECG testing

**>>CORRECT-CHOICE LINE**: Correct answer is b.

**>>CORRECT-CHOICE\_TEXT**: Coronary angiography

**>>REASONING**: Given the co-occurrence of aortic stenosis with atherosclerotic disease, coronary angiography should be performed before aortic valve replacement to assess for coronary artery disease. CT chest is for respiratory disease. HbA1c and TFTs are not indicated without specific suspicion. Treadmill testing is contraindicated in symptomatic severe aortic stenosis.

## Question #:99

**CLINICAL SCENERIO**: A 75-year-old male with a history of stable angina, hypercholesterolaemia, and hypertension presents with increasing chest pain on exertion. He is taking bisoprolol and nifedipine. An angiogram shows stenoses in his left circumflex artery, distal right coronary artery, and mid-left anterior descending artery.

**QUESTION LINE**: What is the most appropriate long-term treatment of choice for his angina?

**OPTIONS**: - 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-CHOICE LINE**: Correct answer is a.

**REASONING**: NICE guidelines recommend using no more than 2 antianginals before considering reperfusion therapies. For this patient, CABG is favored over PCI because patients with complex anatomy, triple vessel disease, or proximal left mainstem disease report better long-term survival and freedom from MI with CABG, according to AHA and ESC guidelines.

**>>DESCRIPTION**: 75-year-old male with stable angina, hypercholesterolemia, and hypertension presents with increasing chest pain on exertion while on bisoprolol and nifedipine. Angiogram shows stenoses in left circumflex, distal right coronary, and mid-left anterior descending arteries.

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

**>>CORRECT-CHOICE LINE**: Correct answer is d.

**>>CORRECT-CHOICE\_TEXT**: Coronary artery bypass graft

**>>REASONING**: CABG is favored over PCI due to the patient’s triple vessel disease. Studies show better long-term survival and less MI with CABG in such cases. NICE guidelines suggest reperfusion therapies after failure of two antianginals.

## Question #:104

**CLINICAL SCENERIO**: A 79-year-old man with a history of ischaemic heart disease and prior CABG presents with severe central chest pain starting 90 minutes ago. He is clammy and vomiting. An ECG is taken.

**QUESTION LINE**: What is the most accurate description of what is shown on this ECG?

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

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: 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 >=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

**>>DESCRIPTION**: A 79-year-old man with ischemic heart disease and prior CABG presents with severe chest pain that started 90 minutes ago. He is clammy and vomiting. An ECG was performed.

**>>OPTIONS**: a) Current anterior NSTEMI b) Current anterior STEMI c) Current inferior NSTEMI d) Current inferior STEMI e) Normal ECG

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Current anterior STEMI

**>>REASONING**: ST elevation in the anterior leads (V2-4) indicates a STEMI. The angiogram confirmed occlusion of a saphenous vein graft to the left anterior descending artery. The inferior leads show changes suggestive of a prior MI, not a current inferior STEMI/NSTEMI.

## Question #:91

**CLINICAL SCENERIO**: An 84-year-old male admitted to CCU post PPCI for acute STEMI developed nausea and vomiting treated with cyclizine. Shortly after, he became increasingly short of breath with sinus tachycardia, respiratory distress, bibasal crackles, and elevated JVP. Observations: BP 186/80 mmHg, HR 120 bpm, Sats 92% on 3L, RR 33/min. ECG shows no new ischaemic changes; chest X-ray reveals bibasal alveolar oedema and bilateral small pleural effusions.

**QUESTION LINE**: What is the cause of the patient’s deterioration?

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

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: The patient developed acute pulmonary oedema, systemic hypertension, and tachycardia after cyclizine administration post-STEMI, indicative of cyclizine-induced heart failure. Cyclizine, an H1 receptor antagonist and anticholinergic, can cause hypertension and tachycardia, potentially worsening a fragile myocardium. Lack of chest pain or ST elevation makes in-stent thrombosis or further ACS unlikely. Hypertension and absence of low voltage ECG make cardiac tamponade unlikely.

**>>DESCRIPTION**: 84M post PPCI for STEMI develops dyspnea, tachycardia, hypertension, and pulmonary edema after cyclizine for nausea. ECG: no new ischemia. CXR: pulmonary edema. What’s the cause?

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

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Cyclizine induced heart failure

**>>REASONING**: Cyclizine (H1 antagonist/anticholinergic) caused hypertension and tachycardia, leading to pulmonary edema in a post-MI patient. No chest pain or ST elevation makes ACS/in-stent thrombosis unlikely. Hypertension argues against tamponade.

## Question #:57

**CLINICAL SCENERIO**: A 54-year-old man with recurrent syncope attends. Respiratory rate 18, SpO2 98%, heart rate 75, BP 114/67 mmHg. ECG shows non-conducted p-waves.

**QUESTION LINE**: What would be the most appropriate intervention?

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

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: 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 d ual 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 nonactivity, 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 stopgap 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.

**>>DESCRIPTION**: A 54-year-old man presents with recurrent syncope. ECG shows non-conducted p-waves, indicating Mobitz type II AV block.

**>>OPTIONS**: a) AAI pacemaker b) Ablation c) DDD pacemaker d) Temporary pacing e) VVI pacemaker

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: DDD pacemaker

**>>REASONING**: DDD pacemaker is indicated for Mobitz type II AV block to sense and pace both atria and ventricles. VVI and AAI pacemakers pace only ventricles or atria, respectively, and are not appropriate. Temporary pacing is for unstable patients. Ablation addresses aberrant conduction, not non-conduction.

## Question #:87

**CLINICAL SCENERIO**: A 28-year-old man presented with heart palpitations. He states that he has had a 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

**QUESTION LINE**: What is the most likely explanation of the ECG results?

**OPTIONS**: - 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-CHOICE LINE**: Correct answer is d.

**REASONING**: 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 WolffParkinson-White (WPW) unlikely.

The QTc is normal making Torsades de pointes unlikely.

**>>DESCRIPTION**: A 28-year-old man with heart palpitations for 3 months presents the following ECG findings: Inverted P wave in lead I, PR interval 130ms, QRS 110ms with loss of R wave progression, QTc 410ms, and right axis deviation.

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

**>>CORRECT-CHOICE LINE**: Correct answer is b.

**>>CORRECT-CHOICE\_TEXT**: Dextrocardia

**>>REASONING**: Dextrocardia is the most likely explanation given the inverted P wave in lead I, right axis deviation, and loss of R wave progression. While lead misplacement can mimic some findings, it wouldn’t typically cause loss of R wave progression. WPW is unlikely given normal PR interval and absence of delta waves. Torsades is unlikely given normal QTc.

## Question #:37

**CLINICAL SCENERIO**: An 84-year-old man with a blood pressure reading of 150/92 mmHg underwent ambulatory blood pressure monitoring (ABPM) and a standard hypertension work-up. His 10-year cardiovascular risk was not calculated due to his age. Lab results: 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. ECG showed sinus rhythm, 72 bpm and first degree heart block.

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

**OPTIONS**: - 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

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: Stage 1 hypertension is defined by an ABPM reading of >= 135/85 mmHg, with stage 2 hypertension having a cut-off of >= 150/95 mmHg.

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

**>>DESCRIPTION**: An 84-year-old man has an ABPM daytime average of 145/80 mmHg. Relevant labs and ECG are unremarkable except for first degree heart block.

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

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Diagnose stage 1 hypertension and advise about lifestyle changes

**>>REASONING**: The patient’s ABPM indicates stage 1 hypertension (145/80 mmHg). Given his age (>80 years), lifestyle advice is the most appropriate initial step.

## Question #:76

**CLINICAL SCENERIO**: A 77-year-old woman with a history of congestive cardiac failure, atrial fibrillation, hypertension, and depression is on bisoprolol, digoxin, amlodipine, furosemide, and citalopram. Digoxin level is 1.1 ng/mL (0.7-2.0).

**QUESTION LINE**: Which agent is responsible for the abnormalities displayed in her ECG?

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

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: 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.

**>>DESCRIPTION**: A 77-year-old woman with CHF, atrial fibrillation, hypertension, and depression is taking bisoprolol, digoxin, amlodipine, furosemide, and citalopram. Digoxin level: 1.1 ng/mL.

**>>OPTIONS**: a) Amlodipine b) Bisoprolol c) Citalopram d) Digoxin e) Furosemide

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Digoxin

**>>REASONING**: The ‘scooped’ ST depression on the ECG is a classic sign of the digoxin effect. Amlodipine and bisoprolol do not cause these specific ST changes. Citalopram can cause QT prolongation, and furosemide can cause flattened/inverted T waves due to hypokalemia, but neither explains the scooped ST depression.

## Question #:11

**CLINICAL SCENERIO**: An 89-year-old gentleman with a history of atrial fibrillation (AF), ischaemic heart disease, a myocardial infarction five years ago and type 2 diabetes mellitus, presented with palpitations despite being on bisoprolol. He has declined anticoagulation. His heart rate is 94/min and irregular. His chest is clear on auscultation and there is no peripheral oedema.

**QUESTION LINE**: How should he be further managed?

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

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: 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 ratecontrol 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.

**>>DESCRIPTION**: An 89-year-old with AF, IHD, prior MI, and DM presents with palpitations despite bisoprolol. He declined anticoagulation. HR 94/min, irregular; chest clear, no edema.

**>>OPTIONS**: a) Amiodarone b) Amlodipine c) Diltiazem d) Dronedarone e) Left atrial ablation

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Diltiazem

**>>REASONING**: Diltiazem is correct as the patient has symptomatic AF not responding to bisoprolol. NICE guidelines suggest either a beta-blocker or a rate-limiting calcium channel blocker. Amiodarone, dronedarone, and ablation are for cardioversion. Amlodipine, while a calcium channel blocker, is not rate-limiting.

## Question #:14

**CLINICAL SCENERIO**: A 54-year-old male with asthma, ischemic heart disease, and TIA presents with palpitations. ECG shows regular, narrow complex tachycardia. Vagal maneuvers failed. IV adenosine 6mg was administered, leading to an 11-second ventricular standstill before sinus rhythm returned.

