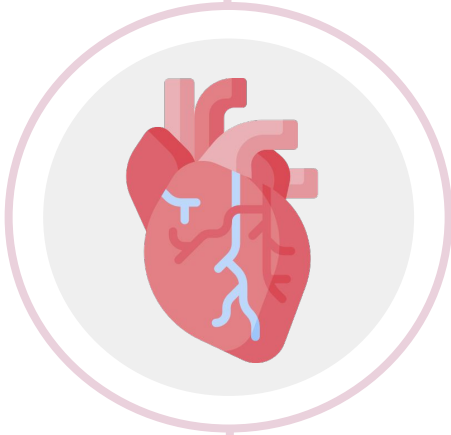




Practice file



Done by :

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Acute coronary syndrome

Q1: A 65-year-old man presents with central crushing chest pain for the first time. He is transferred immediately to the closest cardiac unit to undergo a primary percutaneous coronary intervention. There is thrombosis of the left circumflex artery only. Angioplasty is carried out and a drug-eluting stent is inserted. What are the most likely changes to have occurred on ECG during admission?

- A. ST depression in leads V1–4
- B. ST elevation in leads V1–6
- C. ST depression in leads II, III and AVF
- D. ST elevation in leads V5–6
- E. ST elevation in leads II, III and AVF

Explanation:

Leads	Area	Supplied by
V1–2	Anterior	LCA: Diagonal branch of LAD
V3–4	Septal	LCA: Septal branch of LAD
V5–6	Lateral	LCA: Left circumflex artery
V1–6	Anterolateral	LCA: Left main stem disease
II, III, aVF	Inferior	RCA: Posterior descending branch

Q2: A 61-year-old man presents with a 2-hour history of moderately severe retrosternal chest pain, which does not radiate and is not affected by respiration or posture. He complains of general malaise and nausea, but has not vomited. His ECG shows ST segment depression and T wave inversion in the inferior leads. Troponin levels are not elevated. He has already been given oxygen, aspirin and intravenous GTN; he is an occasional user of sublingual GTN and takes regular bisoprolol for stable angina. What would be the most appropriate next step in his management?

- A. IV low-molecular weight heparin
- B. Thrombolysis with alteplase
- C. IV nicardapine
- D. Angiography with stenting
- E. Oral clopidogrel

Explanation: IV low-molecular weight heparin (A) should almost always form part of the immediate management of an acute coronary syndrome, such as in this case. If the patient were not already on a beta-blocker this would also be given. At this point it would be classified as unstable angina as there is no evidence of actual tissue damage. Thrombolysis (B) probably worsens outcomes in this situation. In the absence of actual thrombus, calcium channel blockers (C) have no proven benefit and at this stage clopidogrel (E) is not indicated, although it would be if stenting (D) is carried out. If the facilities are available, that would be the next step after anticoagulation.

Q3: A 46-year-old man develops sudden severe central chest pain after lifting heavy cases while moving house. The pain radiates to the back and both shoulders but not to either arm. His BP is 155/90 mmHg, pulse rate is 92 beats per minute and the ECG is normal. He is distressed and sweaty, but not nauseated. What would you consider the most likely diagnosis?

- A. Pneumothorax
- B. MI
- C. Pulmonary embolism
- D. Aortic dissection
- E. Musculoskeletal pain

Explanation: The most likely diagnosis, and the one that must be most urgently excluded, is an aortic dissection (D). The location does not indicate a pneumothorax (A), the symptoms and ECG are against an MI (B) and the pain seems too severe for a pulmonary embolus (C), or pain of musculoskeletal origin (E). Chest x-ray may show widening of the aorta, and CT and MRI scans may be diagnostic. If confirmed, BP reduction and dampening of the aortic systolic wave by beta-blockade is indicated and urgent surgical intervention should be considered.

Q4: A 55-year-old man has just arrived in accident and emergency complaining of 20 minutes of central crushing chest pain. Which feature is most indicative of myocardial infarction at this moment in time?

- A. Inverted T waves
- B. ST depression
- C. ST elevation
- D. Q waves
- E. Raised troponin

Explanation: Acute coronary syndrome is a spectrum of cardiac ischaemia-infarction determined by the presence of two out of three factors: chest pain, ECG changes and cardiac enzyme rise. Depending on these results, patients will fall into one of the following categories: unstable angina, NSTEMI or STEMI. Inverted T waves (A) and ST depression (B) are signs of ischaemia. ST elevation, Q waves and raised troponin are indicative of infarction. Initially, 'ST elevation' or 'non ST elevation' ECG changes are used to stratify each patient's risk as the results of blood tests for troponin levels (E) (which should be carried out 12 hours after the pain started) are not known and Q waves have not had time to develop. ST elevation (C) is a very good predictor of imminent infarction (positive troponin). However, if this patient is treated quickly enough with thrombolysis or primary PCI, infarction can be avoided. A patient with STEMI who goes on to have negative troponin is termed to have had an 'aborted MI'. Q waves (D) (indicating full-thickness MI) take time to develop, so 'Q wave' or 'non Q wave' MI is a diagnosis given on discharge.

Acute coronary syndrome

Q5: A 66-year-old woman presents to accident and emergency with a 2-day history of shortness of breath. The patient notes becoming progressively short of breath as well as a sharp pain in the right side of the chest which is most painful when taking a deep breath. The patient also complains of mild pain in the right leg, though there is nothing significant on full cardiovascular and respiratory examination. Heart rate is 96 and respiratory rate is 12. The patient denies any weight loss or long haul flights but mentions undergoing a nasal polypectomy 3 weeks ago. The most likely diagnosis is:

- A. Muscular strain
- B. Heart failure
- C. Pneumothorax
- D. Angina
- E. Pulmonary embolism

Explanation: This patient is most likely suffering from a pulmonary embolism (E), defined as an occlusion of the pulmonary vasculature by a thrombus causing an area of lung that is ventilated but not perfused. Patients most often complain of shortness of breath, pleuritic chest pain and haemoptysis. Clinical signs can include a pleural rub, coarse crackles and atrial fibrillation. In massive pulmonary embolism there can be a raised JVP, respiratory rate, heart rate and hypotension. The Geneva scoring system (see below) is useful for predicting the risk of a pulmonary embolism: a score of ≤ 3 (mild), 4–10 (moderate) and ≥ 11 (high). Muscular strain (A) typically occurs on movement and is not associated with shortness of breath or leg pain and there is usually an indicator of injury or a preceding stressor. Heart failure (B) is unlikely due to the acute presentation of symptoms which tend to occur more insidiously and can be associated with bilateral leg oedema, murmurs, orthopnoea or hepatomegaly, among others. A pneumothorax (C) can present with a similar pleuritic chest pain that occurs in an embolism, however, there is no association with limb pain and a respiratory examination is likely to reveal hyper-resonance. Angina (D) is typically described as a dull or crushing chest pain in the centre of the chest, patients have risk factors such as diabetes, hyperlipidaemia, obesity, smoking and hypertension.

Q6: A 60-year-old man presents to accident and emergency with a 3-day history of increasingly severe chest pain. The patient describes the pain as a sharp, tearing pain starting in the centre of his chest and radiating straight through to his back between his shoulder blades. The patient looks in pain but there is no pallor, heart rate is 95, respiratory rate is 20, temperature 37°C and blood pressure is 155/95 mmHg. The most likely diagnosis is:

- A. Myocardial infarction
- B. Myocardial ischaemia
- C. Aortic dissection
- D. Pulmonary embolism
- E. Pneumonia

Explanation: All of the answer options can present as central chest pain, however the patient describes a very typical description of an aortic dissection (C), usually a severe, tearing pain that radiates toward the back though this can be to the jaw depending on the location of the dissection. An MI (A) is typically described as severe, crushing chest pain with an acute onset, this patient has been suffering from a 3-day history of chest pain which makes an infarction unlikely. Although myocardial ischaemia (B), i.e. angina, can occur for a longer period of time they tend not radiate to the back but more toward the jaw, arms or epigastrium, and again are described as crushing in nature rather than tearing. A pulmonary embolism (D) typically presents with pleuritic chest pain, cough and haemoptysis which are not present in this patient, or preceding risk factors such as long haul travel or surgery. Pneumonia (E) is associated with fever and productive coughing.

Q7: A 49-year-old man is rushed to accident and emergency complaining of a 20-minute history of severe, crushing chest pain. After giving the patient glyceryl trinitrate (GTN) spray, he is able to tell you he suffers from hypertension and type 2 diabetes and is allergic to aspirin. The most appropriate management is:

- A. Aspirin
- B. Morphine
- C. Heparin
- D. Clopidogrel
- E. Warfarin

Explanation: NICE guideline protocols state that in a patient with suspected MI, pain relief in the form of GTN spray or morphine should be administered. Since the patient has had an adequate response to GTN spray, further pain relief in the form of morphine (B) is unnecessary. In patients who are not allergic, 300 mg of aspirin is recommended and ideally should be given in the ambulance. However, if the patient is allergic to aspirin (A) it should not be given since an anaphylactic reaction would compromise the patient's airway and does not overrule the harm from a possible MI. Although heparin (C) and warfarin (E) would provide good anticoagulant cover, they are slower to act and current guidance advises clopidogrel monotherapy (D) in those patients allergic to aspirin.

Acute coronary syndrome

Q8: A 44-year-old woman attends her local accident and emergency department with a history of at least six months of frequent central chest pain in the early morning or during the night. She had no chest pain on exertion. This had been a particularly severe attack, lasting over 2 hours. Her pulse rate is 84/minute in sinus rhythm, and blood pressure is 134/86 mmHg. The ECG shows anterior ST segment elevation, but troponin levels do not rise. Subsequent coronary angiography is normal. What is the most likely diagnosis?

- A. MI
- B. Stable angina
- C. Unstable angina
- D. Anxiety
- E. Variant angina

Explanation: Variant angina, sometimes called Prinzmetal's angina (E), of which this is a typical presentation. Its mechanism is controversial and even its existence has been questioned. The general view is that it is due to vasospasm in small coronary arteries and this is likely to respond to the effects of nitrates and calcium channel blockers such as verapamil. Beta-blockers are not effective and in theory could make it worse by aggravating vasoconstriction, but whether this actually happens is also controversial.

Q9: A 56-year-old man presents to the accident and emergency department with a 2-hour history of central chest pain radiating to the left arm. He is anxious, nauseated and sweaty. His pulse rate is 120/minute in sinus rhythm and the ECG reveals ST elevation in leads II, III and aVF. The troponin level is significantly raised. This is certainly acute MI. Which is the most likely coronary vessel to be occluded?