**QUESTION LINE**: Which of the patient’s medications is most likely to be responsible for this?

**OPTIONS**: a) Phyllocontin b) Dipyridamole c) Bisoprolol d) Simvastatin e) Montelukast

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: 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.

**>>DESCRIPTION**: 54-year-old male with asthma, ischemic heart disease, and TIA presents with palpitations and narrow complex tachycardia. Adenosine 6mg IV caused an 11-second ventricular standstill.

**>>OPTIONS**: a) Bisoprolol b) Dipyridamole c) Montelukast d) Phyllocontin e) Simvastatin

**>>CORRECT-CHOICE LINE**: Correct answer is b.

**>>CORRECT-CHOICE\_TEXT**: Dipyridamole

**>>REASONING**: Dipyridamole potentiates adenosine by blocking its cellular uptake, increasing its concentration at receptors. Theophyllines antagonize adenosine, requiring higher doses. Asthma is a distractor as it’s a relative contraindication but not the primary cause.

## Question #:24

**CLINICAL SCENERIO**: A 30-year-old man presents with fever, headache, and lethargy for three weeks. He has a healed human bite mark on his right forearm from a pub brawl two months ago. Auscultation reveals diastolic and systolic murmurs. A trans-oesophageal echo shows an oscillating mass on a bicuspid aortic valve with aortic regurgitation. Two of three blood cultures grow small colonies of tiny pleomorphic gram-negative bacilli.

**QUESTION LINE**: What is the likely causative organism?

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

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: 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.

**>>DESCRIPTION**: A 30-year-old man has fever, headache, and lethargy for three weeks. He has a healed human bite mark from a pub brawl two months prior. He has diastolic and systolic murmurs with an oscillating mass on a bicuspid aortic valve and aortic regurgitation. Two blood cultures grew gram-negative bacilli.

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

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Eikenella corrodens

**>>REASONING**: Eikenella corrodens is the most likely causative organism because it is a gram-negative bacillus and a commensal of the human mouth, fitting with the human bite injury and blood culture results. Streptococcus viridans and Staphylococcus aureus are gram-positive. Escherichia coli is less likely. Phoenicoparrus andinus (Andean Flamingo) does not cause endocarditis.

## Question #:116

**CLINICAL SCENERIO**: 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.

**QUESTION LINE**: What would be the investigation of choice?

**OPTIONS**: - a) 24 hour ambulatory electrocardiogram - b) Electrocardiogram treadmill tes - c) 24 hour blood pressure - d) 72 hour ambulatory electrocardiogram - e) Event recorder electrocardiogram

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: 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.

**>>DESCRIPTION**: A 58-year-old female with palpitations for six months, last episode four days ago with lightheadedness and chest discomfort. Initial ECG shows sinus rhythm.

**>>OPTIONS**: a) 24 hour ambulatory electrocardiogram b) 24 hour blood pressure c) 72 hour ambulatory electrocardiogram d) Electrocardiogram treadmill tes e) Event recorder electrocardiogram

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Event recorder electrocardiogram

**>>REASONING**: An event recorder ECG is most suitable for suspected atrial fibrillation episodes occurring more than 24 hours apart. A 24-hour ambulatory ECG is for episodes less than 24 hours apart. Treadmill test is for exercise-induced arrhythmias or ischemia, and 24-hour BP monitoring is for suspected hypertension. 72-hour ECG is similar to 24/48-hour, but event recorder is preferred per NICE guidelines.

## Question #:75

**CLINICAL SCENERIO**: A 24-year-old female with a family history of Wolff-Parkinson-White syndrome presents to the ED after binge drinking, complaining of sudden palpitations that started 1 hour ago. Her heart rate is 180 bpm, blood pressure is 100/60 mmHg, saturations are 98% on air, respiratory rate is 26/min, and temperature is 36.8ºC. ECG shows pre-excited atrial fibrillation (AF).

**QUESTION LINE**: Which of the following is an appropriate treatment?

**OPTIONS**: a) Adenosine b) Atenolol c) Flecainide d) Diltiazem e) Digoxin

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: 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.

**>>DESCRIPTION**: A 24-year-old female with Wolff-Parkinson-White syndrome presents with palpitations (HR 180 bpm) after binge drinking. ECG shows pre-excited atrial fibrillation.

**>>OPTIONS**: a) Adenosine b) Atenolol c) Digoxin d) Diltiazem e) Flecainide

**>>CORRECT-CHOICE LINE**: Correct answer is e.

**>>CORRECT-CHOICE\_TEXT**: Flecainide

**>>REASONING**: Flecainide is the appropriate treatment. Avoid AV nodal blockers (adenosine, diltiazem, digoxin, atenolol) in pre-excited AF as they can cause ventricular tachycardia.

## Question #:79

**CLINICAL SCENERIO**: An 89-year-old woman presents 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. She has a past medical history of diabetes mellitus type 2 and hypertension. Examination reveals coarse crackles bi-basally, a pan-systolic murmur loudest over the apex. Investigations show a Haemoglobin of 11g/dl, WCC 6 x 10^9/l, Platelets 178 x 10^9/l, Sodium 139 mmol/l, Potassium 4.2 mmol/l, Urea 8 mmol/l, Creatinine 92 µmol/l, Blood cultures Methicillin-sensitive Staphylococcus aureus. Echocardiogram shows severe mitral regurgitation with large mobile structure on valve leaflet and Chest X-ray shows bilateral blunting of the costophrenic angles and upper lobe diversion

**QUESTION LINE**: What is the best treatment for this lady?

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

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: 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 rightsided endocarditis and with endocarditis due to highly susceptible Streptococcus viridans treated with synergistic antimicrobials.

**>>DESCRIPTION**: 89-year-old woman presents with 1-week of worsening shortness of breath, falls, diabetes, and hypertension. Examination: bibasilar crackles and pansystolic murmur. Labs: MSSA bacteremia. Echo: mitral regurgitation with mobile valve structure. CXR: blunting of costophrenic angles and upper lobe diversion.

**>>OPTIONS**: a) Amoxicillin intravenously and vancomycin intravenously b) Amoxicillin orally and vancomycin intravenously c) Ceftriaxone intravenously d) Flucloxacillin intravenously e) Flucloxacillin orally

**>>CORRECT-CHOICE LINE**: Correct answer is d.

**>>CORRECT-CHOICE\_TEXT**: Flucloxacillin intravenously

**>>REASONING**: The patient has mitral valve endocarditis due to methicillin-sensitive Staphylococcus aureus. Intravenous flucloxacillin is the appropriate treatment. Oral flucloxacillin, ceftriaxone, amoxicillin and vancomycin are not appropriate.

## Question #:81

**CLINICAL SCENERIO**: An 89-year-old woman presents with increased shortness of breath and decreased mobility of 1-week duration. She has shortness of breath that is worse at night and has fallen twice in the last week. She has a history of diabetes mellitus type 2 and hypertension. Examination reveals coarse crackles bi-basally and a pan-systolic murmur loudest over the apex. Investigations show MSSA bacteremia and echocardiogram shows severe mitral regurgitation with large mobile structure on valve leaflet and CXR shows bilateral blunting of the costophrenic angles and upper lobe diversion.

**QUESTION LINE**: What is the best treatment for this lady?

**OPTIONS**: a) Flucloxacillin orally b) Flucloxacillin intravenously c) Amoxicillin orally and vancomycin intravenousl d) Ceftriaxone intravenously e) Amoxicillin intravenously and vancomycin intravenously

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: 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 rightsided endocarditis and with endocarditis due to highly susceptible Streptococcus viridans treated with synergistic antimicrobials.

**>>DESCRIPTION**: An 89-year-old woman presents with worsening dyspnea and reduced mobility. Examination reveals bibasilar crackles and a pansystolic murmur. Labs show MSSA bacteremia. Echocardiogram reveals severe mitral regurgitation with a mobile leaflet structure and CXR with blunting of costophrenic angles.

**>>OPTIONS**: a) Amoxicillin intravenously and vancomycin intravenously b) Amoxicillin orally and vancomycin intravenousl c) Ceftriaxone intravenously d) Flucloxacillin intravenously e) Flucloxacillin orally

**>>CORRECT-CHOICE LINE**: Correct answer is d.

**>>CORRECT-CHOICE\_TEXT**: Flucloxacillin intravenously

**>>REASONING**: The patient has MSSA native valve endocarditis. IV flucloxacillin is the appropriate treatment. Oral flucloxacillin, ceftriaxone, and amoxicillin/vancomycin combinations would not provide adequate coverage.

## Question #:90

**CLINICAL SCENERIO**: An 18-year-old man attends the clinic with blurred vision that has progressively worsened. He has a history of recurrent deep vein thromboses and mild learning difficulty. Examination reveals an increased arm span to body height ratio and scoliosis. Ophthalmological assessment shows a downward lens dislocation.

**QUESTION LINE**: What is the most likely diagnosis?

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

**CORRECT-CHOICE LINE**: Correct answer iis c.

**REASONING**: 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.

**>>DESCRIPTION**: An 18-year-old male presents with progressive blurred vision, recurrent DVTs, and mild learning difficulty. Examination reveals increased arm span, scoliosis, and downward lens dislocation.

**>>OPTIONS**: a) Ehlers danlos syndrome b) Ectopia lentis syndrome c) Homocystinuria d) Marfan’s syndrome e) Multiple endocrine neoplasia type 2B

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Homocystinuria

**>>REASONING**: Homocystinuria is the most likely diagnosis due to the Marfanoid habitus, recurrent DVTs, learning disability, and inferonasal ectopia lentis. Ectopia lentis syndrome lacks vascular complications. Ehlers-Danlos syndrome rarely presents with thrombotic complications or lens dislocation. Marfan’s syndrome typically has superotemporal lens dislocation. Multiple endocrine neoplasia type 2B lacks other supporting clinical features.

## Question #:30

**CLINICAL SCENERIO**: A 34-year-old man presents to the emergency department after a syncope episode during tennis. He experiences shortness of breath and chest discomfort while playing and has had previous light-headed episodes. He is otherwise healthy with no significant medical history. Observations are normal.

**QUESTION LINE**: What is the patient’s most likely diagnosis?

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

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: 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 nonspecific 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 .

**>>DESCRIPTION**: A 34-year-old male with exertional syncope, shortness of breath, and chest discomfort presents to the ED. He has no significant past medical history. Observations are normal. What is the likely diagnosis?

**>>OPTIONS**: a) Arrhythmogenic right ventricular dysplasia (ARVD) b) Brugada syndrome c) Dilated cardiomyopathy d) Hypertrophic obstructive cardiomyopathy e) Wellen’s syndrome

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Hypertrophic obstructive cardiomyopathy

**>>REASONING**: The presentation is consistent with Hypertrophic Obstructive Cardiomyopathy (HOCM), characterized by exertional symptoms and ECG findings like T wave inversions. Brugada syndrome causes electrical disorders (ST elevation), dilated cardiomyopathy presents with heart failure symptoms (not fluctuant), ARVD causes right ventricular issues (T wave inversion in V1-V3), and Wellen’s syndrome indicates LAD stenosis (ischemic symptoms).

## Question #:105

**CLINICAL SCENERIO**: A 32-year-old female collapsed while jogging, reporting subacute chest discomfort, breathlessness, and feeling faint. She experienced a brief blackout and now feels well. She has a history of exertional dyspnea and chest discomfort, with occasional light-headedness but no prior fainting. Observations are normal; ECG is shown.

**QUESTION LINE**: What is the patient’s most likely diagnosis?

**OPTIONS**: - a) Brugada syndrome - b) Dilated cardiomyopathy - c) Hypertrophic obstructive cardiomyopathy - d) Wellen’s syndrome - e) Arrhythmogenic right ventricular dysplasia

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: 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 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.

**>>DESCRIPTION**: A 32-year-old female collapsed while jogging, reporting subacute chest discomfort and breathlessness. She experienced a brief blackout and now feels well. She has a history of exertional dyspnea and chest discomfort. Observations are normal; ECG shows findings suggestive of the diagnosis.

**>>OPTIONS**: a) Arrhythmogenic right ventricular dysplasia b) Brugada syndrome c) Dilated cardiomyopathy d) Hypertrophic obstructive cardiomyopathy e) Wellen’s syndrome

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Hypertrophic obstructive cardiomyopathy

**>>REASONING**: The presentation suggests hypertrophic obstructive cardiomyopathy (HOCM) due to exertional symptoms and ECG findings of left ventricular hypertrophy. Brugada syndrome involves electrical disorders, not structural. Dilated cardiomyopathy presents with heart failure symptoms, not fluctuant symptoms. Wellen’s syndrome shows specific T-wave changes related to LAD stenosis. ARVD primarily affects the right ventricle, with different ECG findings.

## Question #:9

**CLINICAL SCENERIO**: A 75-year-old man recovering from elective knee replacement becomes unable to breathe and goes into cardiac arrest. He has hypertension controlled with medication and refused to wear compression stockings. Initial resuscitation involved adrenaline for pulseless electrical activity (PEA), followed by a DC shock for ventricular fibrillation. A table summarizes the rhythm checks and treatments.

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

**OPTIONS**: - 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-CHOICE LINE**: Correct answer is a.

**REASONING**: 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.

**>>DESCRIPTION**: 75-year-old post-knee replacement patient has cardiac arrest. Initial rhythm was PEA treated with adrenaline, then VF treated with DC shock. What IV drug is indicated after the next DC shock?

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

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: IV adrenaline 1 mg

**>>REASONING**: Adrenaline is indicated every 3-5 minutes after initial administration for PEA/asystole, even if the rhythm changes to VF/VT. Amiodarone is given after 3 defibrillation attempts, and Atropine is not indicated in ALS.

## Question #:1

**CLINICAL SCENERIO**: A 43-year-old lady with schizophrenia presents with palpitations, headaches, and dizziness for 3 days. She feels her heart pounding, becomes dizzy, and feels faint about 3-4 times per day. She was diagnosed with a UTI 4 days ago, treated with ciprofloxacin. Her medications include olanzapine and occasional paracetamol. On examination, pulse is 135 bpm irregular, BP 90/60mmHg, cardiac monitor reveals runs of polymorphic tachycardia. IV lidocaine was ineffective.

**QUESTION LINE**: Which is the most appropriate next step in management?

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

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: 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.

**>>DESCRIPTION**: A 43-year-old female with schizophrenia presents with palpitations, headaches, and dizziness, experiencing faintness 3-4 times daily. She’s on ciprofloxacin for a UTI and olanzapine. Examination reveals irregular tachycardia (135 bpm, BP 90/60 mmHg) with polymorphic tachycardia runs. Lidocaine was ineffective.

**>>OPTIONS**: a) Immediate DC cardioversion b) IV amiodarone c) IV flecainide d) IV labetalol e) IV magnesium sulphate

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: IV magnesium sulphate

**>>REASONING**: The presentation suggests acquired long QT syndrome from ciprofloxacin and olanzapine, leading to torsades de pointes. Magnesium sulfate is the appropriate treatment. DC cardioversion, amiodarone, flecainide and labetalol are not first line treatments for Torsades de pointes.

## Question #:94

**CLINICAL SCENERIO**: A 19-year-old female presents with palpitations, anxiety, dizziness, and one episode of syncope. She has a history of atrial fibrillation. Examination reveals BP 125/85 mmHg and pulse 140 bpm. ECG shows a broad complex regular tachycardia with a short PR interval, slurred QRS upstroke, and a tall R wave in V1.

**QUESTION LINE**: Which of the following would be the most appropriate initial step in medical management?

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

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: The description of the ECG given is that of WPW syndrome, a form of AtrioVentricular 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).

**>>DESCRIPTION**: A 19-year-old female with palpitations, anxiety, dizziness, and syncope presents with a history of atrial fibrillation. ECG shows broad complex regular tachycardia, short PR interval, slurred QRS upstroke, and tall R wave in V1, suggestive of WPW syndrome.

**>>OPTIONS**: a) IV adenosine b) IV digoxin c) IV procainamide d) IV propranolol e) IV verapamil

**>>CORRECT-CHOICE LINE**: Correct answer is c.

**>>CORRECT-CHOICE\_TEXT**: IV procainamide

**>>REASONING**: The ECG description indicates WPW syndrome. AV nodal blocking agents (Adenosine, Beta-blockers, Calcium Channel Blockers, Digoxin) should be avoided as they can paradoxically increase conduction over the accessory pathway, potentially leading to ventricular fibrillation. Procainamide is a more appropriate initial step.

## Question #:115

**CLINICAL SCENERIO**: A 62-year-old woman with type two diabetes mellitus, hypertension, hypothyroidism, osteoarthritis, and dementia was admitted with a lower respiratory tract infection. She is on metformin, ramipril, levothyroxine, and paracetamol. Her metformin was stopped during the first two days in the hospital. She has persistently high blood pressure despite taking ramipril.

**QUESTION LINE**: What is the most appropriate plan to control her hypertension?

**OPTIONS**: - 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

**CORRECT-CHOICE LINE**: The correct answer is to increase the dose of ramipril and to check U&Es within 12 weeks.

**REASONING**: 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.

**>>DESCRIPTION**: A 62-year-old woman with diabetes, hypertension, hypothyroidism, osteoarthritis, and dementia, admitted for a lower respiratory tract infection, has persistently high blood pressure despite taking ramipril.

**>>OPTIONS**: a) Add amlodipine, increase the dose of ramipril and check U&Es within 3-4 weeks b) Add on amlodipine at a low dose c) Increase the dose of ramipril and check U&Es within 1-2 weeks d) Increase the dose of ramipril and check U&Es within 3-4 weeks e) Stop ramipril and start amlodipine instead

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Increase the dose of ramipril and check U&Es within 1-2 weeks

**>>REASONING**: Increase ramipril dose first as the patient is on a low dose. Renal function must be checked within two weeks of altering ACE inhibitor dosage. Amlodipine is less preferred due to the patient’s diabetes.

## Question #:5

**CLINICAL SCENERIO**: A 54-year-old woman presents with a transient rash on her chest, face, and upper arms, mostly occurring in the summertime. She has a history of hypertension, palindromic rheumatism, and heart failure with preserved ejection fraction. Medications include amlodipine, furosemide, indapamide, and hydroxychloroquine. Examination shows a non-itchy maculopapular rash on her chest and distal forearms. ANA is negative.

**QUESTION LINE**: What is the likely explanation for her symptoms?

**OPTIONS**: a) Furosemide b) Hydroxychloroquine c) Indapamide d) Solar urticaria e) Systemic lupus erythematosus

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: 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 nonpruritic 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.

**>>DESCRIPTION**: A 54-year-old woman presents with a summertime rash on her chest, face, and upper arms. History includes hypertension, palindromic rheumatism, and heart failure. Medications: amlodipine, furosemide, indapamide, and hydroxychloroquine. Exam: non-itchy maculopapular rash. ANA negative.

**>>OPTIONS**: a) Furosemide b) Hydroxychloroquine c) Indapamide d) Solar urticaria e) Systemic lupus erythematosus

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Indapamide

**>>REASONING**: Indapamide, a thiazide diuretic, is a known cause of photosensitive rashes, consistent with the patient’s symptoms and sun-exposed rash. Furosemide is associated with bullous reactions. Solar urticaria presents as pruritic urticaria. Hydroxychloroquine treats photosensitivity. SLE is less likely due to a negative ANA.

## Question #:86

**CLINICAL SCENERIO**: A 65-year-old Caucasian man presents with a 3-week history of a new rash, characterized by purplish discolouration of his forearms and hands that initially appeared while on holiday in the south of France. It is mildly itchy but not painful. 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.

**QUESTION LINE**: Which of the following is most likely to be responsible for this patient’s presentation?

**OPTIONS**: - a) Aspirin - b) Digoxin - c) Indapamide - d) Metformi - e) Ramipril

**CORRECT-CHOICE LINE**: Correct answer Is c.

**REASONING**: 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 discoloration 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.