- A. Circumflex artery
- B. Left anterior descending artery
- C. Right coronary artery
- D. Left main coronary artery
- E. Posterior descending artery

Explanation: The answer is right coronary artery (C). This is the artery that supplies the inferior and posterior aspects of the left ventricle. The circumflex artery (A) would affect the anterolateral territory (leads I, aVL, V5-6). The left anterior descending artery (B) supplies the septum (leads V1-V4). The left main coronary artery (D) would include the circumflex artery and left anterior descending artery territory. The posterior descending artery (E) affects a limited portion of the posterior wall, and is associated with tall R waves in V1-2.

Q10: You have been asked to evaluate a 42-year-old white male smoker who presented to the emergency department with sudden onset of crushing substernal chest pain, nausea, diaphoresis, and shortness of breath. His initial ECG revealed ST segment elevation in the anteroseptal leads. Cardiac enzymes were normal. The patient underwent emergent cardiac catheterization, which revealed only a 25% stenosis of the left anterior descending (LAD) artery. No percutaneous intervention was performed. Which of the following interventions would most likely reduce his risk of similar episodes in the future?

- A. Placement of a percutaneous drug-eluting coronary artery stent
- B. Placement of a percutaneous non-drug-eluting coronary artery stent
- C. Beginning therapy with an ACE inhibitor
- D. Beginning therapy with a beta-blocker
- E. Beginning therapy with a calcium-channel blocker

Explanation: This patient's presentation and minimal coronary artery disease are most consistent with Prinzmetal variant angina. Prinzmetal angina is caused by severe spasm of an epicardial coronary artery. The area of vasospasm is often near a non hemodynamically significant atherosclerotic lesion. Patients tend to be smokers and are often younger than patients who present with atherosclerotic coronary artery disease. In this case, the patient's mild LAD stenosis does not explain the degree of ischemia evidenced by the ST segment elevation. Percutaneous intervention has not been shown to be useful in management of Prinzmetal angina, as the culprit is transient vasospasm rather than fixed obstruction. Calcium-channel blockers are the mainstay of therapy to prevent recurrence of spasm. ACE inhibitors and beta-blockers do not prevent acute vasospasm. Of course, the patient should also be counseled to abstain from smoking.

Acute coronary syndrome

Q11: A 55-year-old man comes to the emergency department because of a dry cough and severe chest pain beginning that morning. Two months ago, he was diagnosed with inferior wall myocardial infarction and was treated with stent implantation of the right coronary artery. He has a history of hypertension and hypercholesterolemia. His medications include aspirin, clopidogrel, atorvastatin, and enalapril. His temperature is 38.5°C (101.3°F), pulse is 92/min, respirations are 22/min, and blood pressure is 130/80 mm Hg. Cardiac examination shows a high-pitched scratching sound best heard while sitting upright and during expiration. The remainder of the examination shows no abnormalities. An ECG shows diffuse ST elevations. Serum studies show a troponin I of 0.2 ng/mL. Which of the following is the most likely cause of this patient's symptoms?

- A. Constrictive pericarditis
- B. Early infarct-associated pericarditis
- C. Reinfarction
- D. Cardiac tamponade
- E. Dressler syndrome

Explanation: Postmyocardial infarction syndrome (Dressler syndrome), a type of postcardiac injury syndrome, usually occurs 1–10 weeks after myocardial infarction due to immune-complex mediated damage (type III hypersensitivity reaction) in the pericardium. The exact mechanism of immune-complex formation is not completely understood, but the release of neo-antigens from the infarcted myocardium is suspected to play a role. Patients typically present with features of acute pericarditis such as fever, tachypnea, chest pain that is worse during inspiration and relieved by leaning forward, a dry cough (due to inflammation of the mediastinal pleura adjoining the pericardium), a pericardial rub, increased troponin levels, and diffuse ST elevations on ECG.

Q12: A 64-year-old man is brought to the emergency department 30 minutes after the sudden onset of substernal chest pain and nausea while playing tennis. He has hypertension and hyperlipidemia. Current medications include hydrochlorothiazide and atorvastatin. He does not smoke cigarettes or drink alcohol. He is diaphoretic and distressed. Pulse is 110/min, respirations are 31/min, and blood pressure is 85/55 mm Hg. Examination shows jugular venous distention. Crackles are heard over the lower lung fields bilaterally. Capillary refill time is delayed; the extremities are cool to touch. A 12-lead ECG shows ST elevations in leads II, III, and aVF. Which of the following is the most appropriate next step in management?

- A. Perform a percutaneous transluminal coronary angioplasty
- B. Administer sublingual nitroglycerin
- C. Start single antiplatelet therapy
- D. Start intravenous metoprolol therapy
- E. Perform a coronary artery bypass graft surgery

Explanation: Percutaneous transluminal coronary angioplasty (PCI) is the preferred method of revascularization in patients with myocardial infarction. Emergency revascularization is the most important step in the management of patients with acute STEMI because it restores the patency of the occluded coronary artery, limiting a further increase in the size of the infarction. Since early reperfusion is associated with improved clinical outcomes, door-to-PCI time should be < 90 minutes. Fibrinolysis can be considered in patients with acute STEMI if PCI fails, is unavailable, or cannot be performed ≤ 120 minutes after first medical contact and symptom onset is < 12 hours.

Dual antiplatelet and anticoagulation therapy should also be initiated as soon as possible. Therapy with beta blockers is associated with better outcomes (reduced infarct size and decreased mortality) and must be initiated within the first 24 hours of admission unless there are contraindications (e.g., cardiogenic shock, hypotension, features of heart failure). Patients should also be started on high-intensity statin therapy regardless of baseline cholesterol levels.

Arrhythmias

Q1: A 79-year-old woman is admitted to the coronary care unit (CCU) with unstable angina. She is started on appropriate medication to reduce her cardiac risk. She is hypertensive, fasting glucose is normal and cholesterol is 5.2. She is found to be in atrial fibrillation. What is the most appropriate treatment?

- A. Aspirin and clopidogrel
- B. Digoxin
- C. Cardioversion
- D. Aspirin alone
- E. Warfarin

Explanation: Ideally this patient should be started on antihypertensives, a beta blocker and a statin. There is no indication for hypoglycaemics at present. There is no indication that this is acute atrial fibrillation and she does not seem to be compromised in a female of this age, cardioversion (C) is unlikely to be successful. She should be rate-controlled but the beta blockade is more appropriate in light of her ischaemic heart disease. Whether to start anticoagulation (A) is a decision that has to be tailor-made for each individual patient. The CHAD2 score is a quick and dirty but very useful way of predicting risk of subsequent stroke as a result of atrial fibrillation and helps guide the prescription of prophylactic antiplatelets or anticoagulants. Other factors, such as ease of taking and monitoring warfarin, risk of falls and important risk factors, such as vascular disease, should be taken into account. A score of 0 is low risk (2 per cent of patients/year will have a stroke without treatment), aspirin is considered adequate. A score of 1 is moderate risk (3 per cent annual stroke risk) and either warfarin or aspirin (D) is indicated according to the individual. A score of two or above (>4 per cent annual stroke risk) is classified as high risk and warfarin (E) should be started unless there are clear contraindications. A patient with a full-house of risk factors (scores 6) has an almost 20 per cent chance of stroke/year.

Q2: A 21-year-old man is on his way home from a party when he experiences the sudden onset of rapid palpitations. He feels uncomfortable but not short of breath and has no chest pain. He goes to the nearest accident and emergency department, where he is found to have a supraventricular tachycardia (SVT) at a rate of 170/minute. Carotid sinus massage produced transient reversion to sinus rhythm, after which the tachycardia resumed. What would be the next step in your management?

- A. Repeat carotid sinus massage
- B. IV verapamil
- C. IV propranolol
- D. IV adenosine
- E. Synchronized DC cardioversion

Explanation: IV adenosine (D) has a very high likelihood of success, with rapid onset and offset. It may cause very brief chest pain (which is not ischaemic) and very occasionally bronchospasm. Verapamil (B) and beta-blockers (C) may also be effective but have a longer duration of action which is unnecessary here, may cause excessive bradycardia, and are in any case less effective than adenosine. If the patient has severe haemodynamic compromise, DC cardioversion (E) could be considered but would be excessive here. Carotid sinus massage (A) is likely to remain ineffective. SVT is common in young people and may be associated with excessive nicotine, caffeine and alcohol and patients should be advised about this, although they may not take much notice!

Q3: A 29-year-old woman goes to see her GP complaining of fatigue and palpitations. She says she has also lost weight, though without dieting. On examination, her pulse rate is approximately 120/min and irregularly irregular. Her blood pressure is 142/89 mmHg and her body mass index is 19. There are no added cardiac sounds. The ECG confirms the diagnosis of atrial fibrillation. What would you suggest as the most useful next investigation.

- A. Thyroid function tests (TSH, free T4)
- B. ECG
- C. Chest x-ray
- D. Full blood count
- E. Fasting blood sugar

Explanation: This clinical picture strongly suggests thyrotoxicosis and therefore the correct answer is TFTs (A). This is probably the most common cause of atrial fibrillation in a young person, particularly in the absence of valve disease. No doubt an ECG (B) will be carried out to exclude this but there are no physical signs to suggest an abnormality: the mitral valve is the one most likely to be associated with atrial fibrillation, probably at least in part because of stretching of the left atrium. It is certain that full blood count (D) and fasting blood sugar (E) will be carried out routinely, while a chest x-ray (C) may form part of the search for a retrosternal goitre if there is any indication of this.

Arrhythmias

Q4: A 62-year-old male presents with palpitations, which are shown on ECG to be atrial fibrillation with a ventricular rate of approximately 130/minute. He has mild central chest discomfort but is not acutely distressed. He first noticed these about 3 hours before coming to hospital. As far as is known this is his first episode of this kind. Which of the following would you prefer as first-line therapy?

- A. Anticoagulate with heparin and start digoxin at standard daily dose
- B. Attempt DC cardioversion
- C. Administer bisoprolol and verapamil, and give warfarin
- D. Attempt cardioversion with IV flecainide
- E. Wait to see if there is spontaneous reversion to sinus rhythm

Explanation: The onset of the arrhythmia is recent, and there is a good chance of successful cardioversion (B) at this point without the need for anticoagulation. Conservative management (E) is also reasonable, though the patient is in some discomfort. 'Chemical' cardioversion (D) may be somewhat less likely to succeed than DC cardioversion but may be preferred by the patient. Digoxin (A) may eventually control resting, but not ambulant heart rate, but would probably take several days before it did so. Option (C) is certainly suitable in cases of persistent or permanent atrial fibrillation where it is decided to opt for rate control.

Q5: In the ICU, a patient suddenly becomes pulseless and unresponsive, with cardiac monitor indicating ventricular tachycardia. The crash cart is immediately available. What is the best first therapy?