**>>DESCRIPTION**: A 65-year-old man presents with a 3-week history of a new, mildly itchy rash on his forearms and hands, appearing after a holiday in the south of France. Examination reveals purplish discolouration of hands up to the elbows bilaterally and mild facial erythema. PMH: hypertension, type 2 diabetes, ischaemic heart disease and atrial fibrillation.

**>>OPTIONS**: a) Aspirin b) Digoxin c) Indapamide d) Metformin e) Ramipril

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Indapamide

**>>REASONING**: Indapamide (a thiazide diuretic) can cause photosensitivity. The patient’s presentation is consistent with drug-induced photosensitivity triggered by UV radiation. Aspirin, metformin, and ramipril do not typically cause photosensitivity. Digoxin can cause skin reactions, but not commonly photosensitivity.

## Question #:93

**CLINICAL SCENERIO**: A 44-year-old woman presents with dyspnoea, dizziness, and shortness of breath for 2 weeks. Pulse is 180/min, blood pressure 100/66 mmHg, and oxygen saturation 98% on room air. Chest is clear and she appears well perfused. ECG obtained.

**QUESTION LINE**: What is the most appropriate treatment?

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

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: 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.

**>>DESCRIPTION**: A 44-year-old woman presents with dyspnoea, dizziness, and shortness of breath for 2 weeks. Pulse 180/min, BP 100/66 mmHg, SpO2 98%. ECG shows irregular wide-complex tachycardia.

**>>OPTIONS**: a) Intravenous adenosine b) Intravenous amiodarone c) Intravenous labetalol d) Intravenous magnesium e) Unsynchronised DC shock

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Intravenous magnesium

**>>REASONING**: The ECG indicates torsades de pointes, a polymorphic ventricular tachycardia often associated with prolonged QT interval. IV magnesium sulfate is the appropriate treatment. Synchronized DC shocks are indicated if the patient shows adverse signs, which are not present in this case.

## Question #:22

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

**QUESTION LINE**: What is shown on the ECG?

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

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: 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.

**>>DESCRIPTION**: A 72-year-old woman with ischaemic heart disease and COPD presents with palpitations, pulse 120/min, and BP 110/76 mmHg. Chest is clear. ECG shown.

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

**>>CORRECT-CHOICE LINE**: Correct answer is c.

**>>CORRECT-CHOICE\_TEXT**: Left bundle branch block

**>>REASONING**: The QRS morphology indicates left bundle branch block (LBBB). Myocardial ischemia should be excluded in acutely unwell patients with LBBB.

## Question #:109

**CLINICAL SCENERIO**: A 55-year-old man presents with 1-day history of central chest pain and progressive shortness of breath over 2 weeks. He had a drug-eluting stent inserted 21 days ago following an anterolateral myocardial infarction. His past medical history includes diet-controlled type 2 diabetes mellitus and hypertension. Examination reveals bibasal crepitations and pitting oedema. An ECG is taken.

**QUESTION LINE**: What is the most likely diagnosis?

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

**CORRECT-CHOICE LINE**: This patient has a diagnosis of a left ventricular aneurysm , a rare but serious complication of an anterior myocardial infarction.

**REASONING**: 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.

**>>DESCRIPTION**: A 55-year-old man presents with chest pain and progressive dyspnea 3 weeks post-MI with stent placement. Examination shows bibasal crackles and edema. ECG was taken.

**>>OPTIONS**: a) Anterior myocardial infarction b) Left ventricular aneurysm c) Papillary muscle rupture d) Pericarditis e) Pulmonary embolism

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Left ventricular aneurysm

**>>REASONING**: The patient likely has a left ventricular aneurysm, a complication of MI presenting >2 weeks after the event. ECG shows persistent ST elevation, QS waves, and upright T waves. Acute coronary syndrome is less likely due to the gradual onset and absence of dynamic ST changes. Papillary muscle rupture would present with acute mitral regurgitation, and pulmonary embolism does not explain ST elevation.

## Question #:6

**CLINICAL SCENERIO**: 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

**QUESTION LINE**: What is the patient’s most likely diagnosis?

**OPTIONS**: - a) Brugada syndrome - b) Hyperkalaemia - c) Left ventricular hypertrophy - d) Pericarditis - e) Wellen’s syndrome

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: 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 conductions 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.

**>>DESCRIPTION**: A 52-year-old with chronic renal artery stenosis and poorly controlled hypertension presents with increasing breathlessness, mild chest discomfort, and a recent productive cough. ECG shows S in V1 plus R in V6= 44mm.

**>>OPTIONS**: a) Brugada syndrome b) Hyperkalaemia c) Left ventricular hypertrophy d) Pericarditis e) Wellen’s syndrome

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Left ventricular hypertrophy

**>>REASONING**: The patient’s symptoms and ECG changes are consistent with left ventricular hypertrophy due to chronic uncontrolled hypertension. Brugada syndrome presents with syncope and specific ECG changes. Hyperkalemia is unlikely given the patient’s renal function. Pericarditis typically presents with pleuritic chest pain and specific ECG findings, and Wellen’s syndrome is associated with ischemic chest pain and T-wave changes.

## Question #:85

**CLINICAL SCENERIO**: A 22-year-old male presents with heart palpitations. He reports episodes of rapid heartbeat and dizziness. A 7-day cardiac Holter monitor shows: PR interval 95ms, QRS duration 105ms, and one episode of tachycardia of 145 bpm with normal QRS morphology and QRS 110ms.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: - 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-CHOICE LINE**: Correct answer is a.

**REASONING**: 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 120200ms. A short PR interval is suggestive of a pre-excitation syndrome. Preexcitation 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 WolffParkinson-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 preexcitation syndrome of the heart characterised by a short PR interval and normal QRS width.

**>>DESCRIPTION**: A 22-year-old male with palpitations, rapid heartbeat, and dizziness. Holter monitor: PR 95ms, QRS 105ms, tachycardia (145 bpm) with normal QRS morphology (110ms).

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

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Lown-Ganong-Levine syndrome

**>>REASONING**: The short PR interval (95ms) and normal QRS width (105ms) are key indicators of Lown-Ganong-Levine syndrome (LGL), a pre-excitation syndrome. Wolff-Parkinson-White syndrome and ventricular tachycardia are ruled out by the normal QRS width.

## Question #:25

**CLINICAL SCENERIO**: A 67-year-old male with hypertension, type 2 diabetes, sick sinus syndrome, and stable angina presents with increasing chest pain on exertion. He previously tried isosorbide mononitrate with side effects and is currently on bisoprolol 5mg OD. ECG shows first-degree heart block with normal QRS complexes at 50 bpm, BP is 140/76 mmHg. GTN spray is increasingly ineffective.

**QUESTION LINE**: What is the most appropriate next step in management?

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

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: NICE recommends nitrates followed by beta blockers or calcium channel blockers for stable angina. Combining both is next. Reperfusion is considered after 2 antianginals, with a third drug if PCI/CABG is not suitable. Calcium channel blockers are contraindicated due to first-degree heart block and bradycardia. Ivabradine is contraindicated in sick sinus syndrome. Ranolazine is contraindicated due to liver dysfunction. Nicorandil is only contraindicated in LV failure and cardiogenic shock and is thus the only appropriate anti-anginal in this scenario.

**>>DESCRIPTION**: 67M with HTN, DM2, sick sinus syndrome, and stable angina presents with worsening exertional chest pain. He takes bisoprolol 5mg OD, but GTN spray is ineffective. ECG shows first-degree heart block, HR 50 bpm, BP 140/76 mmHg. Isosorbide mononitrate caused headaches.

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

**>>CORRECT-CHOICE LINE**: Correct answer is c.

**>>CORRECT-CHOICE\_TEXT**: Nicorandil

**>>REASONING**: Nicorandil is the most appropriate choice because calcium channel blockers, ivabradine and ranolazine are contraindicated due to heart block/bradycardia, sick sinus syndrome, and liver dysfunction respectively. Reperfusion is considered after failure of 2 antianginals.

## Question #:36

**CLINICAL SCENERIO**: A patient post-MI, managed with PCI, is asymptomatic and stable. ECG shows widened QRS complexes and AV dissociation, indicating accelerated idioventricular rhythm (AIVR) at 55 bpm.

**QUESTION LINE**: What management should be advised?

**OPTIONS**: 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-CHOICE LINE**: Correct answer is c.

**REASONING**: Accelerated idioventricular rhythm is common and unconcerning 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.

**>>DESCRIPTION**: Post-MI patient, treated with PCI, presents with asymptomatic AIVR at 55 bpm. What is the appropriate management?

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

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: No additional management is required

**>>REASONING**: AIVR is a benign, self-limiting rhythm post-MI, especially in asymptomatic and hemodynamically stable patients. PCI is not indicated as there is no ischemia. Pacemaker insertion is not needed because AIVR is ectopic. Beta-blockers should not be stopped as they are important for secondary prevention. Atropine is not required due to the patient’s stable condition.

## Question #:42

**CLINICAL SCENERIO**: A patient post-MI, managed with PCI, attends a cardiology clinic. They are asymptomatic, adhering to medication, and have stable vital signs. ECG shows widened QRS complexes and AV dissociation, indicative of accelerated idioventricular rhythm (AIVR) at 55 bpm.

**QUESTION LINE**: What management should be advised?

**OPTIONS**: - 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-CHOICE LINE**: Correct answer is c.

**REASONING**: Accelerated idioventricular rhythm is common and unconcerning 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 selflimiting 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.

**>>DESCRIPTION**: Post-MI patient, managed with PCI, presents with asymptomatic AIVR (55 bpm) on ECG. Vitals are stable, and they are adhering to medications.

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

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: No additional management is required

**>>REASONING**: AIVR is a benign, self-limiting rhythm common post-MI, especially after reperfusion. No treatment is needed given the patient’s asymptomatic and hemodynamically stable condition. PCI, pacemaker insertion, stopping beta-blockers, or atropine are not indicated in this scenario.