- A. Amiodarone 150-mg IV push
- B. Lidocaine 1.5-mg/kg IV push
- C. Epinephrine 1-mg IV push
- D. Defibrillation

Explanation: The standard approach to ventricular fibrillation or hypotensive ventricular tachycardia involves defibrillation (with 200 J, then 300, then 360 if using a monophasic defibrillator; 200 J maximum if using a biphasic defibrillator), followed by epinephrine if needed. Therapy with lidocaine, amiodarone, or procainamide may be warranted if prior interventions fail. Magnesium sulfate may be given in torsade de pointes or when arrhythmia caused by hypomagnesemia is suspected.

Q6: A 36-year-old man presents with the sensation of a racing heart. His blood pressure is 110/70, respiratory rate 14/minute, and O2 saturation 98%. His ECG shows a narrow QRS complex tachycardia with rate 180, which you correctly diagnose as paroxysmal atrial tachycardia. Carotid massage and Valsalva maneuver do not improve the heart rate. Which of the following is the initial therapy of choice?

- A. Adenosine 6-mg rapid IV bolus
- B. Verapamil 2.5 to 5 mg IV over 1 to 2 minutes
- C. Diltiazem 0.25-mg/kg IV over 2 minutes
- D. Digoxin 0.5 mg IV slowly
- E. Electrical cardioversion at 50 J

Explanation: Adenosine, with its excellent safety profile and extremely short half-life, is the drug of choice for supraventricular tachycardia. The initial dose is 6 mg. A dose of 12 mg can be given a few minutes later if necessary. Verapamil is the next alternative; if the initial dose of 2.5 to 5 mg does not yield conversion, one or two additional boluses 10 minutes apart can be used. Diltiazem and digoxin may be useful in rate control and conversion, but have a slower onset of action. Electrical cardioversion is reserved for hemodynamically unstable patients. Lidocaine is useful in ventricular (not supraventricular) arrhythmias.

Q7: A 60-year-old woman develops chest pain, respiratory distress, and confusion after right hip replacement surgery. She is confused and appears in respiratory distress. Blood pressure is 80/50, heart rate of 155/minute. ECG reveals atrial fibrillation. Which of the following is the best management of this patient's arrhythmia?

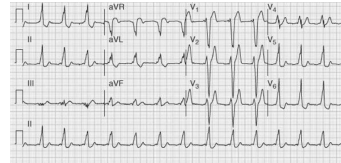
- A. Immediate defibrillation
- B. Intravenous amiodarone
- C. Intravenous metoprolol
- D. Intravenous adenosine
- E. Immediate electric cardioversion

Explanation: This woman has hemodynamically unstable atrial fibrillation which requires immediate electrical cardioversion. Chemical cardioversion with metoprolol or adenosine is not appropriate because these drugs may further worsen her hypotension. Though embolic stroke is a concern during cardioversion, the benefit of promptly stabilizing the patient's hemodynamic compromise outweighs this risk. With atrial fibrillation, synchronized cardioversion (which delivers an electric shock timed to the R wave of QRS complex on the EKG) is preferred. Defibrillation using an unsynchronized electric shock of 360 J is used in ventricular fibrillation and pulseless ventricular tachycardia. In the absence of hemodynamic compromise, the initial goals in the management of atrial fibrillation are (1) ventricular rate control, and (2) prevention of embolic stroke by anticoagulation. Ventricular rate control is best established with beta-blockers and/or calcium channel blocking agents (such as verapamil or diltiazem). These can be given by oral or intravenous route depending on the ventricular rate and clinical status of the patient. Digoxin may be added for rate control. The use of antiarrhythmic such as amiodarone can be instituted once sinus rhythm has been established or in anticipation of cardioversion in an attempt to maintain sinus rhythm. In the long-term management of atrial fibrillation, clinical trials have established no advantage for rhythm control over rate control.

Arrhythmias

Q8: An 18-year-old man military recruit reports several episodes of palpitation and syncope over the past several years. Physical examination is unremarkable. His ECG is shown below. What is the most likely diagnosis?

- A. Prior myocardial infarction secondary to coronary artery disease
- B. Congenital prolonged QT syndrome
- C. Hypertrophic obstructive cardiomyopathy (HOCM)
- D. Preexcitation syndrome (Wolff-Parkinson-White)
- E. Rheumatic mitral stenosis



Explanation: The ECG reveals shortened PR interval and a delta wave causing widening of the QRS. The delta wave is a “slurring” of the upstroke of the R wave caused by the early depolarization of ventricular myocardium. This is consistent with an accessory conduction pathway or WPW. The aberrant conduction tissue bypasses the normal AV node (hence the PR interval of < 0.20 seconds); it leads the electrical impulse directly to the ventricle (bypassing the His-Purkinje fibers and widening the QRS complex). Q waves are not infrequent and can be mistaken for evidence of prior MI. Myocardial infarction, however, does not cause shortened PR interval or delta wave. This patient’s QT interval is normal (< 0.45 second). Patients with HOCM usually have voltage criteria for left ventricular hypertrophy and prominent ST/T-wave changes but may also have large Q waves owing to hypertrophy of the septum. Rheumatic mitral stenosis would cause left atrial enlargement and perhaps atrial fibrillation, not the changes seen on this ECG.

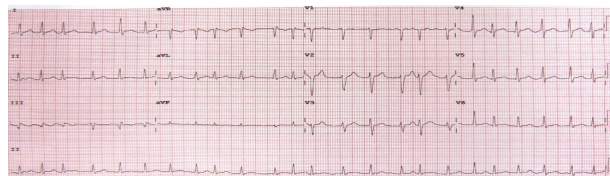
Q9: A 21-year-old woman comes to the physician because of a 2-day history of intermittent palpitations. She is a junior in college, and the symptoms began when she returned to her dormitory after a 4-hour bus trip from home. The palpitations have worsened since then and occur more frequently. She does not have any chest pain or shortness of breath. The patient has smoked 5 cigarettes daily for the past 3 years. She drinks 4–6 alcoholic beverages with friends on weekends and tried marijuana once in her freshman year. She is sexually active with her boyfriend and takes an oral contraceptive pill. She is in no apparent distress. Her pulse is 100/min and irregular, respirations are 25/min, and blood pressure is 140/70 mm Hg. The pupils are 4 mm bilaterally and reactive to light. Physical examination shows a fine tremor in both hands and warm extremities. There is 1+ nonpitting bilateral pretibial edema. Lungs are clear to auscultation. ECG is shown. Which of the following is the most appropriate initial step in management?

- A. Measure serum TSH levels
- B. Obtain urine toxicology screening
- C. Perform CT pulmonary angiography
- D. Perform transthoracic echocardiography
- E. Observation and follow-up in 1 week

Explanation: All patients with new-onset symptomatic Afib should be evaluated for potential causes of arrhythmia. Measurement of serum TSH levels is the best initial step in the diagnosis of hyperthyroidism, a common cause of symptomatic Afib that can manifest with hypertension with wide pulse pressure (140/70 mm Hg), tremor, and warm skin, as seen in this patient. Individuals with Graves disease, the most common cause of hyperthyroidism, can also develop pretibial myxedema, which manifests as nonpitting bilateral pretibial edema, also seen here. Given that this patient has classic features of hyperthyroidism and Graves disease, her TSH levels should be measured at this time.

Q10: A 62-year-old man with a 5-year history of chronic obstructive pulmonary disease comes to the physician for a follow-up examination. He has had episodic palpitations over the past week. His only medication is a tiotropium-formoterol inhaler. His pulse is 140/min and irregular, respirations are 17/min, and blood pressure is 116/70 mm Hg. Pulse oximetry on room air shows an oxygen saturation of 95%. The lungs are clear to auscultation. Cardiac examination shows no murmurs, rubs, or gallops. Serum concentrations of electrolytes, thyroid-stimulating hormone, and cardiac troponins are within the reference range. An electrocardiogram is shown. Which of the following is the most appropriate next step in management?

- A. Synchronized cardioversion
- B. Procainamide therapy
- C. Verapamil therapy
- D. Transesophageal echocardiography
- E. Propranolol therapy



Explanation: Verapamil is a first-line therapy to normalize the ventricular heart rate in patients with stable Afib. This patient’s normal TSH, electrolyte, and troponin levels rule out reversible or treatable causes of Afib. Since this patient is symptomatic with intermittent palpitations and tachycardia, the next appropriate step is rate control. The target resting heart rate for patients with Afib is < 110/min for asymptomatic patients and < 80/min for symptomatic patients. Furthermore, the need for anticoagulation should be evaluated in all patients with Afib. Cardioversion can be considered as an alternative to rate control, but this procedure carries a high risk of thromboembolism. Therefore, patients like this one who have been in Afib for > 48 hours should receive prophylactic anticoagulation therapy for 3 weeks before cardioversion. This period can be shortened if atrial thrombi are excluded by transesophageal echocardiography.

Rheumatic heart disease

Q1: An 18-year-old man complains of fever and transient pain in both knees and elbows. The right knee was red and swollen for 1 day during the week prior to presentation. On physical examination, the patient has a low-grade fever. He has a III/VI, high-pitched, apical systolic murmur with radiation to the axilla, as well as a soft, mid-diastolic murmur heard at the base. A tender nodule is palpated over an extensor tendon of the hand. There are pink erythematous lesions over the abdomen, some with central clearing. The following laboratory values are obtained:

- A. Blood cultures
- B. Antistreptolysin O antibody
- C. Echocardiogram
- D. Antinuclear antibodies
- E. Creatine kinase

Hct	42%
WBC	12,000/ μ L with 80% polymorphonuclear leukocytes, 20% lymphocytes
ESR	60 mm/h

Explanation: This 18-year-old presents with features of rheumatic fever. Rheumatic fever is diagnosed according to the Jones criteria. Evidence of recent streptococcal infection plus two major manifestations or one major and two minor manifestations satisfy the Jones criteria for diagnosis of acute rheumatic fever. Major criteria include carditis, polyarthritides, chorea, erythema marginatum, and subcutaneous nodules. Minor manifestations include fever, polyarthralgia, elevated erythrocyte sedimentation rate, and PR prolongation on ECG. This patient's clinical manifestations include arthritis, fever, and murmur (consistent with mitral regurgitation). The rash suggests erythema marginatum, and a subcutaneous nodule is noted. Rheumatic subcutaneous nodules are pea sized and usually overlie extensor tendons. The rash is usually pink with clear centers and serpiginous margins. Laboratory data include an elevated erythrocyte sedimentation rate. The ECG shows evidence of first-degree AV block. An antistreptolysin O antibody is necessary to document prior streptococcal infection. Endocarditis (for which blood cultures and an echocardiogram would be ordered) might cause fever, joint symptoms, and the tender nodule but would not account for the diastolic murmur or the characteristic skin lesion. There is no evidence of lupus or myocardial infarction.

Q2: A 12-year-old girl is brought to the physician by her mother because of high fever and left ankle and knee joint swelling. She had a sore throat 3 weeks ago. There is no family history of serious illness. Her immunizations are up-to-date. She had an episode of breathlessness and generalized rash when she received dicloxacillin for a skin infection 2 years ago. She appears ill. Her temperature is 38.8°C (102.3°F), pulse is 87/min, and blood pressure is 98/62 mm Hg. Examination shows left ankle and knee joint swelling and tenderness; range of motion is limited. Breath sounds over both lungs are normal. A grade 3/6 holosystolic murmur is heard best at the apex. Abdominal examination is normal. Which of the following is the most appropriate pharmacotherapy?

- A. Vancomycin
- B. Amoxicillin
- C. Clarithromycin
- D. Doxycycline
- E. Ciprofloxacin

Explanation: Clarithromycin, a macrolide, is the antibiotic of choice for rheumatic fever in patients with hypersensitivity to beta-lactam antibiotics, as seen in this patient. Other than antibiotics, treatment consists of bed rest and NSAIDs. Additionally, an echocardiogram should be performed to evaluate for mitral or aortic regurgitation.

Valvular heart disease

Q1: A 32-year-old woman attends her GP for a routine medical examination and is noted to have a mid-diastolic murmur with an opening snap. Her blood pressure is 118/71 mmHg and the pulse is regular at 66 beats per minute. She is entirely asymptomatic and chest x-ray and ECG are normal. What would be the most appropriate investigation at this point?

- A. Echocardiography
- B. Anti-streptolysin O titre
- C. Cardiac catheterization
- D. Thallium radionuclide scanning
- E. Colour Doppler scanning

Explanation: This is typical mitral stenosis and the correct answer is echocardiography

(A). Colour Doppler scanning (E) would almost certainly follow to assess flow and pressure. Cardiac catheterization (C) may be performed prior to surgery, while thallium radionuclide scanning (D) is not relevant. The antistreptolysin O titre (B) can confirm streptococcal infection and the presumed rheumatic fever responsible for the lesion, but only around the time it occurs. It would yield no useful information years later, as would be the case here.

Q2: A patient is admitted with pneumonia. A murmur is heard on examination. What finding points to mitral regurgitation?

- A. Murmur louder on inspiration
- B. Murmur louder with patient in left lateral position
- C. Murmur louder over the right 2nd intercostal space midclavicular line
- D. Corrigan's sign
- E. Narrow pulse pressure

Explanation: A murmur heard loudest on inspiration (A) points to a right-sided valve lesion. The right intercostal space midclavicular line (C) is the anatomical landmark for the aortic valve. The mitral area is over the apex. A murmur louder with the patient in the left lateral position (B) (as opposed to leaning forward) is associated with mitral lesions. If heard, you should determine whether the murmur radiates to the axilla. Corrigan's sign (D) (visibly exaggerated pulsating carotids) is one of the many signs of a hyperdynamic circulation associated with aortic regurgitation (including de Mussetts, Traubes, Quinkes, Duroziez and a whole host of others). A narrow pulse pressure (E) is a sign of aortic stenosis.

Q3: A 49-year-old woman presents with increasing shortness of breath on exertion developing over the past three months. She has no chest pain or cough, and has noticed no ankle swelling. On examination, blood pressure is 158/61 mmHg, pulse is regular at 88 beats per minute and there are crackles at both lung bases. There is a decrescendo diastolic murmur at the left sternal edge. What is the most likely diagnosis?

- A. Aortic regurgitation
- B. Aortic stenosis
- C. Mitral regurgitation
- D. Mitral stenosis
- E. Tricuspid regurgitation

Explanation: This is a typical clinical scenario for an aortic regurgitation (A), with early cardiac failure. Note the wide pulse pressure, and it is also usual for the pulse to be rapidly collapsing. The only lesion producing a diastolic murmur, among those listed, is of course mitral stenosis (D). No other valve abnormality (B), (C) or (E) produces a wide pulse pressure as seen here, but remember that in older people, almost always over the age of 60, similarly wide or even wider pulse pressures may be noted. This would be due to isolated systolic hypertension, i.e. systolic pressure >140 mmHg and diastolic \leq 90 mmHg.

Q4: A 59-year-old man presents for a well person check. A cardiovascular, respiratory, gastrointestinal and neurological examination is performed. No significant findings are found, except during auscultation a mid systolic click followed by a late systolic murmur is heard at the apex. The patient denies any symptoms. The most likely diagnosis is:

- A. Barlow syndrome
- B. Austin Flint murmur
- C. Patent ductus arteriosus
- D. Graham Steell murmur
- E. Carey Coombs murmur

Explanation: This patient is suffering from a mitral valve prolapse (Barlow syndrome, click murmur syndrome) (A). A mid-systolic click followed by a late systolic murmur is heard at the apex as the thickened mitral valve leaflet is displaced into the left atrium during systole. An Austin Flint murmur (B) produces a low pitched, mid-diastolic rumble at the apex. Classically, mitral valve displacement as well as aortic turbulence due to regurgitation qualifies as an Austin Flint murmur. A patent ductus arteriosus (C) produces a constant machinery murmur. A Graham Steell murmur (D) is typically heard best at the left sternal edge, second intercostals space during inspiration. A high pitched early diastolic murmur is heard associated with pulmonary hypertension. A Carey Coombs murmur (E) is a short, mid-diastolic rumble heard best at the apex due to turbulent blood flow over a thickened mitral valve, most often due to rheumatic fever.

Valvular heart disease

Q5: You see a 57-year-old woman who presents with worsening shortness of breath coupled with decreased exercise tolerance. She had rheumatic fever in her adolescence and suffers from essential hypertension. On examination she has signs which point to a diagnosis of mitral stenosis. Which of the following is not a clinical sign associated with mitral stenosis?

- A. Malar flush
- B. Atrial fibrillation
- C. Pan-systolic murmur which radiates to axilla
- D. Tapping, undisplaced apex beat
- E. Right ventricular heave

Explanation: Malar flush (A), atrial fibrillation (B), a tapping apex beat (D) and right ventricular heave (E), which occurs secondary to pulmonary hypertension, are all clinical signs associated with mitral stenosis. On auscultation of the praecordium, a mid-diastolic murmur (±opening snap, representing a mobile valve) is heard rather than a pan-systolic murmur (C) which is usually heard in mitral regurgitation, tricuspid regurgitation and ventricular septal defects.

Q6: An asymptomatic 31-year-old woman has been referred for cardiological assessment. After her ECG she was told that she had mitral valve prolapse and would like further information on this condition. Which of the following statements is correct?

- A. Beta-blocker therapy is indicated
- B. Angiotensin-converting enzyme (ACE) inhibitor therapy is indicated
- C. One or both leaflets of the mitral valve are pushed back into the left atrium during systole
- D. Significant mitral regurgitation will eventually develop
- E. Exercise should be restricted

Explanation: There is no indication for ACE inhibitor therapy (B), while beta-blockers (A) may be used for management of arrhythmias if these occur. Mitral regurgitation (D) is unlikely to occur, although it is a possibility. There is no need to limit exercise (E) in an asymptomatic patient. As mentioned elsewhere, endocarditis is a persistent risk, with the need for antibiotic prophylaxis a topic of current debate.

Q7: A 69-year-old woman complains of intermittent palpitations, lasting several hours, which then stop spontaneously. She also suffers from asthma. Holter monitoring confirms paroxysmal atrial fibrillation. Which of the following statements is correct regarding the management of this patient?

- A. Digoxin effectively prevents recurrence of the arrhythmia
- B. Anticoagulation is not necessary
- C. Sotalol may be effective
- D. Amiodarone should be avoided
- E. Flecainide orally may be an effective as-needed treatment to abort an attack

Explanation: Oral flecainide (E) is now widely recommended to avoid continuous therapy. Propafenone is used in a similar way. Digoxin (A) is not effective in this situation; sotalol (C) may be used but should be avoided because of this patient's asthma. Amiodarone (D) is effective, but has numerous serious adverse reactions including pulmonary fibrosis, liver damage, peripheral neuropathy and abnormal thyroid function. Anticoagulation (B) is very important to prevent strokes, although in low-risk patients aspirin may be adequate. In patients where drug therapy is ineffective or poorly tolerated, ablation therapy can have a high success rate.

Q8: A 76-year-old male is brought to accident and emergency after collapsing at home. He has recovered within minutes and is fully alert and orientated. He says this is the first such episode that he has experienced, but describes some increasing shortness of breath in the previous six months and brief periods of central chest pain, often at the same time. On examination, blood pressure is 115/88 mmHg and there are a few rales at both bases. On ECG there are borderline criteria for left ventricular hypertrophy. Which of the following might you expect to find on auscultation?

- A. Mid-diastolic murmur best heard at the apex
- B. Crescendo systolic murmur best heard at the right sternal edge
- C. Diastolic murmur best heard at the left sternal edge
- D. Pan-systolic murmur best heard at the apex
- E. Pan-systolic murmur best heard at the left sternal edge

Explanation: This is a classical presentation of aortic stenosis, with syncope following a period of increasing shortness of breath on exertion and angina, with relatively narrow pulse pressure and ECG (or echo) indications of left ventricular hypertrophy. The answer is therefore (B). (A) is the murmur of mitral stenosis (with opening snap and loud P2), (C) of aortic regurgitation, (D) of mitral regurgitation and (E) of tricuspid regurgitation. Aortic stenosis is by far the most common valvular lesion likely to be seen in general medicine.

Valvular heart disease

Q9: A 15-year-old student presents to your office on the advice of his football coach. The patient started playing football this year and suffered a syncopal episode at practice yesterday. He reports that he was sprinting with the rest of the team and became light-headed. He lost consciousness and fell to the ground, regaining consciousness within 1 or 2 minutes. He has had no prior episodes of syncope. The patient is adopted and family history unavailable. Physical examination reveals a systolic murmur heard at the left lower sternal border and apex. ECG reveals sinus rhythm with evidence of left ventricular hypertrophy (LVH). What physical examination findings would likely be present?

- A. A systolic ejection murmur heard best at the apex that diminishes with squatting and handgrip, and increases with Valsalva maneuver and standing.
- B. A systolic murmur with mid to late systolic click heard at the apex. The click and murmur occur earlier in systole with squatting and handgrip and are delayed with Valsalva maneuver and standing.
- C. A holosystolic murmur heard best at the apex, radiating to the axilla, which increases with squatting and hand grip, and diminishes with Valsalva maneuver and standing.
- D. A blowing holosystolic murmur heard best at the lower left sternal border which increases with squatting and hand grip and diminishes with Valsalva maneuver and standing.
- E. A low pitched mid systolic murmur radiating to the carotids.