## Question #:88

**CLINICAL SCENERIO**: A 70-year-old woman with type 2 diabetes mellitus and hypertension, with no evidence of diabetic retinopathy, chronic kidney disease, or cardiovascular disease, is reviewed in clinic. She takes simvastatin 40mg, ramipril 10mg, amlodipine 5mg, and metformin 1g bd. Recent blood results: 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. Blood pressure in clinic is 134/76 mmHg.

**QUESTION LINE**: What is the most appropriate course of action?

**OPTIONS**: a) Add gliclazide b) Increase amlodipine c) Increase ramipril d) Add losartan e) No changes to medication required

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: 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 >= 6.5%.

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

**>>DESCRIPTION**: 70-year-old woman with type 2 diabetes and hypertension, no retinopathy, CKD, or CVD. Medications: simvastatin, ramipril, amlodipine, metformin. Labs: HbA1c 6.3%, BP 134/76 mmHg. No proteinuria.

**>>OPTIONS**: a) Add gliclazide b) Add losartan c) Increase amlodipine d) Increase ramipril e) No changes to medication required

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: No changes to medication required

**>>REASONING**: HbA1c is well-controlled (6.3%), and blood pressure is below target (<140/80 mmHg) for diabetics without complications, thus no medication changes are needed. Other options suggesting adding or increasing medications are not indicated.

## Question #:34

**CLINICAL SCENERIO**: A 22-year-old Afro-Caribbean man presents with sharp, right lateral chest pain that worsens on inspiration. He has no past medical history and takes no regular medicines. An ECG is performed:

**QUESTION LINE**: What is the most likely explanation of these ECG results?

**OPTIONS**: - a) NSTEMI - b) STEMI - c) Normal variant - d) Prinzmetal’s angina (variant angina) - e) Stable angina pectoris

**CORRECT-CHOICE LINE**: Widespread T wave inversion in the chest leads can be a normal variant in patients with Afro-Caribbean ethnicity

**REASONING**: 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).

**>>DESCRIPTION**: A 22-year-old Afro-Caribbean man presents with sharp right-sided chest pain, worsened by inspiration. ECG shows widespread T-wave inversions in V1-V6. No ST elevation or depression is noted. What is the most likely diagnosis?

**>>OPTIONS**: a) Normal variant b) NSTEMI c) Prinzmetal’s angina (variant angina) d) Stable angina pectoris e) STEMI

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Normal variant

**>>REASONING**: Widespread T-wave inversion in chest leads can be a normal variant in Afro-Caribbean individuals. Coronary artery disease, STEMI, NSTEMI, and stable angina are less likely due to the patient’s age, lack of risk factors, and absence of ST elevation. Prinzmetal’s angina typically presents with ST elevation.

## Question #:60

**CLINICAL SCENERIO**: 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 |
| --- | --- |
| Platelet s | 260 \* 10 9 /l |
| WBC | 6 \* 10 9 /l |

| Mg | 0.34 µmol/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.

**QUESTION LINE**: What medication is the most likely cause of hypomagnesaemia in this case?

**OPTIONS**: - a) Aspirin - b) Omeprazole - c) Ranitidine - d) Colchicine - e) Ramipril4

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: 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.

**>>DESCRIPTION**: An 80-year-old man with a history of gout, reflux, and ischemic heart disease presents to the ED with atrial fibrillation and rapid ventricular response, now stabilized. Labs show hypomagnesemia (Mg 0.34 µmol/l). Cardiology recommends magnesium correction.

**>>OPTIONS**: a) Aspirin b) Colchicine c) Omeprazole d) Ramipril4 e) Ranitidine

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Omeprazole

**>>REASONING**: PPIs are associated with hypomagnesemia, possibly due to decreased intestinal absorption. Correcting electrolyte imbalances like hypomagnesemia is crucial in managing arrhythmias.

## Question #:32

**CLINICAL SCENERIO**: A 22-year-old woman presents with 1-day history of abdominal pain and vomiting after consuming 7 glasses of wine. She reports a family history of a heart condition. Observations are normal. Examination reveals central and epigastric tenderness. VBG shows lactate of 3.5 mmol/L. ECG shows sinus rhythm, normal axis, QRS duration 110ms, QTc 550ms.

**QUESTION LINE**: What drug is it most important to avoid?

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

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: 5HT-3 receptor antagonists shouldn’t be used as antiemetics in patients with longQT 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 dificile infections, acute interstitial nephritis, but not prolongation of the QTc interval.

**>>DESCRIPTION**: 22F presents with abdominal pain, vomiting after drinking wine. Family history of heart condition. Exam: epigastric tenderness. ECG: QTc 550ms. VBG: elevated lactate.

**>>OPTIONS**: a) Codeine b) Cyclizine c) Hyoscine butylbromide d) Omeprazole e) Ondansetron

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Ondansetron

**>>REASONING**: The patient has a prolonged QTc interval (550ms), increasing the risk of Torsades de pointes. Ondansetron, a 5HT-3 receptor antagonist, prolongs the QTc interval and should be avoided. Codeine, cyclizine, hyoscine butylbromide and Omeprazole do not prolong the QTc interval.

## Question #:107

**CLINICAL SCENERIO**: A 45-year-old woman with a left tibia and fibula fracture develops a left-sided above knee deep vein thrombosis after an open reduction and internal fixation. Subsequently, she experiences sudden onset weakness in her right leg and arm, dysarthric speech, and reduced consciousness. CT reveals a left-sided middle cerebral artery infarct. Carotid Doppler shows minimal stenosis, and a 24-hour tape indicates sinus bradycardia.

**QUESTION LINE**: Which of the following features from further investigations would best explain this woman’s presentation?

**OPTIONS**: - a) Anti phospholipid antibody positive - b) Protein C deficiency - c) Patent foramen ovale (PFO) - d) Hypercholesterolaemia - e) Dilated cardiomyopathy on ECHO

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: This lady developed a thrombus due to recent operation and immobility. Part of it embolised into her left middle cerebral artery. A PFO would explain how a thrombus from the leg would reach the brain rather than the lungs, despite minimal carotid stenosis and lack of atrial fibrillation. Other options are risk factors for stroke but don’t explain the mechanism of embolism with near-normal carotids and sinus rhythm. Dilated cardiomyopathy is not associated with increased embolic stroke risk.

**>>DESCRIPTION**: A 45-year-old woman with a leg fracture develops DVT post-surgery. She then presents with right-sided weakness, dysarthria, and reduced consciousness. CT shows a left MCA infarct, minimal carotid stenosis, and sinus bradycardia.

**>>OPTIONS**: a) Anti phospholipid antibody positive b) Dilated cardiomyopathy on ECHO c) Hypercholesterolaemia d) Patent foramen ovale (PFO) e) Protein C deficiency

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Patent foramen ovale (PFO)

**>>REASONING**: A PFO explains how a DVT thrombus embolized to the brain, bypassing the lungs, given minimal carotid stenosis and sinus rhythm. Other options are stroke risk factors but don’t explain the paradoxical embolism, and dilated cardiomyopathy is not linked to embolic stroke.

## Question #:28

**CLINICAL SCENERIO**: A 58-year-old man with retrosternal chest pain on exertion reports symptoms when walking more than 50 meters or playing with grandchildren. Symptoms have been present for 9 months, with no pain at rest. Bisoprolol and amlodipine were discontinued due to side effects. He has hypercholesterolaemia, takes aspirin and pravastatin, and uses nitrate spray and sildenafil as needed. He quit smoking last year. Examination is unremarkable, BP is 102/72 mmHg. ECG shows sinus rhythm at 58 bpm with non-specific ST segment abnormalities. Echocardiogram is normal, EF 55-60%. Cardiac MRI shows significant ischaemia in 20% of LV myocardium.

**QUESTION LINE**: What is the most appropriate management of the patients chest pain?

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

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: The role of percutaneous coronary intervention in stable angina is controversial. 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.

**>>DESCRIPTION**: 58M with exertional chest pain (9 months). Bisoprolol/amlodipine intolerant. Takes aspirin/pravastatin/sildenafil. Cardiac MRI: 20% LV ischemia. ECG: non-specific ST changes. What next?

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

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Percutaneous coronary intervention

**>>REASONING**: PCI is indicated for stable angina with >10% LV ischemia per ESC guidelines. Nicorandil and ISMN are contraindicated due to hypotension and sildenafil use. Ivabradine is contraindicated due to bradycardia.

## Question #:41

**CLINICAL SCENERIO**: A 43-year-old gentleman admitted with a STEMI develops chest pain on the ward. He is recovering from a percutaneous coronary intervention the day before. The pain is on the left side of the chest and severe when he breathes. He has no cough or fevers and normal observations. Examination shows normal heart sounds, a clear chest, and mild chest wall tenderness. Femoral puncture sites are clean. He is in sinus rhythm with T wave inversion in V1-V3, PR 180ms and flat in most leads . Chest x-ray shows clear lung fields and a normal-sized heart. Labs show elevated Na+, K+, Urea, Creatinine, CRP, Hb, Platelets and WBC.

**QUESTION LINE**: What is the likely diagnosis?

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

**CORRECT-CHOICE LINE**: Correct answer is b.

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

This gentleman develops chest pain in the first 24-48hrs after an MI. A hospitalacquired 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.

**>>DESCRIPTION**: A 43-year-old male, post-STEMI and PCI, presents with pleuritic chest pain within 48 hours. Examination reveals normal vitals, clear lungs, and mild chest wall tenderness. ECG shows sinus rhythm with T wave inversion. Chest X-ray is unremarkable. Labs are notable for elevated Na+, K+, CRP and WBC.

**>>OPTIONS**: a) Coronary artery dissection b) Hospital acquired pneumonia c) Pericarditis d) Pleurisy e) Pulmonary embolism

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Pericarditis

**>>REASONING**: The patient’s pleuritic chest pain occurring within 48 hours post-MI is highly suggestive of pericarditis. Other options, such as hospital-acquired pneumonia and pulmonary embolism, are less likely given the timeline and anticoagulation status, respectively. Coronary artery dissection would likely present with ischemic ECG changes.