Explanation: This patient likely has experienced a paradoxical embolus causing acute embolic stroke. In paradoxical embolism, a venous thrombus (usually from the leg or pelvic veins) passes into the systemic circulation through an intracardiac defect, typically an atrial septal defect (ASD) or less commonly through a ventricular septal defect (VSD). In ASD, a mid-systolic murmur can often be appreciated due to increased flow across the pulmonic valve. During diastole, a mid-diastolic rumbling murmur may be appreciated along the sternal border due to increased flow across tricuspid valve. A prominent right ventricular impulse and palpable pulmonary artery pulsation may sometime be appreciated. The second heart sound is widely split and is relatively fixed in relation to respiration. Ventricular septal defect usually presents as a holosystolic murmur at the mid-left sternal border. Both the murmur of VSD and mitral regurgitation are enhanced by exercise and diminished by amyl nitrate. Aortic insufficiency causes a diastolic decrescendo murmur at the mid left sternal border. A patent ductus arteriosus (PDA) results in a continuous “machinery” murmur heard best at the upper left sternal border. Coarctation of aorta usually presents with a midsystolic murmur over the left interscapular space which may become continuous if the lesion in the vessel is narrowed enough to cause high-velocity jet flow. Classic to this condition are arterial hypertension in the upper extremities and normal or low blood pressure, with diminished or delayed pulsations in the lower extremities. Chest x-ray findings such as sign of “3” due to indentation of the aorta at the site of coarctation with pre and post-stenotic dilatation and rib notching due to rib erosions by dilated collateral vessels are classic findings. Aortic insufficiency, PDA, and coarctation of the aorta are not associated with paradoxical embolism

Q10: A 75-year-old patient presents to the ER after a syncopal episode. He is again alert and in retrospect describes occasional substernal chest pressure and shortness of breath on exertion. His blood pressure is 110/80 and lungs have a few bibasilar rales. Which auscultatory finding would best explain his findings?

- A. A harsh systolic crescendo-decrescendo murmur heard best at the upper right sternal border
- B. A diastolic decrescendo murmur heard at the mid-left sternal border
- C. A holosystolic murmur heard best at the apex
- D. A midsystolic click
- E. A pericardial rub

Explanation: The classic triad of symptoms in aortic stenosis includes exertional dyspnea, angina pectoris, and syncope. Physical findings include a narrow pulse pressure and systolic murmur. The remaining answers describe aortic insufficiency murmur, mitral regurgitation murmur, mitral valve prolapse click, and a rub associated with pericarditis. These conditions are not associated with syncope as a presenting symptom.

Q11: You are called to see a 21-year-old man in the emergency room with new onset of slurred speech and left hemiparesis. On auscultation the patient has a systolic murmur at the pulmonic region with a diastolic rumble along the left sternal border. The second heart sound is split and fixed relative to respiration. What is the likely cause of patient’s symptom?

- A. Ventricular septal defect
- B. Atrial septal defect
- C. Patent ductus arteriosus
- D. Aortic insufficiency
- E. Coarctation of the aorta

Explanation: This patient likely has experienced a paradoxical embolus causing acute embolic stroke. In paradoxical embolism, a venous thrombus (usually from the leg or pelvic veins) passes into the systemic circulation through an intracardiac defect, typically an atrial septal defect (ASD) or less commonly through a ventricular septal defect (VSD). In ASD, a mid-systolic murmur can often be appreciated due to increased flow across the pulmonic valve. During diastole, a mid-diastolic rumbling murmur may be appreciated along the sternal border due to increased flow across tricuspid valve. A prominent right ventricular impulse and palpable pulmonary artery pulsation may sometime be appreciated. The second heart sound is widely split and is relatively fixed in relation to respiration. Ventricular septal defect usually presents as a holosystolic murmur at the mid-left sternal border. Both the murmur of VSD and mitral regurgitation are enhanced by exercise and diminished by amyl nitrate. Aortic insufficiency causes a diastolic decrescendo murmur at the mid left sternal border. A patent ductus arteriosus (PDA) results in a continuous “machinery” murmur heard best at the upper left sternal border. Coarctation of aorta usually presents with a midsystolic murmur over the left interscapular space which may become continuous if the lesion in the vessel is narrowed enough to cause high-velocity jet flow. Classic to this condition are arterial hypertension in the upper extremities and normal or low blood pressure, with diminished or delayed pulsations in the lower extremities. Chest x-ray findings such as sign of “3” due to indentation of the aorta at the site of coarctation with pre and post-stenotic dilatation and rib notching due to rib erosions by dilated collateral vessels are classic findings. Aortic insufficiency, PDA, and coarctation of the aorta are not associated with paradoxical embolism.

Valvular heart disease

Q12: A 78-year-old man is brought to the emergency department because of a 3-week history of productive cough, swelling of the legs and feet, and fatigue. He has had progressive dyspnea on exertion for the past 2 months. Twelve years ago, he received a porcine valve replacement for severe mitral valve regurgitation. He has coronary artery disease, type 2 diabetes mellitus, and hypertension. He has smoked one pack of cigarettes daily for 60 years and drinks one beer daily. Current medications include aspirin, simvastatin, ramipril, metoprolol, metformin, and hydrochlorothiazide. He appears pale. He is 179 cm (5 ft 9 in) tall and weighs 127 kg (279.9 lb); BMI is 41.3 kg/m². His temperature is 37.1°C (98.9°F), respirations are 22/min, pulse is 96/min, and blood pressure is 146/94 mm Hg. Bilateral basilar rales are heard on auscultation of the lungs. Cardiac examination shows a laterally displaced apical heartbeat. A grade 3/6, decrescendo-crescendo diastolic murmur is heard over the apex. There is bilateral pitting edema of the feet and ankles. The remainder of the examination shows no abnormalities. Which of the following is the most likely cause of this patient's symptoms?

- A. IE
- B. COPD
- C. Valve Degeneration
- D. Pneumonia
- E. PE

Explanation: The patient underwent a porcine valve replacement for severe mitral valve regurgitation 12 years ago. Biological valves (both bovine and porcine) typically have a short lifespan due to sclerotic degeneration. If the prosthetic valve becomes dysfunctional, the patient may have to undergo open heart surgery for valve replacement again. Possible complications of the biological prosthetic valve include obstruction/stenosis, as seen in this patient, and regurgitation, both of which may present with signs of heart failure.

Q13: A 75-year-old man comes to the physician for the evaluation of progressive shortness of breath and fatigue over the past month. He reports that he cannot climb more than one flight of stairs without experiencing shortness of breath and dizziness. He has hypertension and hyperlipidemia. He has smoked one pack of cigarettes daily for the past 50 years. He does not drink alcohol. His medications include enalapril, atorvastatin, and low-dose aspirin. His temperature is 37°C (98.6°F), pulse is 70/min, respirations are 18/min, and blood pressure is 100/80 mm Hg. Physical examination shows weak peripheral pulses. Cardiac examination is shown. Which of the following is the most likely diagnosis?

- A. Mitral regurgitation
- B. Aortic stenosis
- C. MVP
- D. HCM
- E. Tricuspid regurgitation

Explanation: This patient's auscultation findings are consistent with aortic stenosis (AS), which causes left ventricular outflow obstruction. The result is dizziness and exacerbation of dyspnea upon exertion, and weak peripheral pulses (pulsus parvus et tardus). Incomplete left ventricular emptying may also cause pulmonary venous congestion, which further exacerbates dyspnea.

Q14: A 60-year-old man comes to the physician because of progressive fatigue and shortness of breath for 2 months. The dyspnea occurs on moderate exertion and during the night; he sometimes wakes up coughing and "gasping for air." He has also had several episodes of heart pounding and palpitations. Two weeks ago, he had a runny nose and a productive cough. He has type 2 diabetes mellitus and peripheral arterial disease. He has never smoked. He drinks one to two beers occasionally. He has a history of intravenous illicit drugs use but has not used in over 25 years. Current medications include aspirin, atorvastatin, and metformin. Vital signs are within normal limits. Examination shows bilateral basilar rales. Cardiac auscultation is shown. Which of the following is the most likely diagnosis?

- A. MS
- B. MR
- C. AR
- D. AS
- E. Pericarditis

Explanation: This patient's auscultatory findings indicate mitral regurgitation as the cause of his left-sided heart failure. Mitral murmurs are heard best during expiration and while the patient lies on the left side. Causes of primary mitral regurgitation include degenerative mitral valve disease (mitral valve prolapse, mitral annular calcification, ruptured chordae tendineae), infective endocarditis, and rheumatic fever.

Heart failure

Q1: A 78-year-old woman is admitted with heart failure. The underlying cause is determined to be aortic stenosis. Which sign is most likely to be present?

- A. Pleural effusion on chest x-ray
- B. Raised jugular venous pressure (JVP)
- C. Bilateral pedal oedema
- D. Bibasal crepitations
- E. Atrial fibrillation

Explanation: Aortic stenosis will first result in left ventricular failure as a result of increased ventricular pressure as the ventricle tries to pump blood across a narrowed valve. Initially the pressure load will cause a backlog of blood into the lungs, resulting in pulmonary oedema – the first sign of which will be bibasal crepitations (D) before enough fluid accumulates as pleural effusions visible on chest x-ray (A). Earlier signs of pulmonary oedema include upper lobe blood diversion and Kerley B lines as fluid infiltrates the interstitium. If the backlog continues back into the right heart, eventually signs of right-sided heart failure will be evident including raised JVP (B) and bilateral pedal oedema (C). Atrial fibrillation (E) may coexist with aortic stenosis, however it is more commonly associated as a result of mitral stenosis as the enlarged atrium disrupts the normal electrical pathways.

Q2: A 78-year-old woman is admitted to your ward following a 3-day history of shortness of breath and a productive cough of white frothy sputum. On auscultation of the lungs, you hear bilateral basal coarse inspiratory crackles. You suspect that the patient is in congestive cardiac failure. You request a chest x-ray. Which of the following signs is not typically seen on chest x-ray in patients with congestive cardiac failure?

- A. Lower lobe diversion
- B. Cardiomegaly
- C. Pleural effusions
- D. Alveolar oedema
- E. Kerley B lines

Explanation: Cardiomegaly (B), bilateral pleural effusions (C), alveolar oedema (D) and Kerley B lines (E) (representing interstitial oedema) are all features that can be seen in a chest x-ray in patients with congestive cardiac failure. Upper lobe diversion is usually seen on chest x-ray and not lower lobe diversion (A).

Q3: A 71-year-old man is being treated for congestive heart failure with a combination of drugs. He complains of nausea and anorexia, and has been puzzled by observing yellow rings around lights. His pulse rate is 53/minute and irregular and blood pressure is 128/61 mmHg. Which of the following medications is likely to be responsible for these symptoms?

- A. Lisinopril
- B. Spironolactone
- C. Digoxin
- D. Furosemide
- E. Bisoprolol

Explanation: These symptoms are characteristic of digoxin (C) (cardiac glycosides). The yellow-tinged vision (xanthopsia) is particular to these drugs. The slow pulse, with probable ectopics, together with the subjective symptoms, suggests toxicity and plasma digoxin should be measured, with lowering of the dosage or withdrawal of the drug, which is not considered first-line therapy in any case in the management of congestive heart failure.

Heart failure

Q4: A 55-year-old man presents with gradually increasing shortness of breath and leg swelling that occurred while on a business trip. He has congestive heart failure, which has caused fatigue and shortness of breath if he walks a block or climbs a flight of stairs. Blood pressure is 140/90; there is no jugular venous distension or gallop, and only minimal pedal edema. An echocardiogram shows left ventricular ejection fraction is 45%. Current medications include aspirin and simvastatin. The patient desires to keep medications to a minimum. What additional treatments are indicated at this time?

- A. Spironolactone
- B. An ACE inhibitor and a beta-blocker
- C. Digoxin
- D. Furosemide
- E. An implantable defibrillator

Explanation: There is very good evidence that ACE inhibitors should be used in patients with symptomatic and asymptomatic congestive heart failure (a depressed left ventricular ejection fraction < 40%). ACE inhibitors stabilize left ventricular remodeling, improve symptoms, reduce hospitalization, and decrease mortality. Beta-blocker therapy represents a major advance in the treatment of patients with congestive heart failure. These drugs interfere with the harmful effects of sustained activation of the adrenergic nervous system (α_1 , β_1 , and β_2) by competitively blocking their receptors. When given with ACE inhibitors, beta-blockers stabilize left ventricular remodeling, improve patient symptoms, reduce hospitalization, and decrease mortality. An aldosterone antagonist is recommended for patients with NYHA class III or IV symptoms who have a left ventricular ejection fraction of less than 35% and who are still symptomatic despite receiving standard therapy with diuretics, ACE inhibitors, and beta-blockers. Likewise, digoxin may improve symptoms of patients with advanced symptomatic congestive heart failure. Neither of these drugs is indicated in this patient with mild symptoms. Furosemide is used to improve symptoms but does not prolong survival. Since this patient wants to minimize medications, an ACE inhibitor and beta-blocker are better first choices because they confer a survival advantage. An implantable defibrillator is indicated in systolic heart failure with left ventricular ejection fraction less than 30% to 35% in order to prevent sudden cardiac death, but is not indicated in this patient whose ejection fraction is 45%.

Q5: A 72-year-old man presents with shortness of breath that awakens him at night. He is unable to walk more than one city-block before stopping to catch his breath. Physical examination findings include normal blood pressure, bilateral basilar rales, and neck vein distention. The patient has diabetes and a known history of congestive heart failure. His last echocardiogram revealed a left ventricular ejection fraction of 25%. The patient has compliant with his medication regimen that includes an ACE inhibitor, beta-blocker, a loop diuretic, metformin, and glipizide. What is the most likely etiology for the patient's heart failure?

- A. Metabolic
- B. Infiltrative
- C. Coronary artery disease
- D. Valvular disease
- E. Infectious

Explanation: Coronary artery disease has become the predominant primary cause of congestive heart failure in industrialized countries, causing 60% to 75% of cases. Coronary artery disease, hypertension, and diabetes mellitus interact to augment the risk of heart failure in many patients, but coronary artery disease is the primary cause in most. In 20% to 30% of patients the exact etiology is not known. These patients are referred to as having nonischemic, dilated, or idiopathic cardiomyopathy. Prior viral infection or toxins (eg, alcohol or chemotherapy) may also lead to a dilated cardiomyopathy. Specific genetic defects such as mutations of genes encoding cytoskeletal proteins (desmin, cardiac myosin, vinculin), and nuclear membrane proteins (lamin) have been identified that may cause dilated cardiomyopathy. The condition is also associated with Duchenne, Becker, and limb girdle muscular dystrophies. Conditions that lead to a high cardiac output (eg, arteriovenous fistula, anemia) are seldom solely responsible for the development of heart failure.

Q6: A 55-year-old woman is brought to the emergency department because of epigastric pain, sweating, and breathlessness for 45 minutes. She has hypertension treated with hydrochlorothiazide. She has smoked 1 pack of cigarettes daily for the past 30 years and drinks 1 glass of wine daily. Her pulse is 105/min and blood pressure is 100/70 mm Hg. Arterial blood gas analysis on room air shows: pH(7.49) pCO₂(32 mm Hg) pO₂(57 mm Hg)

- A. Decreased transpulmonary pressure
- B. Decreased minute ventilation
- C. Increase pulmonary capillary pressure
- D. Increased pulmonary capillary permeability
- E. Decreased total body hemoglobin

Explanation: Increased pulmonary capillary wedge pressure can occur in the setting of myocardial infarction (MI) due to cardiogenic shock. An extensive myocardial infarction, as seen in this patient, may result in decreased cardiac contractility and reduced cardiac output, leading to increased left-sided heart pressures and ultimately backup of blood to the lungs. Cardiogenic pulmonary edema thickens the alveolar-capillary membrane and impairs oxygen diffusion capacity. As a compensatory mechanism, minute ventilation increases, resulting in increased CO₂ elimination. This patient's inability to oxygenate with preserved ventilation, as reflected by the hypoxemia without concurrent hypercapnia in her ABG, is consistent with type 1 respiratory failure.

Heart failure

Q7: A 59-year-old man is brought to the emergency department by his wife because of a 3-day history of worsening shortness of breath. He has a history of long-standing hypertension, type 2 diabetes mellitus, and gout. Current medications include enalapril, amlodipine, metformin, and ibuprofen as needed. His wife reports that he only takes his medications every second or third day. He has smoked one pack of cigarettes daily for 30 years. He stopped drinking alcohol after his first acute gout attack 3 years ago. His temperature is 37.1°C (98.9°F), pulse is 86/min, and blood pressure is 167/95 mm Hg. Pulse oximetry on room air shows an oxygen saturation of 91% on room air. Auscultation reveals coarse crackles over both lower lung fields. Which of the following is the most appropriate next step in management?

- A. Administer oral metoprolol
- B. Administer inhaled albuterol and corticosteroids
- C. Administer intravenous furosemide
- D. Place chest tube
- E. Administer intravenous corticosteroids

Explanation: This patient presents with respiratory distress (acute dyspnea, hypoxemia) and bibasilar crackles on pulmonary auscultation. In conjunction with his history of long-standing poorly controlled hypertension, these findings indicate pulmonary edema due to acute decompensated heart failure (ADHF). As this patient is hemodynamically stable, management includes oxygen supplementation, raising the head of the bed, and intravenous loop diuretics such as furosemide. Diuresis results in a decrease in intravascular volume, thereby reducing the cardiac preload, central venous pressure, and pulmonary capillary wedge pressure, which, in turn, reduces pulmonary edema. Furthermore, in patients with pulmonary edema, furosemide causes transient venodilation, further reducing pulmonary congestion, even before the onset of diuresis. If respiratory distress persists, treatment with vasodilators for ADHF (e.g., nitroglycerin) may be initiated to further reduce preload.

Infective endocarditis

Q1: A 25-year-old woman with known mitral valve prolapse develops a low grade fever, malaise and night sweats within a couple of weeks of a major dental procedure. Examination reveals a pulse rate of 110/minute, which is regular, tender vasculitic lesions on the finger pulps and microscopic haematuria. Which investigation is most likely to provide a definitive diagnosis?

- A. Full blood count
- B. ECG
- C. Autoantibody screen
- D. Blood culture
- E. Coronary angiography

Explanation: The diagnosis here is subacute bacterial endocarditis, probably due to *Streptococcus viridans*. The definitive diagnosis is by blood culture (D) although echocardiography (B) will show vegetations on affected heart valves. Although the lesions described are vasculitic (as are the painless Janeway lesions and the Roth spots in the retina), in this case they are due to antigen-antibody complexes triggered by infection. The issue of routine prophylaxis for patients with valvular disease prior to dental procedures is controversial; in the UK, it is no longer recommended.

Q2: You are volunteering with a dental colleague in a community indigent clinic. A nurse has prepared a list of patients who are scheduled for a dental procedure and may need antibiotic prophylaxis beforehand. Of the patients listed below, who would be most likely to benefit from antibiotic prophylaxis to prevent infective endocarditis?

- A. 17-year-old adolescent boy with coarctation of the aorta
- B. 26-year-old woman with a ventricular septal defect repaired in childhood
- C. 42-year-old woman with mitral valve prolapse
- D. 65-year-old man with prosthetic aortic valve
- E. 72-year-old woman with aortic stenosis

Explanation: Recommendations for prophylaxis of infective endocarditis (IE) from transient bacteremia associated with dental, genitourinary, or gastrointestinal procedures have recently undergone major revision. Only patients with history of prior infective endocarditis (IE), patients with prosthetic heart valves, patients with unrepaired congenital cyanotic heart disease, and patients with prosthetic graft material which has not yet endothelialized (typically 6 months from placement of the graft material) are given prophylactic antibiotics. Therefore, the patients with coarctation of the aorta, repaired VSD, mitral valve prolapse, and aortic stenosis do not require pretreatment. A typical adult prophylactic regimen is a single dose of amoxicillin 2 g orally 30 to 60 minutes prior to the procedure. Any dental procedure that causes bleeding can cause transient bacteremia. Sterile procedures (ie, cardiac catheterization) and procedures with a very low risk of bacteremia (ie, endoscopy without biopsy) do not need preprocedure antibiotics.

Q3: A 43-year-old man with HIV comes to the physician because of fever and night sweats over the past 15 days. During this period, he has also had headaches and generalized weakness. He has no cough or shortness of breath. He has hypertension controlled with lisinopril and is currently receiving triple antiretroviral therapy. He has smoked one pack of cigarettes daily for the past 15 years and drinks one to two beers on weekends. He is a known user of intravenous illicit drugs. His temperature is 39°C (102°F), pulse is 115/min, respirations are 15/min, and blood pressure is 130/80 mm Hg. Examination shows several track marks on the forearms. The lungs are clear to auscultation. A holosystolic murmur that increases on inspiration is heard along the left sternal border. The remainder of the physical examination shows no abnormalities. Laboratory studies show a leukocyte count of 12,800/mm³ and an erythrocyte sedimentation rate of 52 mm/h. His CD4+ T-lymphocyte count is 450/mm³. Which of the following is the most likely sequela of the condition?