## Question #:4

**CLINICAL SCENERIO**: 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.

**QUESTION LINE**: Which of the following should the patient be warned of as a recognised side effect of ivabradine?

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

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: 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 fracture 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.

**>>DESCRIPTION**: A 60-year-old man with congestive cardiac failure and asthma, BP 95/63mmHg, HR 98bpm, and sinus rhythm, is commenced on ivabradine 5mg twice daily after developing wheeze with beta blockade.

**>>OPTIONS**: a) Diaphoresis b) Hypotension c) Neutrophilia d) Phosphenes e) Renal failure

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Phosphenes

**>>REASONING**: Ivabradine is associated with visual disturbances like phosphenes due to inhibition of ionic funny channels in the retina. Hypotension and renal failure are not typical side effects.

## Question #:2

**CLINICAL SCENERIO**: 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.

**QUESTION LINE**: Which coronary territory is likely to be affected?

**OPTIONS**: a) Lateral b) Posterior c) Anteroseptal d) Anterolateral e) Inferior

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: Posterior circulation is the correct answer as tall R waves in V1-2 are suggestive of posterior myocardial infarction.

**>>DESCRIPTION**: 71-year-old with 30-minute crushing chest pain. ECG shows tall R waves in V1-2.

**>>OPTIONS**: a) Anterolateral b) Anteroseptal c) Inferior d) Lateral e) Posterior

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Posterior

**>>REASONING**: Tall R waves in V1-2 suggest posterior myocardial infarction.

## Question #:55

**CLINICAL SCENERIO**: 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:

**QUESTION LINE**: What is the most likely explanation for these changes seen over the heart?

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

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: Pericardial calcification most commonly develops following repeated episodes of acute pericarditis. Calcification of the pericardium often results in constrictive pericarditis.

**>>DESCRIPTION**: A 60-year-old man presents to the ED with acute dyspnoea, and his history is unavailable. Imaging reveals changes over the heart.

**>>OPTIONS**: a) Atrial myxoma b) Left ventricular aneurysm c) Previous episodes of uraemia d) Primary hyperparathyroidism e) Sarcoidosis

**>>CORRECT-CHOICE LINE**: Correct answer is c.

**>>CORRECT-CHOICE\_TEXT**: Previous episodes of uraemia

**>>REASONING**: Pericardial calcification, often leading to constrictive pericarditis, typically follows repeated episodes of acute pericarditis. Therefore, previous episodes of uremia is the most likely explanation.

## Question #:7

**CLINICAL SCENERIO**: 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.

**QUESTION LINE**: What is the likely diagnosis?

**OPTIONS**: a) Acute anterolateral myocardial infarction b) Crescendo angina c) Da Costas syndrome d) Prinzmetal’s variant angina e) Takotsubo cardiomyopathy

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: 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.

**>>DESCRIPTION**: A 45-year-old woman has recurrent severe central chest pain at rest with ST elevation on ECG that resolves. Coronary angiography and echocardiogram are normal.

**>>OPTIONS**: a) Acute anterolateral myocardial infarction b) Crescendo angina c) Da Costas syndrome d) Prinzmetal’s variant angina e) Takotsubo cardiomyopathy

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Prinzmetal’s variant angina

**>>REASONING**: Prinzmetal’s variant angina is the likely diagnosis given the chest pain at rest, transient ST elevation, and normal angiography. The pain resolves as the ST elevation disappears. Acute myocardial infarction is less likely due to normal angiography.

## Question #:21

**CLINICAL SCENERIO**: 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:

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

**OPTIONS**: a) Adenosine b) Digoxin c) Diltiazem d) Verapamil e) Procainamide

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: 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.

**>>DESCRIPTION**: A 26-year-old female with recurrent palpitations, shortness of breath, and chest discomfort. Past medical history and thyroid function tests are unremarkable. ECG shows WPW syndrome.

**>>OPTIONS**: a) Adenosine b) Digoxin c) Diltiazem d) Procainamide e) Verapamil

**>>CORRECT-CHOICE LINE**: Correct answer is d.

**>>CORRECT-CHOICE\_TEXT**: Procainamide

**>>REASONING**: The patient has WPW syndrome. Adenosine, Digoxin, Diltiazem, and Verapamil affect the AV node, increasing conduction via the accessory pathway and the risk of ventricular fibrillation/tachycardia. Procainamide does not act on the AV node, making it the safe choice.

## Question #:78

**CLINICAL SCENERIO**: 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.

**QUESTION LINE**: What is the most appropriate treatment of this patients INR?

**OPTIONS**: - 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-CHOICE LINE**: Correct answer Is d.

**REASONING**: 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’

**>>DESCRIPTION**: 77-year-old with atrial fibrillation on bisoprolol and warfarin, admitted for upper GI bleed with hematemesis. INR is 8.5. Hypotensive (90/45 mmHg) and tachycardic (120 bpm). Resuscitated with 0.9% saline, cross match sent.

**>>OPTIONS**: a) Fresh frozen plasma + stop warfarin b) Prothrombin complex concentrates c) Prothrombin complex concentrates + vitamin K + stop warfarin d) Stop warfarin e) Vitamin K + stop warfarin

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Prothrombin complex concentrates + vitamin K + stop warfarin

**>>REASONING**: The patient is actively bleeding with an INR > 8. The most appropriate treatment is prothrombin complex concentrate, Vitamin K, and stopping warfarin, as rapid correction of Factor X, IX, VII, and II levels is required. FFP is not as effective for rapid correction.

## Question #:13

**CLINICAL SCENERIO**: 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 37.5ºC, 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:

**QUESTION LINE**: What is the most likely diagnosis?

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

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: 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.

**>>DESCRIPTION**: 72-year-old man presents with shortness of breath. Pulse 96/min, BP 100/64 mmHg, respiratory rate 20/min, temperature 37.5ºC, SpO2 96% on room air. ECG: sinus rhythm, rate 94/min, no ST-T changes. Troponin I < 0.05 µg/L. CT chest (with contrast) is ordered.

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

**>>CORRECT-CHOICE LINE**: Correct answer is d.

**>>CORRECT-CHOICE\_TEXT**: Pulmonary embolism

**>>REASONING**: Pulmonary embolism (PE) is likely given the tachypnea (RR > 16/min) and low-grade temperature. These findings are less common in the other listed diagnoses. CT confirmed saddle embolus.

## Question #:33

**CLINICAL SCENERIO**: A 19-year-old student collapses while playing hockey, reporting light-headedness and fainting. She lost consciousness briefly. She has been experiencing shortness-of-breath and central chest pain worsened by coughing for 4-5 days. Her pulse is 120/min, blood pressure 96/60 mmHg, and chest auscultation reveals scattered wheezes. An ECG was done on admission.

**QUESTION LINE**: What is the most likely diagnosis?

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

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: It is usual 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.

**>>DESCRIPTION**: A 19-year-old collapses during hockey with light-headedness and fainting, regaining consciousness quickly. She has 4-5 days of shortness-of-breath and central chest pain worsened by coughing. Pulse is 120/min, BP 96/60 mmHg, scattered wheezes. She uses oral contraceptive pills.

**>>OPTIONS**: a) Acute coronary syndrome b) Asthma attack c) Hypertrophic obstructive cardiomyopathy d) Pulmonary embolism e) Vasovagal attack

**>>CORRECT-CHOICE LINE**: Correct answer is d.

**>>CORRECT-CHOICE\_TEXT**: Pulmonary embolism

**>>REASONING**: Pulmonary embolism is likely due to risk factors (oral contraceptive pill), tachycardia, shortness of breath, and chest pain. Asthma is less likely without prior history. The ECG shows sinus tachycardia and a partial S1Q3T3.

## Question #:59

**CLINICAL SCENERIO**: A 64-year-old man with mitral regurgitation presents with new atrial fibrillation. He denies shortness of breath or chest pain. His BP is 115/98 mmHg and HR is 64 bpm. Examination reveals an audible S3 and a 3/6 pansystolic murmur in the mitral area, and his apex beat is displaced. An echo showed a regurgitant mitral valve with left atrial and ventricular dilatation but preserved ejection fraction. He takes naproxen and vitamin D supplements.

**QUESTION LINE**: From the following options, what would be the most appropriate next step?

**OPTIONS**: 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

**CORRECT-CHOICE LINE**: Refer for mitral valve replacement is the correct answer.

**REASONING**: 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 secondline 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.

**>>DESCRIPTION**: A 64-year-old man with mitral regurgitation has new atrial fibrillation, an audible S3, and left ventricular dilatation with preserved ejection fraction. He is asymptomatic. What is the most appropriate next step?

**>>OPTIONS**: 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

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Refer for mitral valve replacement

**>>REASONING**: The patient has severe mitral regurgitation and now atrial fibrillation. Mitral valve replacement is preferred due to a higher chance of maintaining sinus rhythm compared to cardioversion alone. Bisoprolol and sacubitril-valsartan are not indicated due to preserved ejection fraction. Watchful waiting is not appropriate in severe MR with new AF.

## Question #:49

**CLINICAL SCENERIO**: A 58-year-old woman with a history of recurrent fevers, anorexia, and weight loss was admitted with a stroke. Examination revealed left-sided hemiparesis, facial droop, splinter hemorrhages, and a soft diastolic murmur. An echocardiogram showed an oscillating vegetation on an aortic leaflet, and blood cultures were positive for Streptococci spp. She was diagnosed with infective endocarditis and started on intravenous benzylpenicillin and gentamicin. After 5 days, blood tests showed a Penicillin Minimum Inhibitory Concentration (MIC) of 0.6 mg/L and a prolonged PR interval on ECG.

**QUESTION LINE**: What is the next most important management?

**OPTIONS**: - 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-CHOICE LINE**: Correct answer is d.

**REASONING**: 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.

**>>DESCRIPTION**: 58-year-old woman admitted with stroke, history of fevers, anorexia, and weight loss. Exam: left hemiparesis, facial droop, splinter hemorrhages, diastolic murmur. Echocardiogram: aortic vegetation. Blood cultures: Streptococci spp. Treated for infective endocarditis. After 5 days: elevated Penicillin MIC, prolonged PR interval on ECG.

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

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Refer to cardiothoracic surgeons

**>>REASONING**: The patient has infective endocarditis and a prolonged PR interval, indicating a potential aortic abscess disrupting the AV node. This necessitates urgent cardiothoracic surgery referral. While colonoscopy (for Streptococcus Bovis) and TEE are relevant, they are secondary to addressing the potential abscess. Increasing or switching antibiotics is inappropriate given the potential surgical emergency.

## Question #:77

**CLINICAL SCENERIO**: A 62-year-old man presents with leg pain described as a crampy, uncomfortable feeling in the back of his calves when walking. The pain improves with rest. He has a 40-pack year smoking history and takes amlodipine, paracetamol, and ibuprofen. Examination reveals mild atrophy of thigh and calf muscles, shiny pale skin with hair loss in lower limbs, impalpable pedal pulses, and faint popliteal pulses. Ankle brachial pressure index is 0.70 on the right and 0.95 on the left. Lumbar spine X-rays show joint space narrowing and osteophytes.

**QUESTION LINE**: Which one of the following is the next best step in the management of this patient?

**OPTIONS**: - 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-CHOICE LINE**: Correct answer is c.

**REASONING**: 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 manoeuvers such as leaning forward, and sitting down, whereas vascular claudication does not change with these manoeuvers 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.

**>>DESCRIPTION**: A 62-year-old man has leg pain, described as crampy discomfort in calves during walking, relieved by rest. He has a 40-pack-year smoking history, takes amlodipine, paracetamol, and ibuprofen. Examination: bilateral calf atrophy, shiny skin with hair loss in lower limbs, impalpable pedal pulses. ABPI is 0.70 (right) and 0.95 (left). Lumbar spine X-rays show joint space narrowing and osteophytes.

**>>OPTIONS**: a) Check his HbA1c to screen for diabetes b) MRI scan of lumbar spine c) Refer to vascular surgery for consideration of peripheral arterial stenting or bypass surgery d) Screen for coronary artery disease with ECG stress testing e) Starting him on duloxetine to manage his pain

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Refer to vascular surgery for consideration of peripheral arterial stenting or bypass surgery

**>>REASONING**: The patient exhibits vascular claudication symptoms (leg pain on exertion relieved by rest) and signs of peripheral arterial disease. Referral to vascular surgery is the next step. MRI of the lumbar spine is not indicated as the presentation is not consistent with neurogenic claudication. Addressing PAD symptoms takes precedence over screening for coronary artery disease or managing neuropathic pain with duloxetine.

## Question #:111

**CLINICAL SCENERIO**: A 50-year-old woman with type 1 diabetes, angina, and hypertension presents with sudden vomiting and epigastric discomfort. Her blood glucose is 10.4 mmol/L, and urine dip is ‘+’ for ketones. Respiratory rate is 19/min, oxygen saturation is 96% on room air, heart rate is 96 bpm, and blood pressure is 105/67 mmHg. Blood results are awaited, and an ECG is available.

**QUESTION LINE**: What diagnostic option is most likely to confirm the diagnosis?

**OPTIONS**: - 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-CHOICE LINE**: Correct answer is c

**REASONING**: 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 STsegment changes and so is not correct in this instance.

**>>DESCRIPTION**: A 50-year-old woman with type 1 diabetes, angina, and hypertension presents with sudden vomiting and epigastric discomfort. ECG shows ST elevation in I, aVL, and V6, and ST depression in V3 and V4.

**>>OPTIONS**: 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-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Repeat ECG with posterior chest leads

**>>REASONING**: ECG findings suggest an acute coronary syndrome involving posterior and lateral territories. Posterior chest leads would confirm posterior MI. DKA, intra-abdominal pathology, and adrenal crisis are less likely based on the clinical and ECG findings. Abdominal X-ray would not explain ST changes.

## Question #:97

**CLINICAL SCENERIO**: A 64-year-old caucasian male with a 3-year history of heart failure presents to the clinic with worsening shortness of breath on exertion despite being on ramipril, aspirin, bisoprolol, simvastatin, and spironolactone. He reports using four pillows to sleep. Examination reveals fine crackles at lung bases, pitting oedema up to both knees, RR 16, SpO2 96%, BP 110/85 mmHg, HR 70 bpm, and temperature 37.2ºC. ECG shows normal sinus rhythm. Echocardiogram (2 weeks prior) showed an ejection fraction of 30%. Baseline U&E results are within normal limits.

**QUESTION LINE**: What is the optimum treatment option for this patient?

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

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: 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 if 70 bpm, the guidelines state it should be > 75 bpm for ivabradine to be prescribed.

**>>DESCRIPTION**: A 64-year-old Caucasian male with a 3-year history of heart failure presents with worsening dyspnea despite maximum medical therapy (ramipril, aspirin, bisoprolol, simvastatin, spironolactone). He has orthopnea (4 pillows). Exam: crackles, edema to knees. EF is 30%. U&Es are normal.

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

**>>CORRECT-CHOICE LINE**: Correct answer is e.

**>>CORRECT-CHOICE\_TEXT**: Sacubitril-Valsartan (after stopping ACE inhibitor)

**>>REASONING**: Sacubitril-valsartan is optimal for symptomatic heart failure patients with LVEF < 35% on ACEi and beta-blockers, after ACE inhibitor washout. Digoxin does not improve long-term outcomes. Hydralazine/nitrate is for African-American patients. Ivabradine is contraindicated at HR < 75 bpm.

## Question #:12

**CLINICAL SCENERIO**: 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.

**QUESTION LINE**: Given the likely diagnosis, what is the most characteristic finding on this patient’s

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

**CORRECT-CHOICE LINE**: Correct answer: e

**REASONING**: 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.

**>>DESCRIPTION**: A 29-year-old man collapsed while playing football, with no recollection of the event. He reports intermittent palpitations for 2-3 months. Examination reveals a hypokinetic right ventricle on echocardiogram.

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

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Small positive deflection at the end of QRS complex

**>>REASONING**: The most characteristic ECG finding in ARVD is an epsilon wave, which is a small positive deflection at the end of the QRS complex due to post-excitation of right ventricular myocytes. Left ventricular hypertrophy is seen in HOCM, and Osborn waves are associated with hypothermia and Brugada syndrome. Right bundle branch block and delta waves are not typical findings in ARVD.

## Question #:56

**CLINICAL SCENERIO**: A 54-year-old female presents with heart palpitations, no past medical history, and takes no regular medicines. Her cardiorespiratory exam is normal. An ECG shows a rate of 145 bpm, regular rhythm, no visible P waves, QRS of 145ms with RBBB pattern, QTc of 420ms, and right axis deviation.

**QUESTION LINE**: What is the most likely explanation of these ECG results?

**OPTIONS**: - 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-CHOICE LINE**: Correct answer is b.

**REASONING**: The ECG results confirm a broad complex tachycardia with a regular rhythm, differentiating between supraventricular tachycardia (SVT) with bundle branch block (BBB) and ventricular tachycardia (VT). The presence of RBBB and RAD favors SVT with BBB. The absence of P waves might suggest atrial fibrillation (AF) with BBB, but the regular rhythm excludes this. Normal QTc and regular rhythm preclude Torsades de pointes (TdP).

**>>DESCRIPTION**: A 54-year-old female with palpitations has an ECG showing a rate of 145 bpm, regular rhythm, no P waves, QRS of 145ms with RBBB, QTc of 420ms, and right axis deviation.

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

**>>CORRECT-CHOICE LINE**: Correct answer is c.

**>>CORRECT-CHOICE\_TEXT**: Supraventricular tachycardia with bundle branch block

**>>REASONING**: The broad complex tachycardia with regular rhythm suggests either SVT with BBB or VT. RBBB and RAD favor SVT with BBB. Regular rhythm excludes AF with BBB, and normal QTc excludes Torsades de pointes.

## Question #:96

**CLINICAL SCENERIO**: 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 38.2ºC 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.

**QUESTION LINE**: What is the most appropriate action with respect to antibiotic therapy?

**OPTIONS**: - 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-CHOICE LINE**: Correct answer is a.

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

**>>DESCRIPTION**: A 67-year-old with ischaemic heart disease is admitted with pyrexia. Examination shows a pulse of 96/min, temperature of 38.2ºC, BP of 104/66 mmHg, and a systolic murmur. Echocardiogram three years ago showed no valvular disease. Chest x-ray is normal, urine dipstick shows blood ++, and a petechial rash is noted. Infective endocarditis is suspected, and empirical treatment with IV amoxicillin and gentamicin is started. Two days later, blood cultures show coagulase-negative staphylococcus.

**>>OPTIONS**: a) Make no changes to treatment b) Switch to flucloxacillin c) Switch to flucloxacillin + rifampicin d) Switch to flucloxacillin + vancomycin e) Switch to flucloxacillin + vancomycin + rifampicin

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Switch to flucloxacillin

**>>REASONING**: The BNF recommends flucloxacillin monotherapy for native-valve endocarditis caused by staphylococci. Other options include unnecessary additional antibiotics.

## Question #:19

**CLINICAL SCENERIO**: A 65-year-old man with a history of previous NSTEMI presents with a presyncopal episode. He is lightheaded and feels his heart racing. On examination, he is sweaty, unwell, opens eyes to pain, localizes to pain, and makes incomprehensible sounds. His pulse is 180 bpm with BP 88/54 mmHg. ECG shows wide complex monomorphic tachycardia with prolonged QRS 150msec.

**QUESTION LINE**: What is the most appropriate next step?

**OPTIONS**: - a) Adrenaline - b) Amiodarone - c) Magnesium sulphate - d) Synchronised DC cardioversion - e) Unsynchronised DC cardioversion

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: A synchronised cardioversion is the treatment for a 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.