- A. Pulmonary embolism
- B. Hemorrhages underneath fingernails
- C. Painful nodules on pads of the fingers
- D. Asymptomatic hemorrhages on palms and soles
- E. Retinal hemorrhages

Explanation: IV drug users such as this patient are at an increased risk of infective endocarditis because the use of nonsterile injections commonly leads to bacteremia. Bacterial colonization of the heart valves can lead to the formation of thrombi and subsequent bacterial embolisms. The right heart is only rarely involved in IE. However, IV drug users are at an increased risk of tricuspid or multiple valvular involvement. Thrombi from the right heart most frequently dislodge into the lung, which puts the patient at risk of pulmonary embolism. Patients with left-sided (aortic valve or mitral valve) endocarditis more commonly present with emboli to the retina (Roth spots), extremities (Janeway lesions, Osler nodes, splinter hemorrhages), kidney, brain, and spleen

Infective endocarditis

Q4: Blood cultures are sent to the laboratory. Intravenous antibiotic therapy is started. Transesophageal echocardiography shows a large, oscillating vegetation attached to the tricuspid valve. There are multiple small vegetations attached to tips of the tricuspid valve leaflets. There is moderate tricuspid regurgitation. The left side of the heart and the ejection fraction are normal. Which of the following is the most likely causal organism of this patient's conditions?

- A. *Staphylococcus epidermidis*
- B. *Enterococcus faecalis*
- C. *Streptococcus pyogenes*
- D. *Staphylococcus aureus*
- E. *Streptococcus gallolyticus*

Explanation: *Staphylococcus aureus* is the most common cause of acute infectious endocarditis in almost all groups of patients, including IV drug users. In contrast, subacute endocarditis is characterized by an insidious onset (weeks to months) with less severe symptoms. Subacute IE occurs more often in patients with cardiac valve abnormalities and is most commonly caused by viridans streptococci species. Other notable causes of IE in IV drug users includes *Staphylococcus epidermidis* and fungal infection (e.g., *Candida* species)

Q5: A 34-year-old man comes to the physician because of fatigue and shortness of breath with moderate exertion for the past 2 months. Over the past 10 days, he has had low-grade fevers and night sweats. He has no history of serious illness except for a bicuspid aortic valve diagnosed 5 years ago. He has smoked one pack of cigarettes daily for 10 years and drinks 3–5 beers on social occasions. He does not use illicit drugs. The patient takes no medications. He appears weak. His temperature is 37.7°C (99.9°F), pulse is 70/min, and blood pressure is 128/64 mm Hg. The lungs are clear to auscultation. A grade 2/6 systolic murmur is heard best at the right sternal border and second intercostal space. There are several hemorrhages underneath his fingernails on both hands and multiple tender, red nodules on his fingers. They start empiric treatment with intravenous vancomycin. Blood cultures are sent to the laboratory. Blood cultures grow gram-negative bacilli identified as *Cardiobacterium hominis*. Which of the following is the most appropriate next step in management?

- A. Switch to intravenous gentamicin
- B. Continue intravenous vancomycin
- C. Switch to intravenous ceftriaxone
- D. Switch to intravenous ampicillin
- E. Switch to intravenous cefazolin

Explanation: Empirical treatment for infective endocarditis should be started after obtaining blood cultures. Once the results are confirmed, a targeted antibiotic therapy should be employed to increase the efficacy of therapy and reduce the risk of side effects. In this patient, antibiotic treatment should cover *Cardiobacterium hominis*, a gram-negative bacterium of the HACEK group.

Q6: A 64-year-old man is evaluated for fever, dyspnea, and myalgia 7 days after admission to the hospital for acute traumatic pancreatitis. Since admission, his general condition improved significantly under adequate treatment. His current symptoms started suddenly and worsened over the course of the past 6 hours. He has hypercholesterolemia and type 2 diabetes mellitus. His current medications are fentanyl, insulin, and atorvastatin. He has received lactated Ringer solution via a central venous catheter. His temperature is 38.8°C (101.8°F), pulse is 120/min, respirations are 21/min, and blood pressure is 120/75 mm Hg. Physical examination shows a soft nontender abdomen. Bowel sounds are normal. Cardiac examination discloses a holosystolic murmur heard best at the left sternal border. The lungs are clear to auscultation. There is erythema and purulent discharge at the central venous catheter insertion site at the right side of the neck. Hemoglobin concentration is 13.8 g/dL, leukocyte count is 16,000/mm³, and erythrocyte sedimentation rate is 40 mm/h. Results of blood cultures are pending. Which of the following organisms is the most likely cause of this patient's current condition?

- A. *Pseudomonas aeruginosa*
- B. *Streptococcus gallolyticus*
- C. *Klebsiella pneumoniae*
- D. *Haemophilus aphrophilus*
- E. *Staphylococcus aureus*

Explanation: Staphylococci such as *S. aureus* account for the majority of cases of healthcare-associated infective endocarditis. IE caused by *S. aureus* is characterized by acute onset of symptoms and rapid progression (hours to days). Acute IE due to *S. aureus* can quickly lead to complications such as valvular insufficiency, heart failure, and septic emboli. Therefore, in addition to removing the infected CVC, empiric antibiotic therapy (vancomycin plus beta-lactam for native valve endocarditis) should be initiated after obtaining blood cultures.

Infective endocarditis

Q7: A 41-year-old man comes to the physician because of a 10-day history of generalized weakness. He has also had headaches and night sweats and is a known user of illicit intravenous drugs. His temperature is 39.1°C (102.4°F), pulse is 110/min, respirations are 17/min, and blood pressure is 127/78 mm Hg. There are several track marks on his forearms. A holosystolic murmur that increases on inspiration is heard along the left sternal border. Laboratory studies show a leukocyte count of 13,900/mm³ and an erythrocyte sedimentation rate of 58 mm/h. Which of the following is the most likely consequence of this patient's condition?

- A. Painless macules and papules on palms and soles
- B. Painful nodules on pads of the fingers
- C. Rupture of nail-bed capillaries
- D. Pulmonary embolism
- E. Retinal hemorrhages

Explanation: IV drug users such as this patient are at increased risk of IE because the use of nonsterile syringes can lead to bacteremia. Bacterial colonization of the heart valves can lead to the formation of thrombi and subsequent bacterial emboli. While right heart involvement is rare in IE, IV drug users are at an increased risk of tricuspid or multiple valvular involvement. Thrombi from the right heart most frequently dislodge into the lung, which puts the patient at risk of pulmonary embolism. Patients with left-sided (aortic valve or mitral valve) endocarditis more commonly present with emboli to the retina (i.e., Roth spots), extremities (e.g., Janeway lesions, Osler nodes, splinter hemorrhages), kidney, brain, and spleen.

Q8: An autopsy of a patient's heart who recently died in a motor vehicle accident shows multiple nodules near the line of closure on the ventricular side of the mitral valve leaflet. Microscopic examination shows that these nodules are composed of immune complexes, mononuclear cells, and thrombi interwoven with fibrin strands. These nodules are most likely to be found in which of the following patients?

- A. A 71-year-old male with acute-onset high fever and nail bed hemorrhages
- B. A 54-year-old male who recently underwent dental surgery
- C. A 41-year-old female with a facial rash and nonerosive arthritis
- D. A 28-year-old HIV-positive female intravenous drug user
- E. A 6-year-old female with subcutaneous nodules and erythema marginatum

Explanation: Nonbacterial verrucous thrombi on the undersurface of the mitral valve are suggestive of Libman-Sacks endocarditis. A facial rash and symmetric, nonerosive polyarthritis in a middle-aged female is highly suspicious for systemic lupus erythematosus. Systemic inflammation in SLE can lead to the deposition of circulating sterile immune complexes with platelet thrombi on either side of valve leaflets in what is known as Libman-Sacks endocarditis. Although the vegetations seen in Libman-Sacks endocarditis are usually asymptomatic, they are easily dislodged and can embolize, causing complications. Valvular damage is rare but can sometimes manifest as mitral regurgitation or mitral stenosis.

Q9: Three weeks after undergoing transurethral prostate resection for benign prostatic hyperplasia, a 70-year-old man has fever, malaise, and pain in his extremities. Physical examination shows subungual petechiae and tender red papules on his fingers and toes. A new holosystolic murmur is heard on chest auscultation. The organism does not cause hemolysis on blood agar. Addition of pyrrolidonyl-β-naphthylamide gives the bacterial colonies a cherry red color. Which of the following is the most likely causal organism?

- A. Staphylococcus aureus
- B. Streptococcus gallolyticus
- C. Staphylococcus epidermidis
- D. Haemophilus aphrophilus
- E. Enterococcus faecalis

Explanation: Enterococcus faecalis is a gram-positive, PYR positive, catalase-negative coccus that can grow in 6.5% NaCl and produces variable hemolysis (α-hemolytic or γ-hemolytic) on blood agar. It can cause subacute infective endocarditis in patients who undergo gastrointestinal or genitourinary procedures (e.g., transurethral prostate resection). Enterococcus spp. are resistant to drugs typically used to treat infective endocarditis such as penicillin G, 1st to 4th generation cephalosporins, and vancomycin (in the case of VRE). Enterococcus is sensitive to aminopenicillins and 5th generation cephalosporins (e.g., ceftaroline).

General cardiology

Q1: A 16-year-old boy is diagnosed with a small ventricular septal defect, having been screened by echocardiography because of a family history of hypertrophic obstructive cardiomyopathy. He is entirely asymptomatic, plays several sports regularly and has no growth retardation. The echocardiogram also confirms a small left to right shunt, with pulmonary to systemic flow ratio only just above one. Which of the following is the most likely to be a significant complication of his condition?

- A. Pulmonary hypertension
- B. Heart failure
- C. Dysrhythmias
- D. Endocarditis
- E. Shunt reversal (right to left flow)

Explanation: Large ventricular septal defects (VSDs) may indeed be associated with pulmonary hypertension (A), heart failure (B) and shunt reversal (E), but a small defect is unlikely to lead to these problems and, in general, VSDs are not associated with dysrhythmias (C). Endocarditis (D) is, however, a persistent hazard. Routine antibiotic prophylaxis for dental procedures is no longer recommended.

Q2: A 67-year-old man presents to accident and emergency with a 3-day history of shortness of breath. On examination you palpate the radial pulse and notice that the patient has an irregular heart beat with an overall rate of 140 bpm. You request an electrocardiogram (ECG) which reveals that the patient is in atrial fibrillation. Which of the following would you expect to see when assessing the JVP?