**>>DESCRIPTION**: A 65-year-old man with prior NSTEMI presents with presyncope, lightheadedness, and palpitations. He is sweaty, unwell, and GCS is reduced. Pulse is 180 bpm, BP 88/54 mmHg. ECG: wide complex monomorphic tachycardia (QRS 150msec).

**>>OPTIONS**: a) Adrenaline b) Amiodarone c) Magnesium sulphate d) Synchronised DC cardioversion e) Unsynchronised DC cardioversion

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Synchronised DC cardioversion

**>>REASONING**: The patient presents with unstable VT. Synchronized DC cardioversion is the preferred treatment for unstable patients with tachyarrhythmia displaying life-threatening signs. Adrenaline and unsynchronized cardioversion are for pulseless VT/VF. Magnesium is for polymorphic VT. Amiodarone is not the first line in unstable VT.

## Question #:61

**CLINICAL SCENERIO**: A 38-year-old female of Asian descent presents after a syncopal event. She has a 6-month history of fever, arthralgia, vertigo, and one syncopal event. 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 and MRA confirm a stenotic area along the subclavian. Laboratory tests reveal normocytic normochromic anaemia, elevated CRP and ESR, negative ANA and ANCA.

**QUESTION LINE**: Of the following, what is the most likely diagnosis?

**OPTIONS**: - a) Fibromuscular dysplasia - b) Ehlers-Danlos syndrome - c) Takayasu arteritis - d) Giant cell arteritis - e) Wegener’s granulomatosis

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: 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.

**>>DESCRIPTION**: A 38-year-old Asian female presents with syncope, fever, arthralgia, and vertigo for 6 months. Examination reveals diminished left radial pulse, 14 mmHg BP difference in upper extremities, and a left upper extremity bruit. MRA confirms subclavian stenosis. Labs show normocytic anemia, elevated CRP/ESR, negative ANA/ANCA.

**>>OPTIONS**: a) Ehlers-Danlos syndrome b) Fibromuscular dysplasia c) Giant cell arteritis d) Takayasu arteritis e) Wegener’s granulomatosis

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Takayasu arteritis

**>>REASONING**: Takayasu arteritis is the most likely diagnosis given the patient’s age, ethnicity, systemic symptoms (fever, arthralgia), and vascular findings (unequal pulses, bruit, subclavian stenosis). Fibromuscular dysplasia affects renal arteries, Ehlers-Danlos syndrome involves collagen defects, Giant cell arteritis affects older patients with headaches, and Wegener’s presents with upper respiratory and renal involvement and is usually ANCA positive.

## Question #:38

**CLINICAL SCENERIO**: 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.

**QUESTION LINE**: Given the likely diagnosis, which of the following would be an appropriate management option?

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

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: 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.

**>>DESCRIPTION**: A 56-year-old woman presents with periorbital xanthelasma, present for months. She denies pain, itch or discomfort.

**>>OPTIONS**: a) Topical emollient b) Topical hydrocortisone c) Topical ketoconazole d) Topical trichloroacetic acid e) Urgent referral to secondary care

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Topical trichloroacetic acid

**>>REASONING**: Topical trichloroacetic acid is a treatment option for xanthelasma. Emollients, hydrocortisone and ketoconazole are ineffective. Referral is unnecessary as xanthelasma is not of clinical concern.

## Question #:101

**CLINICAL SCENERIO**: A 53-year-old man with diabetes presents with crushing chest pain, vomiting, and diaphoresis. Initial ECG showed ST-elevation in leads II, III, and aVF, along with complete heart block, and his troponin test is positive. Three days post PCI, he develops severe dyspnoea, cold extremities, and diaphoresis, and auscultation reveals a holosystolic murmur on the left sternal edge.

**QUESTION LINE**: What is the most appropriate next step in management?

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

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: 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.

**>>DESCRIPTION**: A 53-year-old diabetic man post-PCI presents with dyspnea, cold extremities, and a holosystolic murmur after an inferior STEMI complicated by complete heart block. He is hypotensive and hypoxic.

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

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Transthoracic echocardiography

**>>REASONING**: The patient likely has cardiogenic shock and a possible ventricular septal rupture (VSR) or acute mitral regurgitation. Transthoracic echocardiography is the most appropriate next step to delineate the site and extent of the defect. CT angiography, cardiac MRI, and catheter lab are not the immediate next steps. Surgical consultation should follow the echocardiogram.

## Question #:27

**CLINICAL SCENERIO**: 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:

**QUESTION LINE**: What does the ECG show?

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

## e) Trifascicular block

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: RBBB +left anterior or posterior hemiblock + 1st-degree heart block = trifasicular block

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

**>>DESCRIPTION**: A 77-year-old man with dizziness and reduced exercise tolerance has an ECG showing:

**>>OPTIONS**: a) Bifascicular block b) ECG changes consistent with hypokalaemia c) Left bundle branch block d) Long QT syndrome e) Trifascicular block

**>>CORRECT-CHOICE LINE**: Correct answer is e.

**>>CORRECT-CHOICE\_TEXT**: Trifascicular block

**>>REASONING**: The ECG shows RBBB + left anterior hemiblock + 1st-degree heart block, indicating a trifascicular block.

## Question #:8

**CLINICAL SCENERIO**: A 16-year-old female presents with a swollen knee and a history of similar episodes. She also reports excessive bleeding after dental work and mentions that her father has a bleeding disorder. Examination reveals a hot, swollen right knee, and a knee aspirate shows a bloody effusion. Lab results show prolonged aPTT and very low Factor VIII levels.

**QUESTION LINE**: What disorder does she most likely have?

**OPTIONS**: a) Down syndrome b) Edwards syndrome c) Marfan syndrome d) Ehlers-Danlos syndrome e) Turner’s syndrome

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: 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.

**>>DESCRIPTION**: A 16-year-old female presents with a swollen knee, bleeding after dental work, and a family history of bleeding disorder. Exam reveals a hot, swollen knee with bloody effusion. Labs show prolonged aPTT and very low Factor VIII.

**>>OPTIONS**: a) Down syndrome b) Edwards syndrome c) Ehlers-Danlos syndrome d) Marfan syndrome e) Turner’s syndrome

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Turner’s syndrome

**>>REASONING**: The patient likely has hemophilia A due to prolonged aPTT and low Factor VIII. Turner’s syndrome, with its single X chromosome, increases the likelihood of X-linked recessive conditions like hemophilia A in females.

## Question #:120

**CLINICAL SCENERIO**: A 74-year-old man with a history of hypertension and atrial fibrillation, on amlodipine and apixaban, presents with collapse and pre-syncope. A 24-hour tape shows sustained atrial fibrillation with bradycardia (minimum heart rate 20 bpm) associated with pre-syncope. The admission ECG shows slow atrial fibrillation (24 bpm). He recovers, and a permanent pacemaker is planned.

**QUESTION LINE**: What is the appropriate mode to programme the pacing system?

**OPTIONS**: a) AAI b) AOO c) OOO d) VAT e) VVI

**CORRECT-CHOICE LINE**: e

**REASONING**: VVI pacemakers are useful for pure sustained AF. In this mode, the ventricle is sensed and paced. AAI is inappropriate because the atria are not functioning. AOO, OOO, and VAT are also incorrect.

**>>DESCRIPTION**: A 74-year-old man with hypertension, atrial fibrillation (on amlodipine and apixaban), presents with collapse, pre-syncope, and bradycardia. A permanent pacemaker is planned.

**>>OPTIONS**: a) AAI b) AOO c) OOO d) VAT e) VVI

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: VVI

**>>REASONING**: VVI is the correct pacing mode for sustained atrial fibrillation as it paces the ventricle in the absence of intrinsic ventricular activity. Incorrect options: AAI (atria not functioning), AOO, OOO (not suitable for permanent pacing), and VAT (requires functional atria).

## Question #:102

**CLINICAL SCENERIO**: A 55-year-old man presents with a 2-hour history of central chest pain and dizziness, with little improvement after taking paracetamol and ibuprofen. His past medical history includes hypertension treated with amlodipine 10mg daily. He smokes 20 cigarettes/day and drinks 12 units of alcohol per week. An ECG is taken.

**QUESTION LINE**: What is the most likely diagnosis?

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

**CORRECT-CHOICE LINE**: This patient has Wellen’s syndrome .

**REASONING**: 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 STsegment 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 cardiacsounding 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.

**>>DESCRIPTION**: A 55-year-old man presents with 2-hour central chest pain and dizziness, unrelieved by analgesics. He has hypertension, smokes, and drinks alcohol. An ECG was performed.

**>>OPTIONS**: a) Brugada syndrome b) Hypertrophic obstructive cardiomyopathy c) Posterior myocardial infarction d) Pulmonary embolus with right heart strain e) Wellen’s syndrome

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Wellen’s syndrome

**>>REASONING**: The patient’s ECG findings and chest pain history are consistent with Wellen’s syndrome, indicating critical ischemia of the left anterior descending artery. Brugada syndrome, HOCM, posterior MI, and pulmonary embolism do not fit the ECG findings presented.

## Question #:53

**CLINICAL SCENERIO**: A 40-year-old woman presents with a 10-day history of intermittent palpitations, worsening over the last 8 hours with associated lightheadedness. She has no significant past medical history and takes no regular medications. On examination, she appears clammy and warm. Her heart rate is 72 beats/min, and her blood pressure is 125/83mmHg. An ECG is taken.

**QUESTION LINE**: What is the most likely diagnosis?

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

**CORRECT-CHOICE LINE**: This patient has Wolff-Parkinson White syndrome

**REASONING**: 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.

**>>DESCRIPTION**: A 40-year-old woman presents with palpitations and lightheadedness. Her heart rate is 72 bpm, and BP is 125/83 mmHg. ECG shows abnormalities.

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

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Wolff-Parkinson white syndrome

**>>REASONING**: The patient’s ECG shows a delta wave and shortened PR interval, characteristic of Wolff-Parkinson White syndrome. Accelerated junctional rhythm presents with a narrow complex rhythm, atrial flutter presents with a saw-tooth pattern, and SVT lacks delta waves.