- A. Raised JVP with normal waveform
- B. Large 'v waves'
- C. Cannon 'a waves'
- D. Absent 'a waves'
- E. Large 'a waves'

Explanation: The JVP provides clinicians with information regarding right atrial pressures and filling. It mainly consists of five wave forms:

- 1- a wave: representing atrial systole
- 2- c wave: representing closure of the tricuspid valve (this wave is not usually visible)
- 3- x descent : representing a fall in atrial pressure during ventricular systole
- 4- v wave: representing atrial filling against a closed tricuspid valve
- 5- y descent: representing the opening of the tricuspid valve

In atrial fibrillation, the 'a waves' are absent (D) due to dysfunctional atrial systole. A raised JVP with normal waveform pattern (A) is usually seen in fluid overload and right heart failure. Large v waves (B) are usually seen in patients with tricuspid regurgitation. Cannon 'a waves' (C) are seen in patients with complete heart block, single chamber ventricular pacing, ventricular arrhythmias and ventricular ectopics. Large 'a waves' (E) can be seen in pulmonary hypertension and pulmonary stenosis.

Q3: A 56-year-old man presents to your clinic with symptoms of exertional chest tightness which is relieved by rest. You request an ECG which reveals that the patient has first degree heart block. Which of the following ECG abnormalities is typically seen in first degree heart block?

- A. PR interval >120 ms
- B. PR interval >300 ms
- C. PR interval <200 ms
- D. PR interval > 200 ms
- E. PR interval <120 ms

Explanation: The PR interval is usually measured from the start of the P-wave to the start of the QRS and the normal range lies within 0.12–0.2s (i.e. 120–200 ms). In first degree heart block, the PR interval is prolonged, greater than 0.2 s (200 ms) (D). Shortened PR interval (i.e <120 s or <0.12 s) (E) results from fast AV conduction, usually down an accessory pathway seen in Wolff–Parkinson–White syndrome.

General cardiology

Q4: A 48-year-old woman has been diagnosed with essential hypertension and was commenced on treatment three months ago. She presents to you with a dry cough which has not been getting better despite taking cough linctus and antibiotics. You assess the patient's medication history. Which of the following antihypertensive medications is responsible for the patient's symptoms?

- A. Amlodipine
- B. Lisinopril
- C. Bendroflumethiazide
- D. Frusemide
- E. Atenolol

Explanation: ACE inhibitors (e.g. lisinopril (B)) commonly cause a dry cough in some patients. If this occurs, patients are usually taken off the ACEI and started on either an ARB (e.g. irbesartan, losartan, telmisartan) or different class of antihypertensive. Amlodipine (A), bendroflumethiazide (C), frusemide (D) and atenolol (E) do not commonly cause a dry cough as a side effect.

Q5: A 63-year-old male was admitted to accident and emergency 2 days after discharge following an apparently uncomplicated MI. He complained of rapidly worsening shortness of breath over the previous 48 hours but no further chest pain. He was tachypnoeic and had a regular pulse of 110/minute, which proved to be sinus tachycardia. The jugular venous pressure was raised and a pan-systolic murmur was noted, maximal at the left sternal edge. Which of the following is the most likely diagnosis?

- A. Mitral incompetence
- B. Ventricular septal defect
- C. Aortic stenosis
- D. Dressler's syndrome
- E. Further myocardial infarction

Explanation: Ventricular septal defect (B) is the most likely diagnosis and this is potentially a very serious complication which will need endovascular or surgical intervention. The murmur is not where one would expect to locate it for mitral incompetence (A), and there is also the finding of raised jugular venous pressure in this case. Aortic stenosis (C) would have quite different clinical findings, including the murmur, Dressler's syndrome (D) is a type of possibly autoimmune pericarditis and there is nothing pointing to another myocardial infarction (E).

Q6: A 72-year-old man comes to the office with intermittent symptoms of dyspnea on exertion, palpitations, and cough occasionally productive of blood. On cardiac auscultation, a low-pitched diastolic rumbling murmur is faintly heard at the apex. What is the most likely cause of the murmur?

- A. Rheumatic fever as a youth
- B. Long-standing hypertension
- C. A silent MI within the past year
- D. A congenital anomaly
- E. Anemia from chronic blood loss

Explanation: The history and physical examination findings suggest mitral stenosis. Dyspnea may be present secondary to pulmonary edema; palpitations are often related to atrial arrhythmias (PACs, SVT, atrial flutter, or fibrillation); hemoptysis may occur as a consequence of pulmonary hypertension with rupture of bronchial veins. A diastolic rumbling apical murmur is characteristic. If the patient is in sinus rhythm, a late diastolic accentuation of the murmur occurs because of increased flow across the mitral valve with atrial contraction. A loud first heart sound and early diastolic opening snap may also be present. The etiology of mitral stenosis is usually rheumatic, rarely congenital. Hypertension may cause an S4 gallop but not a diastolic murmur. Myocardial infarction may cause mitral regurgitation because of papillary muscle dysfunction and anemia may cause a pulmonic flow murmur; both of these are systolic murmurs.

General cardiology

Q7: Four days after being admitted to the intensive care unit for acute substernal chest pain and dyspnea, an 80-year-old man is evaluated for hypotension. Coronary angiography on admission showed an occlusion in the left anterior descending artery, and a drug-eluting stent was placed successfully. The patient has a history of hypertension and type 2 diabetes mellitus. Current medications include aspirin, clopidogrel, metoprolol, lisinopril, and atorvastatin. His temperature is 37.2 °C (99 °F), pulse is 112/min, respirations are 21/min, and blood pressure is 72/50 mm Hg. Cardiac examination shows a normal S1 and S2 and a new harsh, holosystolic murmur heard best at the left sternal border. There is jugular venous distention and a right parasternal heave. The lungs are clear to auscultation. Pitting edema extends up to the knees bilaterally. An ECG shows Q waves in the inferior leads. Which of the following is the most likely cause of this patient's hypotension?

- A. Papillary muscle rupture
- B. Interventricular septum rupture
- C. Left ventricular free wall rupture
- D. Post-infarction fibrinous pericarditis
- E. Ascending aortic dissection rupture

Explanation: Interventricular septum rupture typically occurs 3–5 days after myocardial infarction. It manifests with sudden onset hemodynamic instability (hypotension, tachycardia, tachypnea) and signs of right ventricular failure (jugular venous distention, parasternal heave, pedal edema, and characteristically clear lungs) due to the development of a left-to-right shunt. This patient's new-onset holosystolic murmur is the result of blood flow across the newly formed ventricular septal defect (analogous to the classic murmur of a ventricular septal defect). Finally, the ECG finding of Q waves in the inferior leads suggests the presence of right ventricular ischemia.

Q8: A 68-year-old woman is brought to the emergency department by her husband because of acute confusion and sudden weakness of her left leg that lasted for about 30 minutes. One hour prior to admission, she was unable to understand words and had slurred speech for about 15 minutes. She has type 2 diabetes mellitus and hypertension. She has smoked 1 pack of cigarettes daily for 30 years. Current medications include metformin and hydrochlorothiazide. Her pulse is 110/min and irregular; blood pressure is 135/84 mmHg. Examination shows cold extremities. There is a mild bruit heard above the left carotid artery. Cardiac examination shows a grade 2/6 late systolic ejection murmur that begins with a midsystolic click. Neurological and mental status examinations show no abnormalities. ECG shows irregularly spaced QRS complexes with no discernible P waves. Doppler ultrasonography shows mild left carotid artery stenosis. A CT scan and diffusion-weighted MRI of the brain show no abnormalities. Which of the following treatments is most likely to prevent future episodes of neurologic dysfunction in this patient?

- A. Aspirin
- B. Warfarin
- C. Clopidogrel
- D. Aortic valve replacement
- E. Atorvastatin

Explanation: In a patient who has recently suffered a TIA and is found to have atrial fibrillation (irregular pulse, no distinct P waves), anticoagulation therapy should be initiated immediately to prevent any future episodes of TIA or stroke that may be caused by the underlying atrial fibrillation (AF). In addition to anticoagulation, new-onset atrial fibrillation should be treated with either rate control or rhythm control strategy. In any patient with newly diagnosed nonvalvular AF, a CHA2DS2-VASc score should be calculated to assess the risk of systemic embolization. This patient's score is 6, which indicates a risk of stroke per year of > 9%. Her score is based on age (+1 if 65–74 years of age), sex (+1 for females), history of hypertension (+1), TIA (+2), and history of diabetes mellitus (+1). In patients with AF and a score ≥ 2 , oral anticoagulation with either warfarin or newer oral anticoagulants is indicated.

Q9: A 66-year-old man comes to the physician because of a 3-week history of shortness of breath with exertion. He has hypertension, hyperlipidemia, and type 2 diabetes mellitus. Current medications include aspirin, losartan, simvastatin, and insulin. His temperature is 37.1°C (98.8°F), pulse is 74/min, and blood pressure is 150/84 mm Hg. Pulse oximetry on room air shows an oxygen saturation of 96%. patient's cardiac examination reveals a third heart sound (S3 gallop). Further evaluation of this patient is most likely to show which of the following?

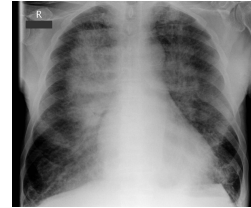
- A. Elevated serum brain natriuretic peptide levels
- B. Calcification of the aortic valve
- C. Bounding pulses of peripheral arteries
- D. Earlier onset of abnormal cardiac sound with standing
- E. Ruptured papillary muscle

Explanation: Elevated levels of brain natriuretic peptide (BNP) and the presence of an S3 gallop both indicate increased ventricular filling pressures, which is seen in congestive heart failure (CHF). BNP, which is released in response to stretching of the cardiac ventricle, is a sensitive parameter to evaluate patients with symptoms of CHF. High levels of BNP (over 400 pg/mL) in patients with classic symptoms of CHF have a high positive predictive value and are associated with a worse prognosis, while low values (under 100 pg/mL) in patients with dyspnea have a high negative predictive value for CHF.

General cardiology

Q10: A 58-year-old man comes to the physician because of a 5-day history of progressively worsening shortness of breath and fatigue. He has smoked 1 pack of cigarettes daily for 30 years. His pulse is 96/min, respirations are 26/min, and blood pressure is 100/60 mm Hg. An x-ray of the chest is shown. Which of the following is the most likely cause of this patient's findings?

- A. Left ventricular failure
- B. Tricuspid regurgitation
- C. Mitral stenosis
- D. pericarditis
- E. Cardiomyopathy



Explanation: This patient's acute onset dyspnea and x-ray findings suggest cardiogenic pulmonary edema due to left ventricular failure. Individuals with left ventricular failure initially develop interstitial edema, which results in smooth reticular opacification and Kerley B lines due to accentuation of the vascular markings. With the further increase in pulmonary capillary wedge pressure, individuals can develop features of alveolar edema (e.g., consolidation with an air bronchogram) and pleural effusion (e.g., blunting of the costophrenic angle). In pulmonary edema, the opacification can often assume a perihilar distribution (a so-called "batwing" or "butterfly-shaped" pattern), which is seen here, because the periphery of the lung has better lymphatic drainage than the central regions